DROSOPHILA

Information Service

46

January 1971

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Prepared at the
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DROSOPHILA INFORMATION SERVICE

JANUARY 1971

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RESEARCH NOTES

TECHNICAL NOTES

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THIRTEENTH DROSOPHILA RESEARCH CONFERENCE will be held May 21, 22 and 23, 1971, in the Department of Biology, Ithaca College, Ithaca, New York 14850. For more information write Ross J. MacIntyre or Steven R. Thompson at the above address.

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EDITOR'S COMMENTS

The financial squeeze affecting all science these days has not left DIS untouched. Increasing costs and its large size brought the total cost of DIS #45 to about nine dollars per copy; in spite of a modest grant subsidy and sales of back issues, we are operating with a substantial deficit at this point.

Among the alternatives open to bridge the gap between income and costs, we have decided on two courses of action. We are strictly limiting the number of pages in the issue and we have made several ecomonies in the method of producing DIS. We trust that this will not appreciably affect its quality.

This issue has been cut in size by omitting several sections - the directories and bibliography - as well as those notes that arrived late. These items will be included in the next issue, which we have already started and which we hope will be ready for distribution during early summer, 1971. Anyone wishing to make a contribution to DIS #47 should not wait for the regular call but should send it at the earliest opportunity. This will enable us not only to produce a more accurate issue, but one that will emerge faster and therefore be more valuable scientifically.

Among the recent changes made to improve DIS is the use of photocopied "proofs" of much of the copy; these not only allow the authors an opportunity to catch our errors, but also to correct errors caused by ambiguities or illegibilities in their original manuscripts. Once again we would like to thank those contributors who send clear and uncluttered originals.

OAK RIDGE, TENNESSEE: OAK RIDGE NATIONAL LABORATORY
Biology Division

Wild Stocks

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

Chromosome 1

b- 1 B/C(1)DX, y f
b- 2 car bb
b- 3 cv v f/C(1)DX, y f
b- 4 dor/ClB
b- 5 f
b- 6 f BB/C(1)DX, y f
b- 7 fa^{g}
b- 8 fu^{59}/C(1)DX, y f
b- 9 l(1)J1 sc^{J1}/Dp(1;f)24
b-10 m f car/C(1)DX, y w f
b-11 mal
b-12 pn
b-13 ptg^{3} v m g^{2} sd f/C(1)DX, y f
b-14 sc cv v f B/C(1)DX, y f
b-15 sc ec cv ptg^{3} v/C(1)RM, y v f car
b-16 sc z ec ct
b-17 spl
b-18 v
b-19 v f su(f)
b-20 w
b-21 w^{a}
b-22 w^{a4}
b-23 w^{a} fa fa^{no} rb/C(1)DX, y f
b-24 w^{a} fa spl
b-25 w^{a} fa^{g} fa^{no} rb/C(1)DX, y w f
b-26 w^{a} fa^{g} spl sn^{3}
b-27 w^{a} fa^{no} spl rb/C(1)DX, y f
b-28 w^{a} pdf
b-29 w^{a} spl rb
b-30 w^{bf}
b-31 w^{co} sn^{2}
b-32 w^{e} bb^{1}/C(1)DX, y f/$B^{S}Y$
b-33 w^{sp}
b-34 y
b-35 y ac sc pn
b-36 y ac sc pn/C(1)DX, y f
b-37 y ac sc pn w rb cm ct^{6} sn^{3} ras^{2} v dy g^{2} f car/C(1)RM, In(1)sc^{S1} + dl-49, sc^{S1} v f car
b-38 y B/C(1)DX, y f
b-39 y bb^{1-3a}/C(1)RM, y w/$y^{+}Y$
b-40 y cv f
b-41 y cv v f
b-42 y cv v f car
b-43 y ec cv ct^{6} v/FM6
b-44 y fa^{n} sn^{3}
b-45 y Hw/C(1)RM, In(1)sc^{S1L} sc^{8R} + S, sc^{S1} sc^{8} w^{a} B
b-46 y l(1)451/FM6, y^{31d} sc^{8} dm B
b-47 y m
b-48 y w
b-49 y w bb^{ds}
b-50 y w fa^{no}
b-51 y w spl sn^{3}
b-52 y w^{a}
b-53 y w^{a} fa^{no} spl/C(1)RM, y w fa^{no}
b-54 y w^{a} m f car
b-55 y w^{a} spl rb
b-56 y^{2}
b-57 y^{2} cv v f
b-58 y^{2} cv v wy car/FM6
b-59 y^{2} sc f
b-60 y^{2} sc f su(f)
b-61 y^{2} sc f su(f)/C(1)DX, y f
b-62 y^{2} sc w^{a} ec
b-63 y^{2} v f car/C(1)DX, y f
b-64 y^{2} v mal^{bz}
b-65 y^{2} w^{cf}
b-66 y^{2} wy car/C(1)DX, y f

Chromosome 2

c- 1 a px or
c- 2 Adh^{D} pr cn
c- 3 Adh^{n1}
c- 4 Adh^{n5}
c- 5 al b c sp
c- 6 al dp b pr c px sp
c- 7 al dp b pr Bl c px sp/CyO
c- 8 al dp spd^{fg}
c- 9 b Adh^{n1} Tft/CyO
c-10 b Adh^{n4};C(1)DX, y f/+
c-11 b cn c bw
c-12 b el rd^{s} Bsh pr Bl/CyO
c-13 b j
c-14 b pr Elp px
c-15 b pr tu-bw^{e} px
c-16 b pu
c-17 Bc Pin^{Yt}/CyO
c-18 Bl L^{2}/SM5, al^{2} Cy lt^{v} sp^{2}
c-19 bw
c-20 bw^{D}
c-21 cl d
c-22 cl fy^{2} j^{676}
c-23 cl Gdh^{R} spd^{fg}
c-24 cn bw
c-25 cn en/SM5
c-26 cui
c-27 d
c-28 da/In(2L)Cy + In(2R)Cy
c-29 fj wt/SM5
c-30 Fo/SM1, al^{2} Cy sp^{2}
c-31 fy^{2}
c-32 j^{676}
c-33 lt stw^{3}
c-34 lys
c-35 lys rc
c-36 lys rc^{2}
c-37 lys^{2} rc^{2}/CyO
c-38 $M(2)e^{S}$/Cy Roi
c-39 $M(2)S2^{10}$/In(2L)Cy + In(2R)Cy + Dp(2;2)41^{2}, Cy pr
c-40 M(2)S7/SM5
c-41 net al ex ds S ast shv ho rub/SM1
c-42 nub^{2}
c-43 nub^{2} b Sco pr cn/CyO
c-44 nub^{2} Bl/SM5
c-45 nub^{2} Bl L/SM5
c-46 nw^{D} Pu^{2} Pin^{Yt}/SM5
c-47 Pin
c-48 Pin^{2}/bw^{V57e} SM1
c-49 Pin^{Yt}/SM1
c-50 Pu^{2}/SM1
c-51 px sp Pin^{2}/SM1
c-52 rc pr
c-53 rk^{crm}
c-54 S Sp Tft nw^{D} Pin^{Yt}/CyO
c-55 Sco/In(2L)Cy + In(2R)Cy, Cy Roi
c-56 sp^{2} bs^{2}
c-57 Sp lys d/SM1
c-58 Sp lys d J/In(2L)Cy + In(2R)Cy
c-59 Sp lys rc J/SM1
c-60 Sp rc^{2}/In(2L)Cy + In(2R)Cy
c-61 spd^{fg}
c-62 spd^{fg} Sp d/SM1
c-63 stw
c-64 stw^{3} c
c-65 Su(H)/In(2L)Cy + In(2R)Cy, Cy pr
c-66 Tft/SM1
c-67 Tft L/SM5
c-68 vg
c-69 vg^{U}/In(2L)t + In(2R)Cy, Roi bw^{45a} sp^{2} or^{45a}

Chromosome 3

d- 1 Bd^{G}/In(3R)C, l(3)e

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

Chromosome 4

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

Multichromosomal Stocks

f- 1 v/Y^{bb} (1;Y)
f- 2 FM7;Pin^{Yt}/CyO (1;2)
f- 3 In(1)w^{m4}; E(var)7/In(2L)Cy + In(2R)Cy (1;2)
f- 4 v;In(2R)bw^{VDel}/SM1 (1;2)
f- 5 y/C(1)DX, y f;dp lys rc pr (1;2)
f- 6 y;cn bw (1;2)
f- 7 y f; nub^2 (1;2)
f- 8 y w^a/bw^+Y; E(w^a) (1;Y;2)
f- 9 y w^a; Frd E(w^a) sp Pin^2/SM1 (1;2)
f-10 y^2; T(2;3)101, al sp^2/Cy L^4 sp^2 (1;2)
f-11 w^e; SM1/In(2LR)bw^{V1}; Sb/In(3LR)Ubx^{130} e^s (1;2;3)
f-12 FM6, y^{31d} sc^8 dm B/In(1)AM, y^2; pr Bl/SM1; Tp(3)Vno, Vno/In(3LR)Ubx^{130} e^s; pol (1;2;3;4)
f-13 +/C(1)DX, y f; bw; st; pol (1;2;3;4)
f-14 y; bw; st; pol (1;2;3;4)
f-15 y^2; TM3, Sb Ser p^p/Ki p^p ry sr e^s (1;3)
f-16 y^2; ve h th ro ca/TM1, Me ri (1;3)
f-17 In(1)sc^{8L} sc^{S1R} + dl-49, y^{31d} sc^- v f (B)/y w^a N^{Nic}/y^+Y w^+; ca K-pn (1;Y;3)
f-18 y^2; ci gvl ey^R sv^n (1;4)
f-19 y^2; ci^D/ey^D (1;4)
f-20 y^2; pol (1;4)
f-21 y^2 w^{cf}; ci gvl spa^{Cat}/ci^D pol (1;4)
f-22 b pr Bl/SM1; Tp(3)Vno, Vno/In(3LR) Ubx^{130}, Ubx^{130} e^s (s;3)
f.23 Bc Elp px/CyO; Tp(3)Vno, Vno/TM6, Ubx^{17005} (2;3)
f-24 bw; st (2;3)
f-25 SM1/In(2LR)bw^{V1}, bw^{V1}; Sb/In(3LR)Ubx^{130}, Ubx^{130} e^s

Inverted chromosomes

g- 1 In(1)65, y f/B^SY
g- 2 In(1)481, y bb^{1-481}/FM6
g- 3 In(1)AM/T(1;3)65, y
g- 4 In(1)B^{M1}/C(1)DX, y f
g- 5 In(1)dl-49, y v f car bb^{amg}/C(1)DX, y f
g- 6 In(1)dl-49, y w lz^s bb/In(1)sc^{L8}, sc^{L8} w^a m car
g- 7 In(1)dl-49, y^2 sc w^a/B^SY
g- 8 In(1)dl-49 + B^{M1}, sc v B^{M1}
g- 9 In(1)dl-49 + B^{M1}, y^2 sc w^a v B^{M1}
g-10 In(1)EN, y bb/y^+Y
g-11 In(1)1-v59, y 1(1)v59/C(1)RM, y w/y^+Y
g-12 In(1)1-v132, y 1(1)v132/C(1)RM, y w/y^+Y
g-13 In(1)1-v231, y 1(1)v231/C(1)RM, y w/y^+Y
g-14 In(1)rst^3, rst^3
g-15 In(1)rst^3, y rst^3 car (bb?)/C(1)RM, In(1)sc^{S1L} sc^{8R} + S, y^{31d} sc^{S1} sc^8 w^a B
g-16 In(1)rst^3, rst^3 ras v f/C(1)DX, y f: bw
g-17 In(1)sc^4, y sc^4
g-18 In(1)sc^4, y sc^4 cv v B/C(1)DX, y f/B^SY
g-19 In(1)sc^{4L} sc^{8R}, y sc^4 sc^8 cv v B/C(1)DX, y f/B^SY
g-20 In(1)sc^{4L} sc^{8R} + S, y sc^4 sc^8 w^a B/C(1)RM, y^2 su(w^a) w^a bb/y^+Y
g-21 In(1)sc^7, sc^7 w^a ec/C(1)DX, y f
g-22 In(1)sc^7, sc^7 w fa^{no}/C(1)DX, y f
g-23 In(1)sc^8, sc^8 v f car
g-24 In(1)sc^8, y^{31d} sc^8 w^a
g-25 In(1)sc^8 + S, y^- ac^- sc^8 w^a/y^+Y
g-26 In(1)sc^9, sc^9 w^a t Bx
g-27 In(1)sc^{S1} + dl-49, y^{62k} sc^{S1} v f car/C(1)DX, y f
g-28 In(1)sc^{S1L} sc^{4R}, sc^{S1} sc^4 cv v B/C(1)DX, y f/B^SY

g-29 In(1)sc^{S1L} sc^{8R} + dl-49, sc^{S1} sc^{8} v B car/C(1)DX, y f
g-30 In(1)w^{m4L} N$^{264-84R}$, y w^{m4} N$^{264-84}$ sn/FM3/B^{S}Yy^{+}
g-31 In(1)y^{3P}, y^{3P} B
g-32 In(1)y^{4}, y^{4}
g-33 In(1LR)sc^{V1}, v.sc^{V1} y^{+}/In(1)sc^{8} + dl-49, y^{31d} sc^{8} v f B
g-34 In(1LR)1-v139, y w^{m139} 1(1)v139/C(1)DX, y w f/Y/Y
g-35 In(2LR)lt^{m3}, lt^{m3}/SM5
g-36 In(2LR)lt^{m12}/SM5
g-37 Basc
g-38 Biny/1(1)J1^{259} y w m f/y^{+}Y
g-39 FM4, w dm B/C(1)DX, y f
g-40 FM6, In(1)sc^{8} + dl-49, y^{31d} w dm B/C(1)DX, y f/y^{+}Y/Y
g-41 FM7, y^{31d} sc^{8} v^{Of} B

Translocated Chromosomes

h- 1 T(1;2)Bld/T(1;2)64, y x Fs(2)Do/T(1;2)Bld
h- 2 T(1;4)A13(18c5)
h- 3 T(1;4)A17(8A2)/C(1)DX, y f
h- 4 T(1;4)A19
h- 5 T(1;4)A20/C(1)DX, y f
h- 6 T(1;4)B^{S}(16A1), B^{S}/C(1)DX, y f
h- 7 T(1;4)B^{S}(16A1), y^{2} cv v B^{S} car/C(1)DX, y f
h- 8 T(1;4)e15
h- 9 T(1;4)h4
h-10 T(1;4)h6
h-11 T(1;4)1-v11(15A), y 1(1)v11 car/y^{+}Y
h-12 T(1;4)w^{m5}(3C3), w^{m5}
h-13 T(1;4)4C3/C(1)DX, y f
h-14 T(1;4)(13B8-9)/C(1)DX, y f
h-15 T(2;3)A, Bl/In(2L)Cy + In(2R)bw^{VDel}
h-16 T(2;3)lt^{m7}(98C), lt^{m7}/SM5
h-17 T(3;4)89E, ss bx bxd/ey^{D}
h-18 T(Y;2)E/b el rd^{s} pr cn
h-19 T(1;4)w^{m5}, w^{m5}/FM4, y^{31d} w dm B
h-20 T(1;4)w^{m5}, B^{S}, w^{m5} v B^{S}/FM4, y^{31d} w dm f

Rings

i-1 R(1)1, y/C(1)DX, y f/y^{+}Y
i-2 R(1)2, f/C(1)DX, y f
i-3 R(1)2, w^{spont} v f/C(1)RM, In(1)sc^{S1L} sc^{8R} + S, sc^{S1} sc^{8} w^{a}/Y
i-4 R(1)2, y B/C(1)DX, y f

X-Y Combinations

j-1 X.Y^{L}, y cv v f car.Y^{L}/C(1)RM, y/Y"
j-2 X.Y^{S}(A3), y w.Y^{S}/C(1)RM, y v f/R(Y)L
j-3 X.Y^{S}(-8b), In(1)sc^{8L}, EN^{R}, y^{+} f y.Y^{S}/C(1)RM, y v f/R(Y)L
j-4 Y^{S}X.(FR1), Y^{S} y cv v f./C(1)DX, y f/Y
j-5 Y^{S}X.(P-7), In(1)EN, Y^{S} y f./C(1)RM, y v f/Y
j-6 B^{S}Y^{S}X.(FR1^{L}, 3a^{R}), B^{S} Y^{S} y cv v f./C(1)RM, y v f/R(Y)L

Compound X Chromosomes

k-1 C(1)RA, 1(1)J1^{259} y/Y^{S} X., 1(1)J1^{259}.y w/B^{S} Y y^{+}
k-2 C(1)RA.Y^{L}, +--In(1)sc^{8L}, EN^{R}, y.Y^{L} y^{+}/Y^{S}X.Y^{L}, In(1)EN, Y^{S}B y.Y^{L}/y^{+} ac^{+}.Y^{L} (FR2)
k-3 C(1)RM, pn/Y^{S}X.Y^{L}, y B/Y

Compound Autosomes

1- 1 C(2L)RM; C(2R)RM
1- 2 C(2L)RM #3; C(2R)RM #3
1- 3 C(2L)RM #4, dp; C(2R)RM #4, px
1- 4 C(2L)RM, b pr; C(2R)RM, cn
1- 5 C(2L)RM, nub^{2} b^{66h} pr; C(2R)RM, cn
1- 6 C(3L)RM; C(3R)RM (Rasmussen No. 26)
1- 7 C(3L)RM #1, h^{2} rs^{2}; C(3R)RM #1
1- 8 C(3L)RM #4, ri; C(3R)RM #4, sr
1- 9 C(3L)RM, se h^{2} rs^{2}; C(3R)RM, sbd gl e^{s} (Rasmussen no. 44)
1-10 +/+/B^{S}Y/(Y?); C(2L)RM #3; C(2R)RM #3
1-11 w/w/B^{S}Y; C(2L)RM #3; C(2R)RM #3
1-12 In(1)AM, y^{2}/FM6; C(2L)RM, dp; C(2R)RM, px
1-13 In(1)AM, y^{2}/FM6/Y; C(2L)RM #3; C(2R)RM #3
1-14 In(1)AM, y^{2}/FM6/Y; C(2L)RM; C(2R)RM
1-15 C(4)RM, ci ey^{R}/gvl sv^{n}

Attached XY Chromosomes

m-1 XY^{L}.Y^{S} (108-9 Parker), y^{2} su(w^{a}) w^{a} Y^{L}.Y^{S}/C(1)RM, y v bb/O
m-2 XY^{S}.Y^{L} (110-8 Parker), y^{2} su(w^{a}) w^{a} Y^{S}.Y^{L} y^{+}/C(1)RM, y v bb/O
m-3 Y^{S}X.Y^{L} (FR-1^{L}, U-8d^{R}), Y^{S} y w^{a} cv v f.Y^{L}/C(1)RM, y^{2} su(w^{a}) w^{a} bb/O
m-4 Y^{S}X.Y^{L}, In(1)EN, Y^{S} B f v y.Y^{L}y^{+}/C(1)RM, y v bb/O
m-5 Y^{S}X.Y^{L}, In(1)EN + dl-49, Y^{S} car f v y.Y^{L}/C(1)RM, y^{2} su(w^{a}) w^{a} bb/O
m-6 Y^{S}X.Y^{L}, In(1)EN + dl-49, Y^{S} f v pn y.Y^{L}/C(1)RM, y^{2} su(w^{a}) w^{a} bb/O

Y Derivatives

n- 1 B^{S}w^{+}Y/y w^{a}
n- 2 B^{S}Yy^{+}/y v; bw
n- 3 B^{S}Yy^{+}/C(1)RM, y/Y^{S}X., Y^{S} y cv v f
n- 4 B^{S}Yy^{+}/In(1)w^{m4L} N$^{264-84R}$, y sn/FM3
n- 5 B^{S}Yy^{31d}/C(1)RM, y w f/Y^{S}X., Y^{S} y cv v f
n- 6 bw^{+}Y/y v; bw
n- 7 bw^{+}Yy^{+}/C(1)DX, y f/y v f
n- 8 Df(Y)bb/In(1)w^{m4}
n- 9 y^{+}Y/C(1)RM, y w/y
n-10 y^{+}Yw^{+}(11a)/y w^{a}
n-11 y^{+}Y^{L}.(FR-2)/C(1)RA, y--In(1)sc^{8}/Y^{S}X.Y^{L}, In(1)EN, y B
n-12 Ymal^{+} no. 2/y^{2} v mal
n-13 Yw^{+} (Y900)/y w^{a}

n-14 $Y^L.ac^+ y^+(sc^8EN$ c.o. Y B-2)X.Y^S, y w.Y^S, y w.Y^S/C(1)RM, y v f
n15 $Y^L.bb^+ ac^+ y^+(sc^8EN$ c.o. Y T-O)/X.Y^S, y w.Y^S/C(1)DX, y f

Extra Y Chromosome Stocks

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

Deficiencies and Duplications

p- 1 Df(2L)GdhA/CyO
p- 2 Df(3R)ry/In(3LR)Ubx^{130}, Ubx^{130} e^s
p- 3 Dp(1;f)3/C(1)RM, y/$XY^L.Y^S$, 1(1)$J1^{259}$ y w $Y^L.Y^S$
p- 4 Dp(1;f)18/C(1)RM, y v f/$XY^L.Y^S$, 1(1)$J1^{259}$ y w $Y^L.Y^S$
p- 5 Dp(1;f)24 (see b-9)
p- 6 Dp(1;f)52/C(1)A/$XY^L.Y^S$, 1(1)$J1^{259}$ y w $Y^L.T^S$
p- 7 Dp(1;f)122/C(1)RM, y v f/$XY^L.Y^S$, 1(1)$J1^{259}$ y w $Y^L.Y^S$
p- 8 Dp(1;f)164/C(1)RM, y v f/$XY^L.Y^S$, 1(1)$J1^{259}$ y w $Y^L.Y^S$
p- 9 Dp(1;f)1492/sc^{53k}
p-10 Dp(1;f)1514/sc^{53k}
p-11 Dp(1;1)B^S(TMG), In(1)$sc^4.B^S$, y sc^4 m f.B^S/In(1)sc^7 + AM, sc^7
p-12 Dp(1;1)B^S(TMG), In(1)sc^{8L}, R(1)$2^R.B^S$, f.B^S/X^D/$B^SY^L.Y^S$
p-13 Dp(1;1)sc^{V1}, y^2 sc v f.sc^{V1} y^+/B^SY
p-14 Dp(1;3)w^{m49a} (Spotter)
p-15 Dp(1;3)51/C(1)RM, y v f/$XY^L.Y^S$, 1(1)$J1^{259}$ y w $Y^L.Y^S$
p-16 Dp(1;3)sc^{J4}/C(1)RM, y v f/O?/$XY^L.Y^S$, 1(1)$J1^{259}$ y w $Y^L.Y^S$
p-17 Dp(1;4)w^{m51c}/C(1)DX, y w f/In(1)w^{m4L}. rst^{3R}, rst^3 car
p-18 Dp(1;4)174/C(1)RM, y v f/$XY^L.Y^S$, 1(1)$J1^{259}$ y w $Y^L.Y^S$
p-19 Dp(2;f)1/sp Pin^2/Px^4
p-20 Dp(1;)C619/SM1, Cy Roi
p-21Dp(3;4)ry^+/st ry^2 sr e^s/st ry^2 sr e^s

Triploid Stocks

q-1 C(1)RM, y w fa^{no}/FM6 females x FM6/B^SYy^+ males
q-2 C(1)RM, y^2 sc w^a ec/FM6 females x FM6/B^SYy^+ males
q-3 C(1)RM, In(1)dl-49, v^{Of} f/FM7 females x FM7 males

SEOUL, KOREA: CHUNGANG UNIVERSITY
Department of Biology

Wild Stocks

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

Chromosome 1

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

Chromosome 2

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

Chromosome 3

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

Chromosome 4

401 bt
403 ci gvl bt
414 ey

Multichromosomal

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

Inversions

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

Translocations

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

CALCUTTA, INDIA: UNIVERSITY OF CALCUTTA
Department of Zoology

Wild Stocks

a2 Oregon-R
a3 P Ceylon
a5 Samarkand

Chromosome 1

b1 B
b2 car
b3 cm
b4 ct
b5 ec dx
b6 Ext/FM6, y^{31d} sc^{8} dm B
b7 f^{36a} odsy f^{tih} & y f:=
b8 ras dy
b9 rb
b10 sn^{3}
b11 v
b12 w
b13 w^{a}
b14 w m f
b15 w^{e} sn/ClB
b16 w^{i} f^{3N} bb
b17 y ac sn^{3}
b18 y ac sn^{3} sx vb^{2} sy/y sc^{1} In dl-49 B v w^{a} sc^{8}
b19 y w ct
b20 y w sn^{3}
b21 z w^{11E4}

Closed X (Unstable)

c2 R(1)2 w^{vc}/In(1)dl-49, y w lz^{s}/sc^{8} Y

Chromosome 2

d1 b
d2 BL L^{2}/SM5, al^{2} Cy lt^{v} sp^{2}
d3 bw
d4 cg c/U
d5 cl
d6 Cy/cg c U
d7 Cy/Pm
d8 dp^{R}
d8 ds S G b pr/Cy, al^{2} lt^{3} L^{4} sp^{2}
d9 ds ft dp^{v2} l(2)M b pr/SM5, al^{2} Cy lt^{v} sp^{2}
d10 dw 24F cl/SM5, al^{2} Cy lt^{v} sp^{2}
d11 fj wt/SM5, al^{2} Cy lt^{v} sp^{2}
d12 ft
d13 ft;Cy/G
d14 ft lt bw
d15 G^{rv}/SM5, al^{2} Cy lt^{v} sp^{2}
d16 lt bw
d17 lt stw
d18 M92)Z/SM5, al^{2} Cy lt^{v} sp^{2}
d19 net al ex ds S ast shv ho rub/SM1, al^{2} Cy sp^{2}
d20 net ed Su(dx^{2})
d21 pr en
d22 px
d23 sm px/SM5
d24 vg

Chromosome 3

e1 ca
e2 Dl H e^{s} cd/In(3R)P
e3 e^{s}
e4 Gl Sb H/Payne
e5 H^{2}/Xa
e6 red
e7 red e
e8 ru e ca
e9 ru st ss ca(rusca)
e10 se h
e11 th
e12 th st Pc Sex p^{p} ss/ TM^{1}, Me^{1} ri

Chromosome 4

f1 a^{r}/ey^{D}
f2 ci^{w}

Multichromosomal

g1 br^{3} dx st;ed Su(dx)^{2}
g2 D tra ca/In CP Dfd In RP ca
g3 ds ft lp^{v2} L(2)Mb pr/ SM5, al^{2} Cy l^{2} sp^{2}
g4 ds S G b pr/Cy, al^{2} lt^{3} L^{4} sp^{2}
g5 ft;h
g6 ft;net
67 tra/In(3LR)Ubx (FMA 3/w^{a} v)
g8 y ac sn^{3};Cy/Pm
g9 y ac sn^{3};Cy/cg c
g10 y ac sn^{3};ft
g11 dp^{T} Sp cn In NSR mr/ Cy;red e

Translocations

h1 T(1;3)05, D/y f:=
h2 T(2;3)S^{L}/In(2L+2R)Cy, Cy E(S)

Altered Y & attached X·Y

j1 (Maxy)L^{J1+}·Y/l^{J1} $sc^{J1(+)}$ In49 ptg oc BM/y sc^{S1} car odsy f g^{2} dy v ras^{2} sn^{3} ct^{6} cm rb ec w pn l sc^{8}
j2 (Maxy-v)l^{J1+}·Y/l^{J1} $sc^{J1(+)}$ In49 v ptg oc B^{M1}/y sc^{S1} car odsy f g^{2} dy v ras^{2} sn^{3} ct^{6} cm rb ec w pn l sc^{8}
j3 Y^{S}·w y·Y^{L}·v^{55} f^{10}/O/y w:=
j4 Y^{S}·Y^{S}#2/y v f·Y^{L} & y f:=
j5 Y^{S}·X·InEN v cv y·Y^{L} y^{+} & y^{2} su-w^{a} w^{a} bb:=
j6 Y^{S}·X·Y^{L}(FR-1L, U-8d^{R})Y^{S}·y w^{a} cv v f·Y^{L} & y^{2} su-w^{a} w^{a} bb:= no free Y
j7 y w f·Y^{L}·Y^{S}/O/y w:=
j8 y w Y^{L}·Y^{S}/O/y w:=

Duplications & Deficiencies

k1 Dp(1;1)B^{S}(TMG), In(1)sc^{8L}, R(1)2^{R}·B^{S}, f·B^{S}/X^{D}/B^{S}·Y^{L}·Y^{S}
k2 Dp(X;4), 174+ T(X;4)174/ $lJ1^{259}$ y w·Y^{L}·Y^{S} & y v f:=

Inversions

l1 In(2L)t esc sp/SM5 sp^{2}
l2 Cy/Gla InLR
l3 dp^{T} Sp cn InNSR mr/S^{2} ls Cy pr Bl cn^{2} L^{4} bw sp^{2}
l4 dp^{T} Sp cn InNSR mr/Cy
l5 Gla InLR/S^{2} Cy cn^{2} pw sp

Triploids

m1 y^{2} sc w^{a} ec:=/FM4, y^{31d} sc^{8} dm B;ru ca

MÜNCHEN GERMANY: ZOOLOGISCHES INSTITUT DER UNIVERSITÄT MÜNCHEN

Wild Stocks

Berlin
Oregon
Zürich
Sevelen

Chromosome 1

cs^{53}/y w bb
ct^{6}
cv
rux^{2}/y
sc z w^{17G2} ec ct^{6}
sc z w^{zm}
sc z w^{z1}
scp t
sn^{3} lz^{46} ras^{4} v/y w f:=
sn^{65a}/y f:=
spl
v
vs^{66a}
w
w^{a}
w^{co} sn^{2}
w^{co} spl cv f
w spl cv f
y
y pn
y sc z w^{a} spl ec/y f:=
y sc z w$^{r,def.}$ spl f/y f:=
y v f
y w spl cv f
y w sn^{3} lz^{50e30}
y z^{a}
z
z w^{11E4}

Chromosome 2

al b c sp
ant (ro)
b dp
Cy/L^{G}
Cy L^{G}/Pm
lgl cn bw/Cy
vg bw
cn bw

Chromosome 3

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

Multichromosomal

y v;bw (1;2)
ptg;px pd;su-pd (1;2;3)
w;se h (1;3)
w;mwh e (1;3)
w^{co} sn^{2};ru^{g} (1;3)
w^{co} sn^{2};ru^{g} jv se (1;3)
w^{c} sn^{2};se h (1;3)
y;se h (1;3)
z;ru^{g} (1;3)
y;D/Sb (1;3)
y/y;D/Sb (1;3)
w;ey (1;4)
b;se (2;3)
bw;e (2;3)
bw;st (2;3)
c;e (2;3)
cn;st (2;3)
Cy/Pm;CxD/Sb (2;3)
SM1, al^{2} Cy sp^{2}/In (2LR)102 ds^{w} sp^{2};In (3LP,3RC)Sb e^{s}/ Ubx^{130} e^{s} (2;3)
ri;ci ey^{R} (3;4)

X with Y Arm Attached

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

Attached XY

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

Closed X

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

Altered Y's

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

Deficiencies

Df(1)N^{8}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
Df(1)N$^{264-105}$/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
Df(1)w$^{258-11}$, y/d1-49, y Hw m^{2} g^{4}
Df(1)w$^{258-48}$, y sc^{5} spl; Dp(1;3)w^{vco}/y f:=

Duplications

Dp(1;3)w$^{m264-58a}$/Y^{S} w y·Y^{L} y^{+}/Y/y w;+$^{Su-V}$
Dp(1;f)101;In(1)sc^{8}, Df (0+ac)·w^{a} sc^{8}

Inversions

In(1)C1B/w
In(1)N$^{264-84}$, y/FM6, y^{31d} sc^{8} dm B
In(1)rst^{3}
In(1)rst^{3}, w m v f/M-5
In(1)rst^{3}, y w^{60i29} car bb
In(1)sc^{4}, y
In(1)sc^{4}, y InS w^{a};S sc^{19i} B1/Cy L^{4}sp;se h
In(1)sc^{7}w^{a};se h
In(1)sc^{8}
In(1)sc^{8}, w^{a};se h
In(1)sc^{9}, w^{a};se h
In(1)sc^{28}, w^{a};se h
In(1)sc^{29}, w^{a}/y;se h
In(1)sc^{S1}f In d1-49 v w & y f:=;se h
In(1)sc^{L8}, car m w^{a}/y w In d1-49 lz^{s};se h
In(1)w^{m4}
In(1)w^{m4}, ct^{6} f
In(1)w^{m4};Cy/En(var)7
In(1)y^{4}, w^{a}
In(1)y^{4} w^{a};se h
Ins(1)sc^{S1L}, d1-49, sc^{8R}, sc^{S1+8} ras-v$^{-(17Cc8)}$B/v
In(2)Cy

Translocations

T(1;3)2B7;84A5 y/sc^{S1}d1-49, sc v f car/B^{s} Y
T(1;3)N$^{264-6}$, y/y w dm
T(1;3)w^{vco}, y f/Cl B^{36d}
T(1;4)w$^{258-18}$(3C4), y/d1-49, y Hw m^{2} g^{4}
T(1;4)w$^{258-18}$(3C4), y/y w dm
T(1;4)w$^{258-21}$/w;ey
T(1;4)w^{m5}/w;ey
T(3;4)A12/CxD

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

Wild Stocks

Algeria
Amherst-3
Boa Esperance, Minas Gerais, Brazil
Canton-S
Crimea
Curitiba
Falsterbo
Florida
Formosa
Gruta, Argentina
Hikone-R (resistant to BHC, DDT, parathione, nicotine)
Karsnäs
Kochi-R (resistant to parathione)
Oregon-R
Salvador, Bahia, Brazil
San Miguel, Buenos Aires, Argentina
Stäket
Tunnelgatan
Ultuna
Örebro

Chromosome 1

1001 B
1002 B/y:=
1003 B car bb/y f:=
1004 BB car/sc^{8}Y y f:=/sc^{8}Y
1005 car
1006 ct
1007 cv
1008 cv sn^{3}
1009 ec
1010 ec ct v f
1011 ec ct v g/y f:=
1012 f
1013 f B os^{o} car/y f:=
1014 f BB/sc^{8}Y, y f:=/sc^{8}Y
1015 f B^{i}B^{i}/y f:=
1016 f os^{o} car
1017 fa
1018 fu/y f:=
1019 g
1020 g^{2} B
1021 gt w^{a}
1022 kz
1023 kz g^{2} B/y:=
1024 lz/ClB
1025 M(1)o f/FM6, y^{31d} sc^{8} dm B
1026 m f
1027 mal/y f:=
1028 os^{o} car
1029 Pgd^{A} F
1030 Pgd^{B} S
1031 pn
1032 pn z is
1033 r^{9}/y f:=
1034 r^{39k} f B/y w f:=
1035 rb
1036 rb$^{27-4}$ cv v f^{3N}/y f:=
1037 rb cx
1038 rb wy
1039 sc z
1040 sc z^{mottled}
1041 sc z ec
1042 sc z w^{sp}
1043 sc z w^{17G2} ec
1044 sc z w^{a}/y w f:=
1045 sc z w^{ch}
1046 sc z w^{e}
1047 sc z w^{h}
1048 sc^{Sl} B InS w^{a} sc^{8}
1049 sc^{Sl} InS w^{a} sc^{8}
1050 sn^{3}
1051 su-w^{a} w^{a}
1052 su-w^{a} w^{a} w^{ch} fa
1053 svr$^{poi-dish}$
1054 t
1055 t^{3}
1056 v g
1057 w
1058 w$^{56\ 1\ 12}$
1059 w cv
1060 w cv sn^{3}
1061 w cv sn^{3} B/y f:=
1062 w sn^{3}
1063 w^{a}
1064 w^{a} su-f
1065 w^{a4}/y f:=
1066 w^{bf} f^{5}
1067 w^{bf2}
1068 w^{bl}
1069 w^{bl} ec
1070 w^{Bwx}
1071 w^{ch} fa
1072 w^{ch} spl
1073 w^{ch} rb wy/y f:=
1074 w^{ch} wy
1075 w^{co}
1076 w^{co} sn^{2}
1077 w^{e}
1078 w^{e2} e(w^{e})/y f:=
1079 w^{h}
1080 w^{m4}
1081 w^{sat}
1082 w^{sp}
1083 w^{sp2}
1084 y
1085 y ac w^{a} ec
1086 y ec ct v f
1087 y f Eb/sc^{Sl} B InS w^{a} sc^{8}
1088 y pn:=/FM6, y^{31d} sc^{8} dm B
1089 y rst^{3} car
1090 y v f:= Y^{S}.Y^{L}, In dl-49 y v f car
1091 y w:= Y^{S}.Y^{L}, y w f
1092 y w bb/X.Y^{S}, y w Y^{S}/Y^{L}.bb^{+} ac^{+} y^{+} sc^{8}
1093 y w^{a} ec
1094 y w^{a} f/y^{+} B.Y
1095 y sc z w^{a} ec
1096 y sc^{Sl} B InS w^{a} sc^{8}
1097 y sc^{4} sc^{8}
1098 y sc^{4} sc^{8}/sc^{8}Y y f:=/ sc^{8} Y
1099 y^{2} sc
1100 y^{2} sc w^{sp}
1101 y^{2} sc w^{sp} spl
1102 y^{2} sc z w^{-} spl
1103 y^{2} sc w^{-} spl
1104 y^{2} sc w^{-} spl B/y w f:=
1105 y^{2} sc w^{a} ec
1106 y^{2} sc w^{a} ec cv sn^{3}/ y w f:=
1107 y^{2} sc w^{a} w^{sp} is/y f:=
1108 y^{2} sc w^{a} w^{ch} fa
1109 y^{2} sc w^{a} w^{ch} spl/y f:=
1110 y^{2} sc w^{bf} spl
1111 y^{2} sc w^{bf} spl sn^{3}
1112 y^{2} sc w^{i} ec/y f:=
1113 y^{2} sc w^{i} spl
1114 y^{2} sc w^{i} w^{ch}
1115 y^{2} sc^{4} InS sc^{Sl}/sc^{8}.Y, y f:=/sc^{8}.Y
1116 y^{2} su-w^{a} w^{a}
1117 y^{2} su-w^{a} w^{a} w^{ch} fa
1118 y^{2} su-w^{a} w^{a2} w^{ch} spl/ y f:=
1119 y^{2} w^{a}
1120 y^{2} w^{a} spl
1121 y^{2} wbf spl sn^{3}
1122 z
1123 z ec
1124 z w^{11E4}
1125 z w^{a} ec
1126 Zw^{A}
1127 Zw^{B}

Chromosome 2

2001 al b c sp
2002 al b c/al^{2} In(2L)Cy
2003 bw
2004 bw^{D}
2005 Cy/bw^{Vl}, ds^{33k}
2006 Cy cn/S
2007 ds dp

2008 ex
2009 fes Alu lt/al^2 Cy lt^3
2010 ho
2011 M(2)1^2/SM1, al^2 Cy sp^2
2012 M(2)S7/SM5, al^2 Cy lt^v sp^2
2013 net
2014 nw^2/In(2L)Cy, IN(2R)NS
2015 pr
2016 rub
2017 S^2 Cy pr Bl cn^2 L^4 bw sp /In(2L)NS In(2R)NS px sp
2018 S Sp Bl bw^D/al^2 Cy lt^3 L^4 sp^2
2019 shr bw^{2b}abb sp/Sm5, al^2 Cy lt^v sp^2
2020 shv
2021 vg
2022 vg bw

Chromosome 3

3001 c(3)G
3002 ca
3003 ca K-pn
3004 cd
3005 D^3/In(3L)P
3006 e^{11}
3007 Est-6^S Est-C^F
3008 Est-6^S Est-C^S
3009 Est-6^F Est-C^F
3010 Est-6^S Lap-A^0
3011 Est-6^F
3012 Est-6^S
3013 Est-6^S/6^F
3014 eyg
3015 Gl Sb/LVM
3016 gl
3017 In(3LR)D cx F/Sb
3018 kar^2
3019 Lap-A^0
3020 M(3)h^y/In(3L)P, Me
3021 M(3)w^{124}/In(3R)C, e l(3)e
3022 ri ss
3023 ri^2
3024 ri^2 ss
3025 ro
3026 ru h st p^p ss e^s
3027 ry
3028 ry^2
3029 ry cd
3030 se
3031 ss
3032 st
3033 st c(3)G ca/ve h th c3G Sb Ubx^{130}
3034 st c(3)G ca/TM1, Me ri sbd^1 (sp^2)
3035 st p
3036 st p e
3037 st ry
3038 st ss e^{11}

Chromosome 4

4001 ci^D spa^{pol}/spa^{Cat}
4002 sv^n
4003 spa^{pol}

Multichromosomal

5001 sc z is;al b c sp
5002 sc z is;Cy/S
5003 sc z is;Cy;Ubx^{130}/Xa
5004 sc z is;D/Sb
5005 sc z is;ru h st p^p ss e^s
5006 sc^{S1}InS w^a sc^8;Cy;Ubx^{130} /Xa
5007 w^{ch};Su-w^{ch}/Cy cn
5008 w^{col};bw
5009 w^e;cru/Cy cn
5010 w^{m4} y^{511};E(var)7/Cy
5011 y^2 sc w$^-$ spl;Cy;Ubx^{130} /Xa
5012 y^2 sc w^a w^{ch} fa;Cy; Ubx^{130}/Xa
5013 bw;st
5014 cn bw;e^{11}
5015 Cy cn/S;D/In(3L)P
5016 L sp;th
5017 L^2/+ sp;th
5018 sp;th
5019 Pod-R

Deficiencies - X

6001 Df(1)bb, y sl^2 bb$^-$/FM4, y^{31d} sc^8 dm B
6002 Df(1)w$^{258-11}$, y/In(1) dl-49, y Hw m^2 g^4
6003 Df(1)w$^{258-42}$, y/FM1, y^{31d} sc^8 w^a lz^s B
6004 Df(1)w$^{258-45}$, sc z/FM4
6005 Df(1)w$^{258-45}$, y/FM4, y^{31d} sc^8 dm B
6006 Df(1)w$^{258-45}$. y w spl dm; Dp(1:3)w^{vco}/y w f:=
6007 Df(1)w$^{258-48}$, y sc^5 spl; Dp(1:3)w^{vco}/y f:=

Deficiencies - Y

6008 Df(Y)Y^{bb-}, y^2 eq
6009 Df(Y)Y^{bb-}, y^2 sc z w$^-$ spl/y pn:=

Deficiencies - 2

6010 Df(2L)al/In(2L+2R)Cy, Cy E(S)

Duplications

6011 Dp(1:1)z^{59d15}, sc z^{59d15}/ y f:=
6012 Dp(1:1), z(w^{a4}/w^a)/y f:=
6013 Dp(1:1)w^a, (w^a/w^a)/y f:=
6014 Dp(1:1), (w^{bf}/w^a)ec
6015 Dp(1:1)112, y f
6016 Dp(1:Y)y w^a/Y·w$^+$ Co

Translocations

7001 T(1;4)B^S/y f:=
7002 T(1;4)w^{m5}/w;ci ey^R
7003 T(2;3)bw^{VDe4}/Cy
7004 T(2;3)Sb^V, Sb^V, In(3R)Mo,$_2$ In(3LR)P35/SM1, al^2 Cy sp^2 ;In(3LR)Ubx^{130}, Ubx^{130} e^S
7005 T(1;Y)y/y^{+Y}

Triploid

8001 y^{2}sc w^{a}ec/FM4, y^{31d} sc^8 dm B

Extra Y

8002 In(1)w^{m4L} N$^{264-84R}$, y sn/ FM3, y^{31d} sc^8 dm B 1/Y; dm sn

Closed-X

8003 X^{c2} f car/y f:=

X with Y fragments attached

8004 FR-1 Y^S y cv v f/y f:=

TURKU, FINLAND: UNIVERSITY OF TURKU
Department of Genetics

Wild Stocks

1 Canton-S
2 Oregon-K
3 Oregon-R-S
4 Samarkand (Inbred)
5 Turku-1
6 Turku-2

Chromosome 1

101 B
102 bb
103 bi ct g^2
104 br w^e ec rb t^4/FM1, y^{31d} sc^8 w^a lz^s B
105 Bx
106 car
107 ct^6
108 cv
109 cv dx sn
110 cv lz/C1B
111 cv sn
112 ec
113 ec ct v g
114 f
115 $f_2$car
116 g_2
117 g^2 f B & y
118 g^2 f sd
119 In(1)rst^3, y rst^3 car(bb?)/C(1)sc^{S1L} sc^{8R} + S, y^{31d} sc^{S1} sc^8 w^a B
120 In(1)sc^8, sc^8
121 In(1)sc^{S1L} sc^{8R} + S, sc^{S1} sc^8 w^a B
122 In(1)w^{m4}, w^{m4}
123 m
124 m g^2
125 m wy g^2
126 ras^2
127 rb
128 rb cx
129 rsc and y f:=
130 s
131 sc
132 sc cv
133 sc cv v f
134 sc ec cy ct^6 v g^2 f/y^2 sc^{S1} B, In-49, v w^a sc^8
135 sc w cv
136 sd
137 sn^3
138 spl
139 t^4
140 v
141 w
142 w^{ch} wy
143 wy
144 y
145 y ac v
146 y cv
147 y In-49 f car & y f:=
148 y sn^3 bb
149 y w
150 y w cv
151 z

Ring X

160 X^{c2}, cv v f/C1B

Chromosome Y

191 f·Y^S/Y^L
192 In(1)w^{m4} & extra Y
193 sc^8·Y/y ac sc oc ptg and y f:=
194 v f B $\overline{XY}$/y^2 su-w^a w^a bb
195 X·Y^L/Y^S and X·Y^L/X·Y^L (Newhaus)

Chromosome 2

201 al dp b pr c px sp/In(2L+2R)Cy, al^2 Cy lt^3 L^4 sp^2
202 ap^4/In(2LR)Rev^B
203 Bl L^2/In(2L+2R)Cy, Cy
204 bw
205 dp b pr
206 ds^{38k}/In(2L)Cy, Cy dp^{lv2} b pr
207 In(2L)Cy, al^2 ast^3 b pr
208 In(2L)Cy, al^2 Cy lt^3/In(2R)Cy, b pr Bl lt^3 cn^2 L^4 sp^2
209 In(2L+2R)Cy, S^2 Cy cn^2/In(2R)Cy, dp^{lv}Sp cn^2
210 In(2L+2R)NS, b mr/In(2L+2R)Cy, Cy
211 In(2L+2R)NS, px sp/In(2R)Cy, cn^2 cg sp^2
212 pr cn ix/SM5, al^2 Cy lt^v sp^2
213 rl
214 stw^2
215 stw^2 vg
216 vg

Chromosome 3

301 Bd^G/In(3R)C, l(3)a
302 ca bv
303 cu e^s
304 Df(3L)in^{61j1}/TM1, Me ri p^p sbd^1
305 dsx^{60} cu sr e^s/TM1, Me ri sbd^1
306 e
307 eg rn^3 p^p bx sr e^s ca/In(3LR)Ubx^{130}, ri Ubx^{130} e^s ca
308 Gl Sb/LVM
309 h Pc^2 sr e^s/TM1, Me ri sbd^1
310 h th st cp in ri p^p sr e^s ca/In(3LR)Ubx^{130}, ri Ubx^{130} e^s ca(hinca)
311 h th st cp in ri Pc^2 sr^{61j2}/In(3LR)Ubx^{130}, ri Ubx^{130} e (cat)
312 In(3L)P, Me Sb/In(3LR)DcxF, ru h D
313 In(3LR)Ubx^{130}, ri Ubx^{130} e^s/TM1, Me ri p^p sbd^1

314 In(3R)AntpB, ss
315 In(3R)Dl^{B}, st Dl^{B}/In(3R)P^{W}, st l(3)W ca
316 In(3R)P^{FLA}
317 k^{D} e^{s}/In(3LR)Ubx^{130}, ri Ubx^{130} e^{s}
318 k^{D} e^{s} Bd^{G}/In(3R)C, l(3)a
319 Ly Sb/LVM
320 rn^{3} pP bx sr e^{s}/TM1, Me ri pP sbd^{1}
321 (ru) h th st cp in ri p^{p} ss^{a} bx^{3} sr e^{s}/TM1, Me ri sbd^{1}
322 ru h th st cu sr e^{s} ca
323 Sb Ubx/T(2;3)ap^{Xa}
324 se app
325 se rt^{2} th/In(3L)P, Me
326 ss bxd k e^{s}/T(2;3)ap^{Xa}
327 st sr H^{2} ca/In(3R)P^{W}, st l(3)W ca
328 th st cp in ri pP
329 th st cp in ri pP bx sr e^{s}/TM1, Me ri p^{p} sbd^{1} (thrie)
330 th st cp in ri pP ss bxd sr e^{s}/TM1, Me ri pP sbd^{1}
331 th st cp Pc^{2}/In(3LR)Ubx^{130}, ri e^{s}(ca±)
332 tra/T(2;3)Me
333 W Sb/In(3LR)DcxF

Chromosome 4

401 ci
402 ci^{W}
403 spa

Multichromosomal

501 bw;e
502 bw;st
503 bw^{V1}, dp b/In(2L+2R)Cy, Cy sp^{2};Sb/In(3LR)DcxF (ru h ca?)

Translocations

701 T(2;3)101, al sp^{2}/In(2L+2R)Cy, Cy L^{4} sp^{2}
702 T(2;3)101;ru h e^{4} ro ca/In(3L+3R)P, Dfd ca
703 T(2;3)A, Bl;ru h D TA ss e^{s}/In(3LR)Ubx^{130}, ri Ubx^{130} e^{s}
--- T(2;3)ap^{Xa} (see 323, 326)
704 T(2;3)B;ru h D TB ss e^{s}/In(3L+3R)P
705 T(2;3)bw^{V4}/SM1, al^{2} Cy sp^{2}
--- T(2;3)Me (see 332)
706 T(2;3)rn (several strains with different marker combinations)
707 T(2;3)spy (several strains with different marker combinations)

In addition, a number of Extra sex comb and Antennapedia mutants in Dr. Hannah-Alava's private collection.

CANTON, NEW YORK: ST. LAWRENCE UNIVERSITY
Department of Biology

Wild Stocks

Canton-S^{2}
Oregon-R
Swedish-b

Chromosome 1

B
f
m
v
W
w m f/ClB
y^{2} w^{a} cv v f B

Chromosome 2

bw
dp
c
L^{5}
vg

Chromosome 3

e
se
st

Chromosome 4

ey^{2}

Multichromosomal

Cy/Pm^{1}

Inversions

Muller-5

SEOUL, KOREA: YONSEI UNIVERSITY COLLEGE OF MEDICINE
Medical Genetics Laboratory

Wild Stocks

001 Oregon-R

Chromosome 1

101 Muller-5

102 y^{2} w^{i} ct^{6} f^{1}
103 y^{3} cv v f
104 sc^{8}.Y.B^{S}/y f:=

Chromosome 2

201 vg bw tu

Multichromosomal

501 bw;st
502 Cy/Pm;Ubx/Sb
503 y sc^{S1} In49 sc^{8};dp bw;st pP
504 y^{S1} sc^{8} InS y^{3}P;al Cy lt^{3} sp^{2}/dp b Pm;ru h D^{3} InCXF ca/Sb ln(3R)

HAREN, GRONINGEN, NETHERLANDS: UNIVERSITY OF GRONINGEN
Genetisch Instituut

Wild Stocks

1 Bacup
2 Bannerdale
3 Davis Oregon
4 Dronfield
5 Finland
6 Groningen I
7 Pacific
8 Israel

Inbreds

51, 52 Bayfordbury 2 strains
53-56 Groningen I, 4 strains
57 M-Oregon
58 Oregon
59-62 Pacific 4 strains

Chromosome 1

101 B
102 f^{55f}
103 g
104 ma-1
105 sc cv v car
106 su^2-s v;bw
107 v
108 w
109 w cv sn
110 w m
111 w^a
112 w^{co} sn
112a w^{co} sn^2
113 w^e
114 y
115 y f car
116 y pn
117 y_2w spl
118 y^2
119 y^2 v mal^{bz}
120 y^{59b}
121 z
122 z^{58g}

Chromosome 2

201 al dp b pr c px sp
202 a px sp
203 b
204 b cn vg bw
205 bw
206 bw^D
207 bw^D cn
208 cn
209 dp
210 dp-1
211 dp b cn bw
212 net
213 px sp
214 vg
215 vg^p

Chromosome 3

302 ca K-pn
303 Cbx
304 e
305 e^{11}
306 e^{ug}
307 ri
308 ru h st cu sr e^S ca
309 ru h th st cu sr e^S
310 ru^g jv se by
311 ry^2
312 se
313 se cp e
314 se sr e^{11}
315 st
316 th cu sr e^S ro ca
317 ve
318 ve^3

Chromosome 4

401 ci^D/ey^D
402 ci^{D-G} spa^{pol}/spa^{Cat}
403 ci^w
404 ci^{57g}
405 ci ey^R/spa^{pol}
406 ey^R
407 spa^{pol}

Multichromosomal

501 bw st
502 bw^{75} st
503 H^{57c}/Ubx^{61d};spa^{pol}
504 Su-er;er
505 Su-er tu bw;st er su-tu
506 v bw^D
507 v cn
508 vg bw st
509 vg spa^{pol}
510 y bw st
511 y bw st p^p

Duplications

701 $Dp(1;3)w^{m264-58a}+$;y w f Y^LY^S/y w/sc^8Y

Inversions

801 al dp b pr c px sp/Cy
802 ClB/fu
803 ClB/sc ec cv ct^6 v s^2 f car bb
804 crc/Pm
805 Cy L/Frd
806 Cy L^4/Pm
807 Cy/Pm Ly/D3
808 Cy/Pm Ly/D3
809 Cy/Pm Ubx/sb
810 Cy/Pm Ubx^{130}/Sb ci^{D-G}/ spa^{Cat}
811 dor/FM6 TM3/Sb
812 dp b pr cn vg_c a px bw mr sp/S Cy lt^3 pr^+ Bl cn^2 L^4 sp^2
813 ec ct^6 s car/FM6, y^{31d} sc^8 dm B
814 FM6/dor TM3/ry
815 FM6/sc ec cv ct^6 v s^2 f car bb
816 $In(1)w^{vC}X^{C2}$/In(1)49 y w lz^S, sc^8Y
817 Ly/In
818 M5 Cy"O"/Pm Ubx^{130}/Sb $ci^{D-J}spa^{pol}/spa^{Cat}$
819 M5 Cy"O"/Pm Ubx^{130}/Sb
820 ru h th st cu sr e^SPr cn/ TM1, Me ri
822 sc ec cv ct^6v g f/FM3, y^{31d} sc^8dm B_1
823 sc^{S1}In S w^a sc^8/FM6;SM5/ Bla TM3/Sb
824 sc^{S1}In S $w^a sc^8$;SM5/Bla TM3/Sb
825 sc^{S1}B In S $w^a sc^8$(=M5)
826 SD5/$SM_1 al^2$ Cy sp^2
827 SD72/SM5 al^2 Cy lt^V sp^2
828 SM5/Bla TM3/Sb
829 Su-H/Cy pr

Rings, attached-X and "sterilizer" Stocks

901 cn bw;e S2 "ster e"
902 lz^{c1} y:=
903 R(1)2 Y^B/C(1)Dx y f
904 R(1)2 w^{spont}v f/C(1)R M In(1)sc^{SIL} sc^8R+ S sc^{S1} sc^8w^a/Y 8-23
905 sc ct v wy f car y:=
906 su^3-S cv v f y f:=
907 X^{C2}f/y f:=
908 X^{C2} cv v f/ClB
909 y f:= y x ry
910 y f:= x mal
911 $Y^{LC}/X{\cdot}Y^S$ "ster +"
912 $Y^{LC}/X{\cdot}Y^S$, bw "ster bw"
913 $Y^{LC}/w{\cdot}Y^S$ "ster w"
914 y f:= ry x ry

Translocations

1001 bx^3 Cbx Ubx bxd pbx/Xa
1003 pbx/Xa
1004 $T(1;4)w^{m258-18}$ y
1005 $T(1;4)w^{m258-21}$ w;ey
1006 $T(1;4)w^{m258-21}$ y w^a/FM4, y^{31d} sc^8 dm B
1008 y w^{ch} spl;Cy;Ubx /Xa
1009 $T(2;3)bw^{VDe3}$Ubx bxd/In(3RL)Cx

VARANASI, INDIA: BANARAS HINDU UNIVERSITY
Department of Zoology

Wild Stocks

a) Canton-S
b) Oregon-R
c) Kerala

Chromosome 1

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

Chromosome 2

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

Chromosome 3

se cu
seh
ss^a
Ly/D_3

Chromosome 4

ey^2
ci^w

Multichromosomal

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

LONDON, ENGLAND: ST. BARTHOLOMEW'S HOSPITAL MEDICAL SCHOOL
Zoology Department, Genetics Laboratory

Wild Stocks

(a) mass mated
Kaduna
Oregon-K
(b) inbred by brother-sister mating
Kaduna
Oregon-K
(c) inbred like (b) and free of yeasts and bacteria

Chromosome 1

dlv
dm
dvr^2
fin
fla
gt w^a
lf
lh B car bb
mgt
mk
pun
rsi
rst^2
sla
sld
smd
slm
ty g^2

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

Wild Stocks	Chromosome 1	Chromosome 2	Chromosome 3
Buenos Aires	pn^2	pr	p
Oregon	w	ltd	e
Leningrado	w^e	cn	se
Brisbane	w^{bl}	bw	e se
Eldorado (Rio Grande do Sul, Brazil)	w^h	pd	p v
Some inbred strains	ras^2	vg	
	f	L	
	sc cv v f	st bw	
	y v		
	y		

CHALFONT ST. GILES, BUCKS, ENGLAND: CHESTER BEATTY RESEARCH INSTITUTE
Institute of Cancer Research, Department of Genetics

Wild Stock

Oregon-K

X-Chromosome

A: Maintained homozygously

1 B
2 cop
3 In(1)sc^{8} w^{a}
4 s
5 sc^{S1} In^{49} sc^{8}
6 sc^{S1} B In^{49} sc^{8}
7 sc^{S1} B InS w^{a} sc^{8}
8 w
9 y fa dwx g pl
10 y In^{49} B^{M1}
11 y sc^{S1} In^{49} sc^{8}
12 y sc^{S1} In^{49} w sc^{8}

B: Maintained heterozygous with sc^{S1} B InS w^{a} sc^{8} (M-5)

13 acc
14 amb^{2}
15 bis
16 br^{59j}
17 brc
18 ccw sma up mal^{bz}
19 cop brc Oce smd
20 dfw
21 dlv
22 drw^{2}
23 dsh
24 dvr^{2}
25 dwg
26 ec dx
27 f mal^{bz} bb
28 ff
29 fin
30 fla
31 gg^{3}
32 lac^{68h}
33 lf
34 mal^{60}
35 mel
36 mel v^{54k}
37 mgt
38 mk
39 nd
40 nd mo
41 nrs
42 obl
43 Oce
44 omm
45 ot^{2}
46 peb na
47 pun
48 r (complementing series)
49 rea
50 rey^{3}
51 rfr
52 rsi
53 rud
54 sc^{Fah}
55 shm
56 sl^{2}
57 sla
58 slc
59 sld
60 slm
61 smd
62 smd rud sl^{2} v^{54k} tc
63 splw^{2}
64 spx
65 sts
66 swb
67 tc
68 thv
69 Tu^{2}
70 unp
71 v^{2} fw
72 wa^{2}
73 ws^{2}
74 w spl
75 y ac fu

C: Maintained against attached-X y v f (or ClB)

76 bi ras amx
77 cm ny un
78 dm rux if
79 ec cv m f Bx
80 g ty r sy
81 kz sd od car
82 pn shf oc ptg
83 rg scp t sbr
84 sc ct wy g f car
85 sc v f car
86 sta rb cx lz
87 svr vs sn
88 tw dy
89 y In^{49} B^{M1}
90 sc v ClB/sc ct v f car

IInd Chromosome

91 al dp b pr
92 al dp b pr cn vg c a px bw mr sp/In(2L+2R)Cy
93 dp b cn bw
94 dp b pr stw(c)px sp
95 lt stw^{3}
96 ltd
97 In(2L+2R)Cy/Bl L
98 In(2L+2R)Cy/In(2LR)Gla
99 In(2L+2R)Cy/In(2LR)Pm^{2}

IIIrd Chromosome

100 Dl^{7}/In(3LR)Ubx^{130} Ubx^{130} e^{s}
101 In(3LR)sep ri p^{p}
102 ru h st e^{s}
103 se cp e^{s}
104 st in ri p^{p}
105 th st pb p^{p} kar su^{2} jvl ss bx sr gl/TM1 Me ri Sb^{1}
106 tu 36^{a} st sr e^{s} ro ca

IVth Chromosome

107 ar/ci^{D}
108 bt
109 ci^{361}
110 ci^{D}/ey^{D}
111 ey^{2}
112 gvl
113 M(4)^{62f}
114 M(4)^{63a}
115 sv^{n}
116 spa^{pol}

Y Chromosome

117 Oregon-K Y^{-bb}

Multichromosomal

118 car bb;Y^{-bb}
119 sc^{S1} B InS w^{a} sc^{8};Y^{-bb}
120 y v f:=;Y^{-bb}/Oregon-K;Y^{-bb}
121 y v f:=;Y^{-bb}/sc^{S1} B InS w^{a} sc^{8}; Y^{-bb}
122 Y^{S} X(FR1)Y^{S} y cv v f/RM y/B^{S} Y y^{+}
123 sc Y^{L}/y^{2} v f.Y^{S} y w f:=
124 In(1)sc^{8} w^{a};In(2L+2R)Cy/ In(2LR)Gla
125 In(1)sc^{8} w^{a};In(2L+2R)Cy/Bl L
126 y sc^{S1} In^{49} w sc^{8};In(2L+2R)Cy/ In(2LR)Gla
127 +/sc^{S1} B In^{49} sc^{8};dp b cn bw
128 In(1)sc^{8} w^{a};tu 36^{a} st sr e^{s} ro ca
129 y sc^{S1} In^{49} sc^{8};tu 36^{a} st sr e^{s} ro ca
130 dp b bw;In(LR)sep ri p^{p}
131 bw;e^{s};pol
132 dp;e^{s};pol

133 y v f;=;bw;e^s;pol
134 y sc^{S1} In^{49} w sc^8;dp;e^s;pol
135 y sc^{S1} In^{49} w sc^8;bw;e^s;pol
136 y v m f;bw;e^s;pol

BUFFALO, NEW YORK: STATE UNIVERSITY COLLEGE AT BUFFALO
Department of Biology

Chromosome 1

1 B_5
2 M^5
3 w
4 w m
5 w^a
6 y
7 y w m

Chromosome 2

8 b
9 b c
10 b vg
11 b vg bw
12 Bl/Cy
13 bw
14 dp
15 vg
16 S/Cy

Chromosome 3

17 In(3L)
18 ss^{a3}
19 st

Chromosome 4

20 ey^2
21 pol

Multichromosomal

22 $Cy/Pm^{ds33k};H/Sb\text{-}C^{(3)}$

VIENNA, AUSTRIA: UNIVERSITY OF VIENNA
Institut für algemeine Biologie

Wild Stocks

Oregon
Wien
Norwegen
Ponza

Chromosome 1

BB
Cl B/v ptg oc sn
fa^n

Muller-5
sn
w
w sn B
w^{bl}
y
y/w
y/f

Chromosome 2

b
b cn
b vg
b cn vg
bl
bw
cn vg
dp
dp b
L^2
L^4
L Cy/Pm
vg
cn

Chromosome 3

Ly/D
st

Chromosome 4

ey

Multichromosomal

bw;e
bw;st

LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY
Department of Zoology

Wild Stocks

1. Lexington, Kentucky, wild type

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

Melanotic Tumor Stocks

1 tu^{36a} st sr e^s ro ca
2 tu^{36a}
3 tu^{47}
4 tu^{47} Cy
5 tu^{48a}
6 tu^{49h} l(2)gl/Pm; se e^{11}
7 tu^{50d}
8 tu^{50i}
9 tu^{50j}
10 tu^{bs}
11 tu^{g}
12 tu^{h}
13 tu^{h} Cy
14 $tu^{i\text{-}21}$
15 tu^{w}
16 tu^{wps}
17 tu bw
18 tu bw^{55g}
19 tu bw B3
20 tu vg
21 tu vg bw
22 tu vg bw
23 tu vg bw sp
24 y^2;tu vg;ca
25 mt^A
26 mt^A sp
27 vg mt^A bw
28 e^{144} Israel
29 $f^{257\text{-}19}/In(1)AM$
30 w^{bf} $f^{257\text{-}5}$ (non-lethal)
31 y $B^{263\text{-}43}$
32 Su-er tu bw;st er Su-tu
33 $b(Su\text{-}er)^+$ bw;st er
34 Oregon-R (MDA 115)
35 Israel wild-type

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Biology

Note: Several hundred additional stocks are maintained.

Wild Stocks

2 Chicago

Chromosome 1

10 Bx^3 ma-1
28 lix
32 $ma\text{-}1^{bz}$
171 pn^{51h8} spl
47 sc z^{59d15}/C(1)DX, y f
48 sc z w^{z1}
49 sc z w^{zm}
67 v^{50j}
192 w^{+u} Etd f/FM-1G
159 y z w^{Bwx} w^{crr}/C(1)DX, y f
153 y ac w^{ch} w^{sp2} spl/C(1)RM, y w
150 w^{Bwx} w^{a} spl
411 y^2 su-w^{a} w^{a} w^{ch} spl/C(1)DX, y f
177 z w^{lle4}

Chromosome 1 Inversions

129 $In(1)sc^{4L}sc^{8R}$, y sc^4 sc^8 cv/C(1)DX, y f
127 $In(1)sc^{4L}sc^{8R}$+dl-49, y sc^4 sc^8 w^a/C(1)DX, y f
120 $In(1)sc^{8L}sc^{4R}$+S, w^a B/C(1)DX, y f; Dp(1;2)sc^{19}/In(2L+2R)Cy, Cy cn^2
406 $In(1)sc^{8L}sc^{L8R}$, w^a/C(1)Dx, y f;Dp(1;2)sc^{19}
407 $In(1)sc^{8L}sc^{S1R}$+S, B/C(1)DX, y f; Dp(1;2)sc^{19}
405 $In(1)sc^{8L}sc^{V2R}$, y^{31d} sc^{V2} f B/C(1)DX, y f
164 $In(1)sc^{L8L}sc^{4R}$, sc^{L8} sc^4 m car/C(1)RM, y w
174 $In(1)sc^{L8L}sc^{8R}$, sc^{L8} sc^8 car/C(1)DX, y f
87 $In(1)sc^{L8L}sc^{S1R}$+S, sc^{L8} B/C(1)DX, y f
203 $In(1)sc^{L8L}sc^{V2R}$, sc^{L8} sc^{V2} m car/C(1)DX, y f
304 $In(1)sc^{S1L}sc^{8R}$, y sc^{S1} sc^8 cv v f/C(1)DX, y f
84 $In(1)sc^{S1L}sc^{8R}$+dl-49, y sc^{S1} sc^8 w^a v f/C(1)DX, y f
110 $In(1)sc^{S1L}sc^{8R}$+S, sc^{S1} sc^8 w^a B bb^{83}/In(1)AM, y/B^SY
214 $In(1)sc^{S1L}sc^{L8R}$, y sc^{S1} w^a m/C(1)DX, y f
212 $In(1)sc^{S1L}sc^{V2R}$, y sc^{S1} sc^{V2}/C(1)DX, y f
409 $In(1)sc^{V2L}sc^{8R}$+dl-49, sc^8 v f B/C(1)DX, y f
408 $In(1)sc^{V2L}sc^{L8R}$, w^a/C(1)DX, y f;Dp(1;2)sc^{19}
316 $In(1)sc^{V2L}sc^{S1R}$+S, B/C(1)DX, y f;Dp(1;2)sc^{19}

Chromosome 1 Duplications and Deficiencies

148 $Df(1)w^{rG}$, w^- spl
138 $Df(1)w^{-59k13}$, w^{-59k13} spl; $Dp(1;3)w^{vco}$
152 y sc z w^{rdf} spl/C(1)DX, y f
154 y^2 w^{rdp} ec f
412 Dp(1;f)1337/X $Y^L.Y^S$, y w f/C(1)RM, y w
156 Dp(1;f)R6094b/Y^S $X.Y^L$, In(1)EN, y w·y^+/y^+Y/C(1)RM, y w

Ring 1 Chromosomes

162 R(1)2, cv v f/C1B

Reversed Acrocentric Chromosome 1

168 C(1)RA60g, + -$In(1)sc^8$+dl-49, y-Hw^- y^- sc^8 B/Dp(1;f)60g, y^{31d}/$Y^SX.Y^L$, y v
169 C(1)RA60g, + -$In(1)sc^8$, y -y^- sc^8 B/su(f)$^+$Y/y

X-Y Combinations

180 $XY^L.Y^S$, y w f/O/C(1)RM, y w
191 $Y^SX.Y^L$, In(1)EN, y w·y^{55f10}/O/C(1)RM, y w
193 $Y^SX.Y^L$, y w^a rb f/O/C(1)RM, y w

Y Derivatives

198 B^SYy^+/y v; bw
199 $bw^{54k}Yy^+$/y v; bw
200 $y^{54e}ac^{54e}$Y/y v; bw
202 w^+Y/y ac br pn w^e spl
205 Ybb^1 (Spofford)
218 $Y^L.sc^{S1}/X.Y^S$, w^a ec sn
221 $Y^L13/X.Y^S$, y f/C(1)RM, y w
222 $Y^L16/X.Y^S$, y ct^6 f/C(1)RM, y v bb;bw
223 $Y^L.sc^{S1}2/X.Y^S$, y f/C(1)RM, y w
224 $Y^L.sc^{S1}/X.Y^S$, y ac w^a ct^6 f/C(1)DX, y f
226 R(Y)L15/X·Y^S, y f/C(1)RM, y w
208 $sc^{V1}.Y^S/X.Y^L$, y v f bb/C(1)DX, y f
209 $Y^Sy^+5/X.Y^L$, B/C(1)RM, y w
211 $Y^Sy^+7/X.Y^L$, B/C(1)RM, y w
213 $Y^S.Y^S2/X.Y^L$, y v f/C(1)DX, y f
215 $Y^Sy^+bb^+8/X.Y^L$, y g^2 B/C(1)RM, y v bb; bw
216 $Y^S/X.Y^L$, y w^a rb

Chromosome 2

227 abo/Cy
250 $In(2LR)1t^{m3}$

Chromosome 3

410 c3G

Multichromosomal

225 C(1)RM; abo/Cy
310 lix; ry^2
311 lix; sf^2
312 ma-1; ry^2
313 pn; ry^2

289 y sn; c3G/D
341 T(2;3)lt^{m29}
342 T(2;3)lt^{m100}/lt stw^{3} l
54 y w; Su(var)
57 y w; Su(var)$^{+}$

S1 $Y^{S}X.Y^{L}$, In(1)EN, y w.y^{+}/C(1)RM, y w; Su(var)$^{+}$ Dp(1;3)N$^{264-58}$, w$^{m264-58}$/+
S2 y w; Su(var) Dp(1;3)N$^{264-58}$, w$^{m264-58}$/ Su(var)

HEVERLEE, BELGIUM: UNIVERSITÉ DE LOUVAIN
Faculté des Sciences Agronomiques, Laboratoire de Génétique

Inbred Temperature Lines

1 line raised at 25°C for 242 generations (Abeele, Belgium)
1 line raised at 25°C for 209 generations (Gabarros, Spain)

Wild Stocks

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

Chromosome 1

B
w
w^{a}

Chromosome 2

b
vg

Chromosome 3

e
ro
ve

Multichromosomal

b;ro
e;vg

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

Wild Stocks

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

Chromosome 1

101 B
102 car
103 cv
104 f
105 m
106 sc ec ct v g f
107 su (s)^{2} w^{a} cv t
108 v
109 w
110 w^{a}
111 w^{bf}
112 w^{e}
113 w sn^{3}
114 w^{co} sn^{2}
115 w^{ch} wy
116 wy
117 y cv v f
118 y w
119 z w^{11e4}

Chromosome 2

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

Chromosome 3

301 bx^{34e}
302 Dfd^{r-L}
303 e^{11}
304 jv se
305 ri
306 ru h th st cu sr e^{s} ca
307 st
308 Tu

Chromosome 4

401 ar/ey^{D}
402 bt ey^{R} sv^{n}
403 ci ey^{R}
404 ci gvl bt
405 Df (4) M/ey^{D}
406 ey^{e}

Multichromosomal

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

Inversions

601 In(1)Cl, sc l(1)t^{2} sl B/+ (=ClB)
602 In(1)dl-49, tyl bb^{1}/C(1)RM, y v f car
603 In(1)dl-49, y Hw w lz^{s}/dor^{1}
604 In(1)sc^{8} + dl-49, y^{Sl} v f B
605 In(1)sc^{8} + dl-49, y^{Sl} v f B;bw;e;spa^{pol}
606 In(1)sc^{SlL} sc^{8R} + S, sc^{Sl} sc^{8} w^{a} B (=M-5)
607 In(1)sc^{SlL} sc^{8R} + dl-49, sc^{Sl} sc^{8} v B car/C(1)DX, y f
608 In(2L)Cy/L^{2}
609 In(2LR)SM5, al^{2} Cy lt^{v} cn^{2} sp^{2}/Bl L^{2}
610 In(3L)D^{3}/Ly
611 In(3LR)TM3, y^{+} ri p^{p} sep Sb bx^{34e} e^{s} Ser/Me^{65d} h th
612 In(1)sc^{SlL} sc^{8R} + S, sc^{Sl} sc^{8} w^{a} B; In(2LR)SMl. al^{2} Cy cn^{2} sp^{2}/In(2LR)bw^{V1}, dp b bw^{V1} ds^{33k};In(3LR)Ubx^{130}, Ubx^{130} e^{s}/ C Sb;spa^{pol}

Attached XX's and XY's

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

Selected Stocks

801 Berlin wild, DDT-resistant 1

AMHERST, MASSACHUSETTS: AMHERST COLLEGE
Department of Biology

Wild Stocks

1 Oregon-R: Inbreeding; generation 588 on 70k11
2 Oregon-R: 100, mass culture, from $ #1 at generation 100
3 Oregon-R: 200, mass culture, from $ #1 at generation 200
4 Oregon-R: 300, mass culture, from $ #1 at generation 300
5 Oregon-R: 400, mass culture, from $ #1 at generation 400
6 Oregon-R: 575, mass culture, from $ #1 at generation 575
7 Samarkand 204: inbred for 2004 generations: mass culture since 53h4

Chromosome 1

8 B
9 Basc: sc^{S1} sc^{8} w^{a} B
10 cm
11 cm ct^{6}
12 cm ct^{6} sn^{2}
13 cm ct^{6} sn^{3}
14 cm ct^{6} sn^{3} oc/y f:=
15 cm ct^{6} sn^{4}
16 cm ct^{6} sn^{4} oc ptg/y f:=
17 cm ct^{6} sn^{5}/y f:=
18 cm ct^{6} sn^{34e}
19 cm ct^{6} sn^{36a}/y f:=
20 ct^{6} oc/y f:=
21 ct^{n} oc/y f:=
22 cx
23 ec rb^{64f14} cv/y f:=
24 Ext/FM6
25 g
26 g_{2} sd
27 g^{2} pl
28 g^{3}
29 g^{3} sd
30 g^{50e}
31 g^{53d}
32 g^{53d} sd
33 g^{w}
34 m g^{53d}
35 oc/y f:=
36 oc ptg/y f:=
37 ras_{2} dy
38 ras^{2} f
39 ras^{2} m f^{69115}/y f:=
40 sc z swb^{62b12} ct^{6}
41 sd^{58d14}
42 sn_{2}/y f:=
43 sn^{2}
44 sn^{2} oc ptg^{3}/y f:=
45 sn^{3}
46 sn^{3} g^{53d}
47 sn^{3} oc/y f:=
48 sn^{4}
49 sn^{4} oc ptg^{3}/y f:=
50 sn^{36a}/y f:=
51 swb^{62b12}
52 un od
53 v
54 w
55 w/+.=
56 w sn^{5}/y f:=
57 w^{a}
58 w^{e}
59 wy^{2}
60 wy^{2} g
61 wy^{2} g^{2}
62 wy^{2} g^{3}
63 wy^{2} g^{4}
64 wy^{2} g^{53d}
65 wy^{2} g^{53d} sd
66 y ct^{6}
67 y ct^{6} ras^{2} f
68 y w^{sp}/FM6
69 y w^{a} m f car
70 y^{2} sn^{3} ras^{4} m/B.Basc

Chromosome 2

71 a px or
72 a px or If
73 a px sp
74 al cl b c sp^{2}
75 al cl nub sp^{2}
76 al lt stw^{3} sp^{2}
77 b
78 b If
79 b Bl vg bw/Cy, bw^{45a} or^{45a} sp^{2}
80 b Bl vg bw/Roi, bw^{45a} or^{45a} sp^{2}
81 Bl/SM5
82 Bl L^{2}/Cy, sp^{2}
83 bw
84 cl
85 cn bw
86 Cy, dp^{lvI} Bl L^{4} sp^{2}/Pm, ds^{33k}
87 ho
88 L^{4}
89 lt stw^{3}
90 ltd
91 net^{38j} b^{38j} cn^{38j} bw^{38j}
92 nub^{2}
93 or^{45a} sp^{2}
94 or^{49h}
95 sca^{65131}
96 sca l(2)C/SM5, al^{2} Cy lt^{v} sp^{2}
97 vg^{51h25}
98 vg^{-B}/Cy L^{4} sp^{2}
99 vg^{nw}/Roi, bw^{45a} or^{45a} sp^{2}
100 vg^{U}/Roi, bw^{45a} or^{45a} sp^{2}

Chromosome 3

101 bar-3
102 cu
103 D/Gl
104 DcxF, ru h ca/Tm3, ru ri p^{P} Sb sep bx^{34e} e^{s} Ser ca
105 D^{3} Sb/Payne
106 Dl^{7}/Ubx^{130} e^{s}
107 gl^{3}
108 h
109 h st
110 h th st pP cu sr e^{s}
111 rsd
112 ru h th st cu sr e^{s} ca
113 ru h st cu sr e^{s} ca
114 ru h st p^{P} ss Su^{3}-ss e^{s}
115 ru sr Dl^{61115} e^{s} ca/DcxF ru h ca
116 ru st ss ca
117 se^{50k}
118 se ss
119 se ss Su^{3}-ss
120 ss
121 ss^{ax}
122 ss bx Su^{2}-ss
123 TM1, Me ri sbd^{1}/D^{3}
124 TM3, ri pP Sb sep bx^{34e} e^{s} Ser/D^{3}

Chromosome 4

125 ci ey^{R}
126 ci^{D}/ey^{D}
127 ey^{2}
128 spa^{pol}

Multiple chromosomes

129 ras^{4} m/y f:=;Bl/Cy, bw^{45a} or^{45a} sp^{2}
130 +/y f:=;bw;e;spa^{pol}
131 bw;e
132 bw;h

133 bw;e;spa^{pol}
134 cn bw;se^{50k} e^{60h}
135 Cy, bw^{45a} or^{45a} sp^{2};TM3, Sb e Ser/T(2;3)bw;h
136 Cy, L^{4}, sp^{2};se^{50k}/T(2;3), Bl vg^{51h25};se^{50k}
137 Cy, SM5;TM1, Me ri sbd^{1}/T(2;3)gl^{63d29}
138 Cy, sp^{2}/Pm, dp b;DcxF/Sb sr
139 net or^{45a} sp^{2};ru bv
140 vg^{51h25};se^{58k}
141 vg^{51h25} bw;se^{50k} e^{60h}
142 vg^{51h25} sp^{2};se^{50k}
143 vg^{51h25};se^{50k};spa^{pol}
144 Ubx^{130} e^{s}/T(2;3)Xa

Deficiencies

145 Df(1)g^{1}, f B/In(1)AM
* Df(2)vg^{-B} in stock 98

Inversions

* In(1)AM in stock 145
* Ins(1)Basc: sc^{S1} sc^{8} w^{a} B(Muller-5) in stocks 9, 70
* In(1)FM6 in stocks 24, 68, 150, 151, 152, 153
* In(2LR)bw^{V1} in stocks 86, 138
* In(2L+2R)Cy in stocks 79, 82, 86, 96, 98, 129, 135, 136, 137, 138
* Ins(2L)t, (2R)Cy in stocks 80, 99, 100
* In(3LR)DcxF in stocks 104, 115, 138
* In(3R)Mo, Sb sr in stock 138
* In(3L+3R)LVM in stock 105
* In(3LR)TM1 in stocks 123, 137
* In(3LR)TM3 in stocks 104, 124
* In(3LR)Ubx^{130} in stocks 106, 144

Trasnlocations

146 T(2;3), bw;h
147 T(2;3)gl^{63d29}, bw
148 T(2;3)gl^{63d29}, bw;e^{60h}
149 T(2;3)gl^{63d29}, se^{50k}
* T(2;3), Bl vg^{51h25};se^{50k} in stock 136
* T(2;3)gl^{63d29} in stock 137
* T(2;3)Xa in stock 144

Triploids

150 cm ct^{6} sn^{3}/FM6, y^{31d} sc^{8} dm B & FM6
151 cv rb^{64c10} ec/FM6, y^{31d} sc^{8} dm B & FM6
152 g^{53d} sd/FM6, y^{31d} sc^{8} dm B & FM6
153 wy^{2} g^{H}/FM6, y^{31d} sc^{8} dm B & FM6

PATRAS, GREECE: UNIVERSITY OF PATRAS
Department of Genetics

Chromosome 1

y
w
pn
Muller-5

Chromosome 2

dp
b
cn
Vg
bw
CyL^{4}/Pm

Chromosome 3

st
ss
e

Chromosome 4

ey

Multichromosomal

dp b;cn bw

IBADAN, NIGERIA: UNIVERSITY OF IBADAN
Department of Zoology

Wild Stocks

Oregon-R

Chromosome 1

ClB/w m f
ClB/cv v f
cv m f
y cv v f
y w^{a} cv v f
w

Chromosome 2

b
b cn vg
bw
cn
dp
vg

Chromosome 3

D/Payne
e
gl
ri
ro
se
se e
se ri e
se ri
st

Multichromosomal

al L^{4} cy sp/Pm;H/Sb sr In(3R)Me (2,3)
bu;e^{4} wo ro (2,3)
bw;st (2,3)
cy sp^{2}/Pm dp b;Sb/D CxF (2,3)
dp;e (2,3)
dp;se e (2,3)
vg;e (2,3)

MILANO, ITALIA: UNIVERSITA' DI MILANO
Istituto di Genetica

Wild Stocks

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

Chromosome 1

18 B
19 N^{B-S}
20 ptg^2
21 sc ec ct v g f
22 v
23 sd
24 w^a
25 w^{bl}
26 w^e
27 y w
28 od
29 abw
30 w m f

Chromosome 2

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

Chromosome 3

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

Multichromosomal

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

X Chromosome Inversions

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

Chromosome 2 Inversions

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

Chromosome 3 Inversions

77 H/Sb sr In(3R)Me
78 ltr/Sb sr In(3R)Me
79 Me ca/ru cu ca
80 ve h th C3 Sb Ubx/st C3 G ca
81 Florida In(3R)Payne

Multichromosomal Inversions

82 Cy L^4 sp/Pm;H/Sb sr In(3R)Me
83 y w;Cy L^4 sp/Pm;H/Sb sr In(3R)Me
84 y sc^{S1} In-49 sc^8;bw;st p^p
85 bsp/bsp;Sb Me/H

Deficiencies

86 $Df(2)Px^2$ Df(2)Px, bw sp/SMl, al^2 Cy sp^2
87 $Df(2)bw^5$ $Df(2)bw^5$ sp^2/Xa
88 Minute (2) Bridges
89 M92)33a/al^2 In Mis Cy cn^2 sp^2

Translocations

90 $T(1;4)B^S(16A_1)$, y^2 cv v B^S car/y f:=

Special Stocks

91 "sz e" $Y^{Lc}/X.Y^S$ & y v f.=;e
92 "szw" $Y^{Lc}/X^w.Y^S$
93 $\frac{y^2\ su(w^a)w^a\ bb}{X.Y}$/v f B,

Stocks selected for tumor manifestation

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

VILLEURBANNE, LYON, FRANCE: FACULTE DES SCIENCES
Biologie générale et appliquée

Wild Stocks	Chromosome 1	Chromosome 2	Chromosome 3	Multichromosomal
Lyon	y	cn	e	st;bw
Algérie	w	bcn vg bw		v;bw
	v			cn;bw

COLD SPRING HARBOR, NEW YORK: CARNEGIE INSTITUTION OF WASHINGTON

Note: Stocks are maintained primarily for distribution to students and teachers interested in performing the experiments outlined in Drosophila Guide, by M. Demerec and B.P. Kaufmann, published by Carnegie Institution of Washington (8th edition, 1968).

Wild Stocks*

1 Canton-S^{2}
2 Oregon-R
3 Oregon-R-EL^{2} (from East Lansing)
4 Swedish-b^{8}

* Superscript numerals refer to successive subcultures (e.g., the eight for Sw-b) from a single pair whose F_1 progeny were examined cytologically to determine absence of gross chromosomal aberrations.

Chromosome 1

5 B
6 bi
7 ec ct^{6} v g^{3}
8 ec ct^{6} v g^{3}/ClB
9 f
10 fw^{H}/y
11 m
12 v
13 w
14 w m f
15 w m f/ClB
16 y^{2} w^{a} cv v f B
17 y^{2} w^{a} spl
18 y^{2} w^{a} spl bi

Chromosome 2

19 bw
20 dp
21 c
22 L^{2}
23 L^{5}
24 vg

Chromosome 3

25 e
26 p^{p} by Sb^{Spi}/In(3R)C, l(3)a
27 se
28 se ss
29 st

Chromosome 4

30 ey^{2}

Multichromosomal

31 Ins(2LR)Cy/Pm^{1}, ds^{33k}; H/In(3R)C, Sb
32 Ins(2LR)SM1, al^{2} Cy sp^{2}/ Pm^{2}, Ubx^{130} e^{s}/Sb
33 y f;bw;e;ci ey^{R}
34 y;bw;e;ci ey^{R}

Inversions

35 In(1)A99b
36 In(1)sc^{S1} B InS w^{a} sc^{8} (Muller-5)
- In(1)ClB (8, 15)
37 In(1)rst^{3}, y rst^{3} car bb
38 Ins(2LR)Cy/L;Pm
- Ins(2LR)SM1, al^{2} Cy sp^{2}/ Pm^{2};Ubx^{130} e^{s}/Sb (32)
39 In(3L)pers
- p^{p} by Sb^{Spi}/In(3R)C, l(3)a (26)

Translocations

40 T(2;3)S^{M} Cy/vg^{nw}
41 Y^{S}X.Y^{L}, +/y^{2} su-w^{a} bb; Ore-R autosomes

Closed-X

42 X^{c2}

Attached-X

- fw^{H}/y (10)
- Y^{S}X.Y^{L}, +/y^{2} su-w^{a} w^{a} bb; Ore-R autosomes (41)
- y f;bw;e;ci ey^{R} (33)

LONDON, ENGLAND: BIRKBECK COLLEGE
Department of Zoology

Wild Stocks

Oregon-R
Oregon +

Chromosome 1

M-5
w
w m f
w^{a}
v
y

Chromosome 2

a px
a px pd bw
bw
bw cn
Cy/Bl L^{2}
Cy/el b pr lt ltd cn a px pd bw
el b
vg

Chromosome 3

ss
ss^{a}
se ss

Chromosome 4

ey^{2}

Multichromosomal

bw;e
bw;ss^{a}

Inversions

In(1)sc^{S1}L$_{sc}^{4R}_{sc}^{S1}_{sc}$^{4}cv v B/C(1)DX y f/B^{s} Y
In(1)sc^{4L}$_{sc}^{8R}_{y}$ sc^{4} sc^{8}cv v B/C(1)DX y f/B^{s} Y

Attached-X

y/w^{bl} wy f

BRIGHTON, ENGLAND: UNIVERSITY OF SUSSEX
Biology School

Wild Types

Edinburgh
Nettlebed
Oregon
Oregon K
Samarkand

Chromosome 1

amx lz^g v/y f:=
C(1)RM y pn ♀ x dor/y ♂
C(1)RM y pn ♀ x v ♂
ct^n oc/FM1, y^{31d} sc^8 w^a lz^s B
ma-1 x y f:=
Muller-5
N^8 Head I - 2
N^{45e}/d149
In 49 lz^s x y f:=
r - 9
r I
r I - IV
r II - III
r IV - V
sn lz v/y f:=
v
w
w m f
w sn m
y pn
y 1308f

Chromosome 2

ab
Edinburgh tu
fs 29 fs(2)E_1 dp^+ Sp/S Cy (In 2LR)cn L^4 sp^2
fs 30 S fs(2)E_1 Alu lt/al^2 Cy(In 2LR)cn^2 L^4 sp^2
1(2)gl: cn bw/SM5, al^2 Cy lt^v sp^2
1(2)gl: a px bw/SM5, al^2 Cy lt^v sp^2
1(2)gd/dp^{tx-1} Cy lns O pr cn^2
1(2)gd: a px or/dp^{tx-1} Cy Ins O pr cn^2
1(2)gl b/SM5, al^2 Cy lt^v sp^2
Sp J L^2 Pin/SM5, al^2 Cy lt^v sp^2
Spd gt-4/SM5, al^2 Cy lt^v sp^2
tu-bw^{55g}
tu 48^a
tu 36e
tu 5od
tu^g
tu-W rc
vg

Chromosome 3

e ss^a
pb/In(3LR)Cx
pp
ry^2 ry^2
Sb/+
se cp e
tet^{Bd} bilat

Chromosome 4

Pacific ey^k

Multichromosomal

ant x ey^k
car K-pn
dp ey^2
Ns-b mr/Cy
SM5/Bl L^2;st/st
tu bw;+ su-tu
tu bw suer;st su-tu er
tu^k TM3/Sb
tu 36a;st sr e^s ro ca

Inversions

In(3R)$Antp^B$, $Antp^B$/TM1, me ri sbd^1

CLEVELAND, OHIO: CASE-WESTERN RESERVE UNIVERSITY
Department of Biology

Chromosome 1

a1 B
a2 m
a3 sc cv v f
a4 w
a5 y w m f/y f:=
a6 y
a7 v
a8 f
a9 y^2 w^a v
a10 y cv v f
a11 y w m f

Chromosome 2

b2 b
b3 bw
b4 cn
b5 cn bw
b6 dp
b8 ltd
b10 vg
b11 b cn c bw
b12 b cn vg

Chromosome 3

c1 e
c3 h
c6 st
c7 ca
c8 e se
c9 ve h th
c10 se
c11 th st

Chromosome 4

d2 ey^2

Chromosomes 1-2

e3 v;bw
e4 f;dp

Chromosomes 1-3

h1 w;e

Chromosomes 2-3

f2 bw;st
f4 ltd;h
f6 ho;ca
f7 vg;ca
f8 b;ca

Chromosomes 1,2,3,4

g1 y;bw;e;ci ey^R

Translocations

t1 T(Y;2)C/pr cn

CAMBRIDGE, ENGLAND: UNIVERSITY OF CAMBRIDGE
Department of Genetics

Note: Only stocks not generally available are listed.

High sternopleural chaetae lines (Thoday):

dp1
dp2
vg4
vg6

Wild stocks

Hd-22
Histon
Barton

Dronfield
Aires

Chromosome 2

b cn vg
dp cn bw

Chromosome 3

h eyg th st cp
se cp e
th cp

th st cp
ve h eyg cp
ve h eyg cp e
ve se cp e

Multichromosomal

y;bw;st
FPU (FM6;Ins(2LR)Px^{4};Ubx^{130} segregating on Oregon background).
YCS (SM5;TM3SbSer segregating on Oregon background).

LEICESTER, ENGLAND: THE UNIVERSITY OF LEICESTER
Department of Genetics

Wild Stock

Oregon K

Chromosome 1

w
w^{a}
y v f
y w (attached X)

Chromosome 2

dp
cn
vg
bw
bw^{75}
bw cn

Chromosome 3

se
st
e

Chromosome 4

ey^{2}

Multichromosomal

bw st
bw^{75} st
bw e

Marker Stocks

M-5
Cy L^{4}/Pm;H/Sb

EDINBURGH, SCOTLAND: INSTITUTE OF ANIMAL GENETICS

Wild Stocks

Pacific
Oregon-K

Chromosome 1

B
ClB/sc v f car
ClB/w sn
ct v g
fw d
m
Pn^{2}
sc w ec cv ct
w^{a}
v
v g f
w
w m
w sn
w sn B
O - 1

y w
y w sn
y m
w^{e}
sc w ec cv ct v f car

Chromosome 2

al dp b pr c px sp
b cn vg
bw vg
cn
Cy - O/Bl L^{2}
bw
dp
ft/ft
a px or
S. O.
sp^{2} bs^{2}
stw
Cy/+
vg

Chromosome 3

Gl Sb/D In Cx F
se
e
st
th
h

Attached X

lz^{3} & y f.=
sc ct v f car & y v f.=
sc rb ct m & y y.=
sc^{S1} B InS w^{a} sc^{8} & y v f.=
w & y w f.=
xx bw st

Y Chromosome

f.Y^{S}/sc.Y^{L} & y w f.=
f.Y^{S}/Y^{L}
y v f.Y^{L}/Y^{S} & f.=

Multichromosomal

y ac sc pr
bw;e
bw;st
Y dp bw st p^{p}
Cy/Bl L^{2};D/LVM
dp;e
sc^{S1} InS w^{a} sc^{8};dp;e
sc^{S1} InS w^{a} sc^{8};vg;e
sc^{S1} InS w^{a} sc^{8} Cy/Bl L^{2}
sc^{S1} & y.=;bw;e;ey
v;bw^{vA}/Bl L^{2}
vg;e
w;bw
y sc^{S1} In49 sc^{8};Cy/Bl L^{2}
y sc^{S1} In49 sc^{8};bw;st
y sc^{S1} In49 sc^{8};dp b cn bw
M-5 dp e
M-5 Cy-e

UTRECHT, THE NETHERLANDS: GENETISCH INSTITUUT DER RIJKSUNIVERSITEIT

Chromosome 1

101 car$^{26-48}$ f^{3N} & y f:=
102 cm ct^{6} sn^{3} & y w f:=
103 cs^{53} & y w bb•=
104 ct^{n} oc lz^{3} v/FM$_{6}$ ct^{6} v
105 Df(1)N^{8}/dl-49, y Hw m^{2} g^{4}
106 fu/ClB
107 In(1)w^{vo}, X^{C2}/In(1)49, y w ly^{s} & sc^{8} .Y
108 mal
109 sc^{S1} B InS w^{a} sc^{8} "Muller-5"
110 sc cv v f
111 sc ec cv ct^{6} v g^{2} f/FM$_{6}$
112 w
113 w cv sn
114 w^{cv} sn^{2}
115 X^{C2}, y B & y f:=
116 y pn
117 y^{2} v mal^{b2}
118 y w^{a} cv v f
119 y w m B
120 y w spl
121 z^{58} g

Altered Y's

150 sc .U/y In-49 B;bw ♂ & y f:=;bw^{D} "Multi ♂"
151 X•Y In EN y;st (no free Y) "Multi ♀"
152 X^{Lc}/X•Y^{S};+ "sterilizer +"
153 Y^{Lc}/X•Y^{S};bw "sterilizer bw"
154 Y^{Lc}/X•Y^{S};dp "sterilizer dp"
155 Y^{Lc}/X•Y^{S};cn bw;e "sterilizer cn bw;e"

Chromosome 2

201 b cn vg
202 b cn vg bw
203 b pr cn vg
204 Bl L/Cy
205 bw
206 cn bw
207 dp
208 dp b cn bw
209 dpTh Cy cn bw/S Sp cn bw
210 dpTh Cy, In-L pr en^{2} In Cy R-O/Ins-NSL Ins-NSR p x sp "Cy O"
211 J/In(2L)t, l(2)B
212 SD-72/SM$_{5}$, al^{2} Cy lt^{4} sp^{2}

Chromosome 3

301 bxd
302 ca K-pn
303 Cbx
304 cn
305 e
306 h ri
307 l tr/e In(3R)In(3L)
308 Dr^{Mio}/In(3R)Sb
309 ry^{2}
310 sbd^{2} bx^{3} pbx/TM$_{1}$ Me ri sbd
311 ss
312 st
313 st e
314 st Sb^{r} e^{s} ro ca "Oster-5"
315 st ss
316 st ss e
317 T(2;3)Ubx^{105}/TM$_{1}$ Me ri sbd^{1}

Chromosome 4

401 ci
402 ci^{D}/spa^{Cat}
403 ci^{D-G1} spa^{pol}/spa^{Cat}

Multichromosomal

501 cn bw;e
502 M-5(B^{+});Cy O/Pm;Ubx^{130}/Sb
503 Cy/Pm;C x, D/In(3R)Sb
504 Dp(1;3)w$^{m264-58a}$/+;y w f Y^{L}•Y^{S}/y w/sc^{8}•Y
505 Df(3)Ubx^{109}/Xa
506 FMA 3, y^{2};sbd^{2} bx^{3}/Xa
507 pbx/Xa
508 T(1;4)w$^{m258-18}$, y
509 T(1;4)w$^{258-21}$/w;ey
510 T(1;4)w$^{m258-21}$ y w^{a}/FM$_{4}$, y^{31d} sc^{8} dm B
511 w^{a};tra/D In sc x F & y v f:=;tra/D In sc x F
512 y;bw;st p^{p}
513 y sc^{S1} In-49 sc^{8};dp b cn bw

Stocks selected for abnormal abdomen

(AA) DC x F/Me Sb
(AA) Cx, D/In(3R)Sb

SEOUL, KOREA: EWHA WOMANS UNIVERSITY
Department of Science Education

Wild Stocks

1 Anyang (Korea)
2 Canton-S
3 Choonchun (Korea)
4 Chungjoo (Korea)
5 Daijun (Korea)
6 Daikoo (Korea)
7 Jungphung (Korea)
8 Koonsan (Korea)
9 Kuje (Korea)
10 Kwangjoo-Chunnam (Korea)
11 Kwangjoo-Kyunggi (Korea)
12 Namhai (Korea)
13 Oregon-R
14 Pusan (Korea)
15 Quelparts (Korea)
16 Mapo-Seoul (Korea)
17 Sinchon-Seoul (Korea)
18 Yusoo (Korea)

Chromosome 1

19 w
20 y

Chromosome 2

21 cn bw
22 vg

Chromosome 3

23 cu
24 e
25 sb^{w}
26 se

Chromosome 4

27 bt
28 ey

Inversion

29 Muller-5

SD

30 SD-72
31 SD^{NH}12
32 R-1
33 R(SD^{NH}-1)-1

LEIDEN, THE NETHERLANDS: GENETISCH LABORATORIUM DER RIJKSUNIVERSITEIT

Wild Stocks

1 Kolmar
2 Leiden
3 Madeira
4 Alicante

Chromosome 1

101 B
102 FM6/sc ec cv ct^{6} v s^{2} f car bb
103 cv
104 ec
105 lz^{cl}/Muller 5
106 m
107 Muller 5
108 sn^{3}
109 v
110 w
111 w B sn
112 w cn sn^{3}
113 w:= & +
114 w:= & y^{2} su-w^{a}
116 y:= & sc ct v w y f car
120 z^{58g}

Chromosome 2

201 al dp b pr c px sp
202 al dp b pr c px sp/Cy
204 Bl L/Cy
205 bw^{60g}
206 bw^{D}
208 cn
209 cn bw
210 cn bw Kr/SM5
211 cn bw vg
212 crc cn/Pm
213 Cu^{270}/Pm
214 dp
215 dp stw^{3} bw
217 M(2)^{58a}/CyO
218 px sp
219 pys
220 sca
222 vg
223 vg bw

Chromosome 3

301 Bd^{G}/In(3R)C 1(3)a
303 Dr^{Mio}/Sb
304 e
305 fz
306 Gl/Ubx^{130}
307 h
308 h gs th
309 H^{57c}/Ubx^{61d}
310 Hu bx^{3} bxd pbx/Tml Me ri sbd^{1}
310a Hu Df(3)bxd^{100}/Tml Me ri sbd^{1}
311 ld
312 ltl/Tm3
312 ltr/Sb sr In(3R)Me
314 Ly/D^{3}
316 pyd
317 R/TM3
318 ro Bd ca/In(3R)C 1(3)a
319 ru h th st cu sr e^{s} Pr ca/TM1 Me ri
321 se ss k e^{s} ro
322 ss^{a}
323 ss^{a} B
324 ss^{ak}
325 st
326 Ubx^{130}/Sb
327 ve

Chromosome 4

401 ar^{57d}/ci^{D}
403 ci^{57g}
404 ci^{D} spa^{pol}/spa^{cat}
405 ci^{D}/ey^{D}
406 ci^{D-G}/ey^{D}
408 ci^{w}
410 ey^{opt}
411 ey^{R}
412 spa^{pol}

Multichromosomal

501 Cy/Pm;D/Sb
502 Cy/Pm;Ly/D^{3}
503 CyO/Pm;Ubx^{130}/Sb;ci ey^{R}
504 dor/FM6;TM3/Sb
505 ec br;ix/Cy
506 eyr/Sb;ey^{4}
507 Hw^{49c}/FM6;TM3/Sb
509 or;ci^{57g}
510 sc^{S1} In(1)S w^{a}sc^{8};SM5/Bla; TM3/Sb
511 SM5/Bla;TM3/Sb
512 v;cn
513 vg;spa^{pol}
514 y^{58a};Cy/Kr
515 y;bw;st
516 y;ec w^{ch} spl;$\frac{Cy;Ubx^{130}}{Xa}$
517 y;se

Altered Y's

551 Y^{Lc}/X·Y^{s} "sterilizer +"
552 Y^{Lc}/X·Y^{s};spa^{pol} "sterilizer spa^{pol}"
553 Y^{Lc}/X·Y^{s};vg;spa^{pol} "sterilizer vg;spa^{pol}"

SANTIAGO, CHILE: UNIVERSIDAD DE CHILE, FACULTAD DE MEDICINA
Departamento de Genética

Wild Stocks

1 Oregon R-c
2 Rapel
3 Santiago
4 Antofagasta
5 Pomaire
6 Chillán
7 Valdivia
8 Las Vizcachas
9 Swedish free of inversions
10 Perú (Lima)
11 Bolivia

Chromosome 1

12 B
13 my
14 sc^{S1} B InS w^{a} sc^{8} Basc
15 v
16 w
17 w m f
18 y
19 sc cv vf
20 car
21 pn^{2}
22 cm
23 t

Chromosome 2

24 b
25 bw
26 dp
27 L^{2}
28 Cy
29 vg

Chromosome 3

30 e^{11}
31 se
32 st
33 ro

Chromosome 4

34 ey^{2}

Multichromosomal

35 dp - e^{11}
36 w vg
37 bu st
38 st sr ro ca tu cs^{5} 36a

READING, ENGLAND: UNIVERSITY OF READING
Department of Agricultural Botany

Wild Stocks

1 Oregon
2 Samarkand

Chromosome 1

3 g^{53d}
4 w
5 w^a
6 w m B
7 y
8 y v f
9 y w^a cv v f

Chromosome 2

11 b cn
12 b cn vg
13 b cn vg bw
14 b pr vg
15 cn
16 el b
17 vg bw

Chromosome 3

18 e
19 eyg
20 ro ca bv
21 ry
22 se e
23 st

Multichromosomal

24 w;vg
25 w;vg;se
26 w;se
27 v;bw^D
28 v;cn;st
29 v f;vg
30 v;se
31 v;vg
32 bw;st
33 vg;se
34 vg;se e

MACOMB, ILLINOIS: WESTERN ILLINOIS UNIVERSITY
Department of Biological Sciences

Wild Stocks

Oregon-R

Chromosome 1

sc^{SI} B^{InS} w^a sc^8

Chromosome 2

bw
c
dp
vg

b pr c px sp
Cy(no Ins)ld/ds^{33k} Pm

Chromosome 3

st
e

STOCKHOLM, SWEDEN: UNIVERSITY OF STOCKHOLM
Institute of Genetics

Revision to DIS 44 (1969): 19-20

Deletions: 102, 110, 140, 142, 218, 225, 309, 313

Additions: 6-17: twelve different wild types.

143b y^2 sc ec ct^6 v f^5 x C(1)DX, y f
143c y^2 sc ec cv ct^6 v f^5 car·Dp(1;1)sc^{Vl}, y^+
205b ap^4/In(2LR)Rev^B
307b ry^2
502b In(2LR)Cy + O, Cy dp^{lvl} pr cn^2/T(2;3), In(3LR)C x F, D f(2R)P, S Sp/Dp(2;3)P, Dl·H e("sifter O")

Alterations

107 F M 7a, y^{31d} sc^8 w^a v^{Off} B
122 R(1)2, y B/y^+ Y x C(1)D X, y f/y^+ Y (Ring O.K. 1969)
143 y^2 sc ec ct^6 v f^5·Dp(1;1)sc^{Vl}, y^+
155 y w^a f/y w^a f/B^S Y y^+ & y w^a f/B^S Y y^+
206 b cn vg;y/y^+ Y
214 In(2L)Cy, al^2 ast^3 b pr cn vg (homoz)
216 In(2LR)Gla, Gla/Cy pr cn sp
503 Cy/T(2;3)ap^{Xa}, ap^{Xa}/T M 2, U bx^{130} e^S

LIVERPOOL, ENGLAND: UNIVERSITY OF LIVERPOOL
Department of Genetics

Chromosome 1

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

Chromosome 2

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

Chromosome 3

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

Multichromosomal

y;bw;st
yy;bw;st
M-S;Ly/Me
y;Cy L^4/Pm;st
Cy L^4/Pm;Sb/H

HELSINKI, FINLAND: UNIVERSITY OF HELSINKI
Department of Genetics

Wild Stocks

3 Haaga
4 Oregon-K
5 Oregon-R-S
6 Porvoo
7 Swedish-b
In addition 10 wild stocks collected from different localities in Finland

Chromosome 1

8 B
10 bi ct^6 g^2
10a ec ct v f
11 f
12 fu/ClB
13 g^2 f B/y
14 In(1)dl-49, y fa^n
16 In(1)sc^4, y sc^4
17 In(1)w^{m4}
18 In(1)sc^{S1L}, s, sc^{8R}, w^a B
21 rb cx
22 s
23 sc cv v f
24 sd (;se)
25 sn^3
26 spl
27 w
29 w^{ch} wy
30 y ac v
31 y In(1)dl-49 f car/y f:=
33 y v f
34 z

Chromosome Y

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

Chromosome 2

39 al dp b pr c px sp/Cy al^2 lt^3 L^4 sp^2
42 Bl L^2/Cy
43 cn^2 InCyR cg sp^2/ InsNS px sp
45 dp^{tx} Sp cn^2/S^2 Cy cn^2 (homoz. InCyR)
48 fj px
50 Ns, b mr/Cy
51 rl
52 rl
53 stw^2
54 vg
89 rn/Cy Bl cn^2 L^4 sp^2
90 rn In(2R)M/Cy cn^2 sp^2

Chromosome 3

55 Bd^G/In(3R)C, l(3)a
56 D^3/Payne
57 app
58 e^{11}
59 Gl Sb/LVM
60 In(3R)Dl^B/In(3R)P^W, stl(3)W ca
61 In(3R)P^{FLA} (homozygous)
62 Ly Sb/LVM
63 Me, InL Sb/ru h D InsCXF
65 se
66 se app
67 se rt^2 th/Me, InL
68 ru h th st cu sr e^S ca
69 tra/Me, T23
70 ry^2

Chromosome 4

71 ci^W
72 spa

Multichromosomal

73 bw;e
75 vg;e
76 w^{m4};Cy/ap^4 vg
77 w^{m4};Cy/blt
77a bw;st

Deficiencies

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

Translocations

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

BIRMINGHAM, ENGLAND: THE UNIVERSITY OF BIRMINGHAM
Department of Genetics

Wild Stocks

1 Edinburgh
2 Wellington
3 Florida

Inbred for 70-700 Generations

4 Oregon
5 Samarkand
6 6 C/L

Chromosome 1

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

Chromosome 2

11 cn
12 vg
13 bw
14 b cn vg
15 dp cn bw

Chromosome 3

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

Chromosome 4

21 ey^2

Multichromosomal

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

Inversions

25 Muller-5
26 Me Sb e/H e
27 y sc^{S1} B dl-49 w^a sc^8
28 Ins(2L+2R)Cy cn^2/In(2LR)Pm al^4 ds^{33k} lt bw^{V1};In(3LR) Dc x F D/Sb
29 y;Ins(2L+2R)Cy cn^2/In(2LR)Pm al^4 ds^{33k} lt bw;In(3L)D, D/Sb

Attached X

30 y x w
31 FM6;TM3 Sb Ser y^+

CLAYTON, VICTORIA, AUSTRALIA: MONASH UNIVERSITY
Departments of Genetics and Psychology

Wild Stocks

Oregon K
Oregon R
Riverside
East African
Bermuda

Chromosome 1

B
w
y
w, y
t
mal
v

Chromosome 2

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

Chromosome 3

e^{11}
e
se
ve
ry

UTRECHT, NETHERLANDS: HUBRECHT LABORATORY

Note: Stocks are as listed in DIS 45: 29 with the following changes:

Removed from the list: No. 10, 11, 14, 23 and 29

Added to the list:

Chromosome 1

30 sc^{10-1}/y Hw
31 $sx\ vb^2\ os^s/FM6,\ y^{31d}\ sc^8$ dm B
32 y sc

Chromosome 2

33 cn en/SM5, al^2 Cy $lt^v\ sp^2$

Chromosome 3

34 $Antp^B\ ss^a/Me$
35 $Antp^B\ ss^a/Ubx^{130}$ ri e ca
36 $Antp^{Yu}/Cy$ Sb
37 $Antp^{Yu}/Ubx^{130}$ ri e ca
38 $Antp^{49}/Ubx^{130}$ (ri) e
39 $Antp^{49}/Ubx^{130}$ ri e ca
40 $Antp^{50}\ p^p\ bx\ sr\ e^s/TM1$, Me ri $p^p\ sbd^1$
41 $Antp^{50}\ ss^a/TM1$, Me ri (p^p)
42 $Antp^{59}/Ubx^{130}$ ri e
43 art
44 bx^{34e}
45 bx^3 Cbx Ubx bxd pbx/$T(2;3)ap^{Xa}$
46 Cbx
47 hl
48 hl tet^{Bd}bilat
49 $In(3R)Antp^B$, $Antp^B/TM1$, Me ri sbd^1
50 Ki
51 $Ns/Antp^B$
52 $Ns/Antp^B$ ld-opht
53 pb/In(3LR)FCx
54 $pbx/T(2;3)ap^{Xa}$
55 $Sb/In(3LR)Ubx^{101}$, Ubx^{101}
56 ss^a
57 ss^{a40a}
58 ss^{aB}
59 tet^{Bd}bilat
60 th st cp in ri $Antp^{59}\ sr\ e^s/TM1$, Me ri (p^p)
61 Ubx $e^4/In(3L-3R)P$, Dfd ca
62 Ubx Cbx

Chromosome 4

63 ci^D/ey^D

Closed-X

64 $In(X^{c2})w^{vc}/In(1)dl-49$, y w lz^s ♀
$In(1)dl-49$, y w lz^s/sc^8.Y ♂

TORONTO, ONTARIO, CANADA: UNIVERSITY OF TORONTO
Department of Zoology

Wild Stocks

Pl I Oregon-R
Ives Oregon-R
M Oregon-R

Chromosome 1

Basc
sc v f
y
y w m
w m f
dp
vg
vg bw

Chromosome 2

b
b pr c
bw

Chromosome 3

e^s
se
ry
st

Chromosome 4

ey

Multichromosomal

Cy/Pm;Ubx/Sb (2;3)
Cy/Pm;H/Sb (2;3)
dp e (2;3)

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

Wild Stocks

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

Chromosome 1

b2 w f
b4 w
b5 y
b9 w f/ClB
b16 y_2 w sn^3
b17 y^2 v
b18 y_2 w
b19 y^2 w^a
b20 sc cv v f B

Combinations of Scute

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

Altered Y's

f1 y/sc^8 Y
f3 y w^{m4}/sc^8 Y
f4 y^2 w^a/sc^8 Y
f10 y^{S1} sc^8 B f In-49 v/sc^8 Y

Chromosome 2

g1 bw
g3 cn
g4 dp
g5 L^2
g6 b vg
g8 Cy L/Pm

Chromosome 3

h1 se ss
h2 st
h3 e
h4 gl bx^D
h8 st c3G ca/In(3LR)Ubx^{130} Ubx^{130}, e^s

Chromosome 4

i1 ey^2

Multiple Chromosomes

j2 Y^S·X·Y^L In(1)EN, Y^S B y·Y^L/ y^2 su-w^abb/0;Cy L/Pm
j3 bw;st
j4 y;bw;e;ci ey^R
j5 "Oster ♀♀" y sc^{S1} In-49 sc^8; bw;st p^p
l7 Cy/Pm;D/Sb
j8 y cv v_2 f car/sc^8 Y;dp
j13 SMI al^2 Cy sp^2/Pm;In(3LR) Ubx^{130}, Ubx^{130} e^s/Sb
j15 y^2w^a/sc^8 Y;e
j18 "Sterilizer" Y^{Lc}/XY^S;bw;st p^p

Attached (Compound) Chromosomes

m1 Y^S·X·Y^L In(1)EN, Y^S B y·Y^L/ y^2 su-w^abb/0
m2 "Oster ♂♂" X^{c2} y B/sc^8 Y (♂♂) sc^8Y/y f:= (♀♀)
m3 sc cv v f B/y f:=

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

Wild Stock

Oregon-R

Chromosome 1

B
Basc
f
g^2
m
m^D (homozygous)
m^DB (homozygous)
$m^D/FM3$, y^{31d} sc^8 dm B
w
w^a
w^e
w B
w f
w m f
y
y ac v
y cv v f

Chromosome 2

al
al dp b pr c px sp
b
b vg
bw
ds dp
L
mr $bs^2/In(2LR)bw^{V1}$, ds^{33K}
Sp J L Pin/SM5, al Cy lt sp^2
Tft/Sml, al^2 Cy sp^2

Chromosome 3

e
In(3R)C, Sb/TM3, y^+ ac^+ ri p^p bx^{34e} Ser
Dl H e^s cd/In(3R)P, spr
Pr Dr/TM3, y^+ ac^+ ri p^p bx^{34e} e^s
R Ly/In(3L)P, gm
ru h th st cu sr e^s ca
st

Multichromosomal Stocks

Basc;SM1, al^2 Cy sp^2/In(2LR)bw^{V1} dp b bw^{V1} ds^{33K};In(3R)C, Sb/IN(3LR) Ubx^{130}, Ubx^{130} e^s;spa^{pol} (1;2;3;4)
SM5, al^2 Cy lt sp^2/Sp;In(3R)C, Sb/TM3, y^+ ac^+ ri p^p bx^{34e} Ser (2;3)

NEW HAVEN, CONNECTICUT: YALE UNIVERSITY
Department of Biology

Corrections to list in DIS 44: Stock No. 76 is ltd^{37b} vg
Stocks no longer available: Nos. 32, 48, 53, 54, 57, 93.

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Zoology

Wild Stocks

3 Samarkand
5 $+^3$

Chromosome 1

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

Chromosome 2

212 bw
215 cg c/U
232 vg

Chromosome 3

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

Chromosome 4

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

Multichromosomal

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

Triploid

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

SEOUL, KOREA: SEOUL NATIONAL UNIVERSITY
Department of Zoology

Wild Stocks

001 Canton-S
002 Oregon-R
003 Seoul-5

Chromosome 1

101 t
102 v
103 w
104 w^a
105 w^e bb^1/ClB
106 y se mf^2
107 y^2 cv v f

Chromosome 2

201 bw
202 cn bw
203 Cy/bw^{V1}
204 L^4
205 pr
206 vg
207 Cy L/bw^{V1}
208 rd/SM5 Cy
209 de/SM1 Cy

Chromosome 3

301 cu
302 jv
303 Sb/$In(3LR)Ubx^{101}$

Chromosome 4

401 bt

Multichromosomal

501 bw;st (2;3)
502 Cy/bw^{V1};Sb/Ubx (2;3)
503 Cy/bw^{V1};D/Bd (2;3)

Inversion

601 Muller-5

LEEDS, ENGLAND: THE UNIVERSITY OF LEEDS
Department of Genetics

Note: Stocks are as listed in DIS 44: 32 with the following additions:

Wild Stocks

6 Swedish strains
2 Yugoslav strains
1 Leeds strain

Chromosome 1

Muller-5
v

Chromosome 2

ab
Cy L^4/Pm
Cy L^4/d b
Cy L^4/S
SD72/SM5, al^2 Cy lt^v sp^2
SD5/SM1, al^2 Cy sp^2
1(2)gl/SM5, al^2 Cy lt^v sp^v
1(2)Me/Cy

Chromosome 3

mwh

Special Stocks

v f B XY/y^2 $su(w^a)w^a$ bb

DURHAM, NORTH CAROLINA: DUKE UNIVERSITY
Department of Zoology

dor/ClB dor/FM_4 fu/ClB $fs(1)N/FM_4$

DÜSSELDORF, GERMANY: UNIVERSITÄT DUSSELDORF
Institut für Allgem. Biologie

Wild Stock

1 Berlin

Chromosome 1

2 B
3 Df(1)bb^{-}, y sl^{2} bb^{-}/FM4-1, y^{31d}sc^{8}dm B 1/sc^{8}.Y
4 Muller-5, sc^{S1}B InS w^{a}sc^{8}
5 sc^{8}bb w^{61a}
6 w
7 y/Y & w/Y
8 y w f
9 y^{2}su-w^{a} w^{a}bb & v f B.Y (no free Y's)
10 X^{C2}, y^{2}su-w^{a}w^{a}bb/sc^{8}.Y & X^{C2}, y v/sc^{8}.Y
11 w^{a2}
12 sn^{3}

Chromosome 2

13 vg
14 al
15 dp^{ovN}

Chromosome 3

16 D/Gl
17 ss^{a}
18 cu
19 e^{11}
20 st

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

Wild Stocks	Chromosome 1	Chromosome 2	Chromosome 3
1 Oregon K	1 w 2 sc^{S1} B InS w^{a} sc^{8} (Muller-5)	1 vg	1 ru cu ca

KALYANI, NADIA, INDIA: KALYANI UNIVERSITY
Department of Zoology

1. Wild (Oregon R)
2. se h
3. vg
4. y
5. y w lt
6. u B

ANNOUNCEMENTS

Permits for shipment of Drosophila stocks within the U.S.A. The following is an excerpt from a letter to Prof. M.R. Wheeler, University of Texas, by C.E. Andrews, Head, Permit Section, Plant Quarrentine Division, U.S.D.A., 209 River St., Hoboken, N.J. 07030.

"Under a recent change in policy we are discontinuing the issuance of courtesy permits and labels for insects which are not classified as plant pests. Since Drosophila spp. are not plant pests, no formal permit is required for interstate movement or importations under the Federal Plant Pest Act. We have, therefore, cancelled permit number INS-1089. Notice of cancellation is enclosed.

Several states have agricultural pest laws which may be applicable to the movement of Drosophila spp. into their states. Therefore, we are enclosing a list of the State Regulatory Officials of the states in question. We suggest that you write to those officials and determine what, if any, restrictions may be imposed on shipments of Drosophila spp. not accompanied by the discontinued courtesy label."

Division of Plant Industry
Alabama Department of Agric. and Indus.
P.O. Box 220
Montgomery, Alabama 36101

Plant Industry Division
Michigan Department of Agriculture
Lansing, Michigan 48913

Division of Agriculture
Alaska Dept. of Natural Resources
P.O. Box 300
Palmer, Alaska 99645

Bureau of Plant Quarantine
California Department of Agriculture
1220 N. Street
Sacramento, California 95314

Division of Plant Industry
Colorado Department of Agriculture
406 State Services Building
1525 Sherman Street
Denver, Colorado 80203

Division of Plant Industry
Florida Department of Agriculture
P.O. Box 1269
Gainesville, Florida 32601

Division of Ent. & Plant Industry
Georgia Department of Agriculture
Agriculture Bldg., Capitol Square
Atlanta, Georgia 30334

Chief Plant Inspector
Hawaii Department of Agriculture
P.O. Box 2520
Honolulu, Hawaii 96804

Bureau of Plant Industry
Nebraska Department of Agriculture
and Economic Development
Lincoln, Nebraska 63509

Division of Plant Industry
New York Department of Agriculture
and Markets
Building 8, State Campus
Albany, New York 12226

Assistant State Entomologist
South Carolina Crop Pest Commission
Clemson, South Carolina 29631

Division of Plant Industries
Tennessee Department of Agriculture
Melrose Station, Box 9039
Nashville, Tennessee 37204

Division of Plant Industry
Wisconsin Department of Agriculture
4802 Sheboygan Avenue
Madison, Wisconsin 53702

Plant Quarantine Section
Puerto Rico Department of Agriculture
P.O. Box 10163
Santurce, Puerto Rico 00908

The "Second European Drosophila Research Conference" will be held April 1st-3rd, 1971 at the Swiss Federal Institute of Technology, Zürich, Switzerland. For Drosophila workers who cannot attend the conference, the Abstract Book will be available at a price of US $1.00. Address for all correspondence: Prof. Dr. F.E. Würgler, Department of Zoology, Swiss Federal Institute of Technology, Universitätstrasse 2, CH - 8006 Zürich, Switzerland.

E.B. Basden, Institute of Animal Genetics, West Mains Road, Edinburgh EH9 3JN. I will be quite pleased to purchase unwanted collections of Drosophila-Drosophilid reprints. All languages and dates. This helps in an easy way towards the checking of all records for the world catalogue.

PERSONAL AND LABORATORY NEWS

Karl H. Ytterborn, of the Genetics Institute, University of Stockholm, Sweden, is spending a sabbatical year in Oxford at the New Genetics Institute led by Prof. Bodmer.
Takashi Narise moved from Josai University, Sakado-Machi, Saitama-ken, Japan, To Josai Dental University and is continuing works on behavior and population genetics.
Akishi Fukatami and Kendo Tsuno moved from Tokyo Metropolitan University of Josai Dental University and are continuing works on enzyme polymorphism.
Sumiko Narise moved from Tokyo Metropolitan University to Josai University and is continuing work on biochemical genetics of isozyme.
Oswald Hess now heads a newly founded Laboratory for General Biology at the University of Düsseldorf, Germany (from the Biological Laboratory I of the University Freiburg, Germany).
Rollin C. Richmond is now Assistant Professor at the Department of Zoology, Indiana University Bloomington, Indiana (from the Rockefeller University, New York, New York).
Peter S. Carlson, who completed his doctoral work in the Department of Biology, Yale University, is now Assistant Professor of Biology at Wesleyan University, Middletown, Connecticut.
Kugao Oishi has returned to the National Institute of Genetics, Misima, Japan, following completion of his doctoral work in the Department of Biology, Yale University, New Haven, Conn.
H. Gloor has accepted a professorship, Department of Genetics, University of Geneva, Switzerland (from Genetisch Laboratorium der Rijksuniversiteit, Leiden, The Netherlands).

Report of G.E.W. Thörig and W. Scharloo

r^{66j}: $rose^{66j}$ Scharloo 66j. Present in offshoot of Pacific cage population (see F.W. Robertson, 1960) with frequency of 10-20%. When reared at 25° eyecolor is translucent brownish-pink and the ocelli are colorless immediately after hatching. In the first days after hatching discrimination from wildtype is difficult. Eyecolor is darkening and ocelli become pigmented with age. Eyes are sepia-brown in flies of several weeks old. At lower temperatures (below 20°) eyecolor approaches wildtype and ocelli are pigmented when the flies hatch. At higher temperatures (30°) eyes are more brownish and ocelli are colorless. Chromatography revealed that compared to wildtype there is a strong decrease of xanthopterin and a small decrease of the drospterins. Sepiapterin and biopterin show higher concentrations. Temperature increase causes a decrease of pteridin content without appreciable change of the relative concentrations.

Report of G. Lefevre Jr.

Cytological information regarding mutants listed in Lindsley and Grell 1968:

N^{5419}: Deficient from 3C6 to 3C11. In combination with Df w^{67k30} (3C2-3C6) is viable and expresses a strong vt phenotype, but is not rough-eyed.

kar^{31}: Deficiency extending from 87C2 or 3 to 87D1 or 2.

ry^{27}: Deficiency extending from 87D2 or 3 to 87F2 or 3.

ry^{54}: Heterochromatic rearrangement, probably an inversion, with breaks in the 87D8-12 interval and in the chromocenter.

ry^{74}: A very short deficiency restricted to the 87D8-12 interval and probably involving only one band. This mutant delimits the ry locus to the region between 87D8 and 87D12.

ry^{75}: Deficiency from 87D2 to 87D14 or 87E1.

ry^{76}: Deficiency from 88B12-14 to 87D13 or 87E1.

Report of S. Gossi and R. Moree

bwn^{G}: brown of Gossi, 3- Spontaneous in Dr(35-CS) (Drop, backcrossed by Drop females to Canton-S males, repeatedly, generation after generation, for 35 generations). Not allelic to mah. Eye color: dull, reddish orange in young flies, darkening to dark brown (darker than bw or Pm); has slight Moire-like sheen and occasionally a very faint eye spot. bwn^{G}/bwn^{G};bw/bw flies have orange eye, only slightly dull, lighter than bwn^{G}/bwn^{G}, and darkening slightly if at all with age; no eye spot. Viability and fertility of males and females good.

Report of K. Valentin

cm67j: carmine 67j From offspring of X-rayed Canton-S male (3600r) mated to Muller-5 female. Allelism shown with cm. RK 1 L.

Report of J.N. Thompson, Jr. and G. Braver

Correction of an error occurring in a new mutant report in DIS 44: 43. In the first line of the report on m^{66d}, it states that the mutant was induced in a y-Muller-5 stock. The mutant was actually induced in a Canton-S wild stock and balanced in the y-Muller-5.

Report of E. Gooskov

px^{70}: plexus 70 Chromosome 2. Spontaneous from wild stock collected in Rostov on Don which arose after 3 generations. Recessive. Wing venation like plexus.

A^{70}: Abnormal abdomen 70 Chromosome 1. Spontaneous after 2 generations. Doninant. Irregular reduction of abdominal tergites, sternites, pigmentation. Penetrance 10%.

Abl: Abnormal legs Linkage group not yet established. Dominant. Irregular reduction of legs of the 2 or 3 pairs shortened and either femur or tibia bowshaped. Highly variable. Penetrance 1%.

Hemithorax from A^{70} Through inbreeding of the wild population of Drosophila melanogaster line A^{70} was obtained. Initial penetrance of this trait was 10%. Selection to increase penetrance gave rise to the appearance of flies with phenotype hemithorax resembling that described by Ludwig in 1936 (DIS 7: 18, 1937) from a vestigial stock.

From 6 generations arose 12 flies hemithorax, 9 females and 3 males. All flies were fertile and gave progeny with wild or A^{70} phenotype. From this

same stock arose two flies with deformed thorax - doublehemithorax. These flies had their thorax devided into two parts and every part carried one normal wing. This deformity is not inherited.

Report of Y.S. Kang and E.H. Park

Srf: Surf wings 2-66.8±0.7 Found as a dominant, visible and viable mutation from the wild population in Seoul, Korea (near the Atomic Energy Research Institute). In heterozygous distal half of wings upturned about 40° from normal wing axis as in Si or j, but usually not diverse (Fig. 1). Penetrance is not perfect

Fig. 1. Srf: Surf wing (♀)

at 25±1°C: female, 96%; male, 84.2%. Crossing over data analysis based upon method of Kojima and Dalebroux (DIS 40: 49-52). Homozygous perfectly lethal. Heterozygous fully viable and fertile. Balanced lethal with bw^{Vl}. Combination with Cy express Cy phenotype and viable, fertile. Post-scutellar and other external phenotypes normal. Salivary chromosome not yet examined. Data now in press. RK2

Report of G. Trippa

Pgm^A: phosphoglucomutase^A 3-43.6 Trippa, G., Santolamazza, C. and Scozzari, R. 1970.
Pgm^B: phosphoglucomutase^B 3-43.6 Trippa, G., Santolamazza, C. and Scozzari, R. 1970.

These are two co-dominant electrophoretic alleles of the structural gene of phosphoglucomutase, which have been demonstrated in larval, pupal and imaginal homogenates of single individuals of Drosophila melanogaster by an adaptation of the method of Spencer et al. (Nature 204: 742-745, 1964). Individuals homozygous for Pgm^A exhibit a single anodal band of activity (A phenotype) which migrates faster than a similar, single anodal band found in individuals homozygous for Pgm^B (B phenotype). Heterozygotes Pgm^A/Pgm^B exhibit both the fast and slow phosphoglucomutase bands, and no hybrid bands (AB phenotype).

These three phenotypes have been found at the frequencies expected by the Hardy-Weinberg equilibrium in a natural population collected near Rome, Italy (gene frequencies: Pgm^A = 0.91 and Pgm^B = 0.09).

Twelve laboratory wild-type and mutant strains have been examined (not less than one hundred flies per stock): ten showed only the Pgm^A allele and two only the Pgm^B allele.

This gene was mapped on chromosome 3 at 43.6 on the linkage map of Drosophila. No morphological differences between Pgm^A and Pgm^B homozygous individuals are apparent. RK1.

Report of D.J. Fox and K. Madhavan

$Hex\text{-}3^F$: Hexokinase-3, fast form

$Hex\text{-}3^I$: Hexokinase-3, intermediate form

$Hex\text{-}3^S$: Hexokinase-3, slow form These three forms of hexokinase are resolvable by agar gel electrophoresis using the EBT buffer system of Ursprung and Leone (J. Exptl. Zool. 60: 147-154). Electrophoresis is routinely conducted for 30 minutes at 225 volts and 25-30 milliamps. In this system the "fast" variant migrates more rapidly toward the anode than does the "intermediate" variant which in turn migrates more rapidly than does the "slow" variant. However, because of electroendosmosis in agar gels the whole pattern is shifted toward the cathode. Hexokinase-3 migrates more slowly toward the anode than do two other hexokinase band systems of independent genetic control (hexokinase-1 and hexokinase-2) for which no electrophoretic variants have been found. Using the second chromosome mapping stock al dp b pr c px sp the Hex-3 map position has been determined to be 2-79±.

Report of R. Alicchio

sw: short wing In lines of Drosophila melanogaster selected for short wing a few flies were found having divergent wings very short in length and with the margin partially wrinkled.

Females with the deviant phenotype are completely sterile when crossed either to phenodeviant males or to normal ones.

The character seemed to be connected with sex since more males than females appear during selection. The new phenotype (named "sw") was tested for genetic determination and localization. Crosses were performed between y/y;+/+ females and +/+;sw/- males; F_1 females were phenotypically wild type, F_1 males wild and yellow type. F_1 wild females were backcrossed to y/-;+/- males; wild type and yellow phenotypes were observed on female flies, whereas four classes of male phenotypes were scored as expected in the case of sex linked traits:

yellow body and normal wing normal body and "sw" wing	parental phenotypes
normal body and normal wing yellow body and "sw" wing	recombinant phenotypes

The frequency of recombinant phenotypes observed was 35.6%.

The χ^2 for independence between parental and recombinant phenotypes (179.3 with 3 degrees of freedom) suggests that the loci "y" and "sw" are not segregating independently. It should be concluded that the "sw" phenotype is determined by a single recessive gene located on the X chromosome. Viability of "sw" flies as compared to wild type flies does not show significant difference.

Report of L.I. Borack, R.D. Water and W.H. Sofer

PDH-NAD^F: NAD-dependent beta-hydroxy propionate dehydrogenase, fast form

PDH-NAD^I: NAD-dependent beta-hydroxy propionate dehydrogenase, intermediate form

PDH-NAD^S: NAD-dependent beta-hydroxy propionate dehydrogenase, slow form

The three forms of beta-hydroxy propionate dehydrogenase are resolvable by agar gel electrophoresis using the EBT buffer system of Ursprung and Leone (J. Exp. Zool. 60: 147-154, 1965). Flies are homogenized in .05 M phosphate buffer pH 7.5, and electrophoresed for 30 minutes at 250 volts and 25 milliamps. The staining mix consists of:

20 mls	.05 M	Tris-HCl	pH 8.6
2 mls		NAD	(25 mg/ml)
10 mls		NBT	(5 mg/ml)
1 ml		PMS	(2 mg/ml)
1 ml	1 M	Na B-OH Propionate (K and K Laboratoires)	pH 9.0
2 mls	1 M	Pyrazole (Borack and Sofer, this issue)	

The slow variant (PDH-NAD^S) migrates more rapidly to the anode than does the intermediate variant (PDH-NAD^I) or the fast variant (PDH-NAD^F) as seen by formazan deposition.

Electrophoresis of heterozygotes produces bands intermediate between the two parental forms of the enzyme.

47 strains were surveyed with 42 showing PDH-NAD^I.

B-8 Oregon, Florida, Nyasa Lake and Swedish C showed PDH-NAD^S.

Samarkand shows polymorphism between PDH-NAD^I and PDH-NAD^F.

PDH is electrophoretically distinguishable from beta-hydroxy butyrate dehydrogenase and alcohol dehydrogenase (Borack and Sofer, this issue) as well as octanol dehydrogenase (Courtright, Imberski and Ursprung, Genetics 54: 1251, 1966) and lactate dehydrogenase. PDH-NAD shows no activity with NADP.

Preliminary studies show PDH-NAD not to be on the 1st chromosome.

Supported by N.I.H. Training Grant GM-57 to L.I.B. and N.S.F. Grant GB 7803 to W.H.S.

Report of J.C. Neeley

w^{69c}: white^{69c} Neeley 69c. 1-1.5. Spontaneous appearance in single F_1 male from cross C(1)RM, y f females X Canton-S males. Eyes nearly snow-white; ocelli colorless. Like white.

w^{aS}: white-apricot of San Neeley 68g. 1-1.5. Spontaneous appearance in single F_1 male from cross C(1)RM, y f females X Canton-S males. Eye color light-orange in hue, difficult (impossible?) to distinguish from white-apricot; ocelli colorless.

w^{dt}: white-dirty Neeley 68k. 1-1.5. Spontaneous appearance in single F_1 male from cross C(1)RM, y f females to Canton-S males. Eye color similar to white, but with a slight sooty cast to color; ocelli colorless. w^{dt}/w indistinguishable from w^{dt}/w^{dt}, but slightly "dirtier" or greyer than w/w.

Report of J.C. Moran and J.C. Neeley

mwg: microwing Neeley 67g. 2-L. Arose spontaneously among the progeny of attached-X females (C(1)RM, y w f/Y) mated to Canton-S males. To date localized in the left arm of chromosome 2. Wings reduced in size, extending slightly beyond scutellum; only proximal regions of veins obvious; wings usually curled or curved at posterior margins; wings often blistery, filled with fluid. No significant reduction in fertility or viability of homozygous female. Halteres appear slightly shorter than normal, possibly caused by missing base segment.

Report of G. Trippa, R. Scozzari and C. Santolamazza.

The genetic localization of the phosphoglucomutase (Pgm) locus in Drosophila melanogaster

A natural population of Drosophila melanogaster, collected near Rome, Italy, has shown to be electrophoretically polymorphic for the phosphoglucomutase (Pgm) gene with two autosomal, co-dominant alleles: Pgm^A and Pgm^B (see New Mutants, this DIS). Lines homozygous for each allele were extracted from that population.

In order to obtain a chromosome location of the Pgm gene, virgin females from an isogenic stock bw;st;spa^{pol}, homozygous for the Pgm^B gene, were crossed with wild males homozygous for Pgm^A.

Heterozygous F_1 males were back-crossed with bw;st;spa^{pol}(Pgm^B) females; at the same time virgin F_1 females were back-crossed with bw;st;spa^{pol}(Pgm^B) males.

Progeny from the two back-crosses were analyzed for the presence of recessive markers and for their isozyme patterns. Table 1 shows the results of the two back-crosses: the data on the left indicate that the gene controlling the Pgm is located on the third chromosome linked with st; the data on the right appear to show that the Pgm gene is closely linked to the st locus.

Table 1. Distribution of the progeny Pgm phenotypes in the two backcrosses.

	bw/+;st/+;spa^{pol}/+(Pgm^B/Pgm^A)♂ x bw/bw;st/st;spa^{pol}/spa^{pol}(Pgm^B/Pgm^B)♀		bw/+;st/+;spa^{pol}/+(Pgm^B/Pgm^A)♀ x bw/bw;st/st;spa^{pol}/spa^{pol}(Pgm^B/Pgm^B)♂	
	Pgm AB	Pgm B	Pgm AB	Pgm B
bw st spa^{pol}	--	18	1	46
+ + +	15	--	15	--
bw + +	21	--	30	--
+ st spa^{pol}	--	19	not tested	
bw st +	--	12	--	58
+ + spa^{pol}	15	--	not tested	
bw + spa^{pol}	12	--	not tested	
+ st +	--	19	--	103

For a more precise localization of the gene on the third chromosome, Gl;Sb/LVM(Pgm^A/Pgm^A) males have been crossed to st/st(Pgm^B/Pgm^B) females. Gl + Sb/+ st + (Pgm^A/Pgm^B) F_1 females were backcrossed to + st +/+ st + (Pgm^B/Pgm^B) males. The results (Table 2) indicate that the Pgm gene is located between Gl and st.

Table 2. Distribution of the Pgm alleles among crossovers from females heterozygous Gl + Sb/+ st + (Pgm^A/Pgm^B)

Recombinant chromosomes	Pgm^A	Pgm^B
Gl st +	2	27
+ + Sb	19	6
Gl + +	23	--
+ st Sb	--	21
Gl st Sb	--	--
+ + +	--	--

The results also show that 85.2 percent of the crossovers occurred between Gl and Pgm and that 14.8 percent occurred between Pgm and st. Using these percentages and the standard gene locations of Gl (41.4) and st (44.0) one can calculate the locus for Pgm at 43.6±.

Report of W.J. Ouweneel

hl: halteroptera From the results of crosses with Cy/Pm;Sb/D flies it was concluded that hl is on the third chromosome (see DIS 45: 35). Simple crosses of hl with bx, bx^3 and bx^{34e} strains yielded flies with a very good bithorax phenotype. Neither bx nor hl interacts with pbx. A location of hl at the bx locus seemed plausible. This was tested by measuring the recombination frequency between ss^a (3-58.5) and hl, which was 0.32% (calculated from 11,317 flies). This corresponds to the locus of bx which is 3-58.8. It was concluded that hl is an allele of bx, having one of the most extreme "tetraptera" phenotypes known.

MATERIALS REQUESTED OR AVAILABLE

E. Ortiz, Instituto de Genética y Antropología, Madrid, Spain, would appreciate receiving any strains of D. kuntzei, D. limbata, D. phalerata, D. transversa and D. andalusiaca (=forcipata)

DROSOPHILA SPECIES - STOCKS

CALCUTTA, INDIA: UNIVERSITY OF CALCUTTA
Department of Zoology

D. ananassae
Wild Stock

1 a^{66}Calcutta(a^6)

Chromosome 1

2 y (Hinton)
3 y^{51} f^{49}

4 w^{65}
5 w^{65} f^{49}
6 w^{65} y^{51}
7 w^{65} f^{49} y^{51}
8 w^{65} y^{51} sn^{65}

Chromosome 2

9 b

10 cd ba^{65}
11 cd se^T ba^{65}

Chromosome 3

12 px
13 px^2
14 pc

15 px pc
16 bri Rf px

Other Species

D. kikkawai (2 strains)
D. bipectinata
D. hydei
D. simulans

SYDNEY, AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Animal Husbandry

pseudoobscura Wild strains homozygous for Chromosome 3 inversions

Arrowhead (4 strains) - Pinon, California
Chiricahua (8 strains) - Pinon, California

simulans Mutants

v
st

NEW YORK, NEW YORK: THE ROCKEFELLER UNIVERSITY

D. pseudoobscura stocks now kept by Dr. W.W. Anderson, Yale University, New Haven, Connecticut

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Zoology

D. simulans wild

Zaprionus vittiger

ST. LOUIS, MISSOURI: WASHINGTON UNIVERSITY
Department of Biology

D. melanica
D. euronotus
D. micromelanica
D. paramelanica
D. nigromelanica
D. colorata

D. sordidula
D. lacertosa
D. moriwakii
D. robusta
D. pseudosordidula
D. immigrans

D. carsoni
D. buzzatii
D. mercatorum
D. paranaensis
D. repleta
D. stalkeri

D. virilis
D. drauma
D. funebris
D. multispina
D. subfunebris
D. americana

SEOUL, KOREA: SEOUL NATIONAL UNIVERSITY
Department of Zoology

D. virilis

DÜSSELDORF, GERMANY: UNIVERSITÄT DÜSSELDORF
Institut für Allgem. Biologie

D. hydei

1 wild

Chromosome 1

2 $ch^{to\text{-}1}{}_y tu\text{-}1{}_m tu\text{-}1$
3 f^2
4 g y m
5 $m^{tu\text{-}2}$
6 N/w lt
7 v^1 [T(Y,A)]
8 v^3 (homozygous lethal)
9 v f
10 w
11 w^a
12 w lt
13 w^{m1}
14 w^{m2}
15 w^{m3}
16 v sc sn y m ch bb
17 w lt/Y & +/Y
18 ch y m/Y & w lt/Y
19 v f/Y & w lt/Y
20 w lt/Y & or/Y
21 to^2

Chromosome 2

22 e^{du}

Chromosome 3

23 cn

Chromosome 5

24 red eye, brown thorax

Multichromosomal

25 bb;p;vg
26 scarlet;scabrous;javelin (2;3;5)

several stocks with mutant morphology of Y chromosomal lampbrush loops

many stocks with T(X,Y)

D. fulvimacula

wild
y
y/Y & +/Y

Other Species

D. bifurca
D. nigrohydei
D. virilis
D. eohydei
D. repleta
D. neohydei
D. simulans

VIENNA, AUSTRIA: INSTITUT F. ALLGEMEINE BIOLOGIE

D. ambigua
Wild Stock

one strain

Chromosome 1

or
y
v

Autosomes

pn
Bd

D. subobscura
Wild Stocks

Wien, Austria
Ponza, Italy
Drobak, Norway
Belgrad, Jugoslavia

Chromosome 0

Ba
ch
cu
ch cu
Va
Va ch cu
Va/Ba^{210}

Küsnacht, St -homozygous for all chromosomes

Other Species

D. mercatorum
D. funebris
D. simulans

SÃO PAULO, BRASIL: UNIVERSIDADE DE SÃO PAULO
Instituto de Biociências, Departamento de Biologia

Same as listed in DIS 43: 71, less D. pallidipennis.

ZÜRICH, SWITZERLAND: UNIVERSITÄT ZÜRICH
Zoologisches Museum

D. subobscura

Mutant Stocks

Chromosome A

9 Oc
12 m ct v bnt sc
14 Bx
37 ct^{Eu}

Chromosome I

64 s th int ey mj
69 ni

Chromosome E

8 sj pl pp stp
11 pl pp pt
19 shsc
32 oi
45 vsz
47 dck
53 dp^{2} Eu
56 qm

Chromosome U

13 ho
36 ltr
71 fd^{Mi} nt

Chromosome O

72 ch cu
74 Va

Homokaryotypic Stocks

Chromosomes not mentioned are St/St; chromosomes mentioned are homozygous for the respective gene arrangement.

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

Wild Stocks

Bisperode (Germany)
Effretikon 1968 (Switzerland)
Effretikon 1969) (Switzerland)
Arcegno 1970 (Switzerland)
Gabès (Tunisia)
Tabarka (Tunisia)
Tunis Belvédère (Tunisia)
Tunis Orangerie (Tunisia)

Other Species (All strains are from Switzerland)

D. ambigua
D. bifasciata
D. helvetica
D. obscura
D. silvestris
D. tristis

LINCOLN, NEBRASKA: UNIVERSITY OF NEBRASKA
Department of Zoology

D. affinis: Minnesota, Nebraska; bristle singed (both X-linked), brown (autosomal).
D. algonquin: Minnesota, Vermont; garnet (X-linked).
D. athabasca: "Western" ath.: Manitoba, Minnesota, Oregon, Wyoming; orange (autosomal).
"Eastern" ath.: Massachusetts, Minnesota, New Jersey, New York, Ontario, Pennsylvania, Vermont; early bright (autosomal).
D. azteca: California, Mexico (Chilpancingo).
D. helvetica: Zürich.
D. narragansett: Nebraska, Texas.
D. tolteca: Bolivia, Colombia.

TUCSON, ARIZONA: UNIVERSITY OF ARIZONA
Department of Biological Sciences

cardini group

acutilabella (8)
antillea (1)
arawakana (3)
belladunni (6)
cardini (17)
caribiana (1)
cardinoides (11)
dunni (3)
neocardini (1)
neomorpha (3)
nigrodunni (2)
parthenogenetica (6)
polymorpha (3)
procardinoides (1)
similis (1)

repleta group

aldrichi (2)
arizonensis (6)
fuliginea (1)
hamatofila (5)
hydei (3)
leonis (1)
longicornis (1)
melanopalpa (2)
mercatorum (1)
meridiana (2)
mojavensis (10)
nigrospiracula (9)
spenceri (2)

obscura group

azteca (1)
persimilis (1)
pseudoobscura (11)

others

acanthoptera (1)
nannoptera (1)
pachea (25)
tripunctata (2)
willistoni (1)

SAPPORO, JAPAN: HOKKAIDO UNIVERSITY
Faculty of Science, Department of Zoology

D. busckii (1 strain)
D. testacea (1)
D. brachynephros (2)
D. immigrans (1)
D. virilis (2)
D. lacertosa (6)
D. sordidula (5)
D. auraria race A (1)
D. nigromaculata (1)
D. angularis (1)
D. funebris (2)
D. moriwakii (2)
D. okadai (3)
D. pseudosordidula (4)

DROSOPHILA SPECIES - NEW MUTANTS

virilis

Report of S. Ohba

In DIS 45: 56-57, new esterase isozyme variants of D. virilis were reported. Recent thorough re-examination of linkage relationships revealed that these variants were controlled by two, not ten, multiple loci on the second chromosome, named Esterase-α and Esterase-β. Consequently, following corrections to the designation are required.

Multiple alleles at Esterase-α locus

New designation	
Est-α^1: Esterase-α^1	(formerly Est-1)
Est-α^2: Esterase-α^2	(" Est-3)
Est-α^3: Esterase-α^3	(" Est-4)
Est-α^4: Esterase-α^4	(" Est-5)
Est-α^5: Esterase-α^5	(" Est-6)
Est-α^6: Esterase-α^6	(" Est-7)
Est-α^7: Esterase-α^7	(" Est-8)

Est-α^8: Esterase-α^8 (" Est-9)

Est-α^9: Esterase-α^9 (" Est-10)

Est-α^0: Esterase-α^0 silent allele

Multiple alleles at Esterase-β locus

Est-β^A: Esterase-β^A (formerly Est-2^A)

Est-β^B: Esterase-β^B (" Est-2^B)

Est-β^C: Esterase-β^C (" Est-2^C)

Est-β^D: Esterase-β^D (" Est-2^D)

Est-β^0: Esterase-β^0 (" Est-2^0, silent allele)

ananassae

Report of D. Moriwaki (3) (1 - 43: 79; 2 - 45: 58)

(X Chromosome)

ty: tiny Moriwaki 68l11. Derived from a cross +al5 x Bn px-b. Bristles thin and short-like of Minute.

kk^c: curly bristles Moriwaki 70f7. Spontaneous in cd Dl bw stock. Bristles curled upward. Allelic to kk.

(Chromosome 2)

pe: peach Tobari, Y.N. 69a. Spontaneous in +Ph5 wild stock. Eye color translucent yellowish pink. Ocelli colored.

Dl: Delta Moriwaki 69h1. Spontaneous in a strain containing px. End of L2 vein and costa are fused together. Dominant. Homozygous lethal. When combined with one px gene, the manifestation is exaggerated by extra veins. In extreme case, combined with homozygous px, additional balloon-like expression appears.

ext: extended Moriwaki 69l25. Appeared in the culture of a Dl pair. Wings extending at about 75° from body axis, usually showing shortened L3 vein. Occasionally only one of those characters is expressed, Dl ext often manifesting only extended wings.

(Chromosome 3)

mot: mottled Moriwaki 69l18. Recovered as a single ♂ in M-b bn-c stock. Eyes mottled, dark-spotted and rough. Expression variable.

Snp: Snipped Moriwaki 69b5. Spontaneous as a single female from a cross referring to bri M-b ru. Wing tips snipped. Dominant, with low penetrance. Homozygous sterile, with low viability and vg-like expression.

M-d: Minute-d Moriwaki 70f. Spontaneous in w ty y stock. Minute bristles. Dominant. Homozygous lethal.

ri: radius interruptus Moriwaki 70i13. Spontaneous in the culture of a M-d px^2 pair. Vein L2 interrupted.

(Chromosome 4)

bb^2: $bobbed^2$ Moriwaki 67k21. Spontaneous in +F2 wild stock. Bristles shortened. Allelic to bb^{67}.

Some of the gene loci reported before (DIS 43: 79) will be revised. The linkage group of M-b is misprinted in DIS 45: 58. Read 3- for 2-.

simulans

Report of J. Puro

f^2: forked^2 Puro 60h12. Two males appeared in a culture from mating of an X-irradiated male and non-irradiated females more than 18 days after the mating, hence were probably F_2 flies of the treated male. Much like sn^3 melanogaster. However, female hybrids between sn^3 melanogaster and f^2 simulans have wild type bristles whereas those between f melanogaster and f^2 simulans are phenotypically f^2. In addition, recombination is fairly free (45%) between w and f^2, as could be expected if the locus of f^2 is the same as that of original f (56.0 according to Sturtevant, 1929, Carnegie Inst. Wash. Publ. 399).

Px: Plexus wing Puro 60g31. Chromos. 2. X-ray induced. In heterozygous flies an extra vein is usually found between the posterior crossvein and the distal part of L4 which is bent in such a way that a bullet-shaped cell is formed between the 1st and 2nd posterior cell. Short branches may be present in other longitudinal veins. Homozygotes are humpy, poorly viable and completely sterile and have short legs and short and spread wings with a thick network of extra veins. Small tests indicate quite free crossing over with py^2 and up (50% and 45% respectively). Lost.

py^2: polychaete^2 2-74 (the locus of py in Sturtevant, 1929). Puro 60h24. X-ray induced. Like py for both the bristle and eye effect. py^2/py show the mutant phenotype.

py^{Mis}: Michrochaetaless Puro 60i1. X-ray induced. A dominant allele of py. Thoracic microchaetae largely disappeared. Ocellars and one or two of the scutellars occasionally missing. Homozygous lethal. The evidence for the allelism with py comes from tests showing that py^{Mis}/py^2 besides being phenotypically py^{Mis} express the eye effect of py^2 and have arc wings and are male sterile. In addition, linkage tests with up (14.0 ± 1.4% crossing over) suggest the same locus for py^{Mis} and py^2. Lost.

up: wings up Puro 60h30. Wings held upright, slightly curved like a spoon or a screw. In combination with py^2 the curvature of the wings is usually reversed, the tips pointing downwards. Not allelic with sd. 14.1 ± 0.6% crossing over between up and py^2.

Dl^2: Delta^2 3-64 (the locus of Dl in Sturtevant, 1929) Puro 62b. Spontaneous. Heterozygotes show a typical Delta phenotype with thickened veins and irregular hairs. Homozygous lethal. Linkage data indicate the same locus as that of original Dl (2.1 ± 0.2% crossing over between H^h and Dl^2 as compared to 2.6 ± 0.3% between H and Dl in Sturtevant, 1929).

rd: rugged Puro 60h27. Chromos. 3. X-ray induced. Eyes rough at the posterior margin. Two independent tests agree in showing that the locus is 10 units to the left of jv mapped to 0 by Sturtevant (1934, DIS 1). (Pooled results indicated 9.6 ± 0.4% and 7.5 ± 0.4% crossing over respectively at the rd-jv and jv-se regions.) Accordingly, the 3rd chromosome map of Sturtevant needs to be revised. Hybrids from mating of ru ("rucuca") melanogaster with rd show rough eyes to a variable extent. The allelism with ru melanogaster was questioned, however, by a test which showed that hybrids between "rucuca" melanogaster and wild type simulans also have rough eyes.

Ubx: Ultrabithorax 3-71 (using the map of Sturtevant, 1929. The locus is 10 and 7 units to the right of H^h and Dl^2, respectively, as measured on the basis of two independent tests.) Puro 60g30. X-ray induced. Corresponds to Ubx of melanogaster both in appearance and in being homozygous lethal. Dominant in hybrids with melanogaster. Hybrids with bx^3 melanogaster are like melanogaster Ubx/bx^3 (Hannah-Alava).

Zamburlini, P. University of Padua, Italy.

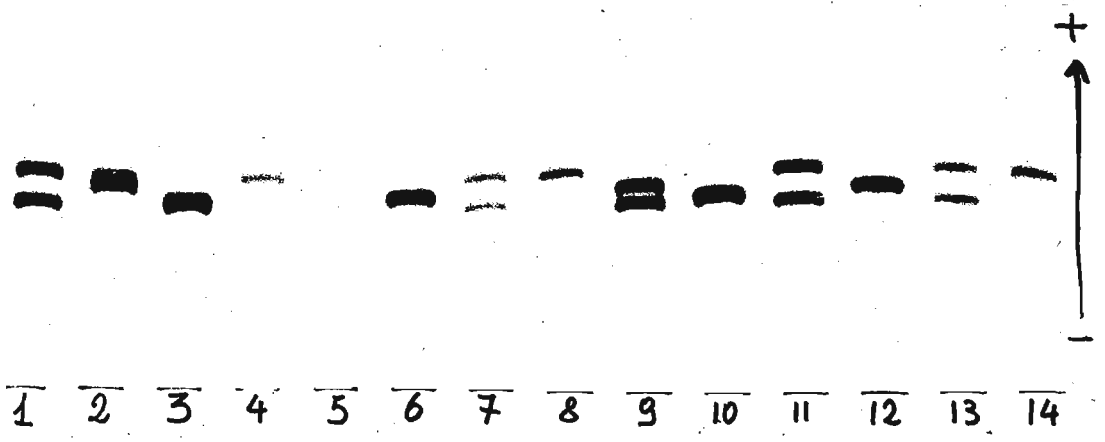
The polymorphism of Est-6 in a wild population of Drosophila simulans.

The polymorphism of the enzymatic system Esterase-6, described in Drosophila melanogaster and Drosophila simulans by Wright and MacIntyre (1963) was investigated in a wild population of Drosophila simulans by acrylamide-gel electrophoresis. The traps were placed in a garden biotope in the surroundings of Udine (North-East of Italy) during the summer. Single flies were homogenized in Tris-EDTA-borate buffer (pH 9.0) and contrifuged at 15,000 rpm; the supernatant was used for the electrophoretic analysis of the enzyme.

Electrophoresis was carried out in vertical acrylamide-gel slab in continuous Tris-EDTA-borate buffer, (pH 9.0) at constant current of 40 mA for 70 minutes, in the cold (4°C).

Except for a few slight modifications, the technique used for the detection and the characterization of the enzyme was the same as that described by Wright (1963). Zymograms of single-fly homogenates show that the wild population of Drosophila simulans investigated contains four forms of the enzymatic system Esterase-6, each showing different electrophoretic mobility; from the cathode to the anode: Est-6^4, Est-6^3, Est-6^2, and Est-6^1.

Figure 1 shows zymograms of single-fly homogenates: in the same slab, homozygous Est-6^3/Est-6^3 (samples 3, 10), Est-6^2/Est-6^2 (samples 6, 12), Est-6^1/Est-6^1 (samples 4, 8, 14) are presented, along with the heterozygous Est-6^1/Est-6^4 (samples 1, 5, 7, 11, 13), Est-6^1/Est-6^2 (sample 2) and Est-6^2/Est-6^4 (sample 9). The four forms of Esterase-6 system just described



are heat-stable at 60°C for 10 minutes; they are also insensitive to 10^{-2} M $AgNO_3$, 10^{-3} M $CuSO_4$ 10^{-2} M EDTA; they are slightly inhibited by 10^{-4} M eserine sulfate.

Unfortunately, a direct comparison with the Est-6 forms described by Wright and MacIntyre (1963) in Drosophila simulans is impossible at the moment.

Table 1 shows the allele frequencies found in the wild population investigated.

Table 1

Est-6^1	Est-6^2	Est-6^3	Est-6^4
0.057	0.545	0.093	0.303

Table 2 shows the observed values for the 10 genotypes tested against the values expected in a Hardy-Weinberg distribution, in a total sample of 252 individuals.

Table 2

Est-6^1/Est-6^1	Est-6^2/Est-6^2	Est-6^3/Est-6^3	Est-6^4/Est-6^4	Est-6^1/Est-6^2	
2	81	10	35	16	observed
0.75	74.84	2.01	22.93	15.62	expected
Est-6^1/Est-6^3	Est-6^1/Est-6^4	Est-6^2/Est-6^3	Est-6^2/Est-6^4	Est-6^3/Est-6^4	
3	6	22	75	2	observed
2,52	8.56	25,20	83.16	14.11	expected

The disagreement between the observed and the expected distributions is very significant ($P<0.001$). It is mainly due to an excess of homozygous genotypes. This situation is difficult to explain; one possibility is that the sample is, for instance, a mixture of different populations. However the presence of non-random mating cannot be excluded.

References: Wright, R.F. and R.J. MacIntyre, 1963 A homologous gene-enzyme system, Esterase 6, in Drosophila melanogaster and D. simulans. Genetics 48: 1717-1726.

Koliantz, G. University of Tehran, Iran.

Spontaneous changes on second chromosome of Drosophila melanogaster.

A previously localized stock (six years ago by our curator of stocks) which exhibits curvature of wings (Cy), bright red eye color (cn) and a recessive lethal gene on the second chromosome, has been recently relocated.

Virgin females from this stock were crossed with brown males. In the F_1, brown and Curly flies with the ratio of 50-50 appeared. The F_1 was divided into three lines:

First line: F_1 brown females backcrossed with their mothertype flies; in the F_2 Curly-cinnabar, Curly, brown and white eye flies appeared.

Second line: F_1 Curly females backcrossed with their mothertype flies. The offspring of the F_2 were Curly, Curly-cinnabar and brown.

Third line: F_1 Curly females produced wild type and Curly-cinnabar when crossed with the cinnabar (Tehran) pure stock.

Genotypical properties: The existence of a deficiency on the right arm of the second chromosome causes the appearance of brown phenotype in the main cross (Table 1).

Table 1

females	males	F_1 genotypes	F_1 phenotypes
Cy cn +/+ cn Df	bw/bw	Cy cn +/+ + bw + cn Df/+ + bw	Curly wings brown eye color

By the cross of the first line we demonstrated that crossing over occurred in the + cn Df/ + + bw genotype (Table 2).

Table 2

females	males	F_1 genotypes non C.O.	F_1 genotypes C.O.
+ cn Df/+ + bw	Cy cn +/+ cn Df	+ cn Df/Cy cn + cn Df/cn Df* + + bw/Cy cn + + bw/cn Df	+ + Df/Cy cn + + Df/cn Df* + cn bw/Cy cn + cn bw/cn Df

* lethal

By other crosses, when + bw/cn Df x + bw/cn Df produces brown flies and when a crossing-over occurs, we obtain flies with white eyes which have the genotype cn bw/cn Df. All of the white eyed females are sterile but such males show normal fertility.

The results of the second line indicate that no crossing over takes place in the right arm of the second chromosome with the Cy cn +/+ + bw genotype, and in crosses such as Cy cn +/ + + bw x Cy cn +/+ + bw only brown and Curly individuals appear as the offspring (Table 3).

Table 3

parents	offspring	
Cy cn +/+ + bw	2	Cy cn +/+ + bw
	1	+ + bw/+ + bw
	1	Cy cn +/Cy cn +*

* lethal

The cross of the third line demonstrates that the genotype of the flies is Cy cn +/+ + bw.

By a comparison of the given data, we conclude that there is a suppressor of crossing over on the right arm of the second chromosome. Therefore final genotype of the stock would be Cy cn C +/+cn + Df(2R)59D2-5;59E1-3.

The cultures were kept at $24^{\circ}\pm1$ C under constant light on Mostashfi medium.

References: Koliantz, G., 1968 The frequency of spontaneous visible mutations in Iranian natural populations of D. melanogaster; Lindsley, D.L. and E.H. Grell, 1968 Genetic Variations of Drosophila melanogaster.

Sloane, C. and E.B. Spiess. University of Illinois, Chicago, Illinois. Stimulation of male courtship behavior by female "odor" in D. pseudoobscura.

Evidence in favor of a sex pheromone in some Diptera has been demonstrated as a factor in stimulating courtship behavior in at least four laboratories: Rogoff et al., 1964, in Musca domestica; Adams & Mulla, 1968, in Hippelates collusor (eye gnat); Ehrman, 1969, in D. pseudoobscura; and Shorey & Bartell, 1970, in D. melanogaster. In contrast, Ewing & Manning, 1963, were unable to demonstrate the effect of scent from one sex upon the other in D. melanogaster. Using the method of Shorey & Bartell, we set about testing three strains of D. pseudoobscura for the influence of females upon male courtship elements. The olfactometer was blown from glass tubing (1" diameter) outfitted with 2 Teflon stopcocks so that air could be routed through one chamber or the other (see Shorey & Bartell's Fig. 1). Flies to be tested as an odor source were placed in a glass tube 5/8" in diameter open at both ends covered with cheesecloth and the tube was inserted in one of the entering air chambers. The males to be observed were placed in a similar tube inserted from the other end of the olfactometer. All flies were unetherized. The air source was the air jet on the lab bench filtered through distilled water containing activated charcoal in suspension; and the air flow was calibrated as close to 500 ml/min. as possible. The 3 strains of flies were obtained from Mr. Richard Sherwin in this laboratory from his 20th generation of selection for fast or slow mating propensity (Sherwin, 1970). One PP and one AR strain (FP2 and FA2) had been selected for fast mating, while one AR strain (SA1) had been selected for slow. Flies to be tested were sexed and isolated on emergence in lots of 6 or 30, depending on their use either in the observation chamber (6) or as the odor source (30).

Observations consisted of counting orientations and wing variations between males in a 6 minute period. Counts were made at 15 second intervals. The first 3 minutes of each test simply allowed air to flow through the empty chamber, then the stopcocks were reversed to allowed air to flow from the odor source (either 30 males or 30 females). Between trials the olfactometer was aired for at least 3 minutes. The first set of experiments was done using the same strain for both sexes while the second set interchanged fast and slow AR (FA ♂♂ had SA ♀♀, or SA ♂♂ with FA ♀♀). Observed orientations and wing vibrations during the 3 minutes of air flow (initial) served as a control for each test. All tests were replicated ten times.

Table 1
Means and st. errors of courtship elements in 3 minutes for 10 replicates per strain. ♀♀ or ♂♂=30 as odor source. Control = 3 initial min. air flow.

Strain Males	Orientations				Wing Vibrations			
	♀♀	Control	♂♂	Control	♀♀	Control	♂♂	Control
FA2	19.1±214	22.0±3.2	12.0±2.4	24.1±2.9	13.5±1.6	15.3±2.7	11.1±2.0	20.2±2.7
SA1	6.4±1.0	7.6±1.4	8.0±2.2	12.3±1.7	3.0±1.1	4.0±0.8	4.2±1.4	5.8±0.8
FP2	12.0±2.4	12.9±2.5	10.9±2.5	14.6±2.6	7.6±1.5	5.5±0.8	5.6±1.3	6.7±1.9

Mean courtship elements (orientations and wing vibrations) with standard errors are given in Table 1. Generally it is evident that the FA2 (AR) strain is most active, SA1 (AR) the least, with FP2 (PP) intermediate in both courtship elememts. Both fast strains (FA and FP) show an increase in these elements when the odor source is virgin females as opposed to males as a source, while the slow strain showed the reverse. Using "t" tests for significance between treatments of 30 ♂♂ vs. 30 ♀♀ as odor source, only in the fast AR (FA2) strain orientation ($t=2.08$) or in the pooled totals of orientations plus wing vibrations ($t=2.18$) were the differences significant (5% level), though the pooled totals for fast PP (FP2) approached significance ($t=1.68$, $p=.11$).

Controls were higher in each case than the odor source. It seems that in the initial 3 minutes before each odor trial flies were active merely in response to the air flow. Once accustomed to that, the values approached a more usual base level of activity for the odor tests.

Table 2
Means and st. errors of courtship elements of AR strains when tested with females of the opposite strain. (Controls, always greater, are omitted).

Strain	Orientations		Wing Vibrations	
Males	SA ♀♀	FA ♀♀	SA ♀♀	FA ♀♀
SA1	6.4±1.0	7.0±1.7	3.0±1.1	6.1±1.6
FA2	13.8±3.7	19.1±2.4	14.1±3.6	13.5±1.6

In the second study, fast and slow AR sexes were interchanged, and the results are given in Table 2. Comparison of males' response with their own strains is included with data from Table 1. FA females raise the orientation level of both types of male, though not significantly for either, while in wing vibration there is only an increment for SA males which approaches significance ($t=1.84$, $p=.07$).

These results indicate some increases in male activity due to presence of females but only for strains which are genetically of high mating propensity. Influence of fast-mating females upon slow-mating males is inconclusive though suggestive. These influences can only be attributed to airborne stimulation, presumably a volatile substance transmitted from the females.

References: Adams, T.S. and M.S. Mulla, 1968 J. Insect Phys. 14: 627-635; Ehrman, L., 1969 Evol. 23: 59-64; Ewing, A.W. and A. Manning, 1963 Anim. Behav. 11: 596-597; Rogoff, W.M., A.D. Beltz, J.O. Johnson and F.W. Plapp, 1964 J. Insect Phys. 10: 239-246; Sherwin, R. N., 1970 Genetics 64: s59; Shorey, H.H. and R.J. Bartell, 1970 Anim. Behav. 18: 159-164.

Picard, G. and Ph. L'Héritier.
Laboratoire de Génétique, Clermont-Ferrand, France. A maternally inherited factor inducing sterility in D. melanogaster.

It was found out recently that a laboratory strain bearing the gene sepia gives puzzling results when crossed with a standard Oregon strain. As shown in the table, females from the cross ♀ se x ♂ + lay eggs which hatch with a very low probability. Males from the same cross and both sexes in the reciprocal cross ♀ + x ♂ se behave normally.

Using flies from some other laboratory strains or flies caught in the wild in the crosses with the sepia strain leads to similar results, but with a somewhat variable degree of sterility.

original cross	back cross	total	eggs hatched	percentage of hatching
♀ se x ♂ +	♀ F_1 x ♂ se	360	34	9.4
♀ + x ♂ se	♀ F_1 x ♂ se	100	96	96.0

Eggs which do not hatch have been fecundated, since segmentation nuclei are readily observed, but most of them die before blastoderm stage. The probability of hatching for an egg laid by a sterile female has been found to be dependent upon two factors:

1) It rises with mother aging.

2) It gets nearly normal when temperature is raised to 30°C for a period of 48 hours located at the end of ovogenesis. Female offspring, which are allowed to reach imaginal stage owing to such a thermal treatment, are not genetically cured, and may show the same kind of sterility as their mothers.

The genetic determinism of the female sterility is presently under active investigation. The following points can be held as demonstrated:

1) The probability of hatching of an egg depends but poorly if at all upon its own genotype, the major factor being the genetic origin of the mother.

2) For a sterile female to arise, two genetic requirements must be filled. A maternally inherited factor of unknown nature normally propagated in the sepia strain and a genotype heterozygous for certain genes must be present.

3) Quite a number of genes located on all of the three major chromosomes seem to be involved in the phenomenon.

Voss, R. The Hebrew University, Jerusalem, Israel. A common suppressor for a lethal mutation and a forked mutation.

The reversion of lethal mutations that were induced in the proximal segment of the X-chromosome is now being studied. Most revertants are sterile. One revertant (in an X-ray induction series) of 1^{3DES}, a lethal induced by diethyl sulfate (Lifschytz & Falk, 1968) was fertile. The reversion of the lethal effect was found to be due to a suppressor mutation on the X-chromosome. The mutant also suppresses the forked mutation and was found to be allelic to su-f. The new suppressor is recessive, the heterozygous female 1^{3DES} su-$f^v/1^{3DES}$ being inviable. The hemizygous male 1^{3DES} su-f^v has outstretched wings and a shortened body. Homozygous females 1^{3DES} su-$f^v/1^{3DES}$ su-f^v are sterile and show the same abnormal phenotype as the hemizygous males; the pigmentation of the abdomen of these females resembles that of bobbed females, but on crossing su-f^v to bb they were found to be non-allelic. Experiments to determine the range of other mutations suppressed by su-f^v (especially other lethals at loci nearby in the proximal segment of the X-chromosome) are now under way. However, no interaction between w^a and su-f^v was found.

The fact that mutations at the su-f locus affect more than one kind of mutant loci suggests the involvement of this gene in the synthesis of some basic protein being a common precursor to more than one metabolic pathway.

Chatterjee, S.N. and A.S. Mukherjee. University of Calcutta, India. DNA replication pattern of the puffing sites in the X-chromosome of Drosophila hydei.

DNA replication pattern in polytene chromosomes of Drosophila hydei confirms the earlier observations (Berendes 1966; Lakhotia and Mukherjee 1970; Mulder et al. 1968; and Rodman 1968) supporting the continuous to discontinuous pattern of replication. The pattern observed shows that the sites replicating faster include most of the puffing sites.

Studies on the replication pattern of the X-chromosomal and autosomal puffs at a particular "puff stage" provides some information on the relation of replication sequence and transcribing activity and its bearing on dosage compensation in Drosophila. For this study, the excised glands were incubated in 0.05ml Ringer containing 5μCi of H^3-TdR (Sp. Activity 5.27 Ci/mM) for 20 minutes. Ten days exposure and usual methods of autoradiography were followed. The presence of a minimum of 8 grains was considered as a criterion for designating a puff labeled.

The replication patterns of 14 autosomal and 18 X-chromosomal puffs arranged in a continuous-to-discontinuous sequence are presented in Table 1. It is clear that at the beginning of

Table 1. Replication pattern of 14 autosomal and 18 X-chromosomal puffs of Drosophila hydei

No. of labeled puffs in autosome (male and female)	No. of labeled puffs in X	
	female	male
14	18	13 - 18
9 - 12	15	10 - 12
5 - 8	12 - 13	2 - 10
3 - 4	9 - 12	1 - 2
1 - 2	0 - 9	0 - 1

replication cycle the pattern is same in both the sexes, but as the replication proceeds the puffing sites of the X in male show less and less incorporation when compared with the puffing sites in the female X's (Table 1). For example, when 9 autosomal puffs are labeled on the 4th chromosome, the number of labeled puffs on X in the male and the female are 10 and 15 respectively. Conversely, when same puffs on the X in the male and the female are labeled, the number of autosomal puffs labeled are more in the male than in the female.

These results reveal that the single-X of the male shows a differential replication pattern with respect to the pattern in the female X's and early completion of the process as well. In addition, individual puffs of the male-X present a faster rate of replication when compared with those in the female X. The data are compatible with hyperactive male X model of dosage compensation in Drosophila.

References: Berendes, H.D., 1966 Chromosoma 20: 32-43; Lakhotia, S.C. and A.S. Mukherjee, 1970 J. Cell. Biol. 47: 18-33; Mulder, M.P., P. van Duijin and H.J. Gloor, 1968 Genetica 39: 385-428; Rodman, T.C., 1968 Chromosoma 23: 271-287.

Posch, N.A.* University of California at Los Angeles, California. Development time of D. melanogaster: dependence on yeast content, pH, and consistency of the medium.

Two types of media were used in this study. Banana agar medium, in ready to prepare form, was obtained from General Biological Supply House. This medium (pH 4.8) consisted of dried banana flakes, agar, brewer's (killed) yeast, white corn syrup, distilled water, and n-butyl parahydroxybenzoate as a mold inhibitor. The concentration of killed yeast was 38 mg/ml medium. The second type of medium used (pH 3.4), contained cornmeal, agar, brewers yeast, unsulphured molasses, distilled water, and propionic acid as a mold inhibitor. The concentration of killed yeast was 10 mg/ml medium.

The natural food of Drosophila larvae is live yeast. The inclusion of killed yeast in a Drosophila medium is to provide the nutrition normally supplied by living yeast. We decided to determine the effect of live yeast on the development time of Drosophila raised in media containing killed yeast. Development time refers to the length of time between introduction of the adult flies into the culture, and the time the first progeny emerge. Of the two media we used, the cornmeal medium contained only one fourth the concentration of killed yeast contained by the banana medium. The first experiment was to determine the amount of live yeast necessary to add to the cornmeal medium to prevent delayed development, as results from inadequate nutritional value of the medium (described by Northrop, 1917, J. Biol. Chem. 30: 181.)

Six male-female pairs of Oregon-R flies, aged 4-6 days, and pre-fed on cornmeal medium with 500 mg of added live yeast (Fleischmann's active dry yeast), were placed in each culture, a 250 ml glass bottle with 30 ml of medium, and maintained at 23°C ±1°. Varying amounts of live yeast had been sprinkled on the surface of the cultures a few hours before introduction of the flies. Cultures were checked four hours after the introduction of the adults to be sure egg laying had commenced. The results are shown in Table I and indicate that addition of 100 mg of active dry yeast to each culture is more than sufficient to provide adequate nutrition for normal rate of development. The length of the pupal period remained essentially constant, regardless of yeast content; only the time period of larval development varied.

Table I

	Active dry yeast added to medium (mg)					
	None	3.1	6.2	12.5	25.0	50.0
Average development time (days)	15.3	13.7	13.0	11.7	12.0	11.7
Number of cultures	3	3	3	3	3	3

From the previous experiment, it was apparent that the cornmeal medium without added live yeast was nutritionally deficient. We then determined the effect of raising successive generations on the cornmeal and banana media, and both media with added live yeast. Wild type, red eye flies, obtained from General Biological Supply House, were used. The parental generation of flies was pre-fed for three days on live yeast seeded media. Six pairs of flies were used to start each culture and left for five days. Flies were raised at 22°C ±2°. The results are shown in Table II.

Table II

Medium	Generation Development time (days) F_1	F_2	F_3	F_4	Number of Cultures	Average Development Time (days)
Banana	12.3	14.3	13.4	13.8	48	13.5
Banana + 100mg live yeast	12.5	12.4	12.6	12.5	24	12.5
Cornmeal	14.1	15.2	17.3	18.3	68	16.2
Cornmeal + 100mg live yeast	11.5	11.3	11.7	11.3	24	11.5

From the results, several conclusions may be drawn: 1. The banana medium, with 38 mg killed yeast/ml, was nutritionally slightly sub-optimal, but sufficient to maintain continuous generations of healthy larvae with consistent development times.

2. The nutrition of the adult (female) affects the development time of its progeny. This is shown by the significantly shorter development time for F1 than for subsequent generations on both cornmeal and banana medium due to the pre-feeding of the parental generation on yeasted media.

3. The cornmeal medium, with 10mg killed yeast/ml, was nutritionally quite deficient, with development time increasing with each successive generation (although the data are not presented, total progeny also decreased for successive generations).

4. Addition of live yeast to both media, in an amount previously determined to be sufficient for normal development, resulted in consistent development times for successive generations, but significantly different between the two media (i.e., compare the average development time on the two yeast supplemented media).

We performed a third experiment to determine if the difference in pH of the two live yeast supplemented media was responsible for the difference in average development times. Batches of banana medium were prepared in the usual manner, with the addition of varying amounts of hydrochloric acid. The acidity of each batch of medium was determined with a pH meter. Adult flies of the same stock as the previous experiment were pre-fed on yeast supplemented banana medium, and then transferred to the acidified cultures, to which no live yeast had been added. Six pairs of flies were placed in each culture and removed after four days. Temperature was 22°C ± 2°. The results of this experiment are shown in Table III.

Table III

	pH of acidified medium							
	4.80	4.60	4.45	4.30	3.80	3.50	3.25	2.55
Average development time (days)	12.5	12.6	12.6	11.8	12.6	12.4	12.6	12.8
Number of cultures	5	5	5	5	5	5	5	5

pH of banana medium: 4.8 pH of cornmeal medium: 3.4

From these results, it is apparent that the difference in average development time on banana and cornmeal media is not due to pH difference. Indeed, the pH has no pronounced effect between 2.55 and 4.80. Sang (1956, J. Exp. Biol. 33: 45) reported that early growth of D. melanogaster is slightly retarded in a gel medium with 10% killed yeast, as compared to growth on live yeast alone. He suggested this was due to difficulty of the first and early second instar larvae in feeding on a non-particulate surface as opposed to the particulate nature of living yeasts and bacteria, the natural food. In our case, the living yeast is equally accessible to larvae in both types of media (on the surface). However, our cornmeal medium is much softer and more particulate than the banana medium, allowing the larvae to move through it more easily. This suggests that the larvae develop faster on the yeast supplemented cornmeal medium than on the yeast supplemented banana medium, either because they can move through the cornmeal medium faster, and therefore eat at a faster rate, or because they expend less energy in pushing through the medium, and therefore need to eat less volume in order to attain the necessary size for pupation, and therefore, pupate sooner.

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Studies on the ecology of Drosophila in Korea.

Collections of Drosophilid flies were made during a period ranging from May to August in 1970 at Mt. Sok-ri (Chung book Province) and Mt. Kae ryong (Chung nam Province) in Korea. Most of these flies were collected by sweeping method. Six species of wild yeasts were isolated from the crops of Drosophilid flies. Among those yeasts Saccharomyces florentinus and Saccharomyces cerevisiae grew well on the medium for the Wagner Y-2 strain and also D. auraria

Table 1. Wild yeasts isolated from the crops of Drosophila.

Drosophila species	wild yeasts
Drosophila (Sophophora) rufa	Saccharomyces florentinus
Drosophila (Drosophila) brachynephros	Saccharomyces florentinus
D. (D.) nigromaculata	Saccharomyces cerevisiae
D. (D.) immigrans (female)	Trichosporon capitatum
D. (D.) immigrans (male)	Trichosporon fermentans
Leucophenga (Trichiasphiphenga) argentosa	Torulopsis salmanicensis
Leucophenga (Leucophenga)	Torulopsis dattila

D. immigrans, D. brachynephros, and D. busckii bred well on cornmeal media with these yeasts (Saccharomyces floretinus, Saccharomyces cerevisiae).

Godbole, N.N., R.M. Kothari and V.G. Vaidya. University of Poona, India. Studies on the nutritive value of different indigenous cereals for the larvae of D. melanogaster.

Cornmeal-agar medium, for cultivation of Drosophila, together with its variations has been described by Bridges and Darby (1933). These media are now widely used in Drosophila laboratories throughout the world. However, the necessity of developing new media suitable for quantity production, healthy flies and low mortality rate of weak mutants, still persists. In addition to the above well recognised criteria, easy availability and low cost of chosen ingredients depending upon the location cannot be overlooked. The present study is an attempt to select a suitable indigenous cereal as an ingredient from amongst the following - bajra (Pennisetum typhoideum), barley (Hordeum vulgare), corn (Zea mays), jowar (Sorghum vulgare), rice (Oryza sativa) and wheat (Triticum vulgare) - on the basis of their nutritive value judged by pupal weights.

The Oregon-K strain of D. melanogaster was used in the present investigation. Corn was replaced by a particular cereal under consideration in the following basic formula for the medium, keeping all the other ingredients constant: 18 g cornmeal, 16 g jaggery, 6 g yeast, 6 g agar in 320 ml distilled water. For a particular cereal used in the medium, a set of 25 glass vials (size 7.5 cm X 2.5 cm) was prepared by placing in each vial 6 g ± 50 mg of the medium concerned. Eggs were obtained in large numbers by using the method introduced by Delcour (1969). Ten freshly laid eggs were then transferred to each vial under sterile conditions and were allowed to develop further at 22±1°C. In general, the larval period varied from 10-12 days. The pupal weights were recorded and mean pupal weights were calculated separately for each set. The differences observed between mean pupal weights of different sets were then tested for their significance by using the standard statistical procedure.

It is clear from Table 1 that the mean pupal weights vary significantly for each cereal used in the medium keeping other ingredients constant. In order of their suitability as ingredients in the medium judged on the basis of mean pupal weights, the cereals can be arranged in the following series - jowar, wheat, barley, bajra, corn and rice.

Table 1. Summary of net results obtained by comparison of mean pupal weights.

Cereal used	Number of pupae	Mean pupal wt, mg	Variance of the mean	Difference between mean pupal weights, mg	Normal deviate	Probability 5%	Probability 1%
Jowar	216	1.879	0.000496				
				0.127	5.125		
Wheat	219	1.752	0.000118				
				0.101	4.106		
Barley	206	1.651	0.000487			1.960	2.576
				0.093	3.723		
Bajra	212	1.558	0.000137				
				0.073	3.311		
Corn	202	1.485	0.000349				
				0.158	7.612		
Rice	222	1.327	0.000082				

The biochemical data (Table 2) showing the composition of different cereals used in this investigation substantiates the results obtained (Aykroyd, 1966). Jowar is rich in carbohydrates, fats as well as minerals, all of which are essential metabolites for the synthesis of ATP. It furnishes the maximum amount of total hydrolysable starch, wheat being second amongst the cereals used (Sahasrabuddhe, 1948). Its mineral content is also relatively high. The role of minerals like Mg^{++} in ATP synthesis, RNA synthesis and activation of amino acids prior to protein biosynthesis is well known. Obligatory requirement of Ca^{++} in lipid metabolism is also known. In fact, these are the index reactions for growth. Trace metal ions as activators of different enzymes regulating overall metabolism and physiological processes are of vital importance. Jowar also provides adequate fat content necessary for the synthesis of membranes of different cell organellae which are basic sites of enzyme action. The protein content of jowar is relatively low. However, the amino acid composition reveals that it is richer in leucine, isoleucine, histidine, methionine, valine, phenylalanine, threonine and

tryptophane (F.A.O., 1968) than the rest of the cereals used. These amino acids are essential for the normal growth and development of Drosophila (Lafon, 1939; Hinton et al, 1951). Thus, jowar fulfills all the requirements for boosting up BMR, subsequently yielding healthy pupae as judged from higher pupal weights.

Table 2. Composition of cereals used (Aykroyd, 1966)

Cereal name	Moisture	Fat	Protein	Carbohydrates	Minerals
Jowar	11.9	1.9	10.4	72.6	1.6
Wheat	12.8	1.5	11.8	71.2	1.5
Barley	12.5	1.3	11.5	69.6	1.2
Bajra	12.4	5.0	11.6	67.5	2.3
Corn	14.9	3.6	11.1	66.2	1.5
Rice	13.7	0.5	6.8	78.2	0.6

Although wheat has more protein content than jowar, it is a poor source of tryptophane, methionine, leucine, isoleucine, phenylalanine, threonine, etc. The amount of total hydrolysable starch is also less than that in jowar. Wheat therefore furnishes comparatively less energy for boosting up BMR. Though almost identical to jowar in fat and mineral content, the above-mentioned major deficiencies in wheat have culminated in pupae with lower weights.

Barley and bajra rank third and fourth respectively in order of their nutritional value as judged from pupal weights. Barley is found to be superior to bajra, perhaps due to its high content of total hydrolysable carbohydrates. In protein contents, both barley and bajra are almost identical as far as the quantity is concerned, but the former has more hydrolysable protein than the latter (Aykroyd, 1966). Further, barley is richer in essential amino acids (isoleucine, lysine, phenylalanine, threonine, valine and histidine) than bajra (F.A.O., 1968). Although bajra is richer in fat than barley, it seems that this alone does not become a decisive factor for growth in the early stages of development.

Corn, which is very widely used in the preparation of Drosophila medium, ranks fifth in order of its nutritive value, judged from pupal weights, amongst the cereals tested here. It has less minerals and lesser amount of hydrolysable carbohydrates. Although it is quite rich in protein quantitatively, it is inferior in being lysine deficient, poor in tryptophane and other essential amino acids (Block et al, 1951).

Rice comes last in its nutritive value. It is very poor in mineral content and poorest in fat content. Although rice affords appreciably higher carbohydrate content than the rest, its total hydrolysable carbohydrate is comparatively less. Rice is not only low in protein content, but also poor in leucine, isoleucine, histidine, tryptophane, etc. It seems from the lowest pupal weights observed, that due to lack of these essential amino acids, the available energy cannot be chanelised for larval growth.

References: Aykroyd, W.R., 1966 in The Nutritive Value of Indian Foods and the Planning of Satisfactory Diets, Revised VI Ed. by Gopalan, C. and S.C. Balasubramaniam (Indian Council of Medical Research, New Delhi); Block, R.J. and D. Bolling, 1951 in The Amino Acid Composition of Proteins and Foods: Analytical Methods and Results, Charles Thomas Pub., Springfield, Ill.; Bridges, C.B. and H.H. Darby, 1933 Amer. Nat. 57: 437; Delcour, J., 1969 DIS 44: 133; F.A.O., 1968 Amino Acid Content of Foods and Biological Data on Proteins by Food Consumption and Planning Branch, Nutrition Division; Hinton, T., D.T. Noyes and J. Ellis, 1951 Physiol. Zool. 24: 335; Lafon, M., 1939 Ann. Physiol. Physiochem. Biol. 15: 215; Sahasrabuddhe, D.L. 1948 in Research Papers on Food Problems, N.W. College, Poona.

Moth, J.J. and J.S.F. Barker. University of Sydney, N.S.W., Australia. Estimation of relative fecundity of two genotypes (or species) in mixed populations.

There is ample evidence that the fitness of genotypes or species in mixed populations cannot generally be predicted from estimation of fitness (or fitness components) in pure populations (e.g. Barker and Podger 1970). We are interested in analytical analyses of competitive outcome between various strains of D. melanogaster and D. simulans, and particularly in the effects of variation in population density and relative frequency of the two species on components of fitness. As the eggs of these species are indistinguishable, analyses of

fecundity in mixed populations would be simplified if those laid by one species were marked in some way. A suitable technique is presented here. By rearing Drosophila on media containing the isotope ^{32}P it is possible to obtain adult females which lay 100% labelled eggs. This technique could be readily used for identifying eggs to species (or genotypes) in any competitive situation where eggs are otherwise indistinguishable.

Table 1. Exposure times for eggs tested immediately.

Time after eclosion of egg laying (hrs)	Exposure time in days	Film type
0 - 18	1.5	AA*
19 - 30	2.5	AA
31 - 42	2.5	AA
43 - 54	2.5	AA
55 - 66	8.5	AA
67 - 78	8.5	AA
79 - 90	9.5	AA
91 - 102	9.5	AA
103 - 114	4.5	IND**
115 - 126	4.5	IND
127 - 138	6.5	IND
139 - 150	6.5	IND
151 - 162	8.5	IND
163 - 174	8.5	IND

* Kodak industrial X-ray film type AA.
** Kodak industrial X-ray film.

The isotope ^{32}P is obtained as orthophosphate in a dilute HCl solution with high specific activity. A known activity is diluted with water so that 1 ml of solution has approximately 35 microCuries of activity. 1 ml of this diluted solution (i.e. 35 μCi) is thoroughly mixed into 30 ml of Medium F (Claringbold and Barker 1961) in a quarter pint cream bottle before solidification. (Note: No more than 1 ml should be added to the medium or it will become too watery.)

After the medium has cooled, twenty five pairs of adults (which have been fed yeast for two days previously) are allowed to lay eggs in each bottle for twenty four hours. Each bottle will produce between 1,000 and 2,000 labelled, optimally reared adults from these eggs. For at least the first seven days after eclosion 100% of eggs laid by females so reared will be labelled.

Table 2. Exposure times for eggs held four days after laying before testing.

Time after eclosion of egg laying (hrs)	Exposure time in days	Film type
31 - 42	2.5	IND
43 - 54	3.5	IND
55 - 66	6	IND
67 - 78	6.5	IND
79 - 90	7	IND
91 - 102	7	IND
103 - 114	9	IND
115 - 126	9	IND
127 - 138	9	IND
139 - 150	10.5	IND
151 - 162	10.5	IND
163 - 174	10.5	IND

Labelled eggs are detected by their ability to expose X-ray film plates. Exposure times and types of film plate used are given in Table 1 for eggs tested immediately after collection.

If eggs are not tested immediately but held for a period of time, the length of exposure must be increased because of a rapid loss of activity. (The half-life of ^{32}P is 14.3 days.) Table 2 gives exposure times and film plate type for eggs held four days. This delay before testing is necessary when one wishes to identify dead and infertile eggs (Stalker 1954).

Development, for these two types of plate, is according to standard procedures outlined by the manufacturer.

In our experiments, cohorts of labelled D. simulans and unlabelled D. melanogaster adults (and vice versa) were allowed to lay eggs on agar slabs coated with a thin suspension of dead yeast. Every 12 hours this agar slab was removed and all eggs and yeast thoroughly dispersed in a 14.5 g sucrose/100 ml water solution at 20°C. After a few seconds the yeast settles and the eggs remain suspended in the sucrose solution. A sample of approximately 250 eggs was taken, and with the aid of a Buchner funnel the sucrose solution removed and eggs collected on a 7 cm dark green filter paper. (Note: No more than 300 eggs should be spread on a 7 cm paper or the individual egg exposures will merge and be indistinguishable.) Each filter

paper has attached a 2 cm by 2 cm label of thin white paper. Onto this label is written, in heavy black (e.g. with a Pentel pen), the treatment number. This filter paper was then put against a film plate but separated from it by a thin plastic sheet (0.01 mm) to prevent eggs sticking to it. The film plate, with filter papers held firmly against it by a sheet of clear glass, was then exposed to a 15 watt incandescent light source 120 cm above for 7-9 seconds. This blackens the part of the film plate not covered by a filter paper, making a clear outline around each paper, and permanently marks onto the plate the treatment number of the filter paper. (Light penetrates the white paper label but not the black writing on it, thereby forming an image of clear writing on a grey background.) Resultant exposures according to Tables 1 or 2 indicate eggs laid by D. simulans and by subtraction from the total, the number laid by D. melanogaster was determined. (Work supported by Australian Research Grants Committee.)

References: Barker, J.S.F. and R.N. Podger, 1970 Ecology 51: 170-189; Claringbold, P.J. and J.S.F. Barker, 1961 J. Theoret. Biol. 1: 190-203; Stalker, H.D., 1954 Genetics 39: 4-34.

Rushton, J. and J.A. Metcalfe. University of York, England. A behavioral mutant of Drosophila melanogaster: "Amiel".

The mutant to be described here was found in a dumpy-oblique (dp^o) stock during a comparative analysis of the courtship behaviour of this stock with a wild type strain. This mutant apparently affects the behaviour of the males only (irrespective of whether he courts dp^o or wild type females) since dp^o females show normal courtship behaviour with wild type males.

Observations were made on 4 day old flies in perspex mating chambers of 2 cm. diameter. The behavioural sequence was recorded up to the time of copulation or for the first hour. The pairs of flies which did not mate within this time were classed as non-mating.

A continuous and permanent record of the mating behaviour was made using a kymograph and 5 pointers which were manipulated through a battery by a 5-way switch. Each pointer corresponded to a particular element of mating behaviour viz., orientation, scissoring, vibration, licking and attempted or successful copulation.

The mutant males differ from wild type males in the following features:

1. The mutant males are less successful at stimulating females as indicated by the facts that the duration of courtship is significantly longer for dp^o than wild type males, and that the percentage of unsuccessful matings is also much higher (dp^o 24/41 = 59%; + 6/46 = 13%).

2. Mutant males always initiate courtship upon entry into the mating chamber by wing vibration and not, unlike wild type males, by orientation although orientation after this initial bout does not seem to be affected.

3. Mutant males have fewer rest periods (percentage of time inactive being significantly lower) despite the fact that the total courtship time is longer.

4. The amount of scissoring is significantly reduced but both vibration and attempted copulations are much increased (percentage of time spent and the number of bouts per minute being significantly different for all 3 behavioural elements).

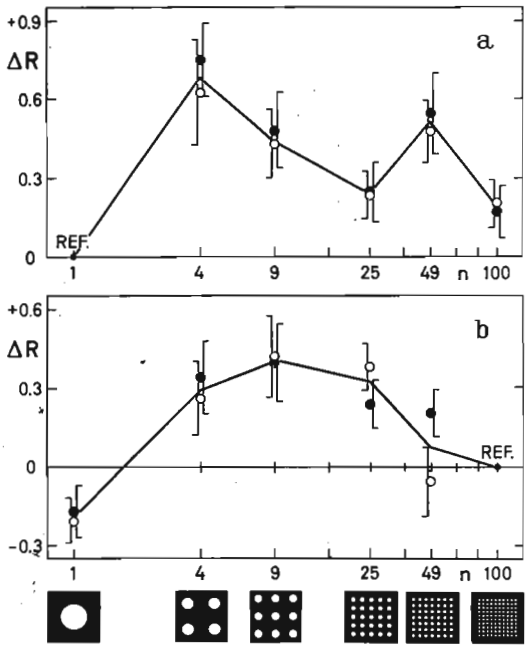
Thus, although the mutant males are more active than wild type males they are in fact less successful. The reduced amount of scissoring, which normally plays an important part in the stimulation of the female, is not compensated by an intensification of other elements of mating behaviour such as vibration and attempted copulation. Or, possibly, the female regards the high activity of the mutant males, in combination with the different pattern, as aggressive rather than courtship behaviour. Outcrossing, reciprocally, the dp^o stock to wild type, showed that the mutant is recessive and not sex linked since both types of heterozygotes behaved as wild type. However, of the 17 progeny of heterozygotes tested 2 showed the same abnormal behaviour.

That the abnormal behaviour does not result from the oblique wing is indicated by the fact that both dp^o males which manifest an oblique wing and those which manifest a normal wing behave abnormally. Furthermore, although the courtship time of dumpy (dp^{o2}) is significantly longer than that of wild type their behaviour pattern is normal.

Behavioural mutants such as described here may prove to be useful in analyzing which elements or pattern thereof of the male's mating behaviour produce the greatest response in the female.

Götz, K.G. Max-Planck-Institut für biologische Kybernetik, Tübingen, Germany.
Spontaneous preferences of visual objects in Drosophila.

Spontaneous preferences in a choice between two visual objects were established by the following experiments with Drosophila melanogaster. The test fly was allowed to walk for several hours on top of a ball, where it was maintained in stationary position and orientation to a 5x5 cm pattern on either side. The patterns were presented on a dark background in 10 cm distance to equivalent anterior regions of the left and the right eye. All patterns were equal with respect to the area and the luminance of the bright objects. The fly was mounted to a minute sledge, which prevented flight and monitored the translatory and rotatory displacements to a servo system. The servo system counteracted the displacements by appropriate rotations of the ball, which were then further evaluated.



The preferences ΔR in the two diagrams denote the revolutions per meter pathlength, which are made by the flies in the direction toward the n dots of the selected pattern if either 1 dot (a) or 100 dots (b) are used as the reference pattern. The means and the standard errors refer to experiments with 183 flies, which covered a distance of 7549 meters on top of the tread compensator. The following can be derived from the data:

1. The preference reaches a maximum at about n=4. The subsequent decrease correlates with the limited acuity of the visual system.

2. The spontaneous preferences are scarcely different during the initial (o) and the final (•) time period of the experiments.

3. The curves a and b coincide within the limits of error if allowence is made for the different origin of the ordinates. The third observation suggests that the additivity of the preferences is roughly accomplished in the present set of patterns. This is to say that the equation $\Delta R_{AB} + \Delta R_{BC} = \Delta R_{AC}$ holds for any three patterns A, B and C of the set. The underlying process of pattern discrimination appears to be simple, whenever the additivity of the preferences is established. Additivity cannot be expected if the spontaneous preference is a function of two or more independently variable parameters of the visual objects.

Chester, B. Flinders University, Adelaide, Australia.
Factors affecting the expression of eyelessness (ey^2).

In selection experiments with eyeless Drosophila melanogaster (ey^2) the frequency of flies with one or both eyes absent declines with increasing age of culture, (Guthrie 1925; Morgan 1919; Spofford 1956; Chester 1969). Morgan (1929) proposed this effect was due to an alteration of the medium by the larvae.

Hunt and Burnet (1969) found deficiencies of thiamine to produce a significant increase in eye size in ey^2 flies. However, Sang and Burnet (1963, 1964) observed that shortages of folic acid and biotin produced significant reductions of eye size, and that eye size increased with increasing amounts of caesin and RNA (up to 0.1%) in the diet.

These results of Sang and Burnet appear contradictory to the selection experiment results where possible medium deficiencies with culture age produce increases in eye size. Therefore, the following experiments attempt to resolve this apparent contradiction of results.

For the first experiment, eggs were collected within two hours of being laid by $eyeless^2$ flies from stocks which had been subjected to selection for eyeless expression for twelve generations. The eggs were placed in three sets of tubes of yeasted medium - one set containing 300-350 eggs per tube, another set containing 100-120 eggs per tube, while the third set con-

tained 18-25 eggs per tube. The first set of tubes should be grossly overworked and hence the medium depleted, while the larvae in the third set of tubes should be subjected to far smaller medium deficiencies - if any, at all. The results are shown in Table 1.

Table 1. Effect of larval density upon frequency of "eyes absent"

Density	Replicates	Total Flies	Percent eyes absent
Very high (300-350 eggs/vial)	2	338	20.9
High (100-120 eggs/vial)	5	396	30.9
Low (18- 25 eggs/vial)	10	160	34.7

Heterogeneity $\chi^2_2 = 27.67$ $p < 0.001$

"Very high c.f. "High" $\chi^2_2 = 19.11$ $p < 0.001$

"High" c.v. "Low" $\chi^2_1 = 1.48$ $p > 0.05$

$$\text{Percent eyes absent} = \frac{100 \times \text{number of eyes completely absent}}{2 \times \text{number of flies}}$$

(Individual χ^2 values calculated from 2 x 2 contingency tables)

The significantly lower percentage of eyes absent in the "very high" density table shows that a depletion of the medium does not increase the expressivity of eyeless^2.

In the second experiment eyeless^2 flies were reared on medium previously worked for 9 days by eyeless^2. The medium was carefully removed from the culture vials, separated into the upper worked-portion and the lower unworked-portion, and all larvae removed. The two portions were placed in different sets of tubes, and further eyeless^2 flies reared on them. The results were compared with eyeless^2 flies reared on normal medium as a control. The results are shown in Table 2.

Table 2. Percent eyes absent in eyeless^2 flies reared on medium removed from previously used culture vials and separated into "worked" and "unworked" portions.

Treatment	Total flies	percent eyes absent
"worked" medium (W)	184	30.2
"unworked" medium (U.W.)	225	24.9
Normal medium - control (C)	2136	25.5

Heterogeneity $\chi^2_2 = 5.8$ $p > 0.05$

W. c.f. C $\chi^2_1 = 3.82$ $p > 0.05$

U.W. c.f. C $\chi^2_1 = 0.084$ $p > 0.05$

W. c.f. U.W. $\chi^2_1 = 2.67$ $p > 0.05$

(Individual χ^2 values calculated by 2 x 2 contingency tables)

No significant difference of results is observed indicating that the working, and hence depleting of the medium by larvae, does not have any significant effect on the adult eye formation.

This lack of medium effect on the adult eye disagrees with the results of both Sang and Burnet (1963, 1964), and Hunt and Burnet (1969). However, Hunt and Burnet (1969) state that "the relevant environmental factors must be, to a great extent if not entirely, density dependent". From this, and the results of experiment 1 where the "high density" tubes gave a significantly lower percent of eyes-absent than the lower density tubes, it appears that possibly population size rather than medium effect is affecting the percent of eyes absent in adults. Whether this "crowding effect" operates through a rapidly lost volatile substance or by some mechanical effect is not known, but considerable evidence has been accumulated (Chester, 1969) to show that the effect does operate. Therefore, it is necessary to control rigorously the larval density when studying the expressivity and penetrance of eyeless.

References: Chester, B.M., 1969 Unpublished B.Sc. (Hons.) Thesis, Flinders University of South Australia; Guthrie, D.D., 1925 J. Expt. Zool. 42: 307-314; Hunt, D.M. and B. Burnet, 1969 Genetics Res. 13: 251; Morgan, T.H., 1929 Publ. Carneg. Inst. 399: 139-168; Spofford, J.B., 1956 Genetics 41: 938-959; Sang, J.H. and B. Burnet, 1963 Genetics 48: 1683; Sang, J.H. and B. Burnet, 1964 Heredity 19: 170.

Leenders, H.J. University of Nijmegen, The Netherlands. Temperature induced puffs in Drosophila: their possible physiological origin.

A sudden rise in environmental temperature (25-37°C) induces characteristic changes in the chromosomal puffing pattern of Drosophila[1,2,3]. Various suggestions have been made with respect to the intracellular mechanism responsible for the activation of the responding loci. Since in vitro treatments of larval salivary glands with 10^{-3}M dinitrophenol (DNP) affected the puffing pattern in essentially the same manner as a temperature treatment[1], it was supposed that either a deficiency in ATP or an accumulation of ADP might be the trigger for the activating mechanism of the responding loci.

In order to test this possibility, the effect of in vitro administration of ATP, ADP, malate and succinate to mid-third instar glands of D. hydei was studied. All incubations were performed under Arachis oil and the response of the loci 2-48C, 2-36A, 2-32A and 4-81B (temperature puffs) was taken as an indication for the activation of the induction mechanism. Neither addition of 10^{-2}(or 10^{-3})M ATP, nor of 10^{-2}(or 10^{-3})M ADP to the incubation medium resulted in the activation of these loci. Addition of 10^{-3}M DNP resulted, as expected, in their activation. However, a slightly increased concentration 3.10^{-3}M DNP appeared to be ineffective. If 10^{-3}M ATP is present in the medium 10^{-3}M DNP has no effect upon the puffing pattern, whereas addition of 3.10^{-3}M DNP to the same medium results in the induction of the puffs. A further increase in DNP concentration ($>5.10^{-3}$M) appears to be ineffective again.

Incubation media containing ATP were also used to investigate the effect of a temperature treatment, CO_2 and N_2 treatments. These treatments all resulted in the appearance of temperature puffs[4]. Thus it seems that, though a deficiency of ATP (or accumulation of ADP) cannot be excluded as possible intracellular trigger for puff induction, it may well be that the change in the level of these metabolites is a consequence of an enhanced metabolism.

Support for this assumption came from experiments in which malate (10^{-2}M), which upon metabolization may supply hydrogen to the coenzyme NAD^+, or succinate (10^{-2}M) which may support the hydrogenation of quinones, was supplied to the incubation medium prior to the addition of 10^{-3}M DNP. These substances did inhibit puff formation of the loci 4-81B, 2-32A and 2-36A but not of 2-48C. It was further found that oxymethylene blue (10^{-2}M) and menadione (10^{-3}M) (vit. K3), compounds which may serve as intermediates in the electron transfer system can induce temperature puffs in vitro. These data may be interpreted as support for the assumption that temperature puffs are induced by an increase in oxidation rate, leading to a temporary shortage of hydrogenated metabolites of the respiratory chain (or a temporary accumulation of oxidized compounds). The effect of DNP may be explained in terms of a common property of uncouplers, the stimulation of oxidation.

References: 1. Ritossa, F.M., 1964 Exptl Cell Res. 35: 601; 2. Berendes, H.D., F.M.A. van Breugel and T.K.H. Holt, 1965 Chromosoma 16: 35; 3. Ashburner, M., 1970 Chromosoma 31: 356; 4. van Breugel, F.M.A., 1966 Genetica 37: 17.

Schalet, A.* University of Connecticut, Storrs, Connecticut. Suppressor of forked: insertion into a sc^8 chromosome; high frequency of X-ray-induced deficiencies.

Since the proximal heterochromatic region of the X chromosome in Drosophila melanogaster, especially when it is distal or (partly) absent, has been a favorite playground for Drosophila geneticists of many persuasions, the insertion of the heterochromatically located mutant su(f) into a sc^8 inversion chromosome should prove to be a useful tool in different types of experiments. When ♀♀, y^+ sc^8 Df(1)mal^{10} B/1J1 y^{J1} sc^{J1} v f mal^1 su(f) are crossed to ♂♂, Y/y v sw mal^2, the only non-crossover class to survive are ♀♀ carrying the y^{J1} marker. This is because mal^{10} which includes sw is lethal in the combination mal^{10}/sw, and ♂♂ are inviable because of mal^{10} or 1J1. Regular offspring marked with y^+ come from a crossover chromosome that must have had one exchange between y^+ and mal^{10}. Of 59 ♂♂ observed, 1 proved to be y^+ sc^8 su(f)mal^1 f v. Of 75 y^+ ♀♀, 51 gave sufficient offspring in crosses to B^SY/y ac In49 v f mal^1 su(f) ♂♂ to show that only 1 carried su(f) in the sc^8 chromosome. The 2 crossovers of independent origin were found among a crudely estimated total of 57,000 chromosomes.

A number of lines of genetic evidence, (Lindsley and Sandler, 1958; Zimmering, 1959; Herskowitz, Schalet, and Reuter, 1962; Schalet, 1963; Schalet and Finnerty, 1968), have pointed to the probable penultimate position of su(f), i.e., close to the left of bb, in the proximal X. The mitotic X cytology of Cooper (in Lindsley and Grell, 1968) and the polytene

X cytology of Lefevre (Schalet, Lefevre, and Singer, 1970) has indicated that su(f) is located in hD of the mitotic X and division 20 of the polytene X. Accordingly, this locus should show a high frequency of X-ray-induced "mutation" long known for loci placed by rearrangements, close to polytene division 20. When y^+ sc^8 $su(f)^+$ mal^2 f ♂♂ were treated with 4,000r and crossed to y ac In49 v f mal^1 su(f) ♀♀, the following incidence of whole-body changes were observed among 2,900 ♀♀ offspring derived from sperm utilized during the first 6 days of egg laying: 12 $su(f)^-$; 7 y^- ac^-; 4 y^- ac^- $su(f)^-$. In addition there were 9 y^+ ac^+ v mal ♂♂ derived from a deleted X, but only 1 of these was $su(f)^+$. The use of the su(f) marker, as in the above experiment, in conjunction with y, ac, bb and the lethal loci localized to division 20, should prove to be a useful tool in the analysis of the "fine-structure" of induced breakpoints in division 20.

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Lakhotia, S.C.[1] and A.S. Mukherjee. University of Calcutta, India. Hyperactivity of the polytene X-chromosome in male D. kikkawai and D. bipectinata.

The functional morphology and the transcriptive activity of the polytene X-chromosome in male and female Drosophila kikkawai and Drosophila bipectinata have been examined. Both the species belong to the melanogaster species group and while D. kikkawai (related to D. montium, Burla, 1954) has an acrocentric X, D. bipectinata (related to D. ananassae, Patterson and Stone, 1952) has a submetacentric X-chromosome, displaying a long (XL) and a short (XR) arm in the salivary gland nuclei. In both the species, the single X-chromosome in larval salivary glands of male is pale stained and is, like that in D. melanogaster, as wide as the paired autosomes or the two X's in female. H^3-uridine autoradiography shows that in both the species, the relative rate of RNA synthesis by the single X of male is similar to that by the two X's of female. The data on grain counts are presented in Table I. The results indicate

Table I. H^3-uridine Incorporation in Male and Female Salivary Gland Nuclei

SEX & SPECIES	MEAN NO. OF GRAINS ± S.E.			MEAN GRAIN RATIO ± S.E.	
A. D. kikkawai	X	3R		3R/X	
Female (16)	233±22	141±13		0.61±0.015	
Male (18)	293±33	195±24		0.65±0.016*	
B. D. bipectinata	XL	XR	3L	3L/XL	3L/XR
Female (11)	242±25	162±27	257±27	1.05±0.03	1.56±0.07
Male (12)	115±19	72±12	113±22	0.98±0.04*	1.62±0.06*

* Ratios in male are non-significantly different from corresponding ratios in female. Numbers in parentheses indicate the number of nuclei examined.

that: (a) the enlargement and pale staining of the single X-chromosome in larval salivary glands of male is of general occurrence in the genus Drosophila; (b) despite the changes in the configuration and organization of the X-chromosome in these species (that have taken place during their evolution, see Patterson and Stone, 1952), the hyperactivity of the male X, and therefore, dosage compensation for X-linked genes (Lakhotia, 1970; Lakhotia and Mukherjee, 1970), has remained unchanged. (Work supported by UGC Fellowship to S.C.L.).

References: Burla, H., 1954 Rev. Brasil. Biol. 14: 41; Lakhotia, S.C., 1970 Ph.D. thesis, University of Calcutta; Lakhotia, S.C. and A.S. Mukherjee, 1970 J. Cell Biol. 47: 18 Patterson, J.T. and W.S. Stone, 1952 Evolution in the genus Drosophila, MacMillan & Co., N.Y.

[1]Present address: S.C. Lakhotia, Cytogenetics Lab., Department of Zoology, University of Delhi, Delhi - 7, India.

Pipkin, S.B. and N.E. Hewitt. Howard University, Washington, D.C. The influence of the X chromosome on specific activity of alcohol dehydrogenase of Drosophila.

The electrophoretic mobilities of Drosophila alcohol dehydrogenase (ADH) fast and slow isozyme variants, respectively, are correlated with the specific activities (SA) of these variants, measured spectrophotometrically. The assay mixture consisted of 0.05ml 2-butanol, 0.05ml NAD^+ (10 mg/ml), 0.2ml enzyme homogenate taken from 10 flies 4-6 days old, ground in 1ml 0.004 M K_2HPO_4 buffer and filtered; 0.8ml of the same buffer. SA is expressed as micromoles of NAD^+ reduced/1.1 ml/min/mg live weight.

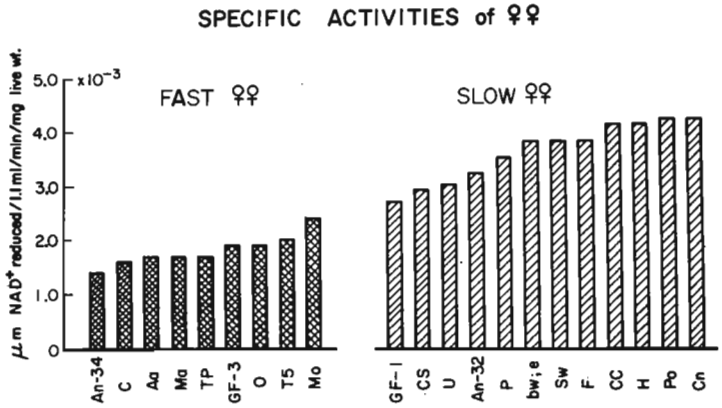


Fig. 1

Eight fast strains of diverse geographic origin have significantly lower SA than 12 slow ADH strains (Fig. 1). The range of SA of the fast strains is less than that of the slow strains. Fast ADH strains studied include those from Oahu, Hawaii; Canton, Ohio; Australia; Malvern, Arkansas; Austin, Texas; and Montgomery County, Great Falls, and Takoma Park, Maryland. Slow strains include those from Hikone, Japan; Ponape, Caroline Islands; Sweden (Swedish-b); Lima Peru; Urbana, Ill.; Florida; Cool Springs and Great Falls, Maryland; Austin and Corpus Christi, Texas; and the laboratory strains cinnabar and brown ebony. In both fast and slow strains males show higher SA than females, but the difference between the sexes is greater in the slow strains. Two experiments show that the X chromosome has a regulatory influence on the SA of ADH, the structural locus of which is located in the second chromosome (Grell et al., 1965). In the progeny of T5 fast triploid females with slow ADH males of four different strains, the 2X3A intersex progeny displayed SA similar to that of their respective 1X2A diploid male siblings and higher than that of 3X3A and 2X2A female siblings (Table 1). Triploid female progeny with 2X's derived from the T5 strain and one from the paternal strain showed SA not differing significantly in 3 of the 4 crosses, but the SA of the corresponding diploid (2X2A) female siblings varied significantly and was in each case higher than that of the 3X3A triploid siblings. The cause of this variability is in part due to the fact that diploid females included both those inheriting their two X's from the T5 triploid female parent and those inheriting one maternal X and one paternal X chromosome. In a second experiment the SA of hybrids between slow and fast strains was found to be intermediate between the SA of parental strains but sometimes differed from that expected on the hypothe-

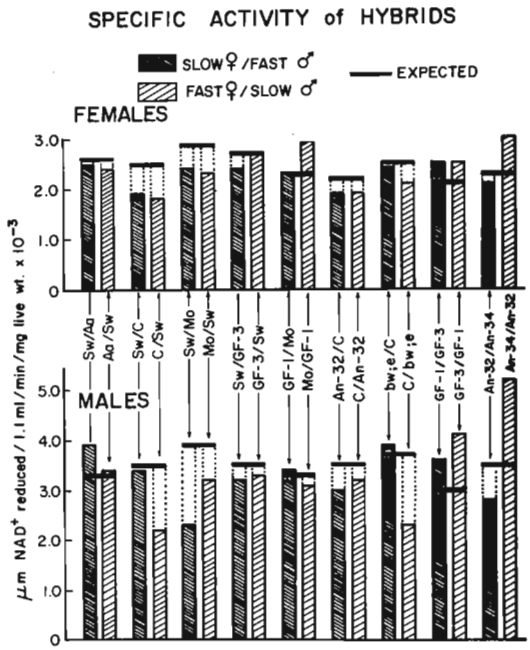


Fig. 2

Table 1

T5 FAST TRIPLOID ♀ PARENT

♂ PARENT ↓	PROGENY 3X3A ♀	2X3A intersex	2X2A ♀	1X2A ♂
T5 fast	1.47 ± .06	3.12 ± .20	1.91 ± .10	2.72 ± .13
ADH_{n_1}	0.9 ± .03	1.8 ± .21	1.1 ± .10	1.9
CC slow	2.46 ± .04	3.69 ± .23	2.96 ± .12	4.05 ± .19
H slow	2.03 ± .20	4.42 ± .45	4.36 ± .16	5.09 ± .35
Sw slow	2.12 ± .10	3.01 ± .37	2.47 ± .11	4.07 ± .49
Cn slow	2.11 ± .16	4.05 ± .06	2.91 ± .21	4.09 ± .24

μm NAD^+ reduced/1.1ml/min/mg live wt x 10^{-3}

sis of simple additive contribution to SA by the parental strains. The X chromosomes of different strains have a special effect on ADH activity of slow/fast hybrids. This is shown by the greater variability in SA of hybrid males (each carrying a single X of maternal origin) than of hybrid female progeny of reciprocal crosses of slow and fast strains (Fig. 2). Further, female hybrids of reciprocal crosses show SA differing less than those of male hybrid siblings from the SA values expected on the hypothesis of simple additive contribution to ADH activity of hybrids by the parental strains. In a cross of a mutant strain with no ADH activity (Grell's ADH_{nl}) with T5 triploid females, all progeny (3X3A, 2X2A, 2X3A and 1X2A) had about the same SA as expected on the hypothesis that each active ADH^F allele derived from the T5 triploid parent is coding for protein at the same rate as in corresponding control T5 triploid, intersex, and diploid forms, respectively (Table 1). In the progeny of ADH_{nl} males with T5 triploid females, the 2X3A intersexes and 1X2A diploid males have similar activities, about twice that found in the 3X3A and 2X2A females. This suggests that the ratio of X to autosomes is not only responsible for determining the sex type but also for determining a male or female like character of ADH activity. Supported by NSF Grant GB 8770.

Reference: Grell, E.H., K.B. Jacobson and J.B. Murphy, 1965 Sci. 149: 80-82.

Parkash, O. Institut für allgemeine Biologie, University of Vienna, Austria.

The behavioural changes produced by thymidine-induced temperature-sensitive lethal factors in D. melanogaster.

In some of the earlier publications, the author reported on the induction of absolute and incomplete (temperature-sensitive) lethals by feeding D. melanogaster on thymidine-containing culture medium. Further, the special significance of the temperature-sensitive lethals in general biological research was pointed out. The existence of the modifier genes, for example, was inferred from the analysis of one of these temperature-sensitive lethals (1969). This particular lethal manifested its lethal effect at 16°C, whereas at 26°C it behaved as a non-lethal. The experiment has been continued and some more such factors have been analysed in the mean time. Because of their general interest, it is thought worthwile to report on two of such factors in the present note.

One of these lethal factors $l^{ts}(1)$ 42.3±0.5 showed its effect in the direction opposite to that of the one reported earlier. This factor manifests its lethal effect at 26°C, whereas at 16°C it behaves as a non-lethal. This, incidently, is interesting as it indicates that thymidine is capable of inducing temperature-sensitive lethal mutations, some of which manifest their effect at lower while the others do so at higher temperatures.

The second one of the two factors, so far as the lethal effect is concerned, resembles the one reported earlier. This has been maintained in a balanced stock against y sc^{S1} In49 sc^{8} at 16°C. An attempt was made to obtain a pure stock of this l^{ts}. The cultures were kept at 26°C to obtain the normal-looking males carrying the lethal factor. The repeated attempts failed, as it turned out that the l^{ts} males were very sensitive to the ether anesthesia and the slightest amount was detrimental to them. The CO_2 anesthesia was less injurious; however it left these males so weak that they died an hour or so later. In a last attempt, the l^{ts} males were separated by employing a suction tube and no anesthesia at all. This method of separation succeeded but it appeared as if the males were infertile. Their mating behaviour was watched by the author and it was found that these males did not respond to the females (aversion to mating?). This abnormal behaviour was very conspicuous and this led the author to search for other physiological reasons. Consequently the males were carefully scrutinised but no macroscopic anomaly was detected. A dissection was then carried out and the testes examined under a microscope. This examination revealed a complete absence of sperm, the testes themselves being normal in size and appearance. The abnormal mating behaviour can, therefore, be traced back to the absence of the sperm. Here we have a case of genetically determined "aspermia" and the consequent abnormal mating behaviour and this may have its parallels in other animal domains. The attempts to localise this factor have, so far, been unsuccessful. Most probably, this is a compound lethal involving more than one locus.

This note points in favour of the study of the temperature-sensitive lethals. The work is being continued and will be reported elsewhere. Thanks are due to Prof. Mainx and Doz. Sperlich of the University of Vienna for their generous help.

Heisenberg, M. Max-Planck-Institut für biologische Kybernetik, Tübingen, Germany.

Isolation of mutants lacking the optomotor response.

A multiple-Y-maze was used to select D.m. mutants lacking the optomotor response. The procedure for mutagenesis which yields sex-linked recessive mutations was the same as used by S. Benzer (Proc. Nat. Acad. Sci., 1967, 58: 1112), for the isolation of non-phototactic mutants. From 60,000 flies tested, 17 mutants were obtained which belong to 5 complementation groups.

Group	Mutants	Location on X-chromosome
I	opm 3,4,5,9,10,14	7 ± 1
II	opm 8 (tan)	27.5 (tan)
III	opm 1,7,13,15,16	29 ± 1
IV	opm 6,11,12	v < X < f
V	opm 2,17	56 ± 5

All mutants have abnormal electroretinograms (ERG's) and belong most likely to the same complementation groups as the ERG mutants isolated by S. Benzer and co-workers (in preparation).

In Group I mutants de- and/or re-polarisation of the retinula cells seem to be disturbed. No ERG has been found in mutants opm 4 and 9; mutants opm 3, 10 and 14 show a comparitively small and extremely slow de- and re-polarisation. In mutant opm 5 depolarisation is fast but repolarisation is very slow. The ERG of this mutant consists of a large lamina potential in addition. In all mutants of this group the optical axes of the retinula cells as judged by the deep pseudopupil are properly oriented but in contrast to wild type no green reflecting screening pigment is observed at the rhabdomers 1-6 under strong illumination. The mutants opm 3 and 5 are not completely blind behaviorally.

The ERG's of Group II and V mutants show an altered lamina potential. The receptor potential, the orientation of the rhabdomers and the pigment migration mechanisms are about normal. Light sensitivity determined by slow phototaxis is diminished by a factor of 50. The optomotor response is not completely lost. (A detailed description is in preparation.)

Group III and IV: The ERG's range from less than 1/50 to about 1/5 the normal amplitude with the lamina potential missing. Mutants without a detectable ERG do not show any slow phototaxis nor an optomotor response. In most cases the deep pseudopupil is greenish-grey and less clearly visible than in wild type. No pigment migration is observed.

Young flies of mutant opm 12 have a normal pseudopupil which, however, does not show pigment migration. The optomotor response is normal at high light intensity but absent in dim light. The ERG has about 1/5 the normal amplitude and consists of the receptor potential only. More than 10 day old flies have no pseudopupil, no ERG and no optomotor reaction. The retinula cells of this mutant seem to degenerate.

Gvozdev, V.A., V.J. Birstein, L.G. Polu-Karova and V.T. Kakpakov. Kurchatov's Institute of Atomic Energy, Moscow, U.S.S.R.

Expression of the sex-linked genes in the established aneuploid sublines of Drosophila melanogaster.

A number of aneuploid sublines of embryonic cells of Drosophila melanogaster were obtained by cloning of the tetraploid subline. 70 - 90% cells in these sublines were characterized by their specific karyotypes remaining unchanged during at least 60 - 80 cell generations. The karyotypes of these aneuploid sublines may be represented as 4X+6A and 5X+5A as compared to diploid karyotype 2X+4A (X-sex chromosomes, A-large autosomes without taking into account the microchromosomes of the fourth pair). The cell size and protein content per cell is equal for both aneuploid and diploid cells.

The increase in the number of X-chromosomes in both aneuploid sublines is not accompanied by the raising of the specific activities of the X-linked 6-phosphogluconate and glucose-6-phosphate dehydrogenases. However in flies with different doses of corresponding structural genes, due to the duplications or deletions of a part but not whole X-chromosome the specific activities of these enzymes have increased almost proportionally to gene dose (Seecoff et al., 1969; Gvozdev et al., 1969).

The absence of proportionality of specific activities of 6-phosphogluconate and glucose-6-phosphate dehydrogenases to the number of X-chromosomes carrying their structural genes may be therefore attributed to a phenomenon of dosage compensation for sex-linked genes which may take place not only in whole flies but also at the cell level in culture.

References: Gvozdev, V.A., V.J. Birstein and L.Z. Faizullin In: Structure and genetical functions of Biopolymers, Moscow, 1969, Vol. I: 137-165 (in Russ.); Seecoff, R.L., W.D. Kaplan and D.G. Futch, 1969 Proc. Natl. Acad. Sci. 62, 2: 528-36.

Alexandrov, I.D. Research Institute of Medical Radiology, Academy of Medical Sciences of U.S.S.R., Obninsk, U.S.S.R.

Mutation isoalleles or modification of frequencies of radiation-induced viable point mutations by attendant chromosome rearrangements in D. melanogaster?

First Timofeeff-Ressovsky (Biol. Zbl. 52: 468-476, 1932) for w^+ locus and then Lefevre (Genetics 40: 374-387, 1955) for y^+ locus reported the existence of alleles with significantly different rates of mutations following X-irradiation of adult males. Such alleles were termed "mutation isoalleles". In both cases the detected frequencies of viable point mutations served essentially as an estimate of mutability of the wild-type homoalleles.

In a previous note (DIS 44: 78) the preliminary data on comparative mutability of five wild-type genes in two stocks of D. melanogaster (D-32 and D-18) induced gamma-irradiation (4000 r) of adult males were presented. The frequencies of point mutations permitted to suggest the existence of the mutation isoalleles for y^+, w^+ and cn^+ loci. Genetics and cytogenetics analyses allowed further to determine more precisely the frequencies of these mutations. They are listed in the Table below. It may be seen that the detected frequencies of viable point mutations for each locus are nearly three times as high in one stock (in D-18 for the y and w but in D-32 for the cn) as in the other. The inter-stock difference for w is statistically significant. The w^+ locus is known to be one of the highly mutable loci. Therefore, lack of the significant difference for y and cn point mutations may well depend on the insufficient samples of examined flies. All the same, this finding, in principle, is parallel to those reported by Timofeeff-Ressovsky for w^+ alleles and Lefevre for y^+ alleles.

However, if y, w and cn mutations being taken into consideration with attendant physiological effects (sterile F_1 mutations, mutations with recessive lethality) and viable chromosomal rearrangements, the mutation rates for each of the loci are practically the same in our two stocks. It seems reasonable to suppose that many of these physiological effects are associated with chromosomal rearrangements of all kinds (for example, large deletions or translocations for the sterile mutations according to Lefevre, Genetics 55: 263-276, 1967; ibid., 63: 589-600, 1969; Lindsley, Proc. XII Intern. Congr. Genet. I: 144, 1968).

Thus the same gene mutation may be accompanied by rearrangements of some kinds occurring with definite but different probability in each of the two stocks. Therefore, the rearrangements occurring with different frequencies in different genotypes may differently modify the frequency of true point mutations. If this is the case, the detected frequencies of viable point mutations in different stocks may hardly provide evidence of different mutational potentialities of homoalleles thenselves. Do the "mutation isoalleles" exist in this case? At least, all cases of the "mutation isoalleles" in D. melanogaster may be as well explained now by this hypothesis of modificatory effect of independently and simultaneously occurring chromosomal rearrangements as an attendant factor with regard to intragenic mutation.

One more aspect of the finding requires explanation: What does determine in the two stocks the different probability of complication of point mutations by rearrangements? So long as our data were obtained under the same experimental conditions and the pattern of difference in the frequencies of w and cn point mutations in D-32 is opposite to that in D-18 the stochastic of complication of these point mutations by rearrangements seems to depend upon some peculiarities of chromosomal environment neighbouring to homoalleles rather than the peculiarity of genotype as a whole. The nature of these chromosomal differences is still obscure although it can point to a possible role of amount of intercalary heterochromatin adjacent to a particular locus. The high radiosensitivity of this heterochromatin in respect to breakage is known, and the quantity of that in different stocks may be quite different (Lefevre, 1955, loc. cit.).

(Table on next page:)

The different types of y, w, cn mutations following gamma-irradiation (4000 r) of adult males of two wild-type stocks of D. melanogaster.

				Mutations with attendant chromosome rearrangements						
		Point mutations		Sterile F_1 mutations		Recessive lethality	Viable chr.rear.	Total		Overall mutations		Total chr.
Loci	Stocks	Mut.freq.**	P***	Mut.freq.	P	Mut.freq.	Mut.freq.	Mut.freq.	P	Mut.freq.	P	studied
y^+	D-32	(1)*0.69	>0.05	(1) 0.69		(2)1.38	(1)0.69	(4) 2.78		(5) 3.47	>0.05	35,843
	D-18	(3)2.27		(1) 0.76		(0) 0	(2)1.52	(3) 2.27		(6) 4.54		33,000
w^+	D-32	(3)2.09	=0.05	(20)13.94	>0.05	(2)1.39	(0) 0	(22)15.34	>0.05	(25)17.43	>0.05	35,843
	D-18	(10)7.57		(11) 8.33		(1)0.76	(2)1.52	(14)10.61		(24)18.18		33,000
cn^+	D-32	(11)4.10	>0.05	(8) 2.98	<0.05	(3)1.12	(3)1.12	(14) 5.22	>0.05	(25) 9.32	>0.05	67,030
	D-18	(5)1.87		(20) 7.50		(3)1.13	(0) 0	(23) 8.63		(28)10.50		66,614

* In parenthesis the number of mutations found is given.
** The average mutation frequency/locus/r x 10^{-8}.
*** P was estimated by use of the tables of Kastenbaum & Bowman (Mut. Res. 9: 527-549, 1970).

Khovanova, E.M. Research Institute of Medical Radiology, Academy of Medical Sciences, Obninsk, U.S.S.R. Somatic mosaicism in D. melanogaster x D. simulans hybrids.

The frequency of somatic mosaics among D. melanogaster x D. simulans hybrids was studied in two series of experiments. In the first series (♀♀ y/y D. melanogaster x ♂♂ +/Y D. simulans) 158 hybrid females out of 1460 had mosaic spots (1 or 2 yellow macrochaetae) which corresponds to 108 per 1000. In the second series (♀♀ y, Muller-5/y, Muller-5 D. melanogaster x ♂♂ +/Y D. simulans) mosaic spots were observed in 345 out of 2479 hybrid females, i.e. 139 per 1000. This result was quite unexpected, because in our preceding experiments with D. melanogaster heterozygous inversions markedly (by about an order of magnitude) decreased the frequency of somatic mosaics, namely 59 out of 8214 y/+ D. melanogaster females, or 7.2 per 1000, had yellow spots, as compared to only 3 out of 5100, or 0.6 per 1000 y, Muller-5/+ D. melanogaster females. A short inversion dl-49, being heterozygous, exerted only a small effect almost indistinguishable from the results of y/+ D. melanogaster series (23 out of 4121, i.e. 5.6 per 1000 y, In dl-49/+ D. melanogaster females). Mosaic spots in D. melanogaster x D. simulans hybrids may be due to somatic crossing-over, point mutations, deletions, or elimination of X-chromosome, bearing normal allele in yellow locus. It may be suggested that the role of somatic crossing-over in the origin of mosaic spots in hybrids is considerably smaller than in heterozygous D. melanogaster females, in which case most of the spots result from somatic crossing-over, and an inversion in one of the homologous X-chromosomes sharply decreases the frequency of mosaics. It is probable that more important role in the origin of somatic mosaicism in D. melanogaster x D. simulans hybrids is played by elimination of one of the X-chromosomes. The research of the mechanisms of somatic mosaicism in D. melanogaster x D. simulans hybrids is in progress now.

Alexandrov, I.D. Research Institute of Medical Radiology Academy of Medical Sciences of U.S.S.R., Obninsk, U.S.S.R. A preliminary analysis of negative complementation in white locus of D. melanogaster.

In a previous note (of DIS, this issue) the data on quantitatively different antimorphic action of two pseudo-allelic w mutants were presented. This difference was observed in different heterozygotes and that (the first fact) was why it was considered to be fundamentally interallelic. To test if this difference is connected with the site of w mutants on the genetic map of white locus, the further experiments included more w mutants as well as deletions of the whole w^+ locus. The w^1, w^{10gA} (both suppress z), w^{13gA}, w^{15gA}, w^{57gA}, w^{59gA}, w^{60gA}, w^{69gA} (all gamma-ray induced and do not suppress z) mutants and the deletions w^{258-45} and w^- were used. The w^- is a recombinant combining the left end of $In(1)w^{m4}$ with the right end of $In(1)w^{mJ}$. The w^+ allele from D-32 stock was used throughout.

For quantitative determination of red eye pigments in ♀♀ heterozygous for w^{+32} and any one of the mutations the spectrophotometric method was applied, the details of which may be found in a previous note. The quantities of red pigments are also expressed here as the extinction (E) per 10 heads extracted per 1 ml of 30% AEA.

The results of these analyses are listed in Table below. It may be seen that these data confirm the existence of difference in antimorphic action of w^{10gA} and w^{69gA} mutants. They are found to be the extreme items of the series of w mutants studied. It is important that all white mutants have pronounced antimorphic action in contrast to the action of deletions. So, ♀♀ heterozygous for any of two deletions and ♀♀ homozygous for w^+ have practically the same quantities of red pigment. This is the second fact which shows that the decrease in red pigment content in ♀♀ heterozygous for any of the w mutants studied is a result of the action of the w mutants themselves. This effect of the w mutants may be considered as a negative complementation at the phenotypic level. As seen, the negative complementation permits w mutants of the left or the right sections of the white locus to be distinguished more clearly from each other. It is important that w^1 and w^{15gA} positioned in different sections of the locus have the same degree of negative complementation. This fact shows that w mutants with functionally the same effects may occur in both the left and the right sections of the white locus and, thus, no drastic effect of w^{69gA} may be the peculiarity of the left section of the locus. The characteristics of negative complementation in the white locus and the possibil-

Red pigment content in ♀♀ heterozygous for different w mutations as an indication of negative complementation.

w mutations	M*	Conf. limits at $P_{0.05}$	
1. w^-	0.630	0.637 - 0.623	
2. w^{258-45}	0.655	0.677 - 0.633	
3. w^{10gA}	0.579	0.601 - 0.557	suppress z
4. w^1	0.520	0.540 - 0.500	
5. w^{59gA}	0.531	0.550 - 0.512	
6. w^{15gA}	0.520	0.532 - 0.508	
7. w^{57gA}	0.511	0.520 - 0.502	
8. w^{13gA}	0.510	0.518 - 0.502	don't suppress z
9. w^{60gA}	0.487	0.499 - 0.475	
10. w^{69gA}	0.436	0.454 - 0.418	
w^{+32}/w^{+32}	0.632	0.670 - 0.594	

* Means at least of 7 replicas

ities of the use of this phenomenon for investigation of such problems as the structure and function of the locus in question are at present studied.

(I wish to thank Eileen S. Gersh for w^-.)

Alexandrov, I.D. Research Institute of Medical Radiology, Academy of Medical Sciences of U.S.S.R., Obninsk, U.S.S.R.

The functional w^+ isoalleles revealed by w mutations in D. melanogaster.

Quantitative analysis of red eye pigment content in w^+/w ♀♀ was successfully used for the estimate, more sensitive than visual observation, of the difference in phenotypic action of w^+ alleles from two different stocks of D. melanogaster (Green, Proc. Nat. Acad. Sci. 45: 549-553, 1959). After the radiation-induced mutability of w^+ alleles from wild-type stocks D-32 and D-18 (w^{+32} and w^{+18}, respectively) had been studied (cf. DIS, this issue) the quantitative analysis of phenotypic action of these alleles was undertaken. Simultaneously, the analysis of action of w^+ alleles from ten other wild-type stocks of D. melanogaster was carried out. Gamma-ray-induced mutations w^{10gA} (allele of w^1, suppressing z) and w^{69gA} (pseudo-allele of w^1, not suppressing z) obtained in our radiation experiments were used.

For the quantitative determination of red eye pigments the spectrophotometric method of Ephrussi & Herold (Genetics 29: 148-175, 1944) was used with minor modifications. This involves extraction of pigments from 30 heads (from which the clypeus and proboscis were removed and an incision between the eyes was made) for 48 hours at 25°C with 3 ml of 30% ethanol acidified to pH 2.2 with concentrated hydrochloric acid. After extraction, quantitative analyses of the pigments in 2 ml cuvettes were made with an Jurány-Kovács model (Hungary) extinctiometer at wave length of 480 mμ. The data are expressed as the extinction (E) per 10 heads extracted per 1 ml of solvent. In addition, the determinations of red eye pigments of w^+/w^+ ♀♀ from each stock were made. All flies were fed on standard mannacroup - sultana - molasses - agar - yeast medium at the same level of crowding and were aged for at least 5 days before the analysis.

The results of these analyses are the following. The quantities of red pigment are different in homozygous +/+ ♀♀ from stocks of dissimilar origin. If the latter are set in order of decrease in red pigment content they form the series listed in Table 1. This phenotypic variability may well be explained by concomitant consequences of inbreeding according to Lerner (Genetic Homeostasis, N.Y., 1954), taking into account relatively high homozygosity of laboratory wild-type stocks. The overall heterozygosity of w^+/w^{10gA} or w^+/w^{69gA} ♀♀ smooths out the effect of certain genotypic modifiers and reveals clearly the specific action of major locus (w^+) on the phenotypic trait in question. It may be seen from the Table 2 that the action of w^+ alleles from different stocks is quite different. Analysis of variance of these

Table 1. Red eye pigment content in w^+/w^+ ♀♀ of twelve stocks.

Stocks	M* ± S.E.	Stocks	M* ± S.E.
1. Centre-1	0.903 ± 0.0033	7. Canton-S	0.747 ± 0.0033
2. Swedish	0.840 ± 0.0115	8. P-86	0.747 ± 0.0033
3. Sevelen	0.833 ± 0.0127	9. Magarach	0.710 ± 0.0057
4. Graaff-Reinet	0.833 ± 0.0127	10. Stellenbosch	0.669 ± 0.0137
5. Inozemceva	0.817 ± 0.0033	11. D-18	0.626 ± 0.0203
6. Oregon-R	0.760 ± 0.0152	12. D-32	0.622 ± 0.0141

* Means at least of 3 replicas

Table 2. The functional activity of w^+ of dissimilar origin revealed by two w pseudo-allelic mutations.

w^+ alleles of stocks	w^{10gA} M M*± S.E.	w^{10gA} M of groups Conf. limits	w^{69gA} M*± S.E.	w^{69gA} M of groups Conf. limits
1. Canton-S	0.738 ± 0.0180	0.738 / 0.783 - 0.703	0.642 ± 0.0127	0.642 / 0.668 - 0.616
2. Centre-1	0.683 ± 0.0066		0.503 ± 0.0033	
3. Magarach	0.673 ± 0.0033		0.533 ± 0.0066	
4. Inozemceva	0.647 ± 0.0033		0.507 ± 0.0033	
5. Oregon-R	0.637 ± 0.0228	0.639 / 0.689 - 0.589	0.527 ± 0.0033	0.522 / 0.529 - 0.515
6. P-86	0.637 ± 0.0033		0.513 ± 0.0066	
7. Graaff-Reinet	0.620 ± 0.0057		0.527 ± 0.0066	
8. Swedish	0.620 ± 0.0115		0.547 ± 0.0033	
9. Sevelen	0.617 ± 0.0117		0.517 ± 0.0066	
10. D-32	0.561 ± 0.0198		0.449 ± 0.0067	
11. D-18	0.547 ± 0.0033	0.551 / 0.575 - 0.527	0.447 ± 0.0033	0.451 / 0.463 - 0.439
12. Stellenbosch	0.533 ± 0.0033		0.473 ± 0.0033	

* Means at least of 3 replicas

data permits us to distinguish three groups of w^+ alleles with high (allele of stock Canton-S), middle (alleles of stocks ranging from Centre-1 to Sevelen) and relatively low (alleles of the last three stocks) levels of the functional activity. Alleles of three groups are functional iso-alleles as regards to each other. The w^{+32} and the w^{+18} are seen not to be iso-alleles. It is important that the composition of three groups coincides for ♀♀ heterozygous for both w^{10gA} and w^{69gA}. Then it was concluded that: (i) two studied w mutations are the pseudo-alleles with quantitatively different antimorphic action and (ii) the difference is fundamentally interallelic. Is this difference related to the different position of two w mutants on the genetic map of white locus? The results of experiments designed to obtain the answer to this question are presented in the following note.

(I wish to thank D.J. Nolte for stocks Graaff-Reinet and Stellenbosch.)

Graf, U. and F.E. Würgler. Swiss Federal Institute of Technology, Zürich, Switzerland. Influence of the maternal genotype on the rate of apparent X/O males after irradiation of mature sperm.

Two to three day old R(1)2, y B/y^+ Y males were X-rayed (50keV, 520 R/min) in nitrogen (20 min pretreatment) with 2000 R. Males for nonirradiated controls were only treated with nitrogen. Each sample of treated ring-X males was divided into two groups and every group mated for 7 hours to a different type of female. We used y sn^3 females and "Oster" females (Inscy;dp bw;st p^p). At the end of the mating period the males were discarded and the inseminated females transferred to standard culture vials. In order to get similar population densities we used 2 females per vial in the control series

but 5 in the X-ray series. The progeny obtained from a 2 day egg sample was recorded. Apparent X/0 males, which were characterized by a yellow body colour, were tested for sterility. All males could be included in the calculation of the rate of sex chromosome loss (X/0 males/females + males + X/0 males), because none were fertile. The following table contains the pooled data of 3 repeats, which gave very similar results:

dose	y sn^3 females	"Oster" females
0 R	0.71% (11/691+837+11)	0.54% (3/256+294+3)
2000 R	4.37% (44/406+557+44)	2.33% (46/750+1180+46)

With both types of females the sex ratio of the normal progeny (females/males) is decreased in the irradiated group. For the y sn^3 females it falls from 0.82 to 0.73, for the "Oster" females from 0.87 to 0.64. This results from the higher rate of X-ray induced dominant lethals in the ring-X-bearing sperms compared to the Y-bearing sperms (Bauer, H., 1942 Chromosoma 2: 407). In the irradiated series the rate of apparent X/0 males is nearly doubled if y sn^3 females instead of "Oster" females are used for the test cross. Statistically (Kastenbaum, M.A. and K.O. Bowman, 1970 Mutation Res. 9: 527) the difference between 4.37% and 2.33% is significant. On the other hand, our data with the "Oster" females are not different from the corresponding data of B. Leigh (1968, Mutation Res. 5: 432). He used the same type of males and Inscy;bw;st p^p females.

In order to test the possibility that the different rates of X/0 males result from a lower viability of Inscy/0 males compared with the y sn^3/0 males, we crossed females of both stocks to XY/0 (y su(w^a)w^a KS·KL y^+, Parker 110-8) males. With y sn^3 females we got 897 X/XY females and 1414 X/0 males. In the case of "Oster" females, we obtained 793 X/XY females and 925 X/0 males. These data show that with the "Oster" stock a somewhat lower frequency of X/0 males (53.8%) is found compared to y sn^3 flies (61.2%). This viability difference of X/0 males is far too small to explain the different rates of recoverable X/0 males in the X-ray experiment. Therefore we assume that the maternal genome, possibly via a repair system present in the oocytes (Rinehart, R.R., 1964 Genetics 49: 855), influences the rate of recoverable X/0 males after exposure of mature sperm in the male.

Work supported by Schweizerischer Nationalfonds zur Förderung der wissenschaftlichen Forschung.

Fowler, G. University of Oregon, Eugene, Oregon. Suppression of SD by an XY Ring.

Novitski has synthesized an XY chromosome which genetically and cytologically appears to be a ring (XY^c). Since it has been shown previously (Enns, 1970) that XY chromosomes reduce the k value of SD-72/"sensitive" cn bw from 0.99 to about 0.85, it was decided to combine XY^c with SD-72 bw (the SD-72 bw is recovered normally from the "sensitive" background with a k value of 0.99+). Using XY^c/0;SD-72 bw/cn bw males from three different XY^c lines, the following results were obtained:

	Total Progeny		
♂	SD-72 bw	cn bw	k value
Line 1	475	461	0.51
Line 2	124	122	0.50
Line 3	49	46	0.51

It seems clear from the findings that there appears to be a relationship between the recovery of SD-72 bw and XY^c such that in the presence of the ring the normal recovery of SD-72 bw (k = 1.0) is completely suppressed (k = 0.5). The fact that the recovery of SD can be altered by a number of different chromosomal rearrangements is well known. Complete suppression of the recovery of the SD chromosome when the X and Y chromosomes are in the configuration of a ring is an interesting addition to these observations.

Reference: Enns, R.E., 1970 DIS 45: 136.

Basden, E.B. Institute of Animal Genetics, Edinburgh, Scotland. Cataloguing of the world's Drosophilidae.

Every record of speciation, biology and systematics that concerns Drosophila and its allies is being abstracted to cards (8 x 5 inches = 20.3 x 12.7 cm.). There has to be four different, coloured cards: Species, Species-Group, Genus and Family. An example of the Species card is shown below. By this means all biosystematic information concerning one species, genus, etc. contained in any publication is on one card. If the particulars are too voluminous abbreviations must be used, or only the page reference to them is given. The headings are arranged so that information on one aspect can usually be continued up or down into an adjoining line if free.

The punch-card system is impracticable to use as the drosophilid cards are filed chronologically under species, etc. Cross-referencing of subjects is as quickly done by entering

NAME			DATE
REF.			
DESCRIPT.		KEY	FIGS.
SPECMNS.	Det.	Location	
TYPES			
SYNS.			
DISTRIBN.			
BIOLOGY			
		MONTHS	HAZARDS
VARIATN.		Sexl.	MUTANTS
RELATNSHP.			
HYBRIDS			
CHROMS.		PHYLOG.	HIST.
BIOCHEM.			
INT.ANAT.		PHYSIOL.	
MATING		OTHER BEHAV.	CULTURING
EGG	LAR.	PUP.	L-CYCLE
SPP.COMPARED			

SPECIES/SUBSPECIES CARD (DROSOPHILIDAE) E.B. BASDEN, INST. ANIMAL GENETICS, EDINBURGH

this on subject cards arranged alphabetically as it is to punch the appropriate edge, then needle through many thousand cards for the information.

The plan thereafter is for all information on a taxon to be grouped on a master sheet. But first of all the world's literature has to be combed and carded. Advice on electronic light-sorting has been sought from the Nature Conservance, Monks Wood.

Others may use this Species card idea if they wish.

Scharloo, W., G.M. Alink and J. van der Vlist. Rijksuniversiteit, Utrecht, the Netherlands. Stabilization of scutellar bristle number.

In previous experiments stabilizing selection was only successful in decreasing environmental variance when it was practised on the expression of a mutant (Rendel and Sheldon, 1959; Rendel, 1968; Scharloo, 1964; Scharloo, Hoogmoed and ter Kuile, 1967). We attempted to stabilize the number of scutellars on 8 bristles with use of genetic variability present in two "wild" cage populations.

We started selection for increased bristle number from the two unrelated populations. When both selection lines had a mean of about 8 (the response continued to means of 13 and 15 respectively) crosses between the lines were made. Stabilizing selection for 8 bristles was started from the F_3 and was continued for 25 generations. Phenotypic variance decreased to less than half the original value. A progeny test showed that this was partly a consequence of a decrease of the genetic variance. Probit analysis revealed that, while the width of the 7 and 9 bristle classes remained constant the width of the 8 class increased to more than two times the original value. Simultaneously the difference between mean values of ♀♀ and ♂♂ decreased from more than 1 bristle to 0.5 bristle. The predominant pattern of 8 bristles consisted of 4 anterior bristles, 2 posterior bristles and 2 apical bristles.

Bos, M. and Ch. van Dijken. University of Groningen, Haren (Gr), The Netherlands.

The fertility of large and small flies in a disruptive selection experiment with Drosophila melanogaster.

In previous experiments (DIS 44: 105, 1969) disruptive selection with random mating (D^R) on thorax length produced a 10% increase in length. This could be the result of a larger reproductive success of the large flies. Therefore two new D^R lines were started.

After G7 D^R2 showed an increase of thorax length (in G 11 the mean value (sexes averaged) was: in the Control line 102.1, in D^R1 102.2, and in D^R2 104.5 units. 1 unit=1/100 mm); in the D^R1 line the mean value rose slowly after G 11. The phenotypic variance, calculated as squared coefficients of variation (c.v.^{2}), in both D^R selection lines showed an increase (in G 11 c.v.^{2} was: in C 4.7, in D^R1 10.0 and in D^R2 12.2), which declined a little, when the mean value of thorax length started to rise. Egg production of selected females and the number of eggs, which yielded adults was determined in generations 1-5 and 10. The combined results are (Table 1):

Table 1: Mean egg production/♀/24 hr.

(Within brackets the number of eggs which yielded adults)

	D^R1		D^R2		C	
	Production	n	Production	n	Production	n
Large females	16.3 (7.4)	38	21.2 (10.9)	44	21.8 (12.2)	43
Small females	12.6 (7.5)	38	12.0 (8.9)	44	12.5 (6.3)	42

In each line the large females produced more eggs than the small females, but only in D^R1 this did not result in a larger production of adults. Mating success of large and small flies was tested in mating choice experiments. Two pairs of large flies and two pairs of small flies were brought together in a culture vial and the copulation types were scored. (Table 2). All flies were four days old and marked with a just-visible, not inconvenient spot of feltpen ink on the posterior end of the left or right wing. Large flies and small flies (within a culture) did always differ at least 7 units in thorax length.

Table 2: Number of the four possible types of mating recorded in the mating choice experiments (L=large, S=small).

	D^R1			D^R2			C		
♀ x ♂	G5	G10	Combined	G5	G10	Combined	G5	G10	Combined
L x L	6	8	14	10	12	22	6	3	9
S x L	4	10	14	10	12	22	5	2	7
L x S	2	9	11	1	4	5	2	4	6
S x S	5	6	11	1	8	9	3	5	8
Total	17	33	50	22	36	58	16	14	30

In the D^R lines L males are more successful than S males, but this is significant only in D^R2 (in G 5: $P<0.005$; in G 10: $P<0.05$). This and the greater egg-to-adult survival of the eggs of large females in D^R2 suggest indeed, that the increase in thorax length in disruptive selection lines with random mating could be the result of a larger reproductive success of the large flies in relation to the small individuals.

Postlethwait, J.H. University of California, Irvine, California.
Effect of X-rays of the eye of heterozygous Antennapedia flies.

In some Antennapedia ($Antp^R$) individuals irradiated with 1000r of X-rays, irregularities were found in the eye and head region, whereas no such abnormalities were found in unirradiated $Antp^R$ animals. These irregularities consisted of lumps of extra cuticle, bearing bristles which appeared in the region of the verticle or orbital bristles. The morphology of the supernummary bristles and the ground pattern was characteristic of this region of the head. The bristles often encroached upon ommatidia, causing the eye to be smaller. Frequently, a sector in the dorsal anterior part of the eye also contained bristles.

The frequency at which defects occurred in $Antp^R$ flies varied with the time of irradiation (Fig. 1), and

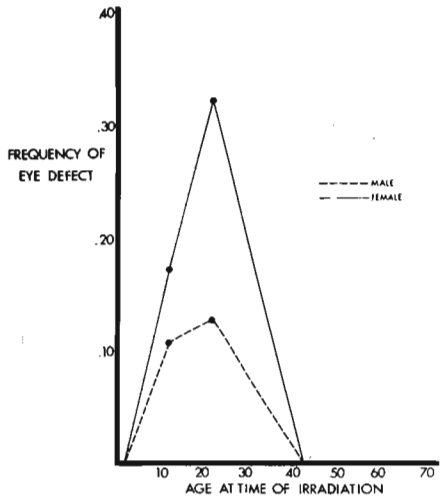


Fig. 1. Frequency of X-ray induced defects in the eye of $Antp^R$ flies vs. time of irradiation.

there was a peak at 24 hours after egg deposition. The penetrance of the eye defect was greater in females than males. In irradiated progeny of the cross y;mwh $Antp^R$/ mwh Sb Ubx X Df(1)sc^8, w^a/Dp(1;3)sc^{J4} and the cross $Antp^R$/Sb Ubx X y sn^3 f^{36a}, 72 of 383 $Antp^R$ individuals irradiated prior to 45 hours had defective eyes, while only 1 of 944 of their Sb Ubx siblings irradiated at this time had a defective eye. This indicates that the factor responsible for the eye defect is on the $Antp^R$ chromosome. Unirradiated $Antp^R$ flies did not show such irregularities in our experiments, nor did progeny of the cross y;mwh X Df(1)sc^8, w^a/Dp(1;3)sc^{J4} and y sn^3 f^{36a} X Oregon R irradiated with 1000r. Haskins and Enzmann[1], however, did obtain eye defects similar to the ones reported here after irradiation of an eosin stock.

Malformations of the head capsul without X-rays occur in D. melanogaster heterozygous for $Antp^{LC}$ [2], D. funebris bearing aristapedia[3], and D. hydei bearing ss^{Anp} [4]. The eye effect in D. funebris is more pronounced in females than males[3], as it was in our experiments. The gene erupt (er), also on the third chromosome, leads to a bristled structure protruding from the eye after irradiation of certain stocks at ten hours after egg laying. It is not known whether the eye defects reported here are due to the $Antp^R$ gene itself, or to some other locus, such as er, on the $Antp^R$ third chromosome.

References: 1. Haskins, D.P. and E.V. Enzmann, 1937 Amer. Nat. 71: 87-90; 2. Le Calvez, J., 1948 Bull. Biol. Fr. Belg. 82: 97-113; 3. Tiniakoff, G., 1939 DIS 11:52; 4. Gloor, H. and H. Kobel, 1966 Rev. Suisse Zool. 73: 229-252; 5. Glass, B., 1944 Genetics 29: 436-446.

Sanjeeva Rao, M. Osmania University, Hyderabad, India.
The alteration of X-ray induced genetic damage by aflotoxin in D. melanogaster.

The treatment of Drosophila flies with certain chemicals, and antibiotics prior to irradiation has altered the genetic damage (Sobels 1961, 1963, 1964, 1965; Burdette 1961, Clark 1963 and M.S. Rao 1965) and one of the methods offered for explanation was the inhibition of protein synthesis.

Aflotoxin is a collective name given to a group of highly toxic substances produced by certain strains of the mould Aspergillus flavus. The biological effects of this substance include: (i) inhibition of protein synthesis and also (ii) inhibition of m-RNA synthesis possibly through RNA polymerase.

With a view to find out whether aflotoxin would be able to alter the genetic damage akin to antibiotics experiments were undertaken to assess the alteration if any.

Oregon-K males of D. melanogaster were injected with 0.2 micro cc of saline solution containing 1 mg of aflotoxin dissolved in 1 cc of saline. The treated flies were exposed to

3000 r X-rays 24 hours after the injection. In the second experiment flies were exposed to 3000 r X-rays and then injected 0.2 micro cc of the saline solution containing 1 mg of aflotoxin dissolved in 1 cc of saline. Twenty-four hours rest was given before they were allowed to mate, in the second experiment (while in the first immediately after exposure to X-rays they were mated) individually with 3 virgin females of Y sc^{S1} In-49 sc^{8};bw;st stock for 3 days only to assess the alteration of genetic damage if any in spermatozoa alone. The F_1 females were mated individually with Y sc^{S1} In-49 sc^{8} males while the males were mated with bw;st females to score for sex-linked recessive lethals and translocations respectively in F_2 generation.

Table 1

Treatment	Sex linked recessive lethals			Translocations		
	T	l	%	T	t	%
3000 r X-rays						
3000 r X-rays	429	28	5.6	429	18	4.2
aflotoxin + 3000 r X-rays	477	17	3.5	411	13	3.1
3000 r X-rays + aflotoxin	468	27	5.7	433	16	4.6

T = total number of X chromosomes or F_1 sons scored
l = lethals recorded
t = translocations recorded

The Chi-square test has been done to compare the following groups: (1) 3000 r X-rays vs aflotoxin + 3000 r X-rays; (2) 3000 r X-rays vs 3000 r X-rays + aflotoxin. The results of the statistical analysis are presented in table 2.

Table 2

Group	Sex linked recessive lethals	Translocations
1. 3000r X-rays vs aflotoxin+3000r	2.139	0.172
2. 3000r X-rays vs 3000r+aflotoxin	0.863	0.137
3. aflotoxin+3000r vs 3000r+aflotoxin	2.358	0.0342

The study indicates that aflotoxin failed to alter the genetic damage induced by X-rays.

Temin, R.G. and R.M. Shore. University of Wisconsin, Madison, Wisconsin.

Heterozygous effects in Drosophila melanogaster following treatment with ethyl methane sulfonate (EMS).

In an effort to assess the populational effects of a general rise in mutation rate, experiments are being conducted in our laboratory to measure the viability of heterozygotes carrying second chromosomes recently descended from flies fed with the chemical mutagen EMS. These experiments utilize special stocks isogenized over a period of several years which enable us to study the comparative effects of treated chromosomes in homozygous and heterozygous backgrounds. The isogenic stocks are: 1) cn bw, maintained by brother-sister single pair matings, 2) cn, maintained by backcrossing a cn female to a cn bw male from the above cn bw stock, and 3) bw, by similarly mating a bw female with a cn bw male. A cross between cn and bw flies from these stocks generates cn +/+ bw males, which are fed with EMS according to the procedure given by Lewis and Bacher (1968). Following treatment, these males are divided, without etherization, into three groups for mass matings with either 1) isogenic bw females, 2) isogenic cn, or 3) M-5 females, for a standard test of the induced frequency of sex-linked lethals. The males are removed after two days in order to sample treated mature sperm. From each cross with the isogenic females, wild type sons (cn +/+ bw) were selected and mated individually to either isogenic cn bw females or to cn bw;e females from a non-isogenic stock. In these cn +/+ bw males either the cn or bw chromosome was the one treated, according to whether the mothers were bw or cn, respectively. In the next generation, the progeny, cn +/cn bw and + bw/cn bw, are counted for each of two broods, in both isogenic and non-isogenic backgrounds, at each dose. Comparing the ratio of cn to bw in cultures where cn is treated to the same ratio where bw is treated gives a measure of the effect of treated chromosomes in heterozygotes, each set providing a control for the other, with the viability effects of these markers cancelling out.

Preliminary results pooled for experiments at 3 doses of EMS are tabulated here; each class is expressed as a mean proportion.

Background	cn treated			bw treated			s	σ_s
	# of tests	cn(A)	bw(B)	# of tests	cn(C)	bw(D)		
isogenic	123	.485	.515	119	.509	.491	.049	.010
non-isogenic	135	.493	.507	131	.512	.488	.039	.009

The s value is the mean reduction in viability of heterozygotes, or the heterozygous load and is derived as follows: if we let x be a measure of the relative survival of the treated class, and p and q the relative viabilities of cn and bw flies, respectively, then the expected ratio of cn:bw where cn is treated is px:q and where bw is treated, p:qx. The value x^2 may therefore be estimated from the ratio AD/BC, where these letters represent the observed proportions of flies in the classes as listed in the table. The load s is approximately 1-x, or with a Poisson correction, s = -ln x.

Thus, in the isogenic (cn bw) background the reduction in viability of heterozygotes carrying a treated chromosome was close to 5%, and in the non-isogenic (cn bw;e) background about 4%. Each of these was significantly different from zero, but not different from each other. If the data is subdivided by broods, the effect is consistent: in the isogenic background, s for brood 1 was .053 ± .011, for brood 2, .050 ± .017. In the non-isogenic background, the s values were .040 ± .010 and .025 ±.015 for broods 1 and 2 respectively. Further experiments are planned in which the heterozygous effects will be correlated with homozygous effects. In particular, lethal heterozygotes will be separated from non-lethal heterozygotes and their viabilities compared. In the preliminary studies reported here, a major fraction of the effect may well be due to lethals, based on extrapolation from measurements of lethals induced on the X, as follows. The standard M-5 tests, with an additional generation tested for the presence of mosaic lethals were carried out to establish the lethal rates at three doses. With .017M EMS, there were 28.9% sex-linked lethals (89/219) in the F_2 and an additional 7.3% lethals in the F_3 (15/191). At .021M EMS, the F_2 rate was 37.9% (143/377) and F_3, 3.5% (7/201). At .023M the lethal frequency in the F_2 was .421 (48/114).

Würgler, F.E. and M. Kälin. Swiss Federal Institute of Technology, Zürich, Switzerland. A "storage" effect with X-rayed mature sperm of Drosophila melanogaster.

Graf and Würgler (this volume) found that the rate of apparent X/O males recorded after anoxic X-irradiation of mature sperm of ring-X males depends on the genotype of the females used for the test crosses. In screening tests, in which several other types of females were used in addition to the y sn^3 and Inscy;dp bw;st p^p flies, another unexpected result was obtained. Data obtained with XY/XY females illustrate this: Two to three-day-old ring-X males (R(1)2, y B/B^S Y y^+) were pretreated with N_2 for 20 min and X-rayed (50 keV, 520 R/min) in nitrogen. Nonirradiated controls were treated with nitrogen in the same way. After the treatment, the males were mated for 7 to 8 hours to 4-day-old virgin females in empty bottles, where the females did not deposit eggs. The females are homozygous XY/XY (Parker 110-8, y^2 su $(w^a)w^a$ KS.KL y^+). At the end of the mating period the males were discarded and the inseminated females transferred to standard culture vials. Every 24 hours the vials were changed until 4 successive broods had been obtained. The progeny from every vial were classified according to the phenotypes: normal B/+ females (F), normal B^S males (M), apparent X/O males (non-Bar, su$(w^a)w^a$) (L), and mosaics for sex chromosome loss (ML). The pooled data of two experiments, which gave very similar results, are given in the table. The percentage of sex chromosome loss is calculated as 100 x (L/F+M+L+ML).

brood (day)	control	2000 R	4000 R
1	2.2% (11/266+227+11+0)	8.7% (49/225+282+49+6)	13.7% (115/286+428+115+9)
2	1.3% (3/125+91+3+1)	3.5% (7/78+113+7+1)	4.5% (12/105+148+12+2)
3	1.9% (6/154+145+6+0)	3.7% (11/138+146+11+1)	8.0% (22/113+139+22+1)
4	1.7% (2/55+63+2+0)	2.2% (4/80+100+4+0)	6.6% (14/67+128+14+3)
2 - 4	1.7% (11/334+302+11+1)	3.2% (22/296+359+22+2)	6.4% (48/285+415+48+6)

The data show that the rates of sex chromosome losses are extremely high in the first brood. In broods 2, 3 and 4 the rates are low, but more or less constant. This finding, which looks like a "storage" effect, could have different causes:

a) Since most sex chromosome losses result from damaged ring-X chromosomes, a preferen-

tial use of X-bearing sperm during the first day after insemination of the females would lead to the observed result. The variation of the sex ratio (F/M) in the controls from brood 1 to 4 (1.17, 1.37, 1.06, 0.87) does not show the systematic variation expected on the basis of this hypothesis. Statistically none of the 4 values is significantly different from the weighted mean of 1.13.

b) The extremely high rate of X/O males might result from the effect that during the first day, Stage-14 Oocytes which had been stored in the virgin females for 2 to 4 days, were inseminated. Physiological differences between stored and non-stored oocytes might be responsible for the high rate of chromosome loss. Experiments to test this possibility are under way.

c) As a third hypothesis one could assume that changes occur in the irradiated sperms during the first day of storage in the females.

Finally it should be stressed here that this "storage" effect is also found with two other types of females, but - as far as can be seen from preliminary data - seems to be absent in experiments with females of two other stocks.

Work supported by Schweizerischer Nationalfonds zur Förderung der wissenschaftlichen Forschung.

Mather, W.B. University of Queensland, Brisbane, Australia. The genus Drosophila at Cebu, Philippines.

An investigation of the evolution of the immigrans species group in South East Asia has made the determination of relative abundance at various stations of considerable interest. Data for Sabah (Mather 1968 and 1969) and Luzon (Mather 1970) have already been recorded. In February 1970 the genus Drosophila was sampled from fermenting banana baits within the grounds of the Cebu Forest Experiment Station at Camp 7. Sorting of the flies yielded the following results:

Species	Number	% of total
D. setifemur	278	16.7
D. pararubida	374	22.4
melanogaster group	1,014	60.9
	1,666	

References: Mather, W.B., 1968 The genus Drosophila in Sabah. DIS 43: 100-101; Mather, W.B., 1969 The genus Drosophila at Sandakan. DIS 44: 98; Mather, W.B., 1970 The genus Drosophila at Mt. Maquiling, Luzon, Philippines. DIS 45: 111.

Limbird, D.L. College of Wooster, Ohio. A test for mutagenicity of MA and its effectiveness in deactivating EMS.

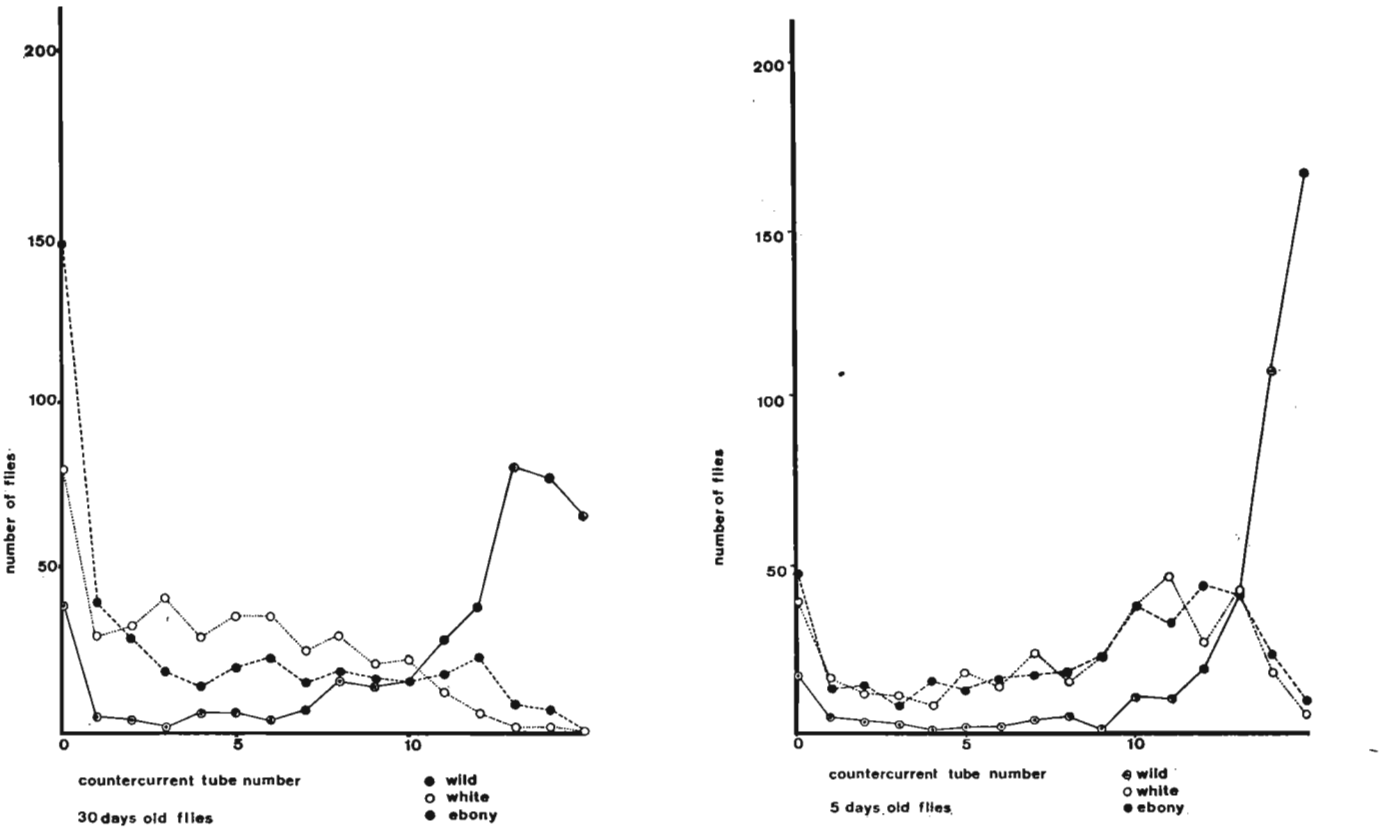
Mercaptoacetic acid (MA) has been recommended as a deactivator of ethyl methanesulphonate (EMS) (Lewis and Bacher, DIS 43) although experimental tests were not reported which would support its effectiveness. In the following experiment, MA was tested for possible mutagenicity and for its effectiveness in deactivating EMS. The experimental procedure involved treating 4-5 day old Canton-S males with one of four test solutions: a) control: 1m KOH in 1% sucrose solution + carmine; b) 0.5% MA: 0.5ml MA/100 ml control solution; c) 0.025M EMS: 0.24ml EMS/100ml control solution; d) EMS/MA: 0.5ml EMS/100ml control solution. Males fed for 24 hours from a pad of Kimwipes saturated with one of the solutions. Only those flies having definitely red guts due to the vital dye carmine were used in M-5 tests for sex linked recessive lethals. According to the results tabulated below, MA should be considered safe to use as a deactivator of EMS, being non-mutagenic itself and effectively cancelling the mutagenic properties of EMS.

Treatment	No. X chromosomes tested	No. X chromosomes lethal	Mutation rate
Control	387	1	.003
MA	309	0	0
EMS	245	35	.143
EMS/MA	306	0	0

Elens, A. and J.M. Wattiaux. Facultés Universitaires N.D. de la Paix, Namur, Belgium. Age and phototactic reactions in D. melanogaster.

The "countercurrent distribution" method has been suggested by S. Benzer (1967) to fractionate populations of Drosophila flies according to their phototactic response. The preliminary results here reported concern wild (Canton S), ebony, and white strains of D. melanogaster, tested according to Benzer.

The flies were grown on cornmeal medium at 25°C and 70% R.H. (10 hrs light per day). Testing was done at 25°C and 70% R.H., in a dark room. The test tubes and light source were



as in Benzer's experiments. Approximately 60 flies of each of the three strains, and of same sex and age, were tested together.

The data here presented result from 4 repetitions (the data concerning both sexes being cumulated).

As shown in Fig. 1 and Fig. 2 the differences between strains are much more marked for the older flies than for the younger ones.

Of course, such behavioural differences can be founded not especially on phototactic but on locomotor characteristics. Other tests are on hand concerning the influence of ageing on the locomotor activity (for flies in a group as well as for isolated individuals) of the same strains.

Reference: Benzer, S., 1967 Proc. Nat. Acad. Sci. 58: 1112-1119.

Cetl, I. J.E. Purkyně University, Brno, Czechoslovakia. The relation between genotypes in a viability test.

Using the Cy/L strain, relative viability of individuals homozygous for 30 independent second chromosomes isolated per 10 from three different natural populations (H, B and M) originating from Moravia was tested by crosses of Cy/+ females with L/+ males. In all 30 chromosome subpopulations studied, the frequencies

of Cy/L, Cy/+, L/+ and +/+ genotypes differed widely and in various manners from the expected 1:1:1:1 ratio. In spite of this diversity, the individual genotypes behaved in some respects similarly, e.g., the highest mean relative viability was ever found in the Cy/+ class. There was, thus, a general tendency of studied natural second chromosomes to be heterotic in viability at least when they were combined with the Cy chromosome.

Table 1. Correlation coefficients (r) of relative viability calculated between the relative viability of +/+ homozygotes and the cumulated relative viability of heterozygotes Cy/+ and L/+ in population samples H, B and M.

Population sample	r ± s	P
H	-0.9392 ± 0.1212	<0.01
B	-0.9482 ± 0.1153	<0.01
M	-0.9653 ± 0.0922	<0.01

If correlations were studied between the relative viability (expressed as chromosome subpopulation means) of +/+ homozygotes and corresponding cumulated relative viability of both heterozygotes, Cy/+ and L/+, high negative values of correlation coefficients significantly differing from zero were found (Table 1). This finding was unexpected. The theorem resulting from classical studies in maize (Jenkins, 1929; Hayes and Johnson, 1939) that the "performance" of genotypes when combined with a "tester" genotype is positively correlated with the "performance" of corresponding "pure" genotypes was not proved here. Evidently, more complicated relations take place if "pure" and "combined" genotypes develop in close mutual contacts as it is realized in a viability test. Wallace (1956) suggested that in these tests specific interactions may exist between larvae of different genotypes during their development and that alterations in the viability of one class may interfere with the relative viabilities of other classes. It is not excluded that the mentioned negative correlation was caused by similar "specific interactions", of course, of competitive character which can substantially change "ideal" ratios determined by "net" viabilities. For this reason, the values of relative viability estimated in a viability test have to be taken as resultants of "net" viability and of the above interactions.

References: 1. Hayes, H.K. and I.J. Johnson, 1939 J. Am. Soc. Agron. 31: 710-724; 2. Jenkins, M.T., 1929 J. Agr. Res. 39: 677-721; 3. Wallace, B., 1956 J. Genet. 54: 280-293.

Kuroda, Y. National Institute of Genetics, Misima, Japan. Effects of various sera and insect blood on the growth of embryonic tissues from D. melanogaster in culture.

To obtain the luxuriant growth in culture of embryonic tissues from the Oregon strain of D. melanogaster, the author has been searching for the supplementation of some macromolecular substances from various natural sources. Dechorionated and surface-sterilized eggs at the time of gastrulation (4 hours after egg laying) were torn into small fragments in balanced salt solution. These fragments were explanted on the glass surface of the culture bottles, incubated in salt solution for 60 minutes, and cultured in the chemically defined medium K-6' (1,2).

Supplementation to medium K-6' of sera from various sources and at various concentrations were examined to obtain better growth of cells under these conditions employed. The results are shown in Table 1.

Table 1. Effects of concentrations of serum on the growth of Drosophila embryonic tissues in culture.

Serum	No. of explants tested	No. of explants in which growth was observed	Percent growth
Calf serum, 3%	16	2	13
5%	19	7	37
10%	34	28	82
20%	5	1	20
Fetal calf serum, 10%	33	2	6

Supplementation of 10% calf serum was found to be the best among sera from various sources and at various concentrations examined.

Effects of silkworm blood on the growth of embryonic cells were also examined in the presence of 10% calf serum. The results are shown in Table 2.

Table 2. Effects of silkworm blood on the growth of Drosophila embryonic tissues in culture.

Source of blood	No. of explants tested	No. of explants in which growth was observed	Percent growth
Control	16	12	75
5th instar larvae, 5%	15	3	20
5th instar larvae, 10%	22	7	32
pupae, 10%	27	3	11

The addition of heat-treated blood collected from fifth instar larvae or pupae of silkworm exhibited no growth improvement in the culture of embryonic Drosophila tissues.

References: 1. Kuroda, Y., 1969 Japan. J. Genetics 44, Suppl. 1: 42; 2. Kuroda, Y., 1970 Exp. Cell Res. 59: 429.

David, J. and J. Bouletreau-Merle. University of Lyon, France. Two levels of egg retention in the genital tract of Drosophila females.

In Drosophila, egg production is controlled by vitellogenic activity, frequency of egg chamber resorptions and retentions. From the study of the relationship between daily egg production and frequency of females with an egg in the uterus, it is concluded that retentions may be initiated at two levels of the female reproductive system.

The results obtained for normally fed, mated or virgin females are plotted on the figure. In spite of a rather significant variability, it appears that the points correspond to an increasing convex curve. So the time spent by each egg in the uterus is quite stable and the variations in fecundity correspond to variations of the duration of absence of intra-uterine egg. Of particular interest is the fact that results for virgin females appear to be

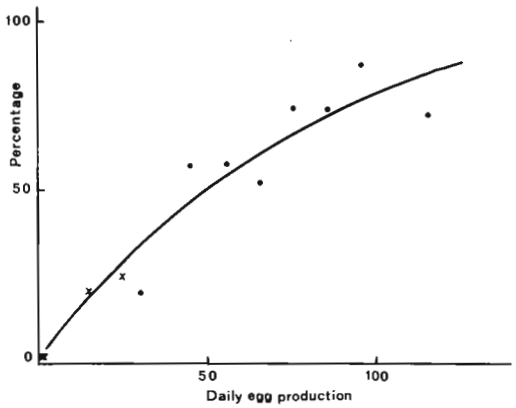


Figure: Relation between daily egg production and percentage of females with an egg in uterus (●: mated females; x: virgin females)

distributed on the same curve as those from mated females. From other studies (Merle and David, 1967) it is known that many stage 14 oocytes are observed in the ovarioles of virgin females. Therefore, this retention takes place in the ovarioles, not in the uterus.

Intra-uterine retention is observed when females are not offered a suitable site for egg laying. For example, properly fed, mated females were given only an agar medium for oviposition. In these conditions, the mean daily fecundity was only 2 eggs, but more than 80% of the females contained an egg in their uterus (data from Mrs. Van Herrewege, 1970). Of course, in such eggs, embryonic development begins and cases of viviparity are observed (see also King, 1963).

In conclusion, retentions may be initiated at two levels. 1) In the uterus, when the conditions for egg laying are unsuitable. This retention involves the oviposition behavior and is probably controlled by the central nervous system.

2) In the ovarioles, when females are not mated. This retention corresponds probably to a lack of ovulation; that is, an absence of contraction of the wall of the ovarioles. Its determinism is not yet known.

References: King, R.C., 1963 D.I.S. 38: 96; Merle, J. and J. David, 1967 C.R. Acad. Sci. Paris 234: 2028-2030; Van Herrewege, J., 1970 C.R. Acad. Sci. Paris 271: 108-110.

David, J. University of Lyon, France.

Utilization of crop content by the Drosophila female.

In most species of flies, the crop is usually considered as a reservoir where food accumulates rapidly during feeding and then is moved progressively into the mid-gut. In laboratory Drosophila, food is usually permanently available to the flies so that the usefulness of the crop could be questioned. The problem was studied in Drosophila females which ingest a great amount of food for egg production.

Highly vigorous F_1 females were dissected and the state of repletion of their crop was determined by grouping them into five qualitative classes (figure 1), ranging from a crop entirely empty (class 1) to a crop full of nutrient (class 5). The experiment was made with 10-day-old flies, the daily egg production of which ranged from 50 to more than 130. For any group of flies, an index of repletion of the crop was calculated as the average value of the classes. The data obtained with two nutritive media are given in the table 1.

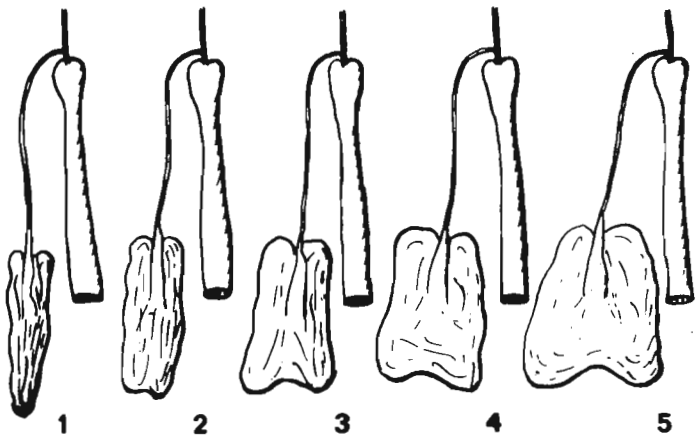


Figure: The five classes of crop repletion.

There is no significant relation between fecundity and the mean index. It is curious to see that the mean repletion of the crop is lower with the live yeast medium, which allows a higher fecundity, but the difference is not significant. Finally, the repletion of the crop is highly variable from one fly to the other. For the totality of the 173 dissected females, the frequencies distribution is the following:

Classes	1	2	3	4	5
frequencies	53	59	28	17	16

Two hypothesis may explain this high variability. First, the classes could correspond to various stages of the cycle of crop utilization: total repletion followed by a progressive emptying into the mid-gut. But in that case it is surprising that most of the females fall into the first two classes, with almost empty crops.

Second, it may be supposed that, when food is permanently present, crop utilization could be facultative: in many females, the ingested food being able to penetrate directly from the oesophagus into the mid-gut. A study of feeding behavior could help to choose between these two hypotheses.

Table I: Mean index of crop repletion in relation to daily egg production and nutrient medium

	Daily egg production				
Medium	50 - 69	70 - 89	90 - 109	110 - 139	Total
killed yeast	2.77 ± 0.26	2.29 ± 0.13	2.50± 0.36	2.50 ± 0.64	2.44 ± 0.11
n	34	82	16	4	136
live yeast		1.71 ± 0.18	1.85± 0.18	3.00 ± 1.00	1.92 ± 0.17
n		7	27	3	37

Budnik, M. and S. Koref-Santibañez. University of Chile, Santiago, Chile.

Temperature, rate of development and inversion polymorphism in D. pavani.

D. pavani Brncic 1957, is a species endemic to Chile and the eastern slope of the Andes Mountains in Argentine; it exhibits chromosomal polymorphism due to the presence of inversions on its second chromosome and in the right and left arms of the fourth chromosome. The polymorphism is of a rigid nature, for the frequency of heterokaryotypes is maintained under most of the environmental and breeding conditions in which the species has been reared. This is most probably due to the adaptive superiority of the heterokaryotypes, that has been measured in some components of fitness, such as longevity (Brncic and del Solar, 1961 Amer. Nat. 95: 211), mating activity (Brncic and Koref-Santibañez, 1964 Genetics 49: 585; Koref-Santibañez and Brncic, 1965 Genetics 52: 453), and rate of development (Brncic, Koref-Santibañez, Budnik and Lamborot, 1968 Genetics 61: 471). The present communication summarizes the experiments performed in order to analyze the behavior of the heterokaryotypes in relation to one of the fitness components, i.e., rate of development, at two temperatures.

A heterogeneous stock, originated from flies collected in the locality of Bellavista near Santiago, with a frequency of heterokaryotypes of around 55% in the left arm and of 48% in the right arm of chromosome IV was used. (The arrangements in the second chromosome were not considered). Eggs were collected every 24 hours, and placed in numbers of 50 in vials containing food medium. One set of vials was incubated at 16°C and another set of vials was left at 25°C. Adults were later sexed every 24 hours until all had emerged. At every temperature two groups were established, according to the time they took to develop from egg to adult: those of fast development, 29 to 35 days at 16°C, and 14 to 16 days at 25°C; and those of slow development: 44 to 65 days at 16°C, and 21 to 29 days at 25°C. The flies from each of these groups were crossed individually to imagoes of the opposite sex from a stock of Standard gene arrangement (D. gaucha) and the salivary gland chromosomes of eight larvae were analyzed in order to determine whether the parent was a homozygote or heterozygote.

The results are summarized in the Table, which shows the number of heterozygotes found in each group.

Temperature	R.D.	No.	Chromosome 4R Heterokaryotypes	χ^2	Chromosome 4L Heterokaryotypes	χ^2
16°C	fast	395	277	48.55*	266	45.31*
16°C	slow	375	170		162	
25°C	fast	238	143	9.53*	142	4.01**
25°C	slow	154	68		76	

*P 1 df < 0.001 **P 1 df 0.02-0.05

The significant Chi-square values between the "fast" and "slow" groups at each temperature reflect the fact that the frequency of heterokaryotypes is much greater among those flies that develop more rapidly, although the difference is significantly higher at 16°C. Thus, the heterozygotes are advantageous in this parameter of fitness, regardless of the environment at which they breed. This provides more evidence for the very rigid nature of chromosomal polymorphism in D. pavani.

(Research financed by grants from the Faculty of Medicine, Grant No. 45 of CONICYT and the Multinational Genetics Program of the O.A.S.)

Benedík, J. J.E. Purkyně University, Brno, Czechoslovakia.

Viability and lethality studies in the natural population of D.melanogaster.

The changes in the mean relative viability and in the frequency of lethals due to the second chromosome genes were studied in a large, isolated natural population. In the experimental period from October to December, average temperature dropped from 16 to 8°C, the consequence of which was change from optimal conditions into stressed ones.

The modified Cy-method was used for studying both second chromosomes of each male. Ten chromosomes in each of twenty males in each of two samplings (Oct. 23 and Dec. 9) were tested by this method for relative homozygote viability and for the presence of lethal genes. The mean relative viability for each male is presented in Table 1. The considerable shift in sampling mean was caused by the decrease of chromosome frequency with high viability values

in the second sampling as given in Table 2.

Obviously, these changes of viability are due to the temperature decrease during the experimental period. The mechanism of this interrelation between the temperature changes and

Table 1. Mean relative viability (per cent of +/+ individuals) for males from both samplings.

Sampling											Sampl. mean
Oct.	9.4	12.4	12.9	14.3	15.6	15.8	16.6	21.3	21.5	21.8	
Dec.	20.9	21.8	29.7	31.3	31.4	33.7	33.7	34.0	35.0	35.3	
Oct. (Cont.)	23.5	23.6	25.5	28.4	32.8	33.1	33.3	34.9	36.9	37.7	23.6
Dec. (Cont.)	35.4	36.8	40.9	41.4	41.6	42.7	43.4	43.8	44.4	44.9	36.1

Table 2. Per cent of chromosomes in different viability groups.

	relative viability (per cent)													
	0	5	10	15	20	25	30	35	40	45	50	55	60	65
	per cent of chromosomes													
Oct.	24.7	3.2	0.5	2.1	7.9	11.6	18.4	17.4	6.8	6.3	1.1	0.0	0.0	0.0
Dec.	7.4	0.6	0.0	1.1	4.0	6.3	14.3	22.3	12.0	10.9	7.4	4.6	5.7	3.4

frequency of lethals can be explained in two possible ways: (1) The drop of temperature leads to the decrease of the effective population size. The subsequent rise of inbreeding is followed by higher degree of elimination in the homozygous condition. Nevertheless in both samplings of this experiment no changes in heterozygosity occurred as it could be judged from steady differences in viability between both chromosomes of the same male. (2) The temperature decrease might be followed by an increase of degree of dominance of lethals and in this way they are eliminated even in the heterozygous condition. The latter hypothesis is being tested.

Ouweneel, W.J. Hubrecht Laboratory, Utrecht, Netherlands. Influence of environment on the development of the homoeotic strain aristatarsia.

During selection of the strain aristatarsia (art) (see DIS 45: 35) it turned out that in the same bottle flies with the mutant phenotype on an average emerge much earlier than flies with the wild-type phenotype. Because most of the flies with the mutant phenotype die rather soon, it could happen that in one bottle on the twelfth day after crossing a penetrance was measured of 60-80%, whereas in another bottle a penetrance of only 10% was found on the twentieth day.

Because in the beginning of these selection experiments the genetic background of the strain was still very heterogeneous, it was supposed that the early-emerging flies differed considerably in their genetic background from the later-appearing flies, which would be expressed in their different phenotype. This could mean that "art" flies developed more rapidly than "wild-type" flies. However, the average total duration of development in these bottles appeared to be longer instead of shorter than that of wild-type stocks (males: 226 ± 2 h; females: 221 ± 2 h; average wild-type stocks: 216 h; all at 25°C). Moreover, although the continuous interbreeding made the art strain more and more homogeneous and the "+" flies could give rise to "art" offspring as well as the "art" flies, the fact of the art phenotype emerging before the + phenotype remained.

Now the parental flies were allowed to mate and oviposit in the bottles during seven days and then they were discarded. Therefore it could be thought that the ageing of the mothers had an effect on the phenotypic expression of their offspring (as is the case in many mutants;

see Ouweneel, 1969 Roux' Arch. 164: 15) in such a way that eggs laid earlier gave rise to flies with a higher penetrance and expressivity of art than eggs laid later. However, here also the results obtained were opposite to those expected: ageing of the mothers had an enhancing effect on penetrance and expressivity. This is shown in the table, in which P is the penetrance (percentage of antennae exhibiting the art effect) and E the expressivity (average expression of the flies), measured according to the following classes: 1-2 unilateral; 3: both aristae less than 3/4 abnormal; 4: one arista more, one less than 3/4 abnormal; 5: both more than 3/4 abnormal.

age of mothers		11h	3d	7d	10d		11h	3d	7d	10d
P	♂	88	98	98	100	♀	77	88	98	96
E		3.7	4.4	4.7	4.7		3.2	4.0	4.3	4.6

Consequently maternal ageing also could not explain the findings. Therefore one solution remained: ageing of the food might alter its constitution in such a way that older food would decrease the penetrance of the art effect in larvae hatching later. This was tested by a simple experiment: a sample of art flies was cultured in a bottle with fresh food; their sons showed a penetrance of 78% and the daughters of 67%. Then another sample of the same strain was cultured in the same bottle on the aged food. Again a large offspring was obtained but now the penetrance was zero. A third sample cultured in the same bottle yielded the same result. This shows the large influence of food constitution on the expression of the homoeotic effect, and again confirms the high sensitivity of homoeotic mutants to environmental influences (see Ouweneel, l.c.).

Sanjeeva Rao, M. Osmania University, Hyderabad, India. The alteration of X-ray induced genetic damage by erythropoietin(s) in D. melanogaster.

Erythropoietin(s) is a polypeptide produced in anaemiated bovine plasma. This substance has altered the X-ray induced dominant lethal frequency in mice both by pre and post-treatment. With a view to find out whether or not the same alteration would be obtained in Drosophila experiments were undertaken.

0.2 micro cc of saline solution containing 1 mg of the substance dissolved in 1 cc of saline was injected into Oregon-K males of D. melanogaster. In the first experiment the treated flies were exposed to 3000r X-rays 24 hours after the injection. In the 2nd experiment flies were exposed to 3000r X-rays and injected 0.2 micro cc of the saline solution containing 1 mg of the substance dissolved in 1 cc of saline. Twenty four hours rest was given before they were allowed to mate in the second experiment while in the first experiment mating was allowed immediately after the exposure to X-rays. Treated males were crossed individually with 3 virgin females of Y sc^{S1} In-49 sc^{8};bw;st stock for 3 days only to assess the alteration in spermatozoa alone. The F_1 females were mated individually with Y sc^{S1} In-49 sc^{8} males while the males were mated with bw;st females to score for sex linked recessive lethals and translocations respectively in F_2 generation. The results are presented in Table I.

Table I

Treatment	Sex linked recessive lethals			Translocations		
	T	l	%	T	l	%
3000 r X-rays	554	48	4.54	542	36	3.50
PPFF + 3000 r	527	33	6.20	370	16	4.30
3000 r + PPFF	296	11	3.80	351	6	1.70

The chi-square test has been done to compare the following groups:
(1) 3000 r X-rays Vs. PPFF + 3000 r X-rays
(2) 3000 r X-rays Vs. 3000 r X-rays + PPFF
The results of the statistical analysis are presented in Table II.

Table II Chi-square values for the differences in sex linked recessive lethals and translocations in groups compared.

Group		Sex linked recessive lethals	Translocations
1.	3000 r X-rays Vs PPFF + 3000 r	1.936	1.968
2.	3000 r X-rays Vs 3000 r + PPFF	7.929	10.640
3.	PPFF + 3000 r Vs 3000 r + PPFF	2.196	3.921

The preliminary studies indicate that erythropoietin(s) failed to alter the genetic damage in pre-treatment while the post treatment studies indicated a significant reduction in both sex linked recessive lethals and translocations.

Chung, Y-J. and K-S. Lee. Ewha Womans University, Seoul, Korea. Further collection record of drosophilid flies from Korea.

When Drosophila melanogaster populations were sampled from four areas in Korea in order to make screening for the SD element, several other drosophilid species were collected which are to be reported here. Collections were made with the use of traps containing peach in orchard areas and by sweeping inside of breweries in the four localities of Korea: Changsungpo, Kuje Island from July 15 to 24, 1970; Taijun, Chungjoo and Jungpyung from Aug. 13 to 16, 1970.

A total of 1,834 flies represented by two genera, 12 species was obtained as given in Table 1. D. immigrans was the most abundant species in Kuje Island located in the South Sea, rather warmer area of Korea and D. suzukii was the second dominant species in the island. D. melanogaster, D. virilis and D. busckii were collected mostly in a brewery and it is noticeable that more individuals of D. busckii were captured compared to D. melanogaster and D. virilis. The most abundant species, D. virilis and the second dominant one, D. melanogaster in Taijun area were captured mostly in a brewery and D. auraria was collected mostly from a peach orchard. The collections in Chungjoo area were made chiefly in the peach orchard and the most predominant species was D. auraria and the second one was D. suzukii. It is interesting that quite a few individuals of D. melanogaster were captured in such an outdoor area. The results of the present collections confirm the following points of the characteristics of the Korean drosophilid fauna: (1) D. auraria is the most predominant outdoor species in Korea; (2) D. suzukii is found mostly in the orchard areas in Korea and this species may be harmful to the fruit trees, apple or peach; (3) D. melanogaster populations are not always found abundantly in every outdoor area in Korea.

Table 1. Number of flies collected in four areas of Korea (♂+♀=total)

Species	Areas				Total
	Kuje	Taijun	Chungjoo	Jungpyung	
Amiota sp.*	---	---	9+13=22	---	9+13=22
Drosophila coracina	---	0+1=1	11+4=15	---	11+5=16
D. busckii	4+6=10	---	2+3=5	---	6+9=15
D. melanogaster	2+1=3	49+60=109	153+63=216	2+2=4	206+126=332
D. suzukii	15+9=24	3+6=9	112+189=301	0+5=5	130+209=339
D. auraria	6+7=13	4+39=43	189+192=381	81+56=137	280+294=574
D. nigromaculata	---	---	---	2+5=7	2+5=7
D. transversa-complex**	---	---	0+2=2	0+2=2	0+4=4
D. immigrans	25+16=41	---	---	1+0=1	26+16=42
D. virilis	2+4=6	238+230=468	---	---	240+234=474
D. sordidula	---	---	---	3+5=8	3+5=8
D. bizonata	---	---	1+0=1	---	1+0=1
Totals	54+43=97	294+336=630	477+466=943	89+75=164	914+920=1834

*not identified. **composed of D. brachynephros, D. angularis, and D. unispina.

O'Brien, S.J. and R.J. MacIntyre. Cornell University, Ithaca, New York. A biochemical genetic map of D. melanogaster.

Presented on the following two pages is a genetic map of the D. melanogaster genome including only those loci whose alleles affect known molecular species, directly or indirectly. At each locus is a locus or gene designation followed by a reference number listed in the bibliography. Under the locus designation is listed the enzyme(s) or other molecular species which is affected by the gene. There is no intended implication that any locus is or is not the structural gene for the molecular species listed. We have also included a list of proteins which have not been mapped, but which have been characterized somewhat in D. melanogaster, and could be mapped by similar techniques to those utilized with previous mappings.

Unmapped systems more or less biochemically characterized in D.m.

Protein-1(49)
Protein-4(50
Protein-5(50)
Protein-8(51)
Protein-9(51)
Protein-13(51)
several enzymes(52)
protease(53)
adenylate kinase(54)
acetyl cholinesterase(55)
choline acetyltraferase(55)
aldolase(56,57)
β-galactosidase(58)
glutamate aspartate transaminase(59)
hexokinase(60)
kynurenine ormamidase(61)
lactate deH(62)

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D. = Drosophila D.m. = Drosophila melanogaster

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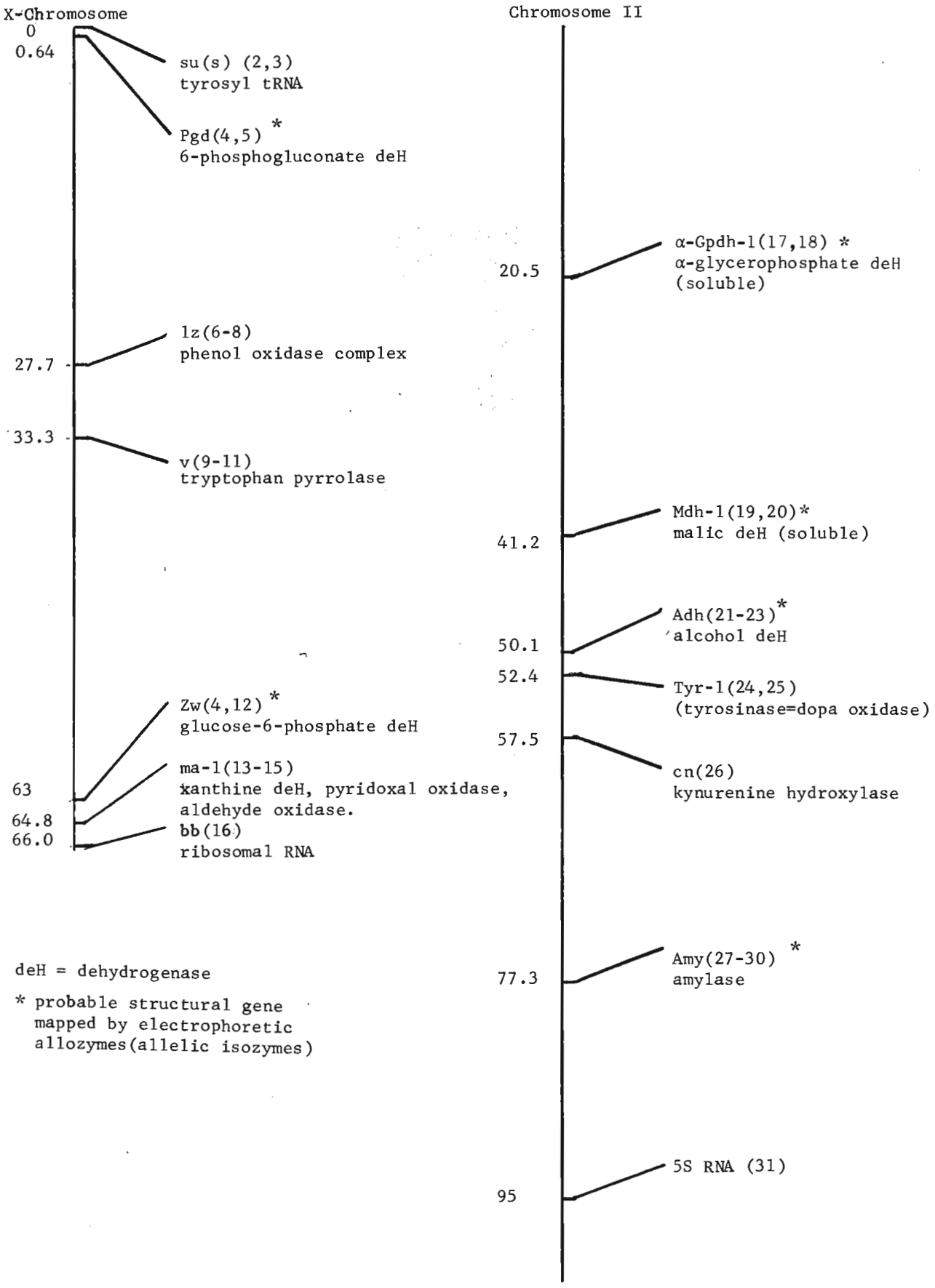
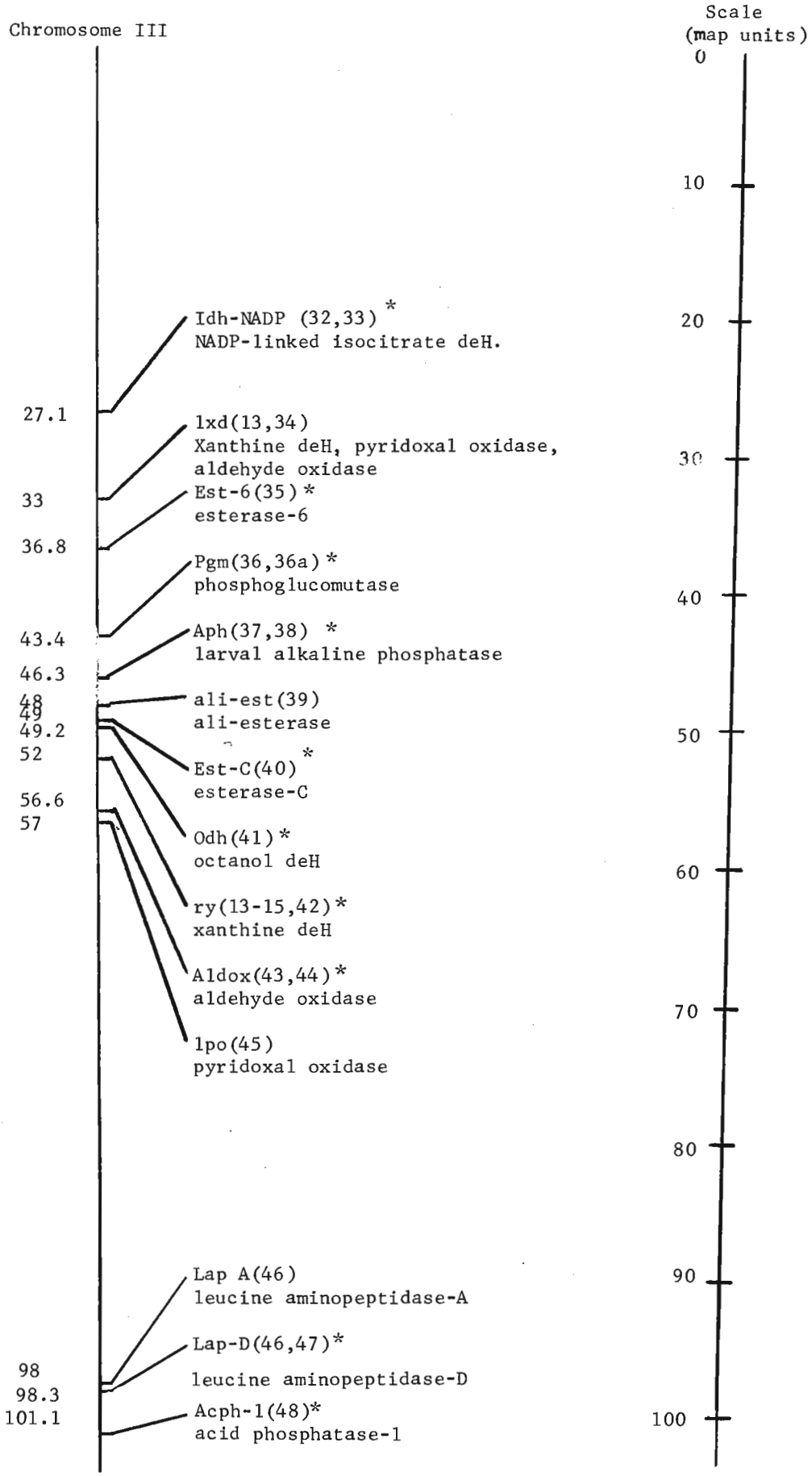


deH = dehydrogenase

* probable structural gene mapped by electrophoretic allozymes(allelic isozymes)


Chromosome III
Scale
(map units)
0
10
20
30
40
50
60
70
80
90
100
Idh-NADP (32,33) *
NADP-linked isocitrate deH.
27.1
lxd(13,34)
Xanthine deH, pyridoxal oxidase,
aldehyde oxidase
33
Est-6(35) *
esterase-6
36.8
Pgm(36,36a) *
phosphoglucomutase
43.4
Aph(37,38) *
larval alkaline phosphatase
46.3
48
49
ali-est(39)
ali-esterase
49.2
52
Est-C(40) *
esterase-C
56.6
57
Odh(41) *
octanol deH
ry(13-15,42) *
xanthine deH
Aldox(43,44) *
aldehyde oxidase
lpo(45)
pyridoxal oxidase
Lap A(46)
leucine aminopeptidase-A
Lap-D(46,47) *
leucine aminopeptidase-D
98
98.3
101.1
Acph-1(48) *
acid phosphatase-1

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Patty, R.A. University of Nebraska, Lincoln, Nebraska. Durations of copulation in some recent "eastern" and "western" strains of D. athabasca.

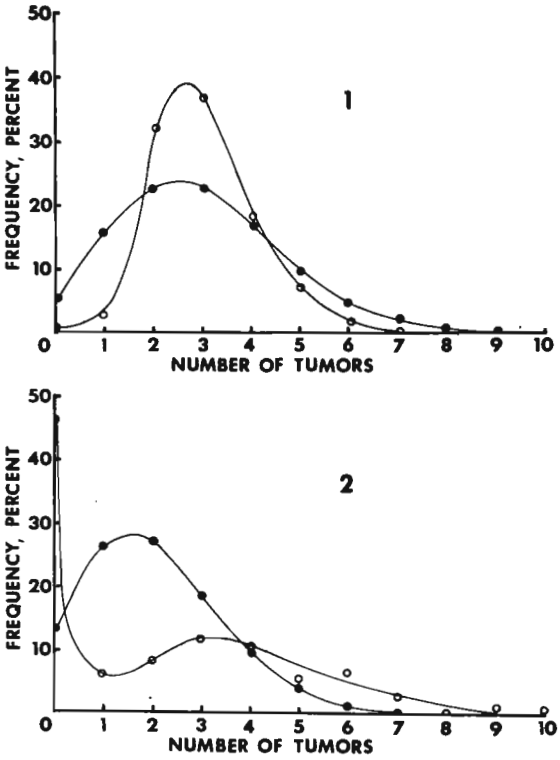
As reported by Miller (1958, Evolution 12: 72-81) and Miller and Westphal (1967, Evolution 21: 471-492), "western" D. athabasca has relatively long durations of copulation, "eastern" athabasca relatively short copulation times, with little overlapping (some long copulation times only in a derivative of the Carbon County, Pennsylvania, strain of "eastern" athabasca -Miller and Westphal, 1967). Strains from several new western and eastern localities have recently been acquired (the latter from collections by Drs. George Hooper and Max Levitan, via Dr. R.A. Voelker); these have been identified as "western" or "eastern" athabasca on the basis of geographical source, Y chromosome type, salivary gland chromosome patterns (by Drs. Miller and Voelker). As part of an attempt to reinvestigate the genetic basis of copulation time variation in athabasca, observations of copulations have recently been made, employing approximately 1-week old flies aged in isolation and observed at 70-80°F. The following table gives observed values (it also includes recent observations with the old Carbon County and Duluth strains used by Miller and Westphal, 1967):

"Western" athabasca:
Minesota (Bass Lake): 6'21", 7'15", 10'18", 12'46", 15'32", 17'56" ($\bar{x}$ = 11'41").
Minnesota (Duluth): 6'37", 7'37", 7'58", 8'00", 8'55", 9'15", 10'50" ($\bar{x}$ = 8'28").
Wyoming (Medicine Bow Natl. Forest): 6'33", 6'52", 8'28", 9'10", 9'46", 9'50", 10'20", 10'55", 11'15", 16'26", ($\bar{x}$ = 9'58").
"Eastern" athabasca:
Massachusetts (South Williamstown): 0'55", 1'07", 1'08", 1'11", 1'22", 1'24", 1'30", 1'34", 1'45", 1'53", 2'09", 2'25", 2'28" ($\bar{x}$ = 1'36").
New Jersey (Englewood): 1'12", 1'20", 1'21", 1'27", 1'36", 1'39", 1'47", 2'22" ($\bar{x}$ = 1'54").
New York (Poughkeepsie): 0'34", 0'45", 0'53", 1'02", 1'03" (2), 1'05", 1'07", 1'09", 1'10" (2), 1'11", 1'14", 1'18", 1'19", 1'33", 1'46", 1'56", 1'57" ($\bar{x}$ = 1'13").
Pennsylvania (Allentown): 1'58", 2'05", 5'54" ($\bar{x}$ = 3'19").
Pennsylvania (Carbon County, Jim Thorpe): 0'41", 0'56", 1'20", 1'21", 1'34", 1'43", 1'46", 1'55", 2'03", 2'07", 2'19", 2'30", 2'37" (2), 2'44", 2'55", 3'40", 3'48", 4'57" ($\bar{x}$ = 2'17").
Pennsylvania (Philadelphia): 1'16", 1'24", 1'25", 1'35", 1'55", 2'03", 2'25", 2'35", 2'39", 2'54", 2'57", 3'19", 6'47" ($\bar{x}$ = 2'33").

The range of copulation times for "eastern" D. athabasca from Allentown, Philadelphia, and Carbon County, Pennsylvania, exceeded the upper limit of 2'29" found by Miller and Westphal (1967) in most of their "eastern" strains. The lower limit for copulation time in "western" strains was observed to be 6'21" (Bass Lake, Minnesota) and the upper limit for the "eastern" strains 6'47" (Philadelphia). Thus, an overlapping of copulation times of "western" and "eastern" athabasca is confirmed, though most strains continue to conform to the earlier impression of nonoverlapping long and short copulation times for "western" and "eastern" athabasca respectively.

Bryant, P.J. University of California, Irvine, California.* Statistical distribution of melanotic tumors.

Many studies of melanotic tumor genes have employed penetrance as a measure of the expression of the gene. However, this becomes inaccurate when penetrance approaches 100%. In such cases, it is necessary to use a parameter related to the number of tumors developed per fly, and in order to do this effectively it is desirable to know how the tumors are distributed throughout the population.



The a priori expectation is that tumor distribution would be of the Poisson type, and this involves assuming that tumors form independently; that is, the probability of tumor formation is not altered by the formation of other tumors in the larva. In some stocks, the distribution of tumors differs markedly from the Poisson distribution. Fig. 1. shows a population of tu bw; $+^{su-tu}$ and Fig. 2. a population of Oregon K, both grown under sterile conditions. These distributions (hollow circles) are compared with Poisson distributions (solid circles) having the same means as the observed populations. tu bw;$+^{su-tu}$ shows a smaller spread than the Poisson curve, and Oregon K shows a wider spread. This is true for all populations of these strains studied, representing a wide range of expression values.

These results indicate that tumors do not form independently in these larvae. The reason for this is not known, but two kinds of explanation are possible. The first kind of explanation is that in tu bw;$+^{su-tu}$, initial tumor formation is inhibitory to further tumorigenesis and that in Oregon K, initial tumor formation stimulates further tumorigenesis. The second possible explanation is that tumors form independently in both stocks, but that they fuse (tu bw;$+^{su-tu}$) or fragment (Oregon K) subsequently, perhaps during metamorphosis. We have been unable to detect any appreciable loss of tumor number during metamorphosis in the tu bw strains, indicating that in those cases there may be real inhibitory effects between tumors during their formation.

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Ayles, B.* University of British Columbia, Vancouver, B.C. Male fertility of wild type stocks of Drosophila melanogaster at different temperatures.

Three years ago, it was decided to screen for mutations on the Y chromosome which produced male sterility at 29°C but fertility at 22°C. However, we found that most wild type strains of D. melanogaster are sterile at 29°C. We therefore screened eight different stocks for fertility at 22°, 28° and 29°C. Twenty males (48-60 hours after eclosion) from each stock were individually mated at each temperature to 3 virgin y/y females. The parents were then discarded after 48 hours and all of the progeny scored.

The percentage of male fertility and mean number of progeny per male is shown in Table 1. The females are fertile at all temperatures as shown by the Amherst^{tr} cross. All but the Amherst^{tr} and Urbana S males were sterile at 29°C. The Urbana S stock produced progeny which died as early pupae at 29°C. At 28°C, all but Samarkand and Swedish C males were highly fertile.

The Amherst^{tr} is a temperature resistant strain selected from the five surviving progeny of a similar test involving an Amberst stock obtained from P.T. Ives of Amherst College in 1967. The Am^{tr} strain has now been maintained in our laboratory by mass mating for over 50 generations at 29°C and it appears to be equally fertile at all three temperatures.

Table 1. Percent male fertility and mean number of progeny per male at each temperature

Stock	% fertile			mean no. progeny per male		
	22°C	28°C	29°C	22°C	28°C	29°C
Urbana S.	100	75	**	37.5	19.0	*
Samarkand	100	20	0	55.2	.8	0
Swedish C	100	20	0	52.3	1.8	0
Lausanne	95	75	0	52.2	18.2	0
Samarkand 204	100	60	35	66.1	21.4	2.6
Oregon 369	95	90	5	66.4	36.6	.3
Canton S.	100	95	40	67.9	70.0	.4
Amherst^{tr}	75	75	60	30.0	47.9	31.9

**flies died in the early pupal stages at 29°C

The restrictive temperature of 29°C is obviously close to the borderline of normal biological function of Drosophila melanogaster. Since all eight mutant X chromosomes which we recovered at 29°C still conferred male sterility at 28°C, we feel that this is a far better restrictive temperature to use.

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(Supported by research grants to D.T. Suzuki from the U.S. A.E.C. and N.R.C. of Canada.)

Miller, D.D. University of Nebraska, Lincoln, Nebraska. On the identity of the "sex ratio" X chromosome of "eastern" D. athabasca.

As reported by Miller and Voelker (1969, Journ. Hered. 60: 231-238, 307-311), the all-female progeny of two wild females of "eastern" D. athabasca collected in Minnesota in 1966 were heterozygous for XL sequence MIII MIX-MX and XS sequence MIII-MIV, suggesting that the X chromosome characterized by these sequences was the carrier of a "sex ratio" factor. Additional evidence for this relationship has been gotten recently from a few single-male matings with strains of "eastern" athabasca from Englewood, New Jersey, and South Williamstown, Massachusetts (from collections of Dr. Max Levitan, via Dr. Robert Voelker). The following table gives the results of four such matings from each of these localities. The symbols represent X chromosome inversions described by Miller and Voelker (1969), except for XL MXIII, a subbasal inversion recently found in several "eastern" strains and to be illustrated and/or described another time.

Mating	Female larvae X chromosomes		Adult Sex Ratio		
	XL	XS	♀♀	♂♂	Total
New Jersey					
-1	MIV MVI MXIII	MVI	55	55	110
-2	MIV MVI MXIII	MVI	26	31	57
-3	MIII MIX-MX (homozygous)	MIII-MIV	61	5	66
-4	MIII MIX-MX (heterozygous)	MIII-MIV	90	0	90
Massachusetts					
-1	MI-MII MVI MVII-MVIII	MI-MII	79	85	164
-2	MI-MII MVI MVII-MVIII	MI-MII	20	22	42
-3	MIII MIX-MX (heterozygous)	MIII-MIV	55	0	55
-4	MIII MIX-MX (homo- and heterozygous)	MIII-MIV	64	3	67

It can be seen that two matings from each locality gave both strongly one-sided sex ratios and female offspring carrying the XL MIII MIX-MX and XS MIII-MIV sequences, some in the homozygous state. The results are consistent with the association of these five inversions with "sex ratio", though, of course, it cannot be claimed that all inversions are necessarily tied to the "sex ratio" factor. "Eastern" D. athabasca would thus seem to differ from its relative D. azteca, in which the "sex ratio" X is characterized by three independent inversions in the long arm (Dobzhansky and Socolov, 1939).

Johnston, J.S. and W.B. Heed. University of Arizona, Tucson, Arizona. A comparison of banana and rotted cactus as a bait for desert Drosophila.

Trap cans, each containing 20 lbs. of bait, were set out on January 15, 1969 in an open desert scrub study area immediately north of Tucson in the southern foothills of the Santa Catalina Mts. at an elevation of 2900' to determine the relative attractiveness of fermenting banana, rotting prickly pear cactus (Opuntia angelmannii) and saguaro cactus (Carnegiea gigantea) to desert Drosophila. The cactus bait was prepared by injecting fresh material (pads or arms) with Erwinia carnegieana, the causal agent of saguaro bacterial necrosis, and incubating in plastic bags at 32°C for 2 to 5 days. Cactus prepared in this way has been shown to attract flies the same as natural rots. Three large trap cans each with a different bait were placed 17 yards apart on the lower one third of the north slope of the hill and this pattern was repeated 75 yards away on the top of the hill and 75 yards away from the top on the lower one third of the south slope. Collections were made on January 16 and 19 in the afternoon. The temperature averaged 65°F, but the north slope was 5°F cooler than other locations late in the afternoon.

The experimental design permitted an analysis of variance of the data (table 1) both for number of species and number of flies of each species. Banana attracted a significantly greater number of species than the cacti (9 vs. 5, $p<.05$). The hilltop attracted fewer species than the north and south slopes, but the difference was not significant. In the analysis of the 5 commonly trapped species, the type of bait, the species of fly, and the bait by species interaction were significant ($p < .05$). In all locations, banana traps attracted more flies of each species than the cactus traps ($p < .001$). Saguaro (host plant for D. migrospiracula) attracted more D. nigrospiracula and nigrospiracula-like than other species, but D. nigrospiracula-like preferred the top location while D. nigrospiracula was equally abundant on the top and south slope. Opuntia (a host plant for D. hamatofila and longicornis and sometimes pseudoobscura) attracted all 5 major species but became more effective north to south for all species ($p<.05$). D. pseudoobscura preferred banana in all locations ($p<.01$) but the majority came to banana on the north slope. Saguaro was actively avoided in all locations by D. pseudoobscura.

	pseudoobscura	nigrospiracula-like	nigrospiracula	hamatofila	longicornis	others	total
Banana							
North	449	36	31	16	9	11	552
Top	134	228	101	69	19	0	551
South	274	237	214	77	35	7	844
Opuntia							
North	2	4	2	1	1	0	10
Top	32	16	9	13	5	1	76
South	58	24	13	21	4	0	120
Saguaro							
North	0	8	4	0	0	0	12
Top	1	84	72	10	0	0	167
South	0	18	61	4	1	0	84
Total	950	655	507	211	74	19	2,416

These data demonstrate the superior attractive ability of fermenting banana both for species and numbers of individuals of Drosophila in the desert. Opuntia is a general but comparatively weak attractant. Saguaro has a very narrow species-attracting range and this is in accordance with our sweeping records from naturally rotting saguaro.

Budnik, M. and D. Brncic. University of Chile, Santiago, Chile. The ability to survive under crowding conditions as an expression of heterosis in inversion heterozygotes in Drosophila pavani.

D. pavani, which is endemic in the central part of Chile, constitutes a good example of a chromosomally polymorphic species, in which the inversion heterozygotes (heterokaryotypes) are at an advantage in most of the environments mastered by the species. Both in the natural populations and laboratory stocks, heterokaryotypes for the fourth chromosome are always in frequencies of about 50%. In searching for physiological properties responsible for the adaptive superiority of these heterokaryotypes, it was found that inversion heterozygotes are superior in longevity (Brncic and del Solar, 1961) exhibit a greater mating activity (Brncic and Koref-Santibañez, 1964) and have a faster rate of development (Brncic, Koref-Santibañez, Budnik and Lamborot, 1969).

A series of experiments was designed to determine whether the inversion heterozygotes in D. pavani in the preadult stages were superior in viability under crowding conditions with respect to the corresponding homokaryotypes. The ability to survive and reproduce under high density conditions represents an adaptive character, and it is well-known that the viability of certain genotypes depends on the density (rev. in Ayala, 1970).

In one series of experiments, eggs from a genetically heterogeneous stock of D. pavani were placed at various densities in small vials with a limited amount of basic cornmeal-agar medium. In a number of these vials the relationship between density and survival from egg to adult was estimated. In the remaining vials, the salivary gland polytene chromosomes of third instar larvae were analyzed in order to investigate the frequency of the different genetic arrangements in the fourth chromosome. In all the experiments there were found a significant increase of the heterokaryotypes in the more crowded vials and, also, a correlation between density and number of adults that emerged (Table 1).

Table 1. Frequencies of heterokaryotypes at various densities in the first series of experiments.

No. eggs x vial	No. of larvae examined	Chromosome IV-R Het.		Chromosome IV-L Het.	
		No.	%	No.	%
10	400	190	47.50	199	49.75
50	400	201	50.25	206	51-50
100	400	231	57.75	248	62.00
TOTAL	1200	622	51.83	653	54.42
Chi-square		9.02		14.15	
P (df 2)		0.01-0.02		<0.001	

In a second series of experiments, eggs of D. pavani at different densities were put together in small vials with an equal number of eggs of the mutant "yellow" of the "sibling" species D. gaucha. In order to determine the chromosomal structure of the D. pavani adults which emerged, each adult was crossed individually with flies which were homozygous for gene arrangements in all of their chromosomes, then the chromosomal arrangements of the F_1 progeny were examined cytologically. It was again found that among the flies that develop in the more crowded vials, the frequency of structural heterozygotes was significantly higher (Table 2). D. pavani and D. gaucha larvae do not compete at low densities, but D. pavani seems to be superior at high densities.

The general conclusion of the experiments reported is that heterokaryotypes in D. pavani

Table 2. Frequencies of heterokaryotypes at various densities in the second series of experiments.

No. eggs x vial	No. of adults examined	Chromosome IV-R Het.		Chromosome IV-L Het.	
		No.	%	No.	%
10	150	62	41.33	70	46.66
50	118	57	48.30	71	60.16
100	167	89	53.29	91	54.49
200	72	48	66.66	52	72.22
TOTAL	507	256	50.50	284	56.01
Chi-square		13.32		13.98	
P (df 3)		0.01-0.001		0.01-0.001	

have a greater survival under crowding conditions than the homokaryotypes. This property, along with their known superiority in longevity, mating, and rate of development, could contribute to the explanation of the high frequency of these heterokaryotypes in all the natural populations of D. pavani investigated.

References: Ayala, F.J., 1970 In: Essays in Evolution and Genetics in Honor of Th. Dobzhansky, M.K. Hecht and W.C. Steere, Eds. (Appleton Century-Crofts, New York): 121-158; Brncic, D. and E. del Solar, 1961 Amer. Nat. 95: 211-216; Brncic, D. and S. Koref-Santibañez 1964 Genetics 49: 585-591; Brncic, D., S. Koref-Santibañez, M. Budnik and M. Lamborot, 1969 Genetics 61: 471-478.

(Research supported by grants from "Comision Nac. Inv. Cientifica y Tecnologica - Chile" and School of Medicine, University of Chile).

Miller, D.D. and A.J. Kleager. University of Nebraska, Lincoln, Nebraska. Some additional data and a summary on interspecific mating in the D. affinis subgroup.

The following table has been put together from some very old unpublished data (circa 1940, D. D.M., designated by "M") on interspecific inseminations between D. affinis subgroup species in 10-day, "no-choice" combinations and some recent similarly derived data on combinations with D. narragansett (by A.J.K., designated by "K"; from Master's thesis, University of Nebraska, 1970). The older work involved various mutant strains, now defunct, the recent work a variety of wild and mutant strains. Frequencies are presented as raw insemination fractions. The data are supplemented by published insemination frequencies with D. tolteca (Ensign, 1960) and by reference to known cases of interspecific hybrids (including one newly reported here, from narragansett ♀ x affinis ♂). Despite the smallness of some of the numbers and the unequal attention to the different combinations, it is believed the table may be worthwhile to persons interested in this species group since it illustrates how at least some insemination has been encountered in 20 out of the 30 possible interspecific combinations of the six major American D. affinis subgroup species, eight combinations of which yield hybrids, of which three kinds manifest some fertility.

D. affinis ♀♀ x algonquin ♂♂: 0/56 "M"; x athabasca ♂♂: 158/431 "M", very few sterile HYBRIDS (Miller, Amer. Nat. 84: 81-93, 1950); x azteca ♂♂: 0/57 "M"; x narragansett ♂♂: 0/50 "M" and 0/87 "K"; x tolteca ♂♂: 31/107 (Ensign, Evolution 14: 378-385, 1960).

D. algonquin ♀♀ x affinis ♂♂: 0/52 "M"; x athabasca ♂♂: 3/144 "M", few fertile ♀♀ and sterile ♂♂ HYBRIDS (Miller, 1950); x azteca ♂♂: 0/52 "M"; x narragansett ♂♂: 10/225 "M" (some cultures gave few matroclinous offspring; nonvirginity?) and 2/62 "K"; x tolteca ♂♂: 1/104 (Ensign, 1960).

D. athabasca ♀♀ x affinis ♂♂: 14/281 "M"; x algonquin ♂♂: 0/159 "M"; x azteca ♂♂: 9/53 "M", sterile HYBRIDS including dwarf ♂♂ (Sturtevant and Dobzhansky, Amer. Nat. 70: 574-584, 1936); x narragansett ♂♂: 0/56 "M" and 3/87 ("western" ath), 2/61 ("eastern" ath.) "K"; x tolteca ♂♂: 19/118, 8/66, 21/59, sterile HYBRIDS (Ensign, 1960).

D. azteca ♀♀ x affinis ♂♂: 0/52 "M"; x algonquin ♂♂: 0/61 "M"; x athabasca ♂♂: 44/59 "M", sterile HYBRIDS including large-winged ♂♂ (Sturtevant and Dobzhansky, 1936); x narragansett ♂♂: 0/54 "M" and 1/55 "K"; x tolteca ♂♂: 35/115 (Ensign, 1960), sterile HYBRIDS (Patterson, Univ. Texas Publ. 5422: 46, 1954), fertile ♀ but sterile ♂ HYBRIDS (Miller and Sanger, Amer. Midl. Nat. 82: 618-621, 1969).

D. narragansett ♀♀ x affinis ♂♂: 0/55 "M" and 2/92 "K" with single sterile, poorly viable ♂ HYBRID; x athabasca ♂♂: 0/57 "M" and 0/68 ("western" ath.), 11/101 ("eastern" ath.) "K"; x azteca ♂♂: 0/52 "M" and 1/68 "K"; x tolteca ♂♂: 2/64 (Ensign, 1960), 0/56 "K".

D. tolteca ♀♀ x affinis ♂♂: 25/105 (Ensign, 1960); x algonquin ♂♂: 12/104 (Ensign, 1960); x athabasca ♂♂: 60/102, 17/60, 34/53 (Ensign, 1960); x azteca ♂♂: 58/109 (Ensign, 1960), fertile HYBRIDS of both sexes (Patterson, 1954); x narragansett ♂♂: 13/98 (Ensign, 1960), 1/66 "K".

Mittler, S. Northern Illinois University, DeKalb, Illinois. N,N' dimethyl 4,4' dipyridinium chloride and radiation induced dominant lethals.

Alekperov et al. (1967) reported that N,N' dimethyl 4,4' dipyridinium chloride, a herbicide, reduced the spontaneous chromosome aberrations in the root tip of Allium fistulosum. To test whether this chemical would protect against radiation induced dominant lethals, approximately 0.1 ul of a solution of 10 mg/liter (same concentration as used by Alekperov et al. (1967) was injected into the dorsal region between the 3rd and 4th tergites of a 24 hour old adult Oregon R male. The control males were injected with 0.85% NaCl. The males were irradiated with 1600 R of X-rays from a G.E. Maximar III, 150 KV, 15ma, 35 cm at 150 R/min. They were mated daily for 11 days to three day old females at a ratio of 1 male to 1 female. The mated females were isolated in plastic tubes of which one end was covered by a nylon mesh through which eggs were deposited onto the media which was darkened with black strap molasses. A cluster of eighteen tubes were used which fitted onto 140 mm petri plate, and was a modification of a method used by Abrahamson and Herskowitz (1957). The females were permitted to lay eggs for only 48 hours, and after 24 hours, eggs were examined to determine whether larvae had emerged. When the females were mated with males that had not been irradiated, 96 per cent of the eggs produced larvae.

The injection of 10 mg/liter of N,N' dimethyl 4,4' dipyridinium chloride did significantly reduce the induction of dominant lethals by X-rays in broods of day 6 and 8, however,

Table 1. Influence of N,N' dimethyl 4,4' dipyridinium chloride upon dominant lethals induced by 1600 R of X-rays in spermatogenesis of Drosophila.

Brood Day	Injection	Undeveloped eggs/ total eggs	Per cent undeveloped	
1	NN'D44'D*	617/2069	29.8	
	Saline**	475/1662	28.6	
2	NN'D44'D	319/1183	27.0	
	Saline	294/1167	25.2	
3	NN'D44'D	436/1612	27.0	
	Saline	370/1303	28.4	
4	NN'D44'D	777/1874	41.5	
	Saline	602/1519	39.6	
5	NN'D44'D	716/1280	55.9	
	Saline	549/1003	54.7	
6	NN'D44'D	639/996	64.2	$\chi^2=18.42$
	Saline	880/1215	72.4	
7	NN'D44'D	921/1343	68.6	
	Saline	721/1030	70.0	
8	NN'D44'D	707/975	72.5	$\chi^2=4.27$
	Saline	817/1064	76.8	
9	NN'D44'D	702/972	72.2	$\chi^2=7.63$
	Saline	512/777	65.9	
10	NN'D44'D	469/943	49.7	
	Saline	337/689	48.9	
11	NN'D44'D	336/900	37.3	$\chi^2=69.96$
	Saline	133/671	19.8	

* 10 mg/liter ** 0.85% NaCl (control)

the percentage of dominant lethals were enhanced on broods of day 9 and 11 (Table 1). The effect of this herbicide was not of radiation protection but to delay the cells about to enter meiosis in spermatogenesis with a resulting shift of the appearance of dominant lethals in the daily broods. The dipyridinium quatunary salts are used as herbicides, for they are readily converted to free radicals during photosynthesis by having a number of positions available for an addition of an electron.

This investigation was supported by a grant from the Environmental Control Administration CPEHS, U.S. Public Health Service EC 00075.

References: Abrahamson, S. and I.H. Herskowitz, 1957 Genetics 42: 405; Alekperov, V.K. A.F. Kalomiets and U.K. Schcherbakov, 1967 Dokl. Akad. Nauk SSSR 176: 199.

Krimbas, C., M. Loukas and E. Diamantopoulou. Agricultural College of Athens, Greece. Gene arrangement and gene frequencies in Mt. Parnes population of D. subobscura.

A sample of D. subobscura taken in April 24, 1970, from Mt. Parnes, Attica, Greece, has been examined for the frequency of the gene arrangements in its five chromosomes and for the frequencies of two polymorphic genes, an Esterase and an Alkaline phosphatase. Table 1 reports on the frequencies of the gene arrangements observed (in percentages, - in parentheses are indicated the maximum and minimum frequencies observed in five previous samples taken in 1964 and 1965, - see Krimbas Mol. Gen. Gen. 99: 133-150). Only 73 chromosomes have been studied for chromosome A, while 100 for each of the other autosomes. This last sample looks in general quite similar to the previous ones.

Table 1. Frequencies in percentages of gene arrangements in Mt. Parnes population 1970. (in parentheses width of estimations in five previous samples of the same population).

A_{St}	A_1	A_2			
55(32-49)	15(21-32)	30(19-38)			
J_{St}	J_1	J_{3+4}			
19(14-18)	81(81-86)	0(0-2)			
E_{1+2+9}	E_8	E_{ST}	E_{1+2}	$E_{1+2+9+12}$*	
46(41-61)	20(21-44)	21(8-13)	12(5-8)	1(1-6)	
U_{1+2+6}**	U_{1+2}	U_{1+2+8}	U_{1+2+7}	U_{1+2+3}	U_{ST}
51(30-53)	34(26-49)	5(1-7)	5(9-19)	1(1-4)	4(1-10)
O_{3+4}	O_{ST}	O_{3+4+1}	O_{3+4+22}	O_{3+4+2}	O_{3+4+7}
49(40-50)	16(4-17)	23(20-28)	7(3-11)	4(2-7)	1(2-7)

*$E_{1+2+9+12}$ and $E_{1+2+9+4}$ considered together.

**U_{1+2+6} and U_{1+2+4} considered together.

Est-6 (previously also noted as gene Est-1 in DIS 44: 71, Est in DIS 45: 105 and Est-1 in Isoz. Bull. 2: 42) is located near the centromere of chromosome O. A sample of 65 individuals (130 genes) revealed the following frequencies of the six known alleles of this gene:

Est-6^0_1	2.3	Est-6^{12}_2	25.4	Est-6^{23}_3	6.9
Est-6^1	19.2	Est-6^2	46.2	Est-6^3	0.0

Some 65% of the flies analyzed were found heterozygotes for this gene. Genotype frequencies did not depart from panmixia.

Aph-4 (previously also noted as gene N,Aph in IB 2: 46) is located in chromosome J. Two active alleles Aph-4^1 and Aph-4^2 were found in 53 individuals (106 genes) examined. Aph-4^1 has a frequency of 56.6 and Aph-4^2 of 43.4. Some 47% of the flies electrophorized were found heterozygotes. Here, too, genotype frequencies did not depart from panmixia.

These frequencies are to our knowledge the first ones reported for natural populations of D. subobscura concerning genes controlling known enzyme polymorphisms.

Tallantire, A.C. and J.H. Buruga*.
Makere University, Kampala, Uganda.
A study of some Drosophilidae of Uganda.

A partial survey of Drosophiladae in many parts of Uganda was carried out, using various trapping methods as well as by breeding the flies out of different organic materials.

Baited traps, using a variety of local cultivated fruits (banana, mango, pawpaw, pineapple, citrus, guava, tomato, cape gooseberry, breadfruit) were set up, the pineapple and banana baits being seeded with yeast. Fruits of a local zingiber, Aframomum sanguineum, were also used. In addition some of the Drosophilidae were obtained by sweeping and others by the use of aspirators and the Malaise trap. A total of 16,991 specimens from at least thirty taxa were trapped over a two-year period, but only in the Kampala area did this represent a continuous sampling throughout this period. The results are presented in Table 1.

Various organic materials were taken to see if they were breeding sites. Bark and rotting stems and leaves of plants, exudates from stems, mature and decaying flowers, ripe and rotting fruits and fungi, as well as cow dung were collected. 11,450 specimens hatched out, representing at least twenty-six taxa, but the majority belonged to only five species viz. Drosophila melanogaster, D. simulans, D. seguyi, Zaprionus tuberculatus and Z. vittiger. Of this total, 9,945 hatched from 28 of the 46 different types of indigenous and introduced fruits collected, the majority of these Drosophilidae being identical with those found in baited traps. Specimens hatched from 15 of the 39 different kinds of flower examined, and from 8 species of fungi, but very few were found from rotting material and only two species, one of Zaprionus and one of Mycodrosophila, from exudates. (See Table 2).

Owing to the existence of a number of poorly-defined species, complicated by the existence of several sibling species and questionable races, it has not been possible to identify all the Drosophilidae collected, but probably about sixty species were obtained, the majority of which belonged to the genus Drosophila. Nearly all of the species hatched out of, or baited on, fruits have been cultured on a corn-meal agar medium, but only one species (of Drosophila) hatching from rotting leaves survived on this medium and then for less than four months.

(This work was part of a thesis submitted by * for the degree of M.Sc. of the University of East Africa, under the supervision of Dr. J.R. Olembo. Grateful acknowledgement is made to E.B. Basden, University of Edinburgh, and L. Tsacas, Gif-sur-Yvette, France, for identifying many specimens.)

Table 1

Genus	Species	Trap used		
		Baited trap	Aspirator	Malaise
Drosophila	D. akai	+	+	-
	D. ananassae	+	-	+
	D. aterrima	-	+	-
	D. framire	+	+	-
	D. lambi	+	+	-
	D. latifasciaeformis	+	-	-
	D. melanogaster/simulans	+	+	+
	D. pruinosa	+	-	-
	D. repleta	+	-	-
	D. seguyi complex	+	+	-
	D. sp. nr. ananassae	+	-	-
	D. sp. nr. mokonfim	+	-	-
Hirtodrosophila	H. sp. nr. akabo or nr. hirticornis	-	+	-
	H. sp.	-	+	-
Leucophenga	L. palpalis	-	+	-
	L. sp. nr. buxtoni	-	+	-
	L. sp. nr. flavipalpis	-	+	-
	L. sp. nr. halteropunctata	-	+	-
	L. sp.	-	+	-
Mycodrosophila	M. ditan	-	+	-
	M. fracticosta	-	+	-
	M. nigerrima sp. A	-	+	-
	M. nigerrima sp. B	-	+	-
	M. sp. nr. nigerrima	-	+	-

	M. sp. nr. ditan	-	+	-
	M. sp. nr. fracticosta	-	+	-
Zaprionus	Z. ghesquierei	+	-	-
	Z. inermis	+	-	-
	Z. koroleu	+	-	-
	Z. tuberculatus	+	-	-
	Z. vittiger	+	-	-
	Z. sp. nr. koroleu	+	-	-

Table 2

Drosophilidae		Organic material				
Genus	Species	Flowers	Fruits	Fungi	Exudate	Stems/leaves
Amiota	A. sp.	-	-	+	-	-
Drosophila	D. akai	-	+	-	-	+
	D. ananassae	+	+	-	-	-
	D. aterrima	+	-	-	-	-
	D. dyaramankana	+	-	+	-	-
	D. framire	-	+	-	-	-
	D. lambi	+	+	-	-	-
	D. melanogaster/simulans	+	+	-	-	-
	D. pruinosa	-	+	+	-	-
	D. repleta	-	+	-	-	-
	D. seguyi complex	+	+	+	-	+
	D. suma	+	-	-	-	-
	D. sp. nr. ananassae	-	-	+	-	-
	D. sp. nr. pugionata	-	-	-	-	+
Hirtodrosophila	H. sp. nr. akabo or nr. hirticornis	-	-	+	-	-
Leucophenga	L. sp.	-	-	+	-	-
Lissocephala	L. punctipennis	-	+	-	-	-
Mycodrosophila	M. bombax	-	-	-	+	-
	M. ditan	-	-	+	-	-
	M. fracticosta	-	-	+	-	-
	M. sp. nr. ditan	-	-	+	-	-
	M. sp. nr. fracticosta	-	-	+	-	-
Stegana	S.? proximata	+	-	-	-	-
Zaprionus	Z. ghesquierei	-	+	-	-	-
	Z. tuberculatus	+	+	-	-	-
	Z. vittiger	+	+	-	-	-
	Z. vrydaghi	+	-	-	-	-
	Z. sp. A	+	-	-	-	-
	Z. sp. B	-	-	-	+	-

Donady, J. City of Hope Medical Center, Duarte, California. Failure to demonstrate genes for nerve or muscle differentiation on chromosome four.

It is assumed that a mutation blocking any step in the differentiation of nerve or nuscle cells will be an embryonic lethal. All known recessive embryonic lethals of chromosome four (received from B. Hochman) have been tested for their ability to give nerve and muscle cell differentiation in vitro.

Egg counts for each mutant [1(4)2h, 5, 7, 23 and ci^D] showed the expected 25% failure to hatch. However, in twenty single-embryo cultures for each mutant, all cultures produced differentiated nerve and muscle cells. Normal differentiation, as compared to control cultures, is based on the appearance of axons (50μ or longer) and ganglion cells for nerve, and pulsating spindle-shaped cells for muscle. Therefore, these mutations are not in the pathway for nerve or muscle cell differentiation. Supported by NIH Grant No. AI05038 and NS09330 to R. Seecof.

Koref-Santibañez, S. and P. Iturra.
Universidad de Chile, Santiago, Chile.

Adult density and mating activity in Drosophila gaucha and Drosophila pavani.

Drosophila pavani and Drosophila gaucha are two sibling species of the mesophragmatica group, whose geographic distribution is mostly allopatric, although they have been found to co-exist in one part of their range (San Luis, Argentine). In nature no hybrids have been found, and in the laboratory the F_1 hybrids are totally sterile. (Koref-Santibañez, 1964).

Their courtship is very similar both qualitatively and quantitatively (Koref-Santibañez and del Solar, 1961), but there exists a certain ethological isolation, revealed by homogamic courtship preferences shortly after males are confronted with females of both species.

Many factors may influence mating propensity, among them variables acting at pre-adult and adult stages as well as interactions among mating adults (Spiess, 1970). The study of these factors may be of aid in analyzing the causes of sexual isolation between sympatric sibling species.

The present communication summarizes the results of a series of experiments performed in order to analyze the density of mating adults in different volumes in relation to mating activity.

5, 10, 20 or 40 pairs of each of the following combinations: ♂ D. pavani x ♀ D. pavani; ♂ D. gaucha x ♀ D. gaucha; ♂ D. pavani x ♀ D. gaucha; ♂ D. gaucha x ♀ D. pavani were placed in vials of the following volumes: 40 cc, 80 cc, 120 cc and 160 cc, giving 64 different combinations. The flies were left for 6 hours under permanent illumination in a constant temperature room at 16°C. The females were then dissected and examined under a microscope in order to detect the presence of sperm within the spermathecae.

Table 1 summarizes the percentage of females inseminated at each of the four volumes. It shows that the activity of males or the receptivity of females are not significantly modified

Table 1. Percentage of D. pavani (p) and D. gaucha (g) females inseminated by D. pavani (p) or D. gaucha (g) males in different volumes (N = 400 pairs in each combination).

	♂ p		♂ g	
Volume	% ♀ p	% ♀ g	% ♀ g	% ♀ p
40 cc	67.50	80	86.00	32.75
80 cc	76.00	69.75	83.25	37.75
120 cc	61.25	75.25	83.75	30.25
160 cc	70.00	82.75	81.75	37.75
χ^2	20.81	22.01	2.73	7.36
P (3 df)	>0.001	>0.001	0.50-0.30	0.10-0.05

by changes in the space allowed for mating. Although D. pavani males seem more sensitive, there is no consistent trend in their activity. D. pavani females always tend to discriminate more against foreign males than do D. gaucha females. This may be due to the great receptivity of these latter females, independently of the male to which they are confronted. Inversely, D. gaucha males have a much lower activity than do D. pavani males.

Table 2, which summarizes the percentage of females inseminated at each density, shows on the other hand, that mating activity is affected by density. There is a decrease in the number of females inseminated with an increase of mating pairs. This is most striking when

Table 2. Percentage of D. pavani (p) and D. gaucha (g) females inseminated by D. pavani (p) or D. gaucha (g) males at different densities (N = 400 pairs in each combination).

	♂ p		♂ g	
Density	% ♀ p	% ♀ g	% ♀ g	% ♀ p
5	76.50	79.75	96.5	54.75
10	75.00	75.50	80.5	33.50
20	71.25	82.00	84.75	25.75
40	52.00	70.50	72.00	24.50
χ^2	71.77	17.36	91.22	103.82
P (3 df)	>0.001	>0.001	>0.001	>0.001

D. gaucha males are confronted with D. pavani females.

These observations on "density effect" upon mating activity of both D. gaucha and D. pavani are different than those found for other species by Spiess (1970) and Spiess and Spiess (1969). These authors observed that females seem to require a courtship summation before mating, and that mating increases when the number of courting pairs is greater. In the present case, there seems to exist some type of interference among mating pairs, which reduces the receptivity of the females independently of the space available for their activity.

References: Koref-Santibañez, S., 1964 Evolution 18: 245-251; Koref-Santibañez, S. and E. del Solar, 1961 Evolution 15: 401-406; Spiess, E., 1970 Evolutionary Biol. 4: 315-379; Spiess, E. and L. Spiess, 1969 Evolution 23: 225-236.

(Research financed by grants from the Faculty of Medicine, Grant No. 41/67 of CONICYT and Multinational Genetics Program of the O.A.S.).

Kuroda, Y. National Institute of Genetics, Misima, Japan. Effects of substances with ecdysone and juvenile hormone activity on the growth of embryonic tissues from D. melanogaster in culture.

Ecdysterone and inokosterone, which have been isolated from plants and previously shown to have ecdysone activity in the differentiation of eye-antennal discs of Drosophila mature larvae in organ culture (1), were tested for their activity to promote the growth of embryonic Drosophila tissues cultured in medium K-6' supplemented with 10% calf serum. The result is shown in Table 1.

Table 1. Effects of ecdysone analogues on the growth of embryonic Drosophila tissues in culture.

Ecdysone analogue	No. of explants tested	No. of explants in which growth was observed	Percent growth
Control	34	28	82
Ecdysterone, 0.1 mg/ml	20	17	85
0.01 mg/ml	23	22	96
0.001 mg/ml	24	16	67
Inokosterone, 0.0001 mg/ml	20	4	20

Among ecdysone analogues tested ecdysterone at the concentration of 0.01 mg/ml had a slight growth-promoting effect, whereas inokosterone at as low concentration as 0.0001 mg/ml had an inhibitory effect.

Dodecyl methyl ether (DME), a substance acting as juvenile hormone, was tested at various concentrations for its growth-promoting effect on embryonic Drosophila tissues. The results are shown in Table 2.

Table 2. Effects of DME on the growth of embryonic Drosophila tissues in culture.

Concentration of DME (mg/ml)	No. of explants tested	No. of explants in which growth was observed	Percent growth
Control	16	12	75
10.0	13	2	15
1.0	5	1	20
0.1	8	7	88
0.01	22	18	82

It was found that 0.1 mg/ml DME stimulated the growth of tissues, whereas with higher concentrations than 1.0 mg/ml an inhibitory effect was observed as compared with the control cultures without it.

Reference: 1. Kuroda, Y., 1969 Japan. J. Genetics 44, Suppl. 1: 42.

Erickson, J. Western Washington State College, Bellingham, Washington. A viable and fertile homozygous Curly.

A scheme for the production of homozygous SD males, developed by Dan Hartl, depends upon the meiotic drive of SD and Curly lethality: the final cross, Cy SD(NH)-2/cn bw ♂♂ x Cy bw/SD-72 ♀♀ produces, presumably, only Cy SD(NH)-2/SD-72 progeny. In fertility checks of males produced by this scheme crossed to cn bw ♀♀, an occasional case turned up of a male considerably more fertile than expected, and with only Curly progeny. These progeny are of four types, in approximately equal numbers: Curly males and females, and Curly males and females with a light brown eye color. (This light brown eye color is also seen in a Cy bw/cn bw^2 stock used in the scheme. Ocelli have slight color - presumably the Cy bw does not carry cn^2.)

A number of possibilities were considered to account for the aberrant males. Although they were not "extreme Curly, dwarf flies" as called for in Bridges and Brehme, the only explanation fitting both their origin and their progeny was that they were Cy SD(NH)-2/Cy bw.

To test this, I mated, first: Cy SD(NH)-2/cn bw ♂♂ x S G/Cy L^4 ♀♀ and S G/CY L^4 ♂♂ x Cy bw/cn bw ♀♀. Next, F_1 Cy SD/S G ♂♂ x Cy bw/S G ♀♀. The progeny were 24 Star Gull and 14 non Star Gull. Among the latter (selected for S$^+$, since Gull is not well-expressed) at least 6 had a definitely aberrant wing posture - but not extreme Curly. The wings were held out and down, at the base, then curled up as usual. I tested four males of this type by crossing to cn bw ♀♀, 3 were fertile. The results were similar to the original case, totalling 24 Cy ♂♂, 27 Cy ♀♀, 22 Cy bw ♂♂ and 21 Cy bw ♀♀.

The Cy bw chromosome used was received by Dr. Hartl from Dr. Hiraizumi, he reports that it is SM1 with bw at the tip of 2R. The Cy SD(NH)-2 chromosome has the Cy gene without inversions derived by Tinderholt.

It appears that the Cy/Cy homozygote, in this case, is quite viable and fertile. The cause of the non-lethality is unknown. As noted at the Pasadena meeting, I would prefer to leave further work on this to someone specifically interested in gene expression, lethality, etc.

Freedman, C. and S.K. Majumdar. Lafayette College, Easton, Pennsylvania. Effect of calcium cyclamate on the productivity of Drosophila melanogaster.

The wild flies were allowed to breed in instant Drosophila medium containing 1, 3 and 5% calcium cyclamate. The F_1 flies from these media were transferred to vials containing instant medium - one female and one male in each. Ten vials were used for each concentration. The five-day brood system was used and four broods were obtained. Each brood was counted eighteen days after oviposition. The experiment was repeated two times. The average results of this testing are compiled in Table I. The results indicate that the percent of productivity was more or less similar in the first two broods, but a decline starts in the treated

Table I. Effect of calcium cyclamate on the productivity of offspring produced by D. melanogaster females.

Treatment	Brood 1		Brood 2		Brood 3		Brood 4		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%
Water	171	42.3	108	26.7	82	20.3	43	10.6	404	30.5
1% Cyclamate	160	46.1	102	29.5	75	21.4	10	2.9	347	26.2
3% Cyclamate	152	50.0	94	30.9	46	15.1	12	4.0	304	22.9
5% Cyclamate	99	36.6	125	46.2	40	14.8	6	2.2	270	20.4

group from the third brood. In the fourth brood, 10.6% of the total number of control flies were born as compared to only 2.2% born in the 5% cyclamate group. In addition, the number of flies born in the control medium averaged 404 while the number of flies born in the 5% cyclamate group averaged only 270. The results thus indicate that high percentages of calcium cyclamate adversely affect the production of offspring of Drosophila melanogaster.

Anderson, B.A.S. University of Oregon, Eugene, Oregon.* Comparison of electropherograms of hemolymph and fat body soluble proteins in larval D. malenogaster.

Electrophoresis of hemolymph and of fat body homogenates was carried out on 7% acrylamide gels using the standard method of Ornstein and Davis (1964). Larvae of an Oregon R strain isogenic for chromosomes I, II, and III provided the tissue. Hemolymph was collected by puncturing 10 - 12 larvae and drawing the fluid which bled out into fine glass tubing. No attempt was made to remove hemocytes. Fat bodies (4 - 5) were isolated by dissection in Drosophila Ringer's. Testes, but not ovaries, were removed. Fat bodies were homogenized with sample gel in a hand held microhomogenizer. After electrophoresis gels were stained with Coomassie Brilliant Blue as described by Chrambach et al. (1967).

Seven major and numerous minor bands were identified in electropherograms of both tissues. Fat body electropherograms consistently showed more background stain than did hemolymph electropherograms. One minor band (between F and G) was commonly seen in fat body samples and not in hemolymph samples. Otherwise the electropherograms from hemolymph and fat body were indistinguishable. Co-electrophoresis of the two tissues did not show any additional protein bands.

The table below summarizes the relative migratory distance of the seven major bands in both tissues. The larva, reared axenically by the method of Keith and Goldin (1968), pupate at 144 - 150 hours; hence all three ages represent third instar larvae. Band B is a double band, though its two components are not always clearly separated. Many minor bands are also seem.

Acrylamide Gel Electropherograms of Hemalymph and Fat Body Homogenates of D. melanogaster Larvae

Larval Age	Bands - Relative Migratory Distances						
	A	B	C	D	E	F	G
4 da.	.04	.17	.49	.55	.59	.79	1.00
	.06, .04	.12, .11	.51, .48	.57, .55	.60, .57	.84, .79	1.00
5 da.	.05± .01	.15± .004	.48± .01	.54± .01	.58± .01	.79± .01	1.00
	.04, .06	.13, .12	.50, .47	.54, .53	.60, .59	.80, .79	1.00
6 da.	.07± .01	.16± .003	.48± .03	.54± .02	.57± .003	.78± .02	1.00
	.06, .04	.17, .13	.51, .49	.57, .53	.61, .57	.80, .81	1.00

For all pairs of values above, the upper figures are for fat body samples and the lower for hemolymph.

References: Chrambach et al. 1967 Anal. Biochem. 20: 150-154; Keith and Goldin, 1968 DIS 43: 178; Ornstein and Davis, 1964 Annals, N.Y. Acad. Sci. 321-349 and 404-427.
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Jacobs, M.E. Goshen College, Goshen, Indiana. Survival of ebony and non-ebony D.m. pupae in low humidity.

e^{11} females were crossed with Oregon-R males. 880 day old F_2 pupae were placed at 92% R.H., and 2640 at 35%, at 25°C. As seen below, at 35% R.H., survival of ebony flies was decreased more than was that of non-ebony.

Adults Emerging

	92% R.H.				35% R.H.			
	Females		Males		Females		Males	
	+	e	+	e	+	e	+	e
O	315.0	115.0	270.0	92.0	687.0	148.0	530.0	117.0
E	322.5	107.5	271.5	90.5	626.1	208.7	485.1	161.7
D	7.5 --- 7.5		1.5 -- 1.5		60.9 -- 60.7		44.9 -- 44.7	
χ^2	0.69		0.03		23.5		16.5	

del Solar, E. University of Chile, Santiago, Chile. The effect of density on locomotor activity in D. melanogaster.

The movement of groups of 50, 100, 150, 200, 250 and 300 males and females was measured over a period of 15 hours in the following apparatus: A translucid plastic tubing 3 cm in diameter and 37 meters long, subdivided into sections one meter in length, coiled in a spiral of one meter in diameter, mounted on a wooden frame with one light bulb in the center of the top and one in the center of the bottom.

Density	% of Migrant	Mean distance of group
50♂	27.3	2.64 ± 1.13
100♂	29.1	1.82 ± 0.49
150♂	44.7	2.88 ± 0.74
200♂	42.4	3.18 ± 1.01
250♂	64.9	5.36 ± 0.84
300♂	61.2	6.09 ± 1.77
50♀	43.6	3.86 ± 0.84
100♀	41.6	3.25 ± 1.16
150♀	51.5	3.81 ± 0.57
200♀	58.8	5.90 ± 0.95
250♀	53.5	6.72 ± 1.80
300♀	60.6	6.15 ± 0.10

The table shows that females exhibit greater locomotor activity than the males; only the individuals which moved over one meter were considered. A correlation between density and the mean distance travelled by the flies in the group gives the following results: 0.369 for females and 0.838 for males, the values being significant in the latter. ($P = 0.05$). The regression lines were of: $x = 0{,}0053y + 4.350$ and $x = 0.014y + 1.210$ for females and males respectively.

If the sections of 5, 10, 15, 20, 25, 30 and 37 meters are added, the frequencies show a density gradient; that indicates that in the group of 200 males, at least one reaches the thirty seventh meter, while this already occurs in the 100 female group.

Minamori, S. Hiroshima University, Japan. One-sided gamete recovery from delta-carrying heterozygous males and females of D. melanogaster.

The extrachromosomal elememt denoted by delta was found and has been studied in our laboratory (Minamori, 1969). Delta is retained and transmitted by certain second chromosome lines denoted by sensitive chromosome and symbolized by S^b. However, it is not retained by the other sensitive lines symbolized by S^c.

In the course of experiments, it was detected that S-Cy/S-5(S-Cy, Cy gene carrying S^c chromosome; S-5, S^b chromosome) heterozygous males and females produced only Curly offspring and no non-Curly offspring when the heterozygous flies carried an appreciable amount of delta. However, the segregation ratio was always normal when non-Curly flies were recovered; Curly and non-Curly flies were recovered in a 1:1 ratio. In these normal recoveries, no instance was found of deviation from a 1:1 ratio. It may be said, therefore, that the progeny recovery from the heterozygotes is either one-sided or normal.

Egg-fly counts were made and the results obtained are shown in Table 1. It is most plausible that the one-sided progeny recovery from male heterozygotes may be due to the pro-

Table 1. Egg-fly counts for recoveries of progeny from S-Cy/S-5 males and females in the mating with homozygotes for a wild second chromosome.

Cross	No. of eggs laid	No. of progeny recovered: Curly flies	No. of progeny recovered: non-Curly flies	Flies/Eggs (%)
S-Cy/S-5(±)♂ x +/+♀ (Control)	706	258	286	77.06
S-Cy/S-5(∂)♂ x +/+♀	884	678	0	76.69
S-Cy/S-5(∂)♀ x +/+♂	841	385	0	45.78

duction of only Cy-bearing spermatozoa, and not due to the inviability of S-5-bearing progeny. A similar interpretation might also be applicable to the one-sided recovery from female heterozygotes. The S-5-bearing spermatozoa may be dysfunctional. Delta appears to cause damage to S-5 chromosomes at an early embryonic stage of the carrier in such a manner that the gametes receiving descendants of this chromosome may become dysfunctional.

Reference: Minamori, S., 1969 Genetics 62: 583-596.

Matsudaira, Y. and T. Yamasaki. Rikkyo University, Tokyo, Japan. Dose-frequency relationship of two types of lethal mutation induced by X-rays in Drosophila.

We reported that for the visible loci of Drosophila, the X-ray induced frequency of fractional-body mutation is well above that of the whole-body mutation in the low dose treatment, whereas in the high dose the relation is reversed (1964 and 1967). In this report, the dose-frequency relationship for two types of lethal mutation [fractional-(gonadal) and whole-(complete) lethal] induced by X-rays will be presented.

Adult males of Oregon-R stock were irradiated with 500, 1000 and 2000R of X-rays, and mated to virgin females from Muller-5 stock. In the F_1 generation from these cultures, pair mating was carried out. In the F_2, the whole-lethal mutations were examined (F_2 test). From each F_1 line, one pair of F_2 was sampled, and their offspring were tested for the lethal (F_3 test). In the F_2 sampling, if the sampled female is of the fractional-lethal carrying line origin, there is equal probability that it is either lethal free or a lethal carrying one. Therefore, 50% of the fractional-lethal mutations must be discarded in the sampling. Consequently, in order to obtain the induced frequency of the fractionals, the observed frequency should be doubled.

The results are summarized in the table below. In the dose region used in this experiment, fractional-lethal mutations did not appear to be induced at a higher rate than whole

Number of fractional- and whole-lethal mutation induced by X-rays.

Dose of X-rays (R)	Total number of chromosomes examined	Number of lethals observed	Classification of lethal type Number and frequency, %	
			Whole	Fractional
500	for F_2 test 5822 for F_3 test 5265	69 98	69(1.19)	29(0.55x2=1.10)
1000	for F_2 test 1079 for F_3 test 1052	20 28	20(1.85)	8(0.76x2=1.52)
2000	for F_2 test 1030 for F_3 test 975	39 47	39(3.79)	8(0.82x2=1.64)

lethals. However, as has been reported, the frequency of fractionals at 0 dose may be higher than that of whole ones from the data of Epler (1966), and others. These results are not contradictory in their general tendency to the results of visible mutations, although quantitatively there is some discrepancy.

Ehrlich, E. University of Oregon, Eugene Oregon. An X chromosome balancer having three scute regions.

Matings of y w spl sn(1)/Muller-5 virgins with "scuteless"/T(Y;1)1E, y sc^+ (Masterson, DIS 43: 161, 1968) males give Muller-5/T(Y;1)1E, y sc^+ male progeny with a greatly reduced frequency. Since the Ins(1)sc^{S1} S sc^8, B w^a chromosome carries a duplication for the scute region, these males possess in their genomes three scute regions.

A non-lethal-bearing X chromosome balancer which can be rendered lethal in males by the presence of a particular Y chromosome is a useful genetic tool. Therefore it seemed advantageous to determine whether or not four scute regions in a male have a lethal effect, and the following synthesis was undertaken.

Virgin females, Ins(1)sc^{S1} S sc^8, w^a/T(Y;1)1E were subjected to approximately 3000 r of X irradiation and subsequently mated to yellow males. The resulting exceptional w^a males were mated individually to both y f:=/Y and y f:=/T(Y;1)1E virgins. Those lines were selected in which the first females produced w^a male progeny but the second gave none.

In the lines thus selected, it appears that the synthesized chromosomes are sc^{S1} S w^a sc^8 ·y sc^+ in composition, and that four scute regions in males do indeed have a lethal effect.

Band, H.T. Michigan State University, East Lansing, Michigan. Lethals and environmental tracking.

In his stimulating book, Levins (1968) postulates that only a genetic system of short memory can track the recent environment. Band (1969) suggested that the lethal and semilethal genetic variants maintained in the South Amherst (Mass.) D. melanogaster population might constitute a system for environmental tracking; the frequencies of these variants recovered from population samples from 1945 through 1959 and later have shown a significant negative correlation with temperature range of the week prior to collection (Band and Ives, 1961). Evidence bearing on the environmental tracking hypothesis is available from the extensive sampling by Ives from the breeding site population in the summer and fall of 1967. The data

Collection	# Chromosomes	% le + sle	Direction of change	t^{o} range	Direction of change
June 7-18	566	22.8		35.4	
June 29	322	25.2	+	25.6	-
July 18	290	27.6	+	22.3	-
August 8	266	21.4	-	26.4	+
October 19	204	26.8	+	16.8	-
October 24	170	20.0	-	22.8	+
October 31	107	22.4	+	24.1	+
November 7	208	27.4	+	20.4	-

have also been included in Ives (1970). Only one sample fails to be in agreement with the expected negative relationship between le + sle frequency and temperature range for successive samples. The sign test for trend changes yields a k value of 3.5, P (one-tailed) = .001, hence highly significant.

The ability of these recessive genetic variants, otherwise severely deleterious to lethal in homozygous condition, to fluctuate in population samples in relation to the environment indicates they play a positive adaptive role in population structure. Further, the likelihood that many such lethals in heterozygous condition can enter the dynamic relationship with the immediate environment removes the restriction that any one lethal need be present in the population in high frequency to be considered heterotic. This gains added support from the finding by Ives (1970) that allelism rate among lethals in the June 7-18 collection was 20.4% and in the November 7 collection only 0.9%. Hence, the level of lethals and semilethals maintained in the population in relation to the immediate environment seems of greater importance in population structure than the frequency of any one individual genetic variant.

References: Band, H.T., 1969 unpublished manuscript; Ives, P.T., 1970 Evolution (in press); Levins, R., 1968 Evolution in changing environments, some theoretical considerations Princeton University Press.

Vogel, E. Zentrallaboratorium für Mutagenitätsprüfung, Freiburg, Germany. Lack of mutagenic effectiveness of 8-Hydroxyquinoline sulfate in D. melanogaster.

8-Hydroxyquinoline sulfate has been utilized as a fungicide and disinfectant. Furthermore, it has medical importance as an ingredient of drugs. In human lymphocytes and in plants 8-Hydroxyquinoline produces gaps and fragmentations of chromatids and chromosomes, respectively (Ref. see Gebhart, E., 1968 Mutat. Res. 6: 308-318).

In order to study the mutagenic action on Drosophila, a 0.1% solution of 8-Hydroxyquinoline sulfate, buffered with phosphate-buffer to keep the pH at 6.8, was administered by feeding on glass filter dishes to wild (Berlin K) males for three days. By means of the Muller-5-technique the rate of sex-linked recessive lethals in three successive broods, each of three day's duration, has been determined. Only two lethals (0.03%) were detected out of 6528 chromosomes total tested.

Hence, under these conditions, 8-Hydroxyquinoline sulfate is not a mutagen for D. melanogaster.

Spiess, E.B. and R. Ritzlin. University of Illinois, Chicago, Illinois. Bacterial infection and cultures of D. persimilis tested for mating propensity.

Fluctuation of apparent random nature in mating speed performance of D. persimilis strains has been bothersome in the past. In addition, when two or more samples are tested for mating from the same culture, there is far more variation between cultures than between replicates from the same culture (Spiess, Sherwin, & Yacher, in press, Genetics). These facts have tended to indicate that microorganism contamination may be detrimental to stability of flies' behavior. It was apparent some years ago that poorly mating flies from fast-mating strains often had a yellow discoloration in the crop. During the summer of 1969, microorganisms from the crops of poorly mating females were isolated and cultured on nutrient agar. The most consistent organism found was tested for diagnostic traits in the laboratories of Drs. E.E. Vicher (University of Illinois at the Medical Center) and M. Silverman (V.A. Research Hospital) with the tentative identification as Achromobacter sp. Its traits are as follows: 1) short Gram-negative rod which forms mucoid colonies on nutrient agar (BHI); mucus growth is greater at low temperature (15°C) than at warm (25°C). 2) Best growth at 25°C, with no growth at 37°C, but good growth at 15°C. 3) Aerobic, tending to overgrow yeast on the surface. 4) Catalase and dextrose positive; negative for oxydase, citrate, indole, methyl red, acetoin; odorless. 5) No flagella visible on electron micrograph after fixation with 10% formalin and uranyl acetate. 6) Sensitive to tetracycline, novobiocin, streptomycin and kananycin, but resistant to penicillin, erythromycin and neomycin.

Flies were cultured on food to which tetracycline and streptomycin had been added in solution to the surface (with about the concentrations listed by Hendrix and Ehrlich, 1965), in three different ways: first, on the assumption that larvae would not ingest much antibiotic unless it was mixed with yeast, food made without yeast was inoculated on the surface with an antibiotic-yeast mixture. This technique suffered from low nutrition to the larvae since the yeast did not grow well and was insufficient to produce normal-sized adults. Second, antibiotics were added in surface solution to food on which parent females were depositing eggs; but females tended to lay eggs on food sources lacking the antibiotics when given a choice, and too few adults were raised to be tested. Finally, the treatment adopted was to add antibiotics to the food after first instar larvae had hatched (on plastic spoons with yeasted food which were then inserted into regular culture food plus 10 drops of antibiotic solution added to the surface). All strains benefitted by that treatment in that productivity increased over untreated, and mating speeds became more consistent and reliable, though they were uniformly lower in propensity (mating index) than the controls. Consequently the antibiotic treatment not only had lowered the bacterial growth, but also depressed the flies' activity.

To test for detrimental effects on preadult stages of the life cycle, eggs and larvae were grown on nutrient (BHI) agar in which there was a two-day growth of bacteria, using the following techniques: parent flies of two strains (D. persimilis), selected for either fast (F2) or slow (S1) mating (KL homokaryotypes from the redwoods population) in their 20th generation of selection, laid eggs on food in plastic spoons. Eggs were washed in 70% ethanol for 15 minutes and planted in lots of 50 on 1) BHI agar with 2-day growth of Achromobacter sp. or 2) on BHI sterile (control). Egg hatch results are as follows with 4 replicates per treatment per strain:

	Control		Bacteria	
Strain	Total Hatch	Range	Total Hatch	Range
F2	173/200=86.5%	79.6%-94.0%	179/200=89.5%	86.0%-96.0%
S1	158/200=79.0%	74.0%-86.0%	172/200=86.0%	84.0%-88.0%

It was surprising to see no real difference between treatments; in fact, there was a slight benefit to hatching when the bacteria were growing on both strains.

For larval-pupal survival, first instar larvae were collected from spoons of food, washed in sterile water on a black cloth which had been sterilized with a strong disinfectant (Microquat) and then rinsed a second time in sterile water. Larvae were then placed in vials on slanted food, 25 per vial, either with a 2-day growth of bacteria or sterile, then were given yeast suspension and sterile paper strips for pupation. Adult emergent results were as follows with 4 replicates per treatment per strain:

Strain	Control		Bacteria	
	Total Adults	Range	Total Adults	Range
F2	70/100	56%-84%	73/100	60%-88%
S1	57/100	48%-68%	71/100	56%-96%
S1 repeat control	60/100	all 60%		

Again there was no significant difference between treatments, though all averages and ranges were higher with bacteria than without it.

Matings of flies from these cultures were unaffected by the bacteria and produced about the same mating propensities as the controls.

In conclusion, this Achromobacter has no detrimental effect on the life cycle under the conditions tested. It still could be detrimental via its mucoid by-product to mire the flies or to decrease their sensitivity to external stimuli after prolonged exposure, but those factors have not yet been tested. Briefly, the basis for unexpected variation between cultures and uncontrolled fluctuations in mating propensity is as yet undetermined.

References: Hendrix, N. and E. Ehrlich, 1965 DIS 40: 99.

Acknowledgments: Contract AT(11-1)-1652, U.S. Atomic Energy Commission.

Schneider, I. Walter Reed Army Institute of Research, Washington, D.C. Embryonic cell lines of D. melanogaster.

Within the past year, Echalier and Ohanessian (C.R. Acad. Sci. 268: 1771, 1969) and Kakpakov et al., (Genetika 5: 67, 1969) have independently reported the establishment in vitro of cell lines from embryos of D. melanogaster. These lines have, in general, not been available for study by individuals from other laboratories. This note reports the existence of 3 additional cell lines from D. melanogaster, subcultures of which are available upon request.

Timed embryos were collected, surface sterilized and allowed to develop almost to the point of hatching. They were then cut into 2 or 3 pieces and placed in 0.2% trypsin solution (1:250, DIFCO) in Rinaldini's salt solution for 30 minutes at room temperature. After a thorough washing the pieces were seeded into glass T-9 flasks with 1.25 ml Schneider's Drosophila medium (GIBCO) containing an additional 500 mg bacteriological peptone per 100 ml bottle and supplemented with 15% inactivated fetal bovine serum. The pH of the medium was 6.7 and the cultures maintained at 27 ± 0.5°C with a gaseous phase of ambient air.

Initially growth took place in the form of hollow spheres issuing from the cut ends of the embryonic fragments. The spheres, each of which consisted of a monolayer of cells, were allowed to grow to a diameter of 1 mm or more before being excised, teased apart (or alternatively treated with trypsin) and returned to the same flask together with the original embryonic fragments. Single cells as well as small cellular masses obtained in this manner usually attached to the bottom of the culture flask and began to multiply. This procedure was repeated until sufficient cells were present in the primary flask to make subculturing feasible. The primary cultures for the 3 lines were initiated in August 1969, December 1969 and February 1970.

The 3 lines are quite dissimilar in appearance. Cells of the 1st line are initially round and vary from 12 to 20μ in diameter when freshly seeded into a new flask. Within 3 to 5 days, however, the majority begin to differentiate in vitro as evidenced by a 2 to 6-fold increase in cell size (due primarily to an increase and/or flattening of the cell cytoplasm) with consequent alterations in cell shape. Once differentiated the cells cease to multiply. However, there are always sufficient numbers of "stem" cells present in each culture so that subsequent subcultures can be made at intervals of approximately 1 week. The chromosome number varies from 2n to possible 8n.

The other 2 lines are much more conventional in appearance and behavior. The cells of both lines are epithelial in shape, predominently diploid and form monolayers but differ from each other in size and generation time. Cells of the 2nd line are approximately 10μ in diameter and 20 to 30μ long and have a generation time of 22 hours at 27°C. Corresponding figures for the 3rd line are 6 to 10μ, 10 to 30μ and 18 hours, respectively.

Definitive identification of the cells' origin(s) has not been made but should be possible once sufficient numbers of cell spheres have been injected into 3rd instar larvae and the differentiated masses examined.

Murnik, M. Rengo. Western Illinois University, Macomb, Illinois. The effects of temperature on the sex ratios observed from OR and SD males.

When the environment of cells which are undergoing spermatogenesis is altered, the functionality or dysfunctionality of the resultant sperm may be affected. Temperature is one of the most obvious environmental factors which can be controlled. The functionality of a mature sperm may also be affected by variations in temperature. To test for such effects, Drosophila melanogaster males were reared at three different temperatures and then given a 24 hour temperature shock at one of these temperatures before mating.

OR and SD-72 males were reared at 18, 22.5 or 29°C until eclosion, collected as virgins and stored at one of these temperatures for 24 hours before mating to virgin OR or cn bw females. The sex ratios of the subsequent progeny were noted, as well as the k (SD/total progeny) of the SD crosses.

The k of the SD progeny was affected in only one category, that of the SD males reared at 18° and heat shocked at 29° before mating. These males exhibited a k of .88. The remainder of the SD progeny exhibited k values of .99 or greater, the value characteristic for our stock of SD-72. Mange (Genetics, 1968) noted a similar effect with low temperature shock treatments of SD-72 during spermatogenesis. The depression of the drive of SD is observed here only with low temperature spermatogenesis and subsequent heat shock of mature sperm.

In both SD and OR crosses, the sex ratios of progeny whose fathers were reared at the three different temperatures are significantly different (see Tables 1 & 2). The higher the temperature during spermatogenesis, the higher the sex ratio (♀♀/total) of the progeny. The

Table 1. Progeny of SD males

	Temp. of Rearing 18°	22.5°	29°
males	720	2821	719
females	1012	3211	841
χ^2	=14.767***		

Table 2. Progeny of OR males

	Temp. of Rearing 18°	22.5°
males	6144	5600
females	6737	6821
χ^2	= 17.362***	

Table 3. 22.5° OR males

	Temp. of Shock 18°	22.5°
males	873	2842
females	977	3396
χ^2	= 1.526	

Table 4. 22.5° OR males

	Temp. of Shock 22.5°	29°
males	2842	1885
females	3396	2448
χ^2	= 4.373*	

Table 5. 22.5° OR males

	Temp. of Shock 18°	29°
males	873	1885
females	977	2448
χ^2	= 7.128**	

Table 6. 29° SD males

	Temp. of Shock 22.5°	29°
males	264	455
females	253	588
χ^2	= 7.700**	

Legend: * = P < .05, that deviation observed due to random variation in a homogenous population
** = P < .01, " " " " " " " " " " "
*** = P < .005, " " " " " " " " " " "

probability of an X chromosome being included in a functional sperm increases with the temperature of meiosis. A Y-bearing sperm which would be rendered dysfunctional at 29° may have an increased chance of being functional at 22.5° or 18°. These data imply a nonrandon recovery of gametes in both OR and SD. This difference in recovery of X and Y sperm may be unrelated to the SD drive mechanism.

The males treated with temperature shock also produced progeny with significantly different sex ratios. The effect was dependent on the genotype of the male. OR males reared at 22.5° produced higher sex ratios (♀♀/total) after 29° shock. Cold shock did not cause any significant effect on sex ratio in OR. (See Tables 3-5.) SD-72 males reared at 29° gave significantly lower proportions of female progeny when exposed to cold shock. SD males reared at lower temperatures were unaffected by heat shock treatment.

Godoy, R. and E. del Solar. University of Chile, Santiago, Chile. Choice of oviposition sites in D. melanogaster over 24, 48 and 72 hours.

Three sets of 40 groups of 25 four day old inseminated females were placed in population cages containing 25 numbered equally distanced vials. After 24, 48 and 72 hours the number of eggs per vial, and the position of the vial within the cage, were registered.

Table 1 summarizes the results of the three types of statistical analysis used to measure aggregation, and the mean number of eggs per cage. These show that aggregation decreases while egg numbers increase with time.

Table 1

	Time in hours 24	48	72
Proportion of eggs in the preferred vial	0.30 ± 0.03	0.28 ± 0.01	0.26 ± 0.04
Mean number of vials occupied	16.1 ± 1.04	16.3 ± 0.80	18.8 ± 0.25
Aggregation index $100\sqrt{s^2-\bar{x}/\bar{x}}$	166.48 ± 12.5	165.91 ± 16.2	144.91 ± 20.8
Mean number of eggs per cage	147.5 ± 17.6	156.8 ± 20.9	191.2 ± 26.0

In table 2 the number of vials divided into groups according to the number of eggs collected during 24, 48 and 72 hours, are shown. It may be seen that increase in time allowed for oviposition does not modify the frequency of vials containing a large number of eggs. This result suggests that there is no competition, and that the highest number of eggs is deposited in vials containing between one and ten eggs.

Table 2. Frequency of vials with different number of eggs counted in 24, 48 and 72 hours.

	24 hours		48 hours		72 hours	
	Number of vials	%	Number of vials	%	Number of vials	%
0	357	35.7	348	34.8	253	25.3
1 - 10	476	47.6	484	48.4	546	54.6
11 - 20	100	10.0	84	8.4	100	10.0
21 - 30	41	4.1	40	4.0	47	4.7
31 - 40	11	1.1	20	2.0	23	2.3
41 - 50	3	0.3	6	0.6	15	1.5
51 - 60	3	0.3	4	0.4	4	0.4
61 - 70	2	0.2	2	0.2	3	0.3
71 - 80	4	0.4	1	0.1	3	0.3
81 - 90	1	0.1	1	0.1	3	0.3
91 - 100	1	0.1	1	0.1	1	0.1
101	8	0.8	2	0.2	2	0.2

A Chi-square test performed in order to investigate whether the females use any of the 25 vials for oviposition, indicates no such discrimination in relation to the position of the vial within the cage. (χ^2_{24} = 17.169, P = .85 - .80, N = 120).

A similar test performed to see whether one of the 25 vials is preferred shows no significance either. (χ^2_{24} = 21.205, P = .70 - .65, N = 120).

Seecof, R.L. City of Hope Medical Center, Duarte, California. Phosphate-buffered saline for Drosophila.

The following saline has proved satisfactory for Drosophila embryonic cells cultured in vitro. It was developed by testing various salt combinations for their ability to maintain pulsations of visceral myocytes, up to 10 hours for this formulation. The integrity and pulsatility of larval organs is maintained very satisfactorily as well.

Na_2HPO_4, 85; KH_2PO_4, 50; NaCl, 620; KCl, 200; $MgCl_2 \cdot 6H_2O$, 130; $CaCl_2$, 25; distilled water 100 ml. Weights given as milligrams, final pH 6.75 to 6.80 without further adjustment. Keep sterile or frozen.

Supported by NIH Grant No. AI05038 and NS09330.

Wright, C.P. Western Carolina University, Cullowhee, North Carolina. Attempts to reverse lethality in some lethal mutants of D. melanogaster by transplanting wild-type fat bodies into lethal larvae.

This investigation involved 17 different lethal mutants of D. melanogaster which were X-ray induced by Novitski (1963). It was found by Novitski that these lethals are non-autonomous, because patches of tissue hemizygous for the lethal genes can survive when present in individuals such as gynandromorphs which also have normal tissue. Presumably some essential chemical substance passes from the normal tissue into the lethal tissue and enables it to develop.

In this work attempts were made to reverse the lethality of whole organisms by supplying presumably missing, essential chemicals to the organism. It was thought that the missing chemicals might be supplied by transplanting fat bodies taken from third-instar, Oregon-R larvae into third-instar, lethal larvae. It is known that insect fat bodies contain many metabolic substances, such as enzymes, which are essential for normal development (Clements, 1959; Kilby, 1963). The method of Ephrussi and Beadle (1936) was used to transplant the fat bodies.

Completely negative results were obtained. The transplanted fat bodies had no noticeable effects on any of the 17 recipient lethals. There are several possible explanations for these results. Perhaps the chemicals which were necessary for normal development could not pass from the wild-type fat bodies to the lethal individuals. On the other hand, perhaps by the third-instar stage of development degeneration of larval tissues had occurred to such an extent that reversal of lethality could not be achieved even if the missing metabolites were supplied.

References: Clements, A.N. 1959 Studies on the metabolism of locust fat body. J. Exptl. Biol. 36: 665-675. Ephrussi, B. and G.W. Beadle 1936 A technique of transplantation for Drosophila. Amer. Nat. 70: 218-225. Kilby, B.A. 1963 The biochemistry of the insect fat body. Advances in Insect Physiology 1: 111-174. Novitski, E. 1963 List of biochemical mutants. DIS 37: 51-53.

Majumdar, S.K. and C. Freedman. Lafayette College, Easton, Pennsylvania. Mutation test of calcium cyclamate in Drosophila melanogaster.

In recent years there has been an increase in the use of cyclamates. For this reason research has been done in connection with possible cytogenetic and mutagenic effects of the artificial sweeteners (Legator et al, 1969; Sax and Sax, 1968) in rats and in onion root tips. On the basis of the results obtained from some of the research the FDA has removed cyclamates from its list of "safe" drugs. No work has been presented thus far on the effects of calcium cyclamate in Drosophila except the work of Sram and Weidenhofferova (1969) who studied the mutagenic effects of saccharin.

The mutagenic activity of calcium cyclamate has been investigated by the Muller-5 technique. The wild flies were raised in instant media containing 1, 3 and 5% calcium cyclamate. The F_1 males from these bottles were tested for the sex-linked recessive lethal mutation. The results are given in the table:

Concentrations %	No. Chromosomes Tested	No. Lethals	Mutation Rate %
Control	1530	1	.06
1% Calcium Cyclamate	1120	1	.09
3% Calcium Cyclamate	1222	2	.16
5% Calcium Cyclamate	1280	9	.70

Sram and Weidenhofferova (1969) found a mutation rate of 2.83% using 5 mM concentration of saccharin. These results show that calcium cyclamate has the ability to produce mutation in Drosophila melanogaster wild flies. Further studies are in progress.

The assistance of Mr. Jack Carty is gratefully acknowledged.

References: Legator, M.S., K.A. Palmer, S. Green, and K.W. Peterson, 1969 Science 163: 1139. Sax, K. and H.J. Sax 1968 Jap. J. Genet. 43: 89. Sram, R. and H. Weidenhofferova 1969 DIS 44: 120.

Narise, S. Josai University, Sakado-Machi, Saitama-Ken, Japan. Biochemical differences between α- and β-esterase isozymes in D. virilis.

It has been reported that 15 electrophoretic variants of esterase were found in D. virilis collected in Japan. Among them, ten were controlled by α-esterase locus, and other five were by β-esterase locus. The former, α-esterase, reacted on α-naphthyl acetate and the latter, β-esterase, had the specific activity on β-form when α- and β-naphthyl acetates were used together as substrates after agar gel electrophoresis (S. Ohoba, DIS 45: 56 and DIS, this issue).

13 of 15 electrophoretic variants of the esterase have been extracted separately from the homozygous strains and purified more than 150 fold of the crude enzymes by means of ammonium sulfate fractionation, DEAE-Cellulose and DEAE-Sephadex column chromatography. The molecular weight of the α-esterase was estimated as 80,000 by Sephadex column technique, and that of the β-esterase was 150,000 respectively. All of α-esterases had the activities on both of α- and β-substrate, while the activities of all β-esterases were restricted to β-substrate only. Both α- and β-esterases were not affected by o-iodobenzoate (10^{-4} M) and EDTA (10^{-4} M), slightly inhibited by eserine (10^{-4} M), and strongly inhibited by DFP (10^{-4} M). However, 10^{-4} M PCMB inhibited (90%) only the activities of α-esterases. Furthermore, α-esterases retained their activities (about 70% of controls) after heat treatment at 60°C for one minute while β-esterases lost greatly their activities by the same treatment even though the degree of heat stability was variable among α-esterases as well as β-esterases.

Moyer, S.E., C. Grenier and D. Arthur. Northeastern University, Boston, Massachusetts. "Genetic assimilation" and other characteristics of a salt resistant population of D. melanogaster.

Some preliminary descriptions have been obtained of a population that is able to reproduce in Carolina Instant Medium to which 8 gm. NaCl/100cc H_2O is added. It was initiated by S.M. ten years ago from an outcross of vg stock to a large sample of a natural collection and made homozygous for the vg marker. Wing length began to increase more than five years ago and has gradually become longer, including a low frequency of normal type wings. Males have a much greater frequency of longer wings than females.

The effect of salt to produce longer wings in a vg strain can be interpreted as a type of genetic assimilation with sexual dimorphism. Inherent variability for length of vg wings, exists, especially during occasional higher temperatures. Males with longer wings probably have a mating advantage, especially under the stress of salt in the food. Hence, the frequency of longer wings has increased due to this selective advantage. Furthermore, "sexual dimorphism" for a greater frequency of longer wings in the males may result from selection primarily in males as part of courtship behavior.

A true breeding sub-population of short vg can be established by selection but the wings become longer again in a few generations. Wings have remained short in two other populations initiated from the same base that have not been subjected to salt food, including one that has developed some resistance to DDT.

Limited data indicated no obvious enlargement of the larval "anal organ" in the vg salt strain as compared to the two other vg strains in the sense of genetic assimilation in a salt resistant strain described by Waddington (1958, 1959). His index for measuring this organ shows a possibly slight enlargement if our salt strain is cultured in normal media for at least two generations. (Details and data, present and future, is available on request).

However, the larvae have developed ability to regulate salinity. Evidence obtained by C.G. is that intestinal bacteria from pupae of the salt strain have no greater salt resistance than from other pupae and are the same microbial species.

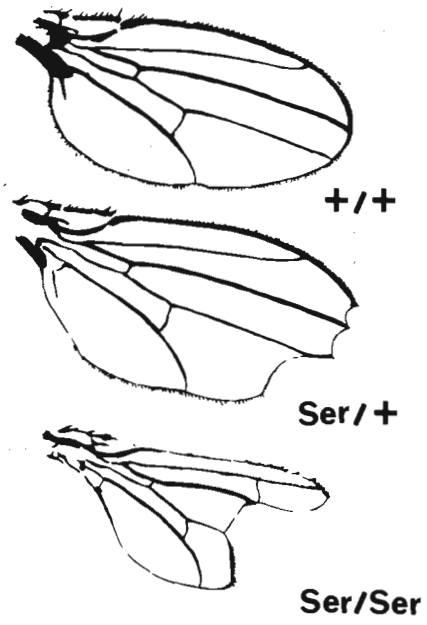
Adults of the vg salt strain die on the 8% NaCl food within two or three days after transfer. Hence, they are required to lay sufficient eggs during this period to establish a new culture. The salt causes generation time to be nearly a week longer and adult body size is smaller.

Limited data suggests that the third chromosome contributes less resistance factors than the X or second.

References: Waddington, C.H. 1958 DIS 32: 163; 1959 Nature 183: 1654.

Belt, A.L. University of Sheffield, England. A non-lethal allele of Serrate?

TM3 Ser (Lewis 1960 D.I.S. 34, Tinderholt 1960 D.I.S. 34) carrying the dominant visible and recessive lethal mutant, Serrate, was used as a third chromosome cross-over suppressor to maintain heterozygosity of the melanotic tumor gene tu-C4 for a selection experiment. The tu-C4 chromosome carried the Stubble mutant to prevent survival of tu-C4 homozygotes. Among the progeny were found flies which were described as 'extreme Serrate'. They were characterised by the absence of the dominant marker Stubble and by an apparent extreme expression of Serrate so that the wings were very much reduced. The phenotype of extreme Ser, heterozygous Ser and Ser^+ are shown in the figure. The development time of the extreme Ser flies was longer than that of normal heterozygotes and they were tumorous. They were isolated from the selection lines and crossed with each other. Stubble did not reappear in the F_1 or subsequent generations and the same extreme Ser expression was seen in all the flies. When they were crossed with wild type, all the F_1 progeny showed the normal heterozygous expression of Ser. In the F_2, extreme Ser, heterozygous Ser and wild type, segregated out.



The possibility of survival of TM3 Ser homozygotes was ruled out because of the tumorous nature of the flies and because they showed no evidence of the recessive mutants also carried by that chromosome. This was confirmed by a salivary-gland chromosome analysis of the extreme Ser flies (carried out by Dr. M. Ashburner, University of Cambridge) which revealed that the Ser chromosome was completely wildtype in sequence.

From this evidence it was concluded that Ser had been transferred from the TM3 Ser chromosome to the tu-C4 chromosome (probably as a result of the double cross-over), and at the same time the lethal effect of the mutant gene had been alleviated. As a result, Ser now segregates as a dominant visible, non-lethal mutant. The most likely explanation for this is that during the cross-over events the dominant visible effects of Ser were separated from a very closely linked recessive lethal mutant. Alternatively, the Ser mutant may have undergone further mutational change to a non-lethal allele which has retained its visible effects.

It is obvious from these findings that the complex inversion system present in TM3 cannot be reliably used as a complete balancer for chromosome three, since cross-over events producing viable gametic products occur at an appreciable frequency. Neither can the recessive lethal mutant Ser be used successfully to maintain heterozygosity of the third chromosome.

Godbole, N.N., R.M. Kothari and V.G. Vaidya. University of Poona, India. An observation on the uric acid content in the excretion of D. melanogaster larvae.

During our studies on the nature of the excretory products in D. melanogaster larvae, a striking variation was observed in the amount of uric acid in the excreta.

Ten larvae of Oregon K strain of D. melanogaster were reared in each of the 25 glass vials each containing 6 g ± 50 mg of the standard cornmeal-agar medium. The vials were placed at 22 ± 1°C during the development of the larvae. After pupation, the medium from each vial was homogenized separately in 100 ml distilled water. Few drops of concentrated KOH solution were added to facilitate maximum extraction of uric acid. Uric acid was quantitatively estimated for each vial by Brown's reaction (Brown, 1945) using Klett and Summerson's photoelectric colorimeter at 660 mμ.

It was observed that the uric acid content of the extracts varied between nil and 330 μg. Similar results were obtained by rearing larvae on six different media. This indicates that composition of the medium plays no role in the observed pattern of uric acid excretion. An alternate pathway resulting from an error in uric acid metabolism is suspected in some flies from our culture.

Reference: Brown, H. 1945 The determination of uric acid in human blood. J. Biol. Chem. 158: 601-608.

Rajaraman, R. and O.P. Kamra. Dalhousie University, Halifax, Canada. Effect of pretreatment with DNA bases and base analogs on the incidence of the sex-linked recessive lethal mutations in irradiated sperms of D. melanogaster.

It is known that the incorporation of the halogenated base analogs in the DNA increases the radiation damage to DNA (Szybalski, 1967) and that U.V. or X-irradiation stimulates the incorporation of bases or base analogs in the DNA strands even in the non-S-phase cells, a phenomenon known as "unscheduled DNA synthesis" or "repair replication" (Rasmussen and Painter, 1966; Painter and Cleaver, 1967; Evans and Norman, 1968). During our studies on the disputed question of radiosensitization by the halogenated base analogs in the premeiotic germ cells of male D. melanogaster (Rajaraman and Kamra, 1970), we also included a study of the effects of these chemicals on the radiosensitivity of the postmeiotic germ cells (mature sperms). This note reports the results obtained with the mature sperms.

The effect of the base Thymidine (Tdr) and base analogs 5-bromo deoxycytidine (BCdR) and 5-bromo deoxyuridine (BUdR) on the radiosensitivity of D. melanogaster sperms was studied by intra-abdominal injection of 0.1% base or analog solution in 0.7% NaCl followed by gamma irradiation (^{137}Cs source at the dose rate of 4.2 R/sec.) with proper controls. Treated 1-day-old $X^{c2}y$ B/sc^{8} Y males were individually mated with six 3-day-old y sc^{S1} In-49 sc^{8};bw;st p^{p} virgins for two days and the frequency of the sex-linked recessive lethal mutations was studied through F_2. The treatments and the results are shown below:

Treatments*	No. Chrom. Tested	No. Leth.	% Leth.
1. NaCl + 1.2kR gamma rays	907	28	3.1
2. BCdR + 1.2kR gamma rays	905	11	1.1
3. BUdR + 1.2kR gamma rays	385	7	1.7
4. TdR + 1.2kR gamma rays	667	9	1.3

$\chi^2 = 8.76$ d.f. = 3 $P = <0.05$

* Unirradiated control experiments in all the four treatments showed a lethal frequency well within the range of the spontaneous mutation frequency.

The results indicate that the presence of the exogenous bases or base analogs reduce the frequency of the sex-linked lethal mutation significantly at 0.05 level. Preliminary studies on the fecundity and dominant lethals did not show any appreciable difference between the different treatments. Hence, it seems unlikely that the reduction in the frequency of the sex-linked recessive lethals was due to a selective elimination of sperms with induced lethals. Subsequent studies using 7-day-old males and a higher radiation dose showed a similar reduction in the incidence of lethals (to be reported elsewhere). Hence, it is highly unlikely to be an artifact.

The mechanism of reduction (repair?) of radiation induced genetic damage is not known. Since the DNA in the sperm nucleus has been synthesized long before the injection of the base analogs, they are not likely to be incorporated in the sperm nuclear DNA at the time of irradiation and hence no radiosensitization. Nevertheless, the "fixation" of the radiation induced genetic damage has been reduced by the presence of the exogenous base or base analogs. This effect may be ascribed to radiation stimulated non-semiconservative incorporation of nucleosides in the damaged sites of the DNA strands (repair replication) in the sperm nucleus. On the other hand, any other indirect metabolic effects (Wolf, 1966; Smets, Hallman, Lause and Kuyper, 1967) may also result in the repair of the chromosomal damage. Studies are in progress to elucidate the mechanism involved in the reduction of radiation-induced sex-linked recessive lethals in the mature sperms of D. melanogaster following the injection of bases or base analogs.

References: Evans, R.G. and A. Norman, 1968 Nature 217: 455; Painter, R.B. and J.E. Cleaver, 1967 Nature 216: 369; Rajaraman, R. and O.P. Kamra, 1970 Can. J. Genet. Cytol. 12: 392; Rasmussen, R.E. and R.B. Painter, 1966 J. Cell Biol. 29: 11; Smets, L.A., P. Hallman, P. Lause and Ch.M.A. Kuyper, 1967 Int. J. Radiat. Biol. 13: 269; Wolf, S., 1966 in Genet. Aspects of Radiosensitivity: Mechanisms of Repair, I.A.E.A., Vienna, 1.

This work was supported in part by the National Research Council of Canada Grant no. A-4892.

Turner, D.C. and C.P. Wright Western Carolina University, Cullowhee, North Carolina. The effects of some human-consumed chemicals on development of Drosophila melanogaster.

A study of the effects of several human-consumed commercial products on the development of D. melanogaster was made. The following commercial products were used in the indicated concentrations: Bayer brand of aspirin - .25%, .5%, 1% and 3%; Accent brand of monosodium glutamate - 1% and 3%; Sweet-10 brand of artificial sweetener (contains 36% calcium cyclamate, 3% calcium saccharin, and 61% acacia) - 1%, 3% and 6%; and Norwich brand 1/4 grain sodium saccharin - 1%, 3% and 6%.

The various concentrations of the chemicals were prepared in distilled water and the resulting solutions were used in preparation of Carolina Biological instant Drosophila medium. In the control, instant Drosophila medium was used which contained distilled water only. The various concentrations of media were placed into sets of ten vials each. Three male and three female five-day-old Oregon-R flies were placed in vials containing the various media, and after five days of egg laying the flies were removed and development of the next generation was observed.

After five days of egg laying the P_1 adults were removed from the vials and counted to determine the number of flies which had survived. Table 1 shows that with increase in concentration of Bayer aspirin there was increasing lethality in the P_1 adults. Such a lethality effect was not observed in P_1 flies exposed to the other chemicals used in the experiment.

Table 1
Effects of Bayer Aspirin on P_1 Generation

Conc. of Bayer Aspirin	No. of Surviving Adults
0%	39
.25%	58
.50%	34
1.00%	7
3.00%	0

Table 2
Effects of Bayer Aspirin on F_1 Generation

Conc. of Bayer Aspirin	No. of Adults Produced
0%	981
.25%	1190
.50%	476
1.00%	58
3.00%	0

Table 3. Effects of Sweet-10 Artificial Sweetener on F_1 Generation

Conc. of Sweet - 10	No. of Adults Produced
0%	981
1.00%	1465
3.00%	1076
6.00%	129

Table 4. Effects of Norwich Sodium Saccharin on F_1 Generation

Conc. of Sodium Saccharin	No. of Adults Produced
0%	981
1.00%	1186
3.00%	776
6.00%	230

Table 5. Effects of Monosodium Glutamate on F_1 Generation

Conc. of Monosodium Glutamate	No. of Adults Produced
0%	981
1.00%	942
3.00%	948

Table 2 shows that with increasing concentration of Bayer aspirin, fewer F_1 adults were produced. Table 3 shows that with increasing concentration of Sweet-10 artificial sweetener, fewer F_1 adults were produced. Table 4 shows that with increasing concentration of Norwich sodium saccharin, fewer F_1 adults were produced. Table 5 shows that there was no significant difference between F_1 adult populations which developed on monosodium glutamate and those which developed in the control.

Band, H.T. Michigan State University, East Lansing, Michigan. Trend changes in rainfall and temperature range in the Amherst, Massachusetts area.

Changes in rainfall and temperature range patterns spanning several decades in Amherst, Mass. have been occurring. The data given below are May-October averages. This interval approximately spans the breeding seasons of D. melanogaster there (see Ives, 1970). The population in that area has been investigated by Dr. P.T. Ives since the 1930's. Genetic changes toward a lower level of lethals and semilethals among second chromosomes in the population were observed in 1947 (Ives, 1954) and multiple genetic changes occurred in the 1960's (Band, 1964; Ives, 1970). Average daily temperature range and average total rainfall for the six months interval in the various periods have been:

	1930-1946	1947-1962	1963-1966	1967-1969
average daily temperature range	23.0	24.0	26.4	24.5

	1930-1946	1947-1961	1962-1965	1966-1969
total rainfall in inches	23.48	22.01	15.22	22.07

Furthermore, average daily temperature range for the summer months shows a regular 0.8^{o} increase per decade, starting with an average of 22.5^{o} between minimum and maximum daily temperatures in the 1930's and climbing to a 24.9^{o} average for the 1960's. No other season shows such regular changes over the four decades though increases in temperature range in other seasons are also observed both in the 1940's and in the 1960's, particularly spring quarter of the latter decade.

The fairly simultaneous changes in both average daily temperature range and rainfall suggest the two are not independent climatic variables. Both had already been found to be significantly correlated with the level of lethals and semilethals found in the population. Summer rainfall is significantly positively correlated with fall le + sle frequency (Band and Ives, 1968) and temperature range prior to collection is highly significantly negatively correlated with le + sle frequency recovered in a sample (Band and Ives, 1961, 1968). The genetic changes detected in the population have been found to occur at each shift in one or both environmental variables.

The above climatic data strengthen the argument that genetic homeostasis accounts for the retention of le + sle frequency at 34% in 1964 (Band, 1969), the level around which these variants have fluctuated since 1947. No significant decline in lethals was detected until June 1966, when the frequency of these variants was only 17% (Ives, 1970). Yet after 1962 rainfall and after 1963 temperature range also maintained respectively their lowest and highest four-year averages during the forty-year period.

More climatic information than that available from averages per climatic period is needed to determine whether retention of le + sle frequency at 33.9% in 1962 is also an example of the resistance of le + sle frequency to decline. However, the increased developmental homeostasis present after 1961 (Band, 1964) does seem to have delayed the decline in le + sle frequency despite the persisting downward selection pressures exerted by both environmental variables from 1963 through 1965.

How widespread or how local these climatic shifts have been is not known at present. Tantawy et al. (1969) in Egypt and Hoenigsberg (1968) in Colombia both reported localities in which D. simulans supplanted D. melanogaster as the dominant species in the mid-1960's. Watanabe (1969) reported a significant decline in le + sle frequency in a Japanese D. melanogaster population in 1966. The possibility of climatic shifts was not considered; alternate hypotheses advanced in each case seemed more appropriate. Time-wise these may all be coincidental with climatic shifts and genetic changes in the S. Amherst D. melanogaster population.

References: Band, H.T., 1964 Evolution 18: 384-404; ____, 1969 Japan. J. Genet. 44 Suppl. 1: 200-207; ____ and P.T. Ives, 1961 P.N.A.S. 47: 180-185; ____ and ____, 1968 Evolution 22: 633-641; Hoenigsberg, H.F., 1968 Amer. Nat. 102: 389-390; Ives, P.T., 1954 P.N.A.S. 40: 87-92; ____, 1970 Evolution, in press; Tantawy, A.O., A.M. Mourad and A.M. Masri, 1969 Amer. Nat. 104: 105-109; Watanabe, T.O., 1969 Japan. J. Genet. 44: 171-187.

Band, H.T. Michigan State University, East Lansing, Michigan. Increased developmental homeostasis in a natural population and climatic changes.

Natural populations or species inhabiting a more variable climate possess greater developmental homeostasis than those in more equable conditions (Tantawy and Mallah, 1961; Dawson, 1968). Experiments by Beardmore and Levine (1963) demonstrated that populations of D. pseudoobscura maintained in fluctuating temperatures developed greater homeostasis than those in constant temperatures although all populations were initiated by the same founders. Band (1964) presented evidence that the S. Amherst D. melanogaster population displayed greater developmental homeostasis in 1962 than in 1961 or 1960, and that the new level was maintained in the population in 1964 (Band, 1969). Prior to the time of onset of increased developmental homeostasis it was known only that the winter of 1960-61 had been unusually severe and that the combined spring and summer quarters of 1962 were the driest on record (i.e. back to 1889). Subsequently Ives determined that total rainfall in the area had declined about 30% below 1889-1960 norms for a period from July 1961 to June 1966 (see Band and Ives, 1968; Band, 1969). This provides evidence that we are dealing with a population in a period of climatic change but fails to explain why an adaptation to drier conditions should also convey a decreased variability in the population to temperature.

In this issue we have reported that there have been near-parallel climatic shifts in daily temperature range and rainfall in the May-October interval from 1930 to 1969. Both display maximum severity during the 1961-62 through 1965-66 span of the past decade.

The summer months constitute a standardized quarter of the year and chromosome collections analyzed in 1960, 1961, 1962 and 1964 were made in September. Average daily temperature range and number of days having narrow (3-20°), intermediate (21-25°) and wide (26-42°) ranges between minimum and maximum daily temperature are given below for summers in the 1960 decade. Summers 1963 through 1966 have a significantly wider range per day between minimum

Table 1. Average daily temperature range per summer

1960	1961	1962	1963	1964	1965	1966	1967	1968	1969
23.5	23.6	25.4	26.4*	25.9*	26.3*	26.6*	23.4	24.2	24.0
±7.4	±7.0	±8.0	±6.7	±7.3	±7.2	±7.2	±7.5	±7.6	±7.8

and maximum temperatures than those in the earlier part of the decade or summer 1967. Average daily range in 1962 misses the significance level, but when total numbers of days with range

Table 2. Number of days with narrow, intermediate or wide ranges.

	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969
N	30	23	23	20	16	21	16	30	21	25
I	23	33	23	17	25	17	22	28	25	20
W	39	36	46*	55*	51*	54*	54*	34	46*	47*

26° or above are considered, then summers 1962 through 1966 all have significantly more days in the wide range category.

The months of June and July in the summer and May, June, and July in the May-October span in 1962 all have an average temperature range of 26° or over. Hence it would seem that elevated ranges in the natural environment as well as drier conditions have contributed to the onset of increased developmental homeostasis in the natural population detected by September 1962.

If developmental homeostasis in the population can be said to be maintained by stabilizing selection, then an increase in developmental homeostasis indicates the efficiency of directional selection to bring about population adaptedness to the more severe climatic conditions.

Further, since the average le + sle frequency found in the population 1947-1961 is 33.7% (2205 chromosomes, 743 lethals and semilethals; from Ives, 1970, table 1), retention of le + sle frequency at 33.9% in 1962 seems evidence for the operation of genetic homeostasis and indicates that such variants play an adaptive role in population structure.

References: Band, H.T., 1964 Evolution 18: 384-404; ____, 1969 Japan. J. Genet. 44 Suppl. 1: 200-207; ____ and P.T. Ives, 1968 Evolution 22: 633-641; Beardmore, J.A. and L. Levine, 1963 Evolution 17: 121-129; Dawson, P.S., 1968 Evolution 8: 119-134; Ives, P.T., 1970 Evolution 24: 507-518; Tantawy, A.O. and G.S. Mallah, 1961 Evolution 15: 1-14.

Scozzari, R., G. Trippa, C. Santolamazza, L. Ulizzi, C. Barberio and G. Modiano.
Istituto di Genetica, Università di Roma, Italia. Enzyme activity in three phosphoglucomutase phenotypes of D. melanogaster.

Natural populations of Drosophila melanogaster present a polymorphism for phosphoglucomutase (PGM) with at least two common electrophoretic alleles: Pgm^A and Pgm^B (Trippa et al., Biochem. Genetics 4, 1970, in press).

The mean PGM activities of the three PGM electrophoretic phenotypes (PGM A, PGM AB and PGM B) have been measured in order to estimate the mean PGM activities associated with the two PGM alleles.

A sample of 295 females from a natural Italian population, collected near Rome, has been examined for PGM electrophoretic phenotype and PGM activity. The PGM electrophoretic phenotypes were determined according to Trippa et al. (1970). The PGM activity has been expressed as micromoles of glucose-6-phosphate (G6P) produced per hour at 37°C per mg of nucleic acids of the single-fly homogenate.

The mean PGM activities were found to be not significantly different in the three PGM electrophoretic phenotypes. This finding shows that the activities depending upon the two PGM alleles (Pgm^A and Pgm^B) are the same, at least in the conditions of the present assay.

In order to get a better estimate of the mean PGM activity the data obtained for the three phenotypes have been pooled together, the mean activity in the whole sample being 77.59±1.17.

Lieb, E. Universität München, Germany. Recombination in gynandromorphs of Drosophila melanogaster.

In 1912 Morgan found that Drosophila melanogaster males, unlike females, do not show genetic recombination. The basis for this difference between the sexes in Drosophila and some other organisms is still unknown. It could be due to factors which are present in the germ-cells themselves. It also seems possible that dissimilarities in the somatic cells of males and females could influence recombination in germ-cells. In order to investigate this latter possibility the rate of recombination in gynanders was registered.

Gynanders heterozygous for the second chromosome markers al, b, and c were selected from the offspring of R(1)2, $In(1)w^{vC}/In(1)dl\text{-}49$, y w lz^s ♀♀ and XY^LY^S, w spl cv f; al b c ♂♂. After loss of the unstable ring-X the XY^LY^S testes are able to produce motile spermatozoa. Six "female" gynanders, i.e. with ovaries and female genitalia, were backcrossed to al b c ♂♂; seven "male" gynanders were backcrossed to al b c ♀♀. In each of the 13 gynanders both gonads were of identical type, i.e. both ovaries or both testes. The mosaic character of the flies was visible in their heads and thoraxes; in some cases in an otherwise female abdomen one dorsal half was male, or vice versa. As a control al b c heterozygous ♀♀ were backcrossed to al b c ♂♂.

The recombination rate in the six female gynanders was the same as in the controls (within the limits of a statistical test for homogeneity; $p = 0.18$). No recombinants were found among the 3853 offspring of six of the male gynanders. Among the 1189 offspring of the seventh male gynander six recombinants (0.5%) were found: 5 al b^+ c^+ and one of the complementary type al^+ b c. This cluster of recombinants can well be explained by a single premeiotic exchange. Under this assumption the overall recombination rate is ca. 0.1%, which is of the same order of magnitude as the spontaneous frequencies of recombinants found for males (e.g. Cooper 1944).

In my experiments only the influence of somatic (i.e. other than gonadal) tissues of the opposite sex on recombination in the gonads of male (or female) gynanders could be studied. No such influence was found. Gynanders with both an ovary and a testis have been found (Sturtevant 1929, Dobzhansky 1931, Tokunaga 1961) and may even be fertile. They are, however, very rare and there were no gynanders of this kind among the 54 whose gonads I investigated. The influence of gonads of one sex on recombination in gonads of the opposite sex has been studied in transplantation experiments by Gloor and Hadorn (1959; D.I.S. 33). They implanted testes in female larvae; the results were not fully conclusive. The corresponding test for males has not been made.

Wasserman, M., H.R. Koepfer and M.J. Geller. Queens College, Flushing, New York. Collections of Drosophilids from New Mexico and Colorado, with new data on the third chromosome arrangements of D. pseudoobscura found there.

During July 1970, collections were made in Ruidoso, New Mexico and the Black Forest, approximately 10 miles north of Colorado Springs, Colorado. The Ruidoso locality, at an elevation of 6900 feet, is mainly a pine and juniper woods with some shrub oak. Pine is the dominant form in the Black Forest, which is at 7500 feet elevation. Both localities were heavily baited with bananas, oranges and pineapple. D. cinerea and a few specimens of D. tenebrosa and D. suboccidentalis were also taken over mushrooms in the Black Forest. At both localities, D. pseudoobscura was a dominant form, the quinaria species group (D. tenebrosa, D. inubila and D. suboccidentalis) making up the bulk of the other Drosophilids (Table 1). The Ruidiso locality is the furthest east that D. arizonensis has been found in the United States.

Table 1. Collection records of Drosophila and Chymomyza

Species	Ruidoso, N.M.	Black Forest, Col.
D. pseudoobscura	957	275
D. tenebrosa	21	22
D. inubila	14	-
D. suboccidentalis	-	338
D. melanogaster	1	7
D. arizonensis	4	-
D. victoria	-	1
D. cinerea	-	1
C. amoena	7	-
TOTAL	1004	644

The results of the analysis of the third chromosome arrangements of D. pseudoobscura males collected at these localities are shown in Table 2. Also are shown the results of the analyses of chromosomes from females obtained from the same 1970 Ruidoso collection (personal communication from Th. Dobzhansky), and from a previous, 1964, collection (Dobzhanshy et al. 1966). There are no significant differences in the frequencies of the chromosome arrangements between the males and the females from the 1970 Ruidoso collection.

Table 2. Percentage of third chromosome arrangements and total chromosomes studied (n).

Locality	Sample	Year	AR	PP	CH	ST	OL	EP	TL	n
Ruidoso, N.M.	males	1970	74.2	18.2	4.6	1.5	1.5	-	-	66
	females*	1970	67.2	27.4	2.9	1.6	0.4	0.4	-	244
	females**	1964	69.5	25.6	1.2	2.4	-	-	1.2	82
Black Forest, Col.	males	1970	60.0	35.0	-	2.0	1.0	-	2.0	100

* Dobzhansky (personal communication) ** Dobzhansky et al. (1966)

Moreover there are no significant differences between the 1964 and 1970 collections from Ruidoso.

References: Dobzhansky, Th., W.W. Anderson and O. Pavlovsky, 1966 Evolution 20: 418-427.
This work was supported by grants from the City University of New York Faculty Research Award Program and from the N.I.H. FR-07064.

Roberts, P.A. Oregon State University, Corvallis, Oregon. Localization of pr to region between gene duplications in chromosome arm 2L.

The location of the gene for purple eyes in D. melanogaster had previously been narrowed to the region between 38B2 and 38F7 (Roberts, P.A. 1968, Genetics 60: 216). Whether this recessive gene is within the proximal duplication on Bridges' map extending from 38E--39E could not be determined at the time. An X-ray induced pr deficiency extending from 37D1--38C1 has since been recovered. This would place pr between bands 38B2 and 38C1 - between the duplicated segments. Had pr been within the duplication, it would have suggested evolutionary divergence of the duplicated genes. As mentioned in the previous report, the large size of recovered deficiencies in this region suggests that the duplicated genes may still retain many functions in common.

Evans, W.H. Western Washington State College, Bellingham, Washington. Preliminary studies on frequency of autosomal nondisjunction in females of D. melanogaster.

As noted by Chadov (1969) aneuploid eggs may be fertilized by complementary sperm from isochromosome-bearing males. By this method he was able to demonstrate the non-homologous pairing of the X and Y chromosomes with the second chromosomes of XXY;Sm1, Cy/+ females. However, quantitative statements about the rates of primary and secondary nondisjunction of the major autosomes have not been possible to date because of the difficulty of recovering all progeny from the virtually sterile matings and because the behavior of isochromosomes in the male parents has not been well-defined.

Table 1 presents the results of an experiment designed to indicate the extent to which the presence of a Y chromosome and/or inversion heterozygosity causes nondisjunction of the major autosomes in D. melanogaster females. Three-to-five day old females of the various genotypes were mated to an excess of attached-3 males. Eggs were collected on charcoal-blackened standard medium by a method modified after Hildreth and Brunt (1961, D.I.S. 36: 128). After counting, eggs developed in a moist chamber at 25°C for 28 hrs. before flat eggs were tallied.

Table I

Hatchability of eggs laid by females of various genotypes after mating to C(3)L, ri;C(3)R, sr males. Second chromosome rearrangement is Sm1, Cy. Third chromosome inversion is In(3LR)CxD.

Maternal genotype	% Hatchability by attached-3 ♂♂*	% Hatchability by w/Y;+/+;+/+ ♂♂	Progeny type
1. XX;+/+;+/+	3.8	93	adult progeny rare, larvae die soon after hatching
2. XX;Cy/+;+/+	4.8	70	intersexes
3. XX;+/+;D/+	5.4	84	matroclinous and patroclinous-3
4. XX;Cy/+;D/+	4.3	61	same as above, also intersexes
5. $XXB^{S}Y$;+/+;+/+	6.1	91	same as 1 above
6. $XXB^{S}Y$;Cy/+;+/+	6.6	59	intersexes
7. $XXB^{S}Y$;+/+;D/+	10.1	65	same as 3 above
8. $XXB^{S}Y$;Cy/+;D/+	9.2	53	see Table II

* Data based on counts of 658-893 eggs.

From Table I it is clear that the hatchability of eggs laid by wild-type females mated to C(3)L, ri;C(3)R, sr males is unexpectedly high. This 3.8% hatchability reflects a toleration for aneuploidy that does not normally extend beyond the first instar. Assuming no major autosome nondisjunction in wild-type females, one would expect that this 3.8% hatchability is the lowest figure that would be obtained in any of the experimental matings to C(3)L, ri;C(3)R, sr males. Assuming further that these attached-3 males produce four sperm types (C(3)L, C(3)R, C(3)L;C(3)R, and nullo-3) in equal numbers (D.L. Lindsley and E.H. Grell, 1969) any hatchability in excess of 3.8% should represent 1/4 of the total diplo-and nullo-3 eggs laid. On this basis estimates of the frequency of third chromosome nondisjunction in the XX females could be made. Estimates of 2^{nd} chromosome nondisjunction due to the presence of Sm1 in the female genome are complicated by the fact that only 1/16 of aneuploid-2 eggs are recoverable in crosses to attached-3 (ri, sr) males. Only the diplo-2 eggs are recoverable, and these only by X-bearing C(3)L;C(3)R sperm, resulting in X/X;Sm1, Cy/+/+;C(3)L/C(3)R/+ intersexes.

Nevertheless, the data in Table I do seem to reliably reflect the number of aneuploid eggs laid by the various experimental females. Thus, the introduction of Sm1 causes an increase in hatchability and adult intersexes are recovered (lines 2 and 5). In the case of XX females which are inversion heterozygous for both major autosome pairs, the reduction in hatchability is presumed due to non-homologous associations of the major autosomes (Forbes, 1962). In the case of similar XXY females the reduction in hatchability is probably due to competition of the major autosomes in pairing with the Y chromosome. The data in the "progeny type" column of Table I are derived from mass matings paralleling the egg count matings.

Table II details the progeny types recovered from the cross XXB^{S}Y;SM1, Cy/+;Cx, D/+♀♀ x C(3)L, ri;C(3)R, Sr♂♂. The number of progeny is remarkable considering the small scale of the experiment. It is noted that the maternal Y is virtually always recovered in patroclinous-3 progeny indicating frequent Y-3 associations. Also, there seems to be a pronounced discrepancy in the recovery of such progeny.

Table II

Progeny from the cross XXB^{S}Y;SM1, Cy/+;In(3LR)CxD/+♀♀ x C(3)L, ri;C(3)R, sr♂♂

Patroclinous-3				Matroclinous-3				Intersexes	
genotype	♂♂	♀♀	total	genotype	♂♂	♀♀	total	genotype	total
B^{S}Y;+/+;(ri, sr)	3	8	11	+/+D/+	8	26	34	+/+/+;C(3)L/C(3)R/+	1
B^{S}Y;Cy/+;(ri, sr)	8	6	14	Cy/+D/+	7	14	21	Cy/+/+;C(3)L/C(3)R/+	8
+/+;(ri, sr)	1	1	2	B^{S}Y+/+D/+	1	1	2		
		total	27			total	57	total	9

In the course of this study it was noted that adult progeny are more readily recovered from matings of SM1/+ females to attached-3 males than from a mating of such females to attached-2 (b, cn) males. It would seem that aneuploid-2 eggs would be best recovered by complementary sperm from C(2)L, b;C(2)R, cn males. As mentioned previously only 1/16 of aneuploid-2 eggs are recoverable in matings to attached-3 (ri, sr) males. This result led to the conclusion that meiosis in C(2)L, b;C(2)R, cn males is not entirely random in regard to segregation of isochromosomes. This is further indicated in that 41% of the eggs laid by these attached-2 females develop into adults. In contrast the figure for the C(3)L, ri;C(3)R, sr stock is 19%. This figure agrees fairly well with a theoretical maximum of 25% if meiosis in females is regular and if segregation of the isochromosomes in males is random.

References: Chadov, B.F., 1969 Non-homologous pairing and non-disjunction of the second chromosomes in oogenesis of Drosophila melanogaster. Genetica 5:190-192; Forbes, C., 1962 The effect of heterozygous inversions on primary non-disjunction in Drosophila melanogaster. Genetics 47: 1301-1311; Lindsley, D.L. and E.H. Grell, 1969 Spermiogenesis without chromosomes in Drosophila melanogaster. Genetics Suppl. 61:1, 70-78.

Dapples, C.C. Rocky Mountain College, Billings, Montana. Ovarian morphology of the singed^{36a} mutant of D. melanogaster.

In this study feulgen-stained whole mounts of ovaries from females homozygous for the singed^{36a} mutation were compared with Oregon-R wild type ovaries of identical age. The singed^{36a} ovary is about half the size of the Oregon-R wild type ovary. The reduced size is due in part to the lack of oocytes in vitellogenic stages. There are also fewer chambers per ovariole and fewer ovarioles per ovary than in wild type ovaries. In wild type there are an average of a little over six egg chambers per ovariole and this number remains constant over the ten day period studied (0-10 days). In singed^{36a} however, there are three egg chambers per ovariole for the first two days, the number then increases to five by day four, but then steadily decreases until there is only an average of one chamber per ovariole by the tenth day. This reduction in the number of egg chambers per ovariole is due to the increasing amount of degeneration present. Although the average number of ovarioles in a singed^{36a} ovary is less than that for wild type the number remains constant over the ten day period studied. The egg chambers within a singed^{36a} ovariole are generally shaped abnormally and packed against one another. This packing is caused by a blocking of the lateral oviducts with degenerating chambers.

Degeneration appears in most chambers at the time when vitellogenesis should start or else develops because yolk synthesis does not commence. In either case degenerating egg chambers are already present in one day old flies. That is, about nine percent of the egg chambers appear pynotic and this degeneracy increases linearly with age up to ten days when most of the egg chambers appear to be dead.

Van Valen, L. and P. Van Valen. University of Chicago and University of Illinois, Chicago, Illinois. A method that does not estimate age in Drosophila.

Perhaps the most important reason for the virtual lack of analytical ecology of Drosophila is the inability to determine individual post-teneral ages. Labeling techniques like that of Richardson et al. (1969) will help but are unsuitable for some purposes. Neville (e.g. 1967 a, b) and others have found daily growth layers in the cuticle of several insects and it seemed possible that they might occur in Drosophila.

D. melanogaster adults were kept at 25° for 6 or 7 hours in the day and at 15° at other times from eclosion. On the 15th to 20th day they were killed and the middle legs fixed in Kahle's fluid. Frozen sections of femora gave no results, probably because of the unfamiliarity of the technician with this method.

Standard embedding and sectioning produced useful sections which were stained with Mallory's or hematoxylin-eosin, or left unstained. No sets of layers were visible at magnifications to 1000X, for both thin and thick cuticle. A control using a wild-caught grasshopper did produce visible layers. It may be that electron-microscopic studies would detect layers in Drosophila, but a method more suitable for routine ecological work did not if layers are produced endogenously or by temperature.

We thank J. McLane for technical assistance and W.L. Doyle, D.B. Wake, E.B. Spiess and W.K. Baker for use of equipment. A grant from NSF supported this work.

References: Neville, A.C., 1967a, Adv. Ins. Physiol. 4: 213-286; Neville, A.C., 1967b Biol. Rev. 42: 421-441; Richardson, R.H., R.J. Wallace, S.J. Gage, G.D. Bouchey and M. Denell, 1969 Stud. Genet. (Univ. Texas) 5: 171-186.

Ehrman, L. and D.L. Williamson. Rockefeller University, New York, and Medical College of Pennsylvania, Philadelphia, Pennsylvania. Differential centrifugation and infectious hybrid sterility in D. paulistorum.

Matings between the D. paulistorum semispecies produce sterile male progeny (Perez-Salas, et al 1970). At least in some of the instances of sterility in this species-complex, transmissible symbionts or commensals are implicated. Genetically pure Mesitas males can sometimes be made sterile by injecting their mothers with material derived from Santa Marta X Mesitas sterile F_1 hybrid males (Ehrman and Williamson, 1969 and references therein). We have previously used homogenates of sterile hybrid male material centrifuged at low temperatures at approximately 1000X gravity; this results in only a crude homogenate. To try to purify the infectious "factor(s)" involved we attempted differential centrifugation in a sucrose solution using assorted speeds up to an including 100,000X gravity. Little or no purification of the self-reproducing entity (presumably a mycoplasma-like microorganism, see Kernaghan and Ehrman, 1970), resulted. But some of the data are worth recording:

1) The percentages of induced male sterility were higher in later broods, i.e., when the recipient females had longer intervals within which to incubate the infectious material injected into them;

2) The source of infectious material providing results closest to that of injected crude homogenates came from that pelleted at approximately 10,000X gravity. This was true in all broods surveyed for sterile males. At 10,000 gravity one may expect structures like mitochondria and/or mycoplasma to sediment;

3) The smallest percentage of induced male sterility occurred when the resuspended pellet from the highest speed, 100,000X gravity, was used to inject females whose sons were tested for sterility.

We realize that much more work remains to be done using the techniques of differential ultracentrifugation on this unique material. With Dr. R.P. Kernaghan of The State University of New York at Stony Brook, we plan to couple ultracentrifugation with the electron microscopy of suitable homogenates.

References: Ehrman, L. and D.L. Williamson, 1969, Genetics 62: 193-199; Kernaghan, R.P. and L. Ehrman 1970, Chromosoma 29: 291-304; Perez-Salas, et al, 1970, Evolution 24: 519-527.

Lee Ehrman is the recipient of USPHS Career Award 2K03 HD 09033-06. David L. Williamson acknowledges the support of NIH Grant AI 09615.

Langjahr, S.W. California State College San Bernardino, California. Further effects of butylated hydroxytoluene on the longevity of D. melanogaster.

The free radical reaction inhibitor 2,6-Di-tert-butyl-p-cresol (BHT) and other chemical antioxidants have been shown to significantly prolong the life span of certain strains of laboratory mice when added to the daily diet (Harman 1957, 1961, 1968). As highly reactive living system intermediates, free radicals can propagate many deleterious reactions contributing to degradation of neighboring tissues. These endogenous reactions, fed by enzymatic or non-enzymatic sources, have been implicated in contributing to the overall process(es) of aging through time (Harman, 1969). Thus, any chemical capable of harmlessly removing free radicals from participation in such reactions would tend to offset autooxidation and decrease harmful changes in the bioplasm.

The experiment at hand was designed to determine the effects of relatively high concentrations of BHT on the mean life span of wild-type Drosophila melanogaster.

TECHNIQUES: Pint-bottle stock cultures of D. melanogaster, existing on a specific agar-base medium (Kalmus, 1943) were used for collection of full-term pupae separated according to sex into sterile shell vials 2 x 8 1/2 cm, ten individuals per vial, and allowed to emerge into adults.

Crystalline BHT (M.P. 70°) was pulverized and added by weight to the agar and inorganic salt medium in the following concentrations: 0.5%, 0.25%, 0.125% and 0.0625%. A drop of 10% baker's yeast suspension was consistently added to all vials to provide necessary protein.

All vials were stored horizontally at 25°C, average humidity 50-60%. Daily observations were made, noting deaths and checking medium dehydration. Dead flies were removed and surviving flies transferred to fresh medium every six days. Populations were not consolidated as the flies died.

Mid-way through the experiment, it was thought that perhaps the flies were sensitive to the atmosphere saturated by the BHT vapor present from the BHT/agar mixture. A quick test was constructed by stocking three vials with ten female pupae each. One vial contained an ordinary Kalmus preparation, another a typically lethal 0.5% dose of BHT added in the medium, and lastly one to which ordinary control medium was added, but with two grams of BHT crystals implanted in the vial plug, in such a way as to be unavailable for consumption but allowing for easy gaseous diffusion of the vapor throughout the tube. This test would quickly ascertain any fumigant qualities of BHT on Drosophila.

RESULTS: BHT as a diet additive failed to lengthen the life span in wild-type Drosophila, and in fact was shown to be toxic in all but the lowest concentrations.

BHT Concentration	No.♂♂	Mean life span	No.♀♀	Mean life span
0 = control	41	39.6 ± 15.6 days	41	50.0 ± 14.7 days
0.0625 %	34	31.9 ± 13.4*	45	35.6 ± 13.5**
0.125 %	16	10.0 ± 5.5	32	15.3 ± 10.4
0.25 %	18	8.9 ± 4.0	21	12.6 ± 6.8
0.50 %	16	7.2 ± 2.4	18	11.1 ± 6.7

* value significantly less than controls, $.02<P<.05$
** value significantly less than controls, $P<.001$

Female life expectancies were invariably greater than males. Their superior tolerance to BHT was shown in an LD_{50} of .108% by weight, compared to the male median lethal dose of .0848% BHT.

The gaseous effects of the antioxidant were demonstrated in the vial stoppered with a BHT crystal-packed plug, where total mortality of the emerging flies was inflicted within only two days. The 0.5% BHT vial showed a typical precipitous population loss with 100% mortality within 20 days, whereas the total control population endured well beyond 25 days.

COMMENTS: The demonstrated reduction in mean life span of Drosophila does not necessarily conflict with mammalian studies, previously cited, in which antioxidants, BHT included, exhibited therapeutic effects by lengthening average life. Recent reports employing much lower levels of .01% and .001% BHT by weight produced a slight gain in mean life spans under similar conditions (Félix, et al, 1970). The cumulative conclusion, therefore, is that while high doses of BHT in the medium of Drosophila are toxic, probably as a result of the respiratory effects of the concentrated chemical, minute quantities significantly prolong the normal life span by acting, as Harman has theorized, in a free-radical inhibitory capacity.

References: Félix, R., J. Ramírez, V.M. Salceda and A. de Garay Arellano, 1970 DIS 45:

121-123; Harman, D., 1957 J. Geront. 12: 257-264; Harman, D., 1961 J. Geront. 16: 247-254; Harman, D., 1968 J. Geront. 23: 476-482; Harman, D., 1969 J. Amer. Ger. Sco. 17: 721-735; Kalmus, H., 1943 Amer. Nat. 77: 376-380.

(Supported in part by NSF Grant BO16310-000).

Eiche, A. and K. Fridman. University of Stockholm, Sweden. Variation in the number of ovarioles in Drosophila melanogaster females as a source of error in estimating oocyte stage.

Many experiments have been made with the view of elucidating frequency of lethal induction and the rate of oviposition. The investigations of King, Rubinson and Smith (1956) (1) on oogenesis and its division into 14 successive stages opened new ways for further experiments in this field.

During the last decade much interest has been devoted to the problem of sensitivity to irradiation during stages 7 and 14. When estimating the oocyte stage researchers have, as a rule, based their calculations on the findings of King et al. concerning Oregon-R flies, namely that the number of ovarioles per female is 24 [e.g. Rinehart 1964 (2) and Sankaranarayanan 1969 (3)].

In our tests two lines of a wild type stock, Karsnäs, were used. One of these lines was a non-irradiated control line (C), and the other one (R) was acutely irradiated for a considerable number of generations with 1120 R/generation at larval stage. Females were irradiated with different doses at the age of 4 days (80 R/min., 15 mA, 170 kV) and then the hatching of the first 24 and the following 24 eggs was studied.

Results obtained in our test differ considerably from those obtained by other researchers when irradiating stage 14 or rather the first 24 eggs. As an example may serve the fact that when irradiating with 2000 R we found that hatching in the first laid 24 eggs in the C-line was 29.6 ± 1.9 per cent and in the R-line 21.5 ± 2.5 per cent. This divergence may have several interpretations.

In some experiments females were presumed to have identical number of ovarioles, which appears not to be the case. Robertson (1957) (4) has found that the number of ovarioles can vary considerably between individual females. Great variations also exist as regards the rate of oviposition. Most probably there also exist variations in the sensitivity pattern within the same stage of oogenesis.

In a count of ovarioles with late oocytes in the posterior chambers in the lines used in the hatchability test the following results were obtained (the females were examined after the hatchability test):

	C - line			R - line		
	Range	Mean	n	Range	Mean	n
Series 0 R	16-25	22.8 ± 0.46	25	16-26	22.3 ± 0.44	25
Series 4000 R	15-26	21.0 ± 0.70	18	16-26	20.8 ± 0.55	24
Series 5000 R	16-24	20.8 ± 0.39	24	18-26	21.2 ± 0.45	23

If these findings - namely that in these series 15-26 eggs may be found simultaneously in stage 14 - are not taken into account, it is easy to arrive at misleading conclusions about the sensitivity pattern. The number of eggs should not be limited to 24 for any stage since considerable variations may exist with regard to the number of ovarioles in individual females. Neither should the hour-interval be considered a criterium for the different stages, since individual females have different rates of egglaying.

Thus when estimating the oocyte stage, consideration should be taken to age, rate of egglaying, the dosage and last but not least, to the fact that individual females have different numbers of (functioning) ovarioles.

References: (1) King, R.C., A.C. Rubinson and R.F. Smith, 1956 Growth 20: 121-157; (2) Rinehart, R.R., 1964 Genetics 49: 855-863; (3) Sankaranarayanan, K., 1969 Mutation Res. 7: 357-368; (4) Robertson, F.W., 1957 Jour. Gen. 55: 410-427.

Pasteur, N. and C.D. Kastritsis. University of Texas (Southwestern) Medical School at Dallas, Texas. Electrophoretic general protein patterns during development of Drosophila pseudoobscura.

If we follow the method of synchronization of Stocker and Jackson (this issue), we find that Drosophila pseudoobscura larvae (stock from Mather, California) go through their first and second molt about 24 and 72 hours after oviposition, while spiracle eversion occurs (on the average) at 165 hours after oviposition.

Samples of different ages were collected and homogenized according to the method of Johnson (1966). We used 500 eggs or first-instar larvae, 100 second-instar larvae, 30 early third-instar larvae, 20 medium third-instar larvae, 10 late third-instar larvae, prepupae or pupae, 15 24-hour, 7-day or 25-day old adults. These were homogenized in 15µl of deionized water in each case. We employed 7% separating acrylamide gels, and used a Canalco Model 1200 disc electrophoresis apparatus. The procedure was carried out in the cold with a buffer of pH = 9.5 at a constant current (4mA per tube) and until the tracking-dye (Bromophenol blue) reached 1 cm from the lower end of the tube. The gels were stained in 7% aniline-blue black for 90 to 120 mn and destained in 7% acetic acid in a Canalco quick gel destainer. Split gels were also used to allow better comparisons between stages as described by Spiegel et al.(1970).

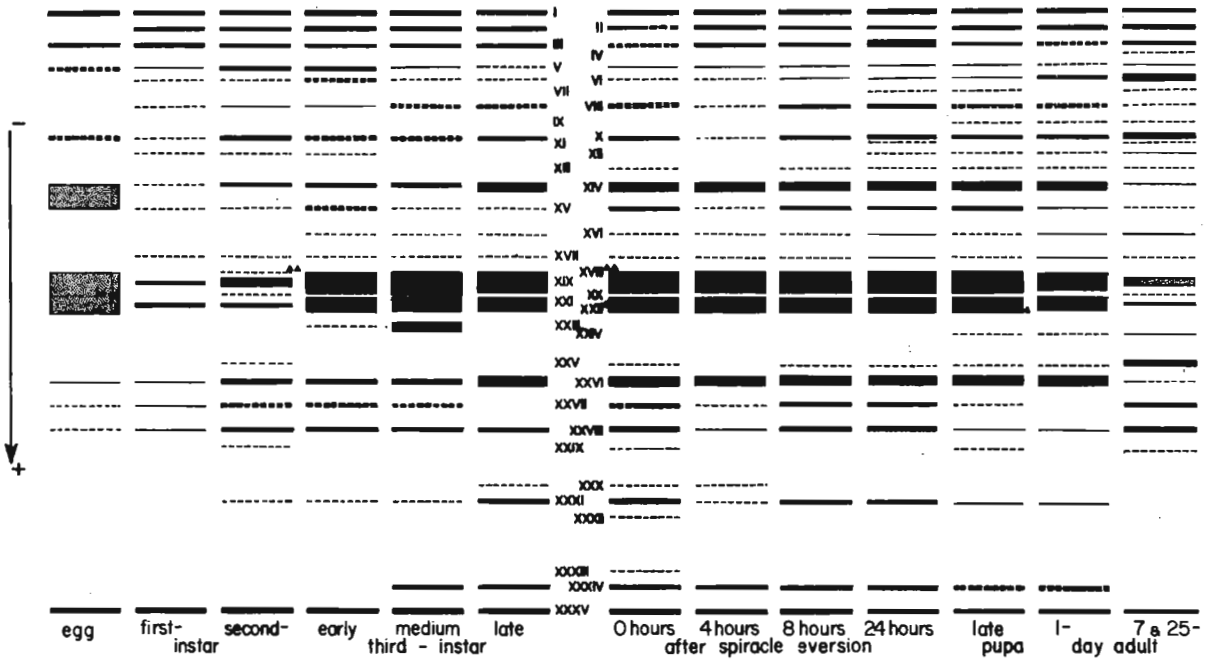


Figure 1. Summary of results concerning the general protein patterns of various developmental stages of Drosophila pseudoobscura. Interrupted thin or thick bands = bands not found in all electrophoretic runs of a particular stage. Stippled bands = diffuse bands. Dark bands of various thickness = bands present in all samples. Roman numerals = band number. XVIII▲▲ and XXII▲ refer to the bands bearing two or one triangles.

A total of 35 bands can be identified from one stage or the other. Among these bands about one-half do not show any change (quantitative or qualitative) regardless of developmental stage. The others show either qualitative or quantitative changes (bands XXIII and XIX or XXI respectively are characteristic).

References: Johnson, F.M., 1966 D.I.S. 41: 193-194; Spiegel, M., E.S. Spiegel and P.S. Meltzer, 1970 Develop. Biol. 21: 73-85.

Frías L., D. and Lamborot CH., M.
University of Chile, Santiago, Chile.

Mating activity, sexual isolation and temperature in the "yellow", "white" and wild type strains of D. gaucha.

The object of the present work was to study the effect of temperature on mating activity and sexual isolation in two mutant strains of D. gaucha, "white" and "yellow" in relation to a heterogeneous wild type stock from which the mutants arose spontaneously a few years ago (Iturra, P.D.I.S. 45, 1970).

Sexual activity and isolation was analyzed by direct observation for a period of two hours in mating chambers designed by Ellens and Wattiaux (DIS 39: 118, 1964). 15 wild type pairs were placed in one chamber with 15 males and 15 females of either the "yellow" or the "white" strains. The experiments were performed under uniform illumination, at 16°C and 25°C.

The results from the study of 40 observations of each type (600 pairs) are summarized in the Table. It shows the number of matings, and the statistical significance of the differences in mating of different types at both temperatures.

In general, the wild type males are the most active at both temperatures, as they inseminate more females than any of the mutant males. "White" males are very little active at 16°C and 25°C, both in relation to their own, as well as to the wild type females. On the other hand, "white" females are highly receptive at both temperatures. The receptivity of "yellow" and wild type females is increased significantly at higher temperatures.

	Temperature				
	16°C		25°C		16/25°C
Type of cross	Total no. of matings	χ^2	Total no. of matings	χ^2	χ^2
♂ n x ♀ n	57	12.668+	104	0.367	17.962+
♂ n x ♀ w	96		96		0.007
♂ w x ♀ n	4	0.671	4	0.126	0.126
♂ w x ♀ w	2		4		0.168
♂ n x ♀ n	52	0.877	101	0.000	20.209+
♂ n x ♀ y	62		102		12.786+
♂ y x ♀ n	30	0.157	52	0.047	2.705
♂ y x ♀ y	34		49		6.229++

n = wild type w = white y = yellow + = 0.001 ++ = 0.02-0.01

As has been described for other species, the activity of males and the receptivity of females are in inverse relationship, a fact that is specially noteworthy in the low activity of the "white" males and great receptivity of the "white" females. These differences must be attributed mainly to the mutation, as the genetic background in the three strains is the same.

The behavioral changes of the "white" and "yellow" mutations in D. gaucha follow a similar pattern to those described for other phylogenetically quite distant species, such as D. melanogaster (Bastook, M., 1956 Evolution 10: 421-439).

The mechanisms by which these mutations modify sexual behavior and discrimination in relation to temperature, must be analyzed in the future.

Valentin, J. University of Stockholm, Sweden.

How much crossing-over occurs within heterozygous inversions?

This has been tested by Sturtevant and Beadle 1936 and by Novitski and Braver 1954, and using X chromosome inversions these authors find a general decrease of crossing-over within the loop (especially near breakpoints). On the other hand, heterozygous inversions can enhance recombination, not only in heterologous bivalents but also often in the inversion chromosome outside the inversion. We wondered whether under particularly suitable conditions, this Schultz-Redfield effect might even act in the middle of an inversion loop. Observation of such intra-inversion effects would have a bearing on hypotheses about the cause of the Schultz-Redfield effect. Such suitable conditions might be expected with In(2LR)Gla, a reasonably symmetric pericentric inversion, superimposed on an

In(2L)Cy lacking the Cy gene. This inversion has a considerable Schultz-Redfield effect on X, and the centromere region, usually the most sensitive part of a chromosome in this sense, lies in the middle of it. Indeed Alexander (Univ. Texas Publ. 5204: 219) has already checked the amount of double crossing-over within In(2LR)Gla, and found none among 2125 offspring. However, at the time of her experiment it was not known that Gla overlaps In(2L)Cy. Instead, it was regarded as a two-break arrangement, and thus she used an unsuitable marker stock.

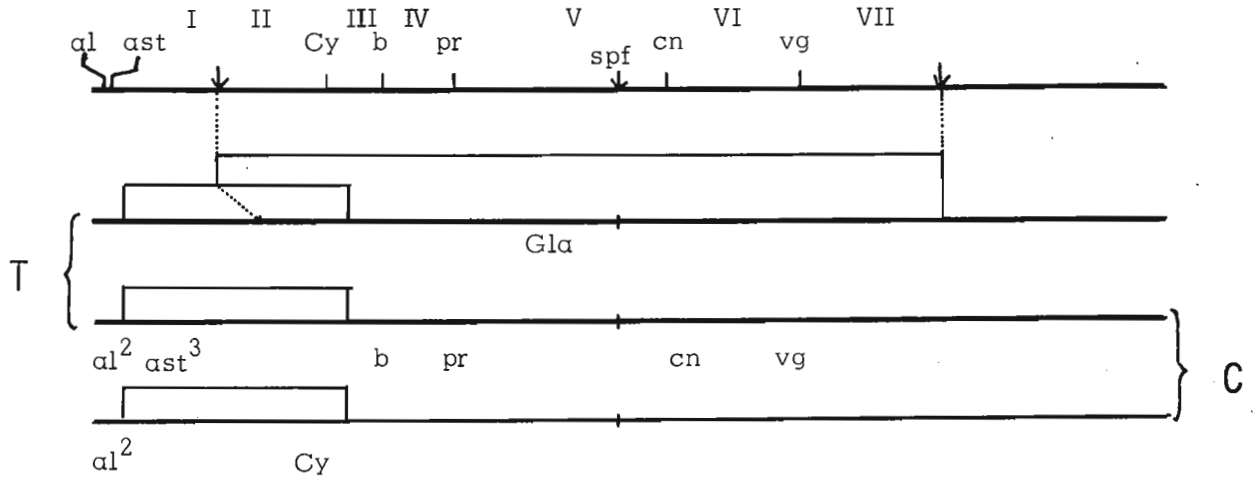


Fig. 1. Approximate salivary distribution of markers and rearrangements, markers spaced as in In(2L)Cy chromosome. T = test female chromosomes, C = control female chromosomes. I - VII = regions where crossing-over was scored.

% recombination in structurally homozygous regions

	I	II	III	IV	V	VI	N counted
Control series	30.0		14.6	7.0	0.6	7.6	1488
Gla series	1.4	-	-	-	-	-	1390

Double recombinants (triples excluded)

	I-II,III	I-II,IV	I-II,VI	III,IV	III,V	III,VI	IV, V	IV,VI	V,VI	
Control series	15	16	27	15	1	14	1	9	1	
Gla series				1				1		6
				II-III,IV				IV,VI		VI,VII

We therefore felt it was worth while to repeat her interesting experiment, but our marker stock contained In(2L)Cy - which was thus rendered homozygous, so that a large single loop could be formed in meiotic prophase. The tested females carried In(2LR)Gla/In(2L)Cy, al^2 ast^3 b pr cn vg, and the control consisted of full sibs to these, carrying In(2L)Cy, al^2 Cy/In (2L)Cy, al^2 ast^3 b pr cn vg, as shown in Fig. 1. Note that in the Gla series, intervals II + III are scored as one region; in the control series I + II are similarly lumped.

The result, as shown in the table, was felt unsuitable for treatment by common statistical methods, but it is evident at sight that no recombination enhancement of the kind envisaged above occurs. The number of VI, VII doubles in the Gla series is high considering the small physical size of region VII, but since all 6 animals appeared in one brood from one vial, they may be the result of a gonial event. Being negative, the result does not entirely rule out the possibility of intra-inversion Schultz-Redfield effects, but it seems probable that the general depression of crossing-over observed by older authors is indeed universal with heterozygous inversions.

Moyer, S.E. and D. Burton. Northeastern University, Boston, Massachusetts.

Incomplete dominance in hybrids from D. melanogaster adapted to DDT or NaCl.

Strains have been selected for adaptation to extreme environments, DDT or NaCl in the food. An ebony (e) strain is adapted to .60g DDT/100cc H_2O of a commercial 50% wettable powder. A vestigal (vg) strain is adapted to 8% NaCl. "Instant medium" (Carolina) is used by adding H_2O or a specified solution of DDT or NaCl to an equal volume of dry medium. Each strain is intolerable to the maximal stress environment of the other strain but grows well on normal food. There is no obvious effect of DDT in surviving e flies. The vg flies are very small when subjected to 8% NaCl and the wings have become much longer than non-adapted vg at any temperature or salinity of food. Disposable culture containers, "cartons" and "cups" used for this work are described in Moyer and Yarbrough (1969).

In this study, virgin e and vg females were collected within the first two days from normal food cultures. After ageing for two or three days mating chambers (paper cartons containing only a small cup of food) were employed for three mating groups: 1. 24 e couples and 24 vg couples; 2. 10 e couples and 40 vg couples; 3. 40 e couples and 10 vg couples.

After 24 hours, the e and vg females were transferred separately (without males) to "egg collection chambers". Eggs were collected on an agar-acetic acid-alcohol mixture (Delcour, 1969) in a plastic "cup" cover with a thick yeast suspension on the surface. Egg laying caps were changed daily. Eggs and the agar from the cap were transferred to half pint paper cartons containing medium with the desired concentration of DDT or NaCl. Preliminary results in the table show the proportions of wild type progeny which resulted from e (DDT) x vg (NaCl) matings as detected from the first brood of eggs on normal food, in relation to females mated by males of their own strain. Of course, the proportion of heterogamic matings was influenced by competitive disadvantages of mutants and the proportions of parents of each strain.

In any case, the most striking consequence is the very low proportion of heterozygotes which survive on DDT in contrast to ebony progeny from DDT adapted parents. Furthermore the body size of these wild types was much smaller than e progeny.

Survival of progeny in various environments.

Daily Brood	Food	Progeny of 24 e ♀♀ + ♂♂ / 24 vg ♀♀ + ♂♂			Progeny of 10 e ♀♀ + ♂♂ / 40 vg ♀♀ + ♂♂			Progeny of 40 e ♀♀ + ♂♂ / 10 vg ♀♀ + ♂♂		
Separated e ♀♀ (after mating)		e	+	vg	e	+	vg	e	+	vg
1	Normal	96	221		14	70		138	87	
2	.35 DDT	115	5		26	7		222	0	
3	.45 DDT	109	11		10	14		99	1	
4	.30 DDT	150	25		23	12		246	16	
5	.40 DDT	152	5		30	13		213	21	
Separated vg ♀♀ (after mating)										
1	Normal		165	244		130	283		205	106
2	6 NaCl		56	79		63	108		47	24
3	8 NaCl		8	4		2	3		1	1
4	5 NaCl		59	34		89	187		65	64
5	7 NaCl		28	31		14	20		21	23

Less clear, so far, is the viability of hybrids on NaCl compared to vg salt adapted progeny in the same cultures.

References: Delcour, J., 1969 DIS 44: 133; Moyer and Yarbrough, 1969 Am. Biol. Teacher 31: 593-596.

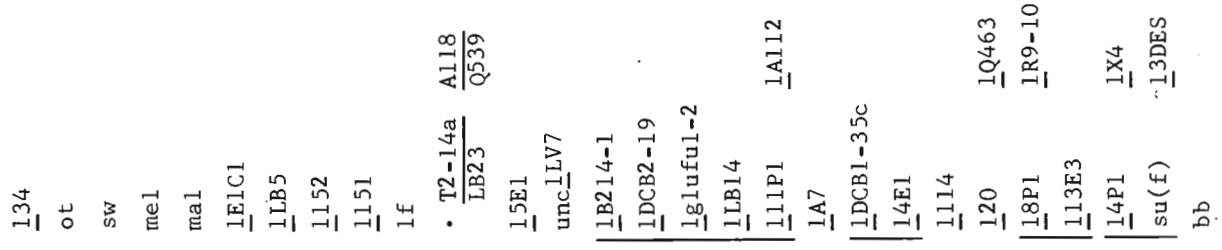
Schalet, A.* and K. Singer. University of Connecticut, Storrs, Connecticut.

A revised map of genes in the proximal region of the X chromosome of Drosophila melanogaster.

Following the initial report that a Y chromosome ($y^+Ymal^+No.2$) with a duplication for the proximal region of the X chromosome covered at least 10 genes located between M(1)n and bb (Schalet, 1963), two earlier maps of this

region have been presented (Schalet and Finnerty, 1968; Chovnik, Finnerty, Schalet and Duck, 1969). The present map adds 12 new sites, indicated by ' , to the one in Chovnick et al. The sites connected by ___ have not been ordered with respect to their left-right positions.

To the corrections and additions given in DIS 44 and 45 add: 1) The 1N5 earlier positioned to the left of 1152 is an allele of 1151. This error was probably due to a stock erroneously labled Df(1)mal^{8}. Subsequent retesting of lethals between mal and lf with one another and with cytologically analyzed deficiencies including mal^{8} has produced the order



shown here. 2) The site indicated by a • represents the visible phenotype displayed by ♀♀ heterozygous for the deficiencies T2-14a/LB23 as well as A118/Q539 and the two other appropriate combinations of these lethals. 3) Since 122 had been lost, it has been eliminated from the map because its relation to the new lethals between 120 and su(f) could not be tested.

The following features of the region may be noted: 1) We have only listed here those mutants that have thus far proved to involve single functional units. We have additional alleles of some of these mutants that also appear to involve single functional units. 2) As reported earlier (Schalet, Lefevre and Singer, 1970), 1A7 and the mutants to the right are in salivary division 20. 3) With one possible exception, su(f) is placed immediately to the left of bb. In consideration of some interesting characteristics associated with this locus, that are being reported elsewhere, this position may not be fortuitous. These attributes permit the speculation that su(f) is an element in the protein synthesizing mechanism. 4) All 16 genetically analyzed deficiencies, with a right break-point apparently between su(f) and bb and a left break-point extending different lengths to the left, have proved to be ♂ fertile. In our own material and from the literature it appears that deficiencies extending from the right or left into the bb locus are usually ♂ sterile. We have tested 14 such deficiencies and 10 were ♂ sterile. Since these deficiencies were not carefully analyzed as to whether the bb locus was completely or only partially removed no further comment is warranted. However, we are aware that ♂♂ with the bb deficient sc^{4}-sc^{8} chromosome are fertile.

Above the main map we have listed a number of lethals obtained from Dr. Raphael Falk. The positions given are in accord with tests for allelism with the mutants listed below them and supported by analysis with our duplications and deficiencies. Lethal 3DES behaves as an allele of su(f) with respect to forked suppression but does not show the phenotype associated with the combination su(f)/su(f)$^{-}$. It has not been tested with bb yet. The following comments apply to the complementation map presented by Lifschytz and Falk, Mutation Research 1969. 1) The entire map lies to the right of mal. Section number 1 lies between 1E1C1 and 1152. 2) The "hot spot", section 17, as well as the "hot spot", section 18, for X-ray induced breaks attributed to intercalary heterochromatin, is in salivary division 20, probably 20A1-2. We have observed these "not spots" in our own material. 3) We note the disagreement with their map in respect to the 4 right lethals listed above the map presented here. The relative positions and section numbers on their map are: X4 (27), 3DES (29), R9-10 (31-32), Q463 (34). We further note the crucial position occupied by Q463 in their analysis of recombination data for the proximal region (Lifschytz and Falk, 1970). That the alleles of Q463 and 3DES, 120 and su(f) respectively, are positioned 120 left and su(f) right, is supported by a large scale recombination experiment. This experiment, which obtained evidence that crossing over at the bb locus in a normal ordered chromosome could generate bb mutants, utilized 120 and su(f). All 9 fertile recombinants bwteen 120 and su(f) proved to be recombinant for the flanking markers mal^{1} and y^{+} of the sc^{V1} duplication.

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Moyer, S.E. and S.P. Stepak. Northeastern University, Boston, Massachusetts. Inheritance of trident and its role in detecting ebony heterozygotes in D. melanogaster.

Bridges and Morgan (1923, Carnegie Inst. #327) describe some experiments conducted by Morgan during 1910-11 on inheritance of gradations of the trident pattern which he named "with" or "super-with". They could not clearly judge whether the trait was inherited as a dominant or a recessive. The mutant tri is credited to Plough and Ives (1934) by Lindsley and Grell (1967) and is described as "semidominant" on chromosome II.

The strains used in the present study were: 1. Nearly non-trident: very low frequency of tridents for at least two years. Homozygous for vestigial wing (vg) and resistant to 8% NaCl in the medium (vg S)

2. Partially trident: Moderate frequency of tridents. Homozygous vg and maintained on normal medium (vg N)

3. Ebony: Homozygous for ebony (e) and resistant to .70g/100cc H_2O of commercial 50% wettable DDT powder in the medium (e D)

All matings were single pairs, unless specified otherwise. Scoring of pigmentation was made after newly emerged flies were aged for at least one day. Temperature was controlled at about 21°C.

Matings of non-trident x non-trident produced different results depending on whether parents were taken from the vg S or vg N stock. F_1 progeny were all non-trident if both parents were from vg S. If one or both parents were from vg N, the F_1 progeny were mostly a darker class of non-trident plus an appreciable frequency of tridents. F_2 samples resulted in an increased frequency of tridents, presumably due to new combinations of modifiers. In a notable number of rare cases when one phenotypically non-trident parent was taken from vg N, nearly equal numbers of trident and non-trident F_1 progeny were produced. That is, these pairs seem to be a testcross of an impenetrant dominant factor for trident of the type Tt x tt.

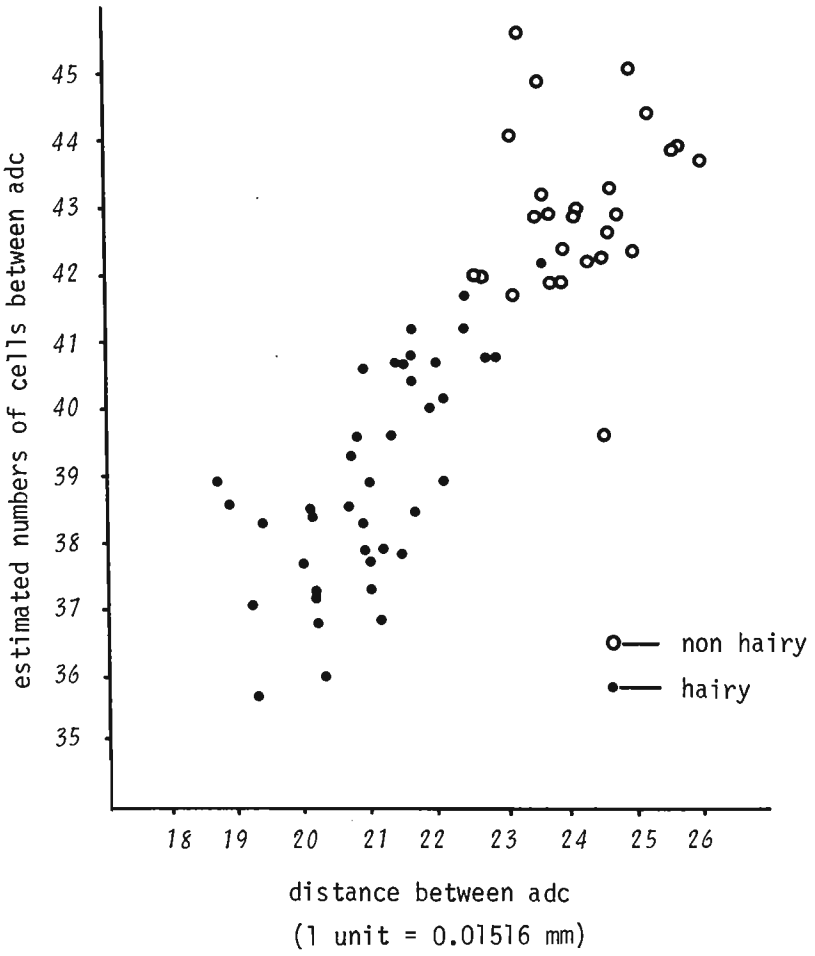
Matings of trident x non-trident produced mostly two types of F_1 results as if it depended on whether the trident parent was homozygous or heterozygous for a dominant factor for trident. However, a general conclusion of the inheritance of trident probably depends on the source of the material.

The main purpose of this study was to determine whether the trident factor is expressed independently of a dark marking on the thorax due to slight partial dominance of ebony in heterozygotes. Mass crosses were made with 100 pairs of parents between e D x vg S. All vg S parents were carefully examined to select light non-tridents. In the F_2, random samples of non-ebony males were scored for trident type and testcrossed singly with ebony females. One-third of the F_2 males tested (54/167) were non-trident and 2/3 were trident, as might be expected. Only seven of the 54 non-trident were heterozygous for ebony and two of the trident males were, in fact, not heterozygous for ebony. If trident was a second chromosome dominant, crosses between trident-bearing ebony and true non-trident, non-ebony could produce an F_2 ratio among the non-ebony progeny of 1/12 non-trident : 11/12 trident. This apparently is not the case in our material.

An immediate application of the above result is the general use of the trident marking for detecting heterozygotes of ebony in estimation of gene frequency. In the F_3 and F_4, there was also good agreement between trident scores and test cross results from populations cultured on normal food. Replicate populations cultured in food having a concentration of .30g. DDT/100cc had a selective disadvantage against genotypes lacking e-bearing chromosomes. Again, tridents were good indicators of heterozygotes and non-tridents were good indicators of non-e homozygotes. In this sense, the trident mark may be at least as useful for detecting ebony heterozygotes as electrophoretic techniques for detecting isozyme genotypes.

Claxton, J.H. University of New England, Armidale, N.S.W., Australia. Cell numbers and acrostichal row numbers in hairy and non-hairy flies.

D. melanogaster normally has eight acrostichal rows of microchaetes running longitudinally between the anterior dorsocentral bristles (adc) on the dorsal mesothorax. It has been suggested that the hairy wing (Hw) mutant, which, among numerous effects, adds extra acrostichal rows, achieves this result by providing extra space near the mid dorsal line, thereby permitting "enlargement of the prepattern (determining) the distribution of chaete for this new



region" (Gottlieb, 1964). This followed the discovery that Hw is partially non-autonomous in Hw mosaics. It would seem reasonable to hypothesize, therefore, that this is a type of secondary non-autonomy whereby an undisturbed prepattern determining mechanism operating over an enlarged field, produces a greater-than-normal number of prepattern elements, each corresponding with an acrostichal row.

Hairy (h) mutants which also commonly have one extra acrostichal row on both sides of the mid dorsal line, provided a simple check on the generality of this hypothesis. In small samples of hairy and non-hairy flies, the numbers of hypodermal cells between adc were estimated. For this purpose, the numbers of cell hairs (and, therefore, cells) were counted in a narrow rectangular area which stretched between the adc, and which had a width 1/10 of its length. Making the assumption that all hypodermal cells are regular hexagons, and that all fit together with no spaces between, it was calculated that the square root of the product (8.66 x number of cells counted) was the maximum number of cells which could lie in a straight line between adc.

With these methods the average estimated numbers of cells between adc was 39.00 (s.d. = ±1.64) in hairy mutants (N = 40), and 42.99 (s.d. = ±1.26) in non-hairy flies (N = 26). From these results there is clearly no evidence that the hairy mutants which had extra acrostichal rows, also had larger than normal numbers of cells between adc.

Generally those hairy flies examined were smaller than the non-hairy individuals. The differences in cell numbers between adc appear to be strongly related to body size. Combining results for both mutant and non-mutant flies, and using (a) the distance between adc, and (b) length x breadth of the scutellum, the numbers of cells between adc was positively correlated with both these parameters [(a) r = +0.86, (b) r = +0.65]. Further, it is apparent (Figure I) that a single regression line relates cell number between adc and distance between adc for both the hairy and non-hairy samples. In these samples then, the numbers of cells between adc has no determinitive influence on the hairy/non-hairy difference in acrostichal row number.

This preliminary comparison anticipates a study of the autonomy (or otherwise) of hairy (with respect to extra acrostichal rows) in non-hairy/hairy mosaics. In this context, the foregoing conclusion might also have important interpretive significance.

Reference: Gottlieb, F.J., 1964 Genetics 49: 739-760.

Alonso, C. University of Nijmegen, The Netherlands. The effect of gibberellic acid on the development of D. hydei.

The effect of injection into larvae of D. hydei of various concentrations of gibberellic acid (GA_3) on the chromosomal puffing pattern, the moment of puparium formation and the number of flies emerging from pupae was investigated.

It was observed that GA_3 can induce a new puff, 4-72B, in 15% of the larvae injected at 140 hours following oviposition. This puff can be observed 9 - 10 hours after injection. If CA_3 is injected at 153 h. a new puff develops within 3 - 4 hours at region 2-21B. These puffs have never been observed in normal development and are absent in animals injected with the solvent (Ringer's). The frequency with which puff 2-21B occurs depends on the concentration GA_3 applied, but never reaches a 100%. Apart from the newly induced puffs, GA_3 appears to affect the occurrence and size of puffs which are characteristic for the period prior to puparium formation. Injection of 1.5 µg/larva delays the appearance of the ecdysone-specific puff 4-78B and inhibits its full development. The same applies for puff 4-77BC. Mixtures of ecdysone and GA_3 injected into intermoult larvae gave a similar effect, a delayed development of the puffs 4-78B, and 4-77BC as compared with larvae injected with ecdysone only (2.10^{-1} µg/larva).

Also puparium formation was delayed if GA_3 was injected at 153 h. or later (pup. formation occurs normally around 160 h.). The delay was increased with increasing concentration of GA_3.

The percentage of flies emerging from pupae developed from GA_3 injected larvae decreased considerably with increasing GA_3 concentration. Injection of 6 µg/larva resulted in 54% lethality.

Schalet, A.* University of Connecticut, Storrs, Connecticut. Two modified crossover-selector systems of general application to fine structure analysis.

Scheme 1 has been used in a fine structure analysis of the vermilion locus, but is applicable to other X-linked and autosomal loci. For the v locus a cross of the following type was used:

♀♀ (A) $\frac{l1 + v^x\ l3 + X}{(B) + l2\ v^y + l4}$ ♂♂ (C) $\frac{+\ l2\ v\ l3 +}{(D) - + v + - Y}$; bw^D

From this cross the only ♂♂ that survive are 1/2 of the crossovers between the outside lethals, l1 & l4. The only ♀♀ that survive, aside from non-disjunctionally produced individuals, are 1/2 of the crossovers between the inside lethals, l2 & l3. If v^x is located to the left of v^y, then v^+ recombinant ♂♂ will survive. If v^x is located to the right of v^y, then v^+ recombinant ♀♀ will survive.

Chromosome (B) was introduced into the cross thru parental ♂♂ carrying a Y chromosome that covered the region from l2 thru l4 (Schalet DIS 44: 123). This chromosome, as well as chromosome (D), was derived from the v^+Y y^+ chromosomes synthesized by Chovnick, DIS 43: 170. The presence of the y^+ region on the Y, chromosome (D), with its Hw effect, and the partial suppression of the bw^D phenotype in ♀♀ carrying a Y chromosome, permitted the detection of XXY ♀♀. Such ♀♀ appeared at an estimated rate of 1 for every 1,500 regularly produced zygotes. Linkage relationships determined from other crosses were as follows: ras--l1, 0.1; l1--l2, 0.4; l2--v, 0.2; v--l3, 0.7; l3--l4, 2.0. Note that the value of 0.7 for the interval between ras and v is closer to the value of 0.59 reported by Lefevre, DIS 45: 40, than the standard value of 0.2.

In the table below the total number of zygotes sampled has been calculated on the basis that each regularly produced ♀ represented approximately 1/444 of the number of eggs laid, (2/1,000 & 7/1,000)/4. Although only 1/2 of the eggs laid represent sampled chromosomes, this scheme provides the advantage that for any two potentially separable alleles, whatever their left-right orientation, only a single cross is required. Consequently, each allele need be inserted in or induced on only one of the two types of lethal bearing chromosomes.

Test	v^+/Total	Order	Test	v^+/Total	Order
2/1	0/307,000		1/E1	1♂/330,000	1-E1
1/k	0/890,000		36f/E1	1♀/460,000	E1-36f
36f/65c	0/250,000		2/E1	3♂♂/167,000	2-E1
1/36f	(Green)	1-36f	36f/2	1♀/195,000	2-36f
48a/36f	(Barish & Fox)	48a-36f	36f/k	2♀♀/350,000	k-36f

All v^+ individuals were fertile and the recombinant chromosomes proved to carry v^+ and the appropriate non-selective lethal markers, except that the 3♂♂ from 2/El were not tested for l3. However, all 3 chromosomes did carry dy which was derived from chromosome (A) of the parental ♀♀. The recovery of v^+ individuals in crosses 1/El and 36f/El indicates that there are 3 separable sites in the order 1-El-36f. The El allele was indiced by EMS and is unsuppressed by su^{51c15}-v. Note that the data in the table present additional examples of the separability of unsuppressed alleles, 36f/El, and the separability of suppressed and unsuppressed alleles, 1/El, 2/El, 2/36f and k/36f. The separability of the left-positioned suppressible alleles, 1, 2 and k has not been achieved.

Scheme 2 was originally devised for a fine structure analysis of the maroon-like locus, but was abandoned in favor of Glassman's purine selector system. The basic scheme is not only applicable to sex-linked visible loci, but an important variation may also be used for any sex-linked lethal locus where an analysis is not facilitated by the existence of temperature sensitive mutants. The basic scheme has recently been utilized by Finnerty and Chovnick (Genetical Research 1970) to recover maroonlike double mutant recombinants. For any visible mutant, m, consider a cross of the type:

$$♀♀ \frac{l1\ m^x\ +}{+\ m^y\ l2}\ ;\ \frac{\text{Ins, Sb or Ubx}}{l(3)26,\ \text{Sb or Ubx}}\ X\ ♂♂\ T(1;3),\ \text{cu kar ry}^{26}\ l(3)26\ \text{Sb Ubx}$$

The translocation used was one selected for good viability and fertility from among a number of X-ray-induced rearrangements between a wild-type X chromosome and a third chromosome carrying the indicated markers. In addition to the zygotic lethality produced by paternally derived aneuploid chromosomal combinations, all ♀ euploid zygotes, aside from those produced by maternal X chromosome non-disjunction, die because of the lethality associated with homozygosity for l(3)26, Sb or Ubx. All euploid ♂ zygotes die except those derived from a lethal-free chromosome produced by crossovers, including those between m^x and m^y, in the small region between the tightly linked lethal markers. For loci in which the double mutant cannot be immediately recognized, it is necessary, at least in the initial stages of the analysis, to make a companion cross in which the distribution of the X chromosome markers is l1 m^y +/+ m^x l2. In addition to the availability of 2 closely linked lethals, it is of course necessary to have a duplication, preferably on the Y, for the region that covers at least one of the lethals in order to introduce into the cross one of the tested alleles thru the ♂ line.

Welshons and Von Halle (Genetics 1962) have used a very effective selector system for the Notch locus to separate alleles that behaved as non-complementary recessive lethals. Their scheme, essentially: ♀♀ N^x/N^y;Ins, DpN^+ l1 l2/+ X ♂♂ fa^{no}/Y;l1/l2, was facilitated because the viable fa^{no} was lethal in the combination fa^{no}/N. A variation of scheme 2 can be applied to any sex-linked lethal locus, m, as follows: ♀♀ m^x/m^y;Dpm^+ Ly Sb/In(3)Ubx^{130} X ♂♂ T(1;3), cu kar ry^{26} l(3)26 Sb Ubx/Ly. Aside from the sex-linked lethal alleles being tested, there are two new elements added to scheme 2. The dominant mutant Lyra behaves as a recessive lethal. The Ly Sb third chromosome carries a duplication for the region of interest. In principle, the production and detection of such a duplication should offer no problems. Again ♀♀ die as in the basic scheme 2, and all ♂♂ with the Dpm^+ chromosome die because they will be homozygous for Ly. The only survivors of the cross will be ♂♂ derived from a crossover, conversion or back-mutation event at the m locus.

Finally, it is recognized that the type of scheme just discussed may be dispensed with, for the most part, where the expense of the purine selector system is not a prime consideration. Since purine will kill maroonlike and rosy mutants, and mal^+ Y chromosomes are available, the following type of selector system is possible for a sex-linked lethal locus: ♀♀ m^x mal/m^y mal;Dpm^+ ry/In(3)Ubx^{130} X ♂♂ mal/mal^+ Y;ry. However, it should also be emphasized that further, relatively simple variations of scheme 2 alone, or in combination with the purine system, may be used for a fine structure analysis of autosomal lethal loci.

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Kastritsis, C.D. University of Texas (Southwestern) Medical School at Dallas, Texas. "Micronucleoli" in D. paulistorum?

During the continuing investigation of the cytology of strains belonging to the Interior semispecies of D. paulistorum it was discovered that, quite frequently, one sees certain structures (fig. 1) which strongly resemble the nucleoli of the salivary gland cells. These structures can be seen both in the strains of the Interior, and in their hybrids with the strain of the Andean semispecies which is used as a "standard"; the latter strain does not exhibit similar structures. Tentatively, we call these structures "micronucleoli" but this is to be taken as an indication of morphological similarity with the nucleolus rather than anything else.

The "micronucleoli" are found to occupy (very frequently) a position in the chromocentric heterochromatin (fig. 2). The number of such structures varies, and we have found as many as three both inside or outside the heterochromatic mass.

When free, the "micronucleoli" are connected with the heterochromatin with strands of material of an unknown nature (arrows in fig. 1). It is not known whether the release of the "micronucleoli" from the heterochromatin is a stage-dependent phenomenon or strictly the result of the application of pressure during the preparation of the slides.

The structures described here have been found both in male and female individuals at the late third instar or spiracle eversion stages of development.

At this time, it cannot be determined whether or not the structures described here

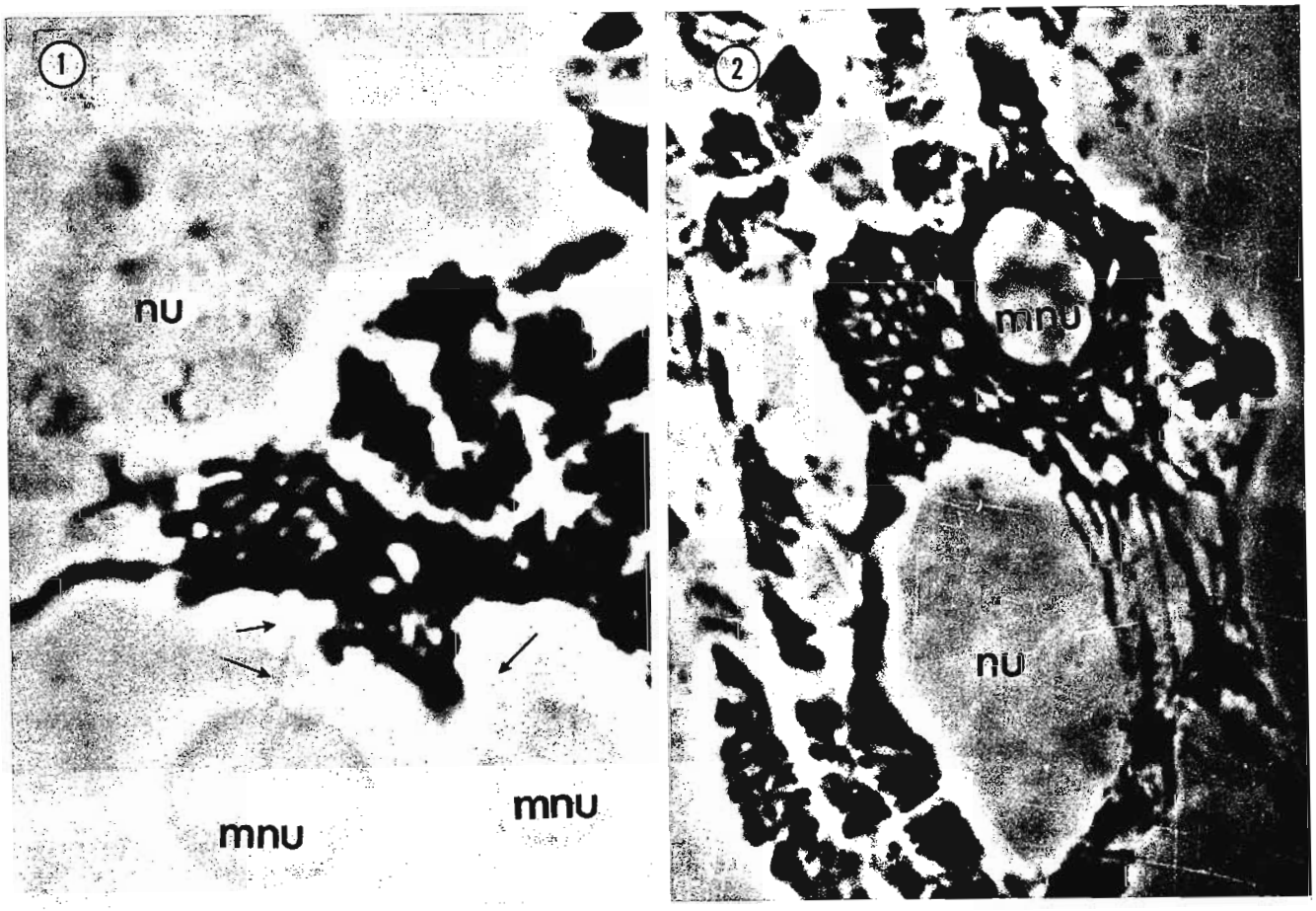


Fig. 1. Phase contrast photomicrograph from a salivary gland cell of a strain of the Interior semispecies of D. paulistorum. Nucleolus (nu). "Micronucleolus" (mnu). Arrows point at the connections of the "micronucleoli" with the heterochromatic mass.

Fig. 2. Phase contrast photomicrograph from a salivary gland cell of a strain of the Interior semispecies of D. paulistorum. Nucleolus (nu). "Micronucleolus" (mnu).

resemble those described by Da Cunha et al. (1967) in their paper on chromosomal diseases of D. willistoni. In no case were these structures found in or near any parts of the chromosomes other than the chromocentric heterochromatin.

Electron microscopic and autoradiographic experiments are being planned to determine the exact nature of the "micronucleoli."

Work supported by grant no. GM 16736-02 from the National Institutes of Health.

References: Da Cunha, A.B., Z.M. Franca, A.M. Amaral Goncalves, A. Hitelman and M. Garrido, 1967 Rev. Brasil. Biol. 27 (2): 113-124.

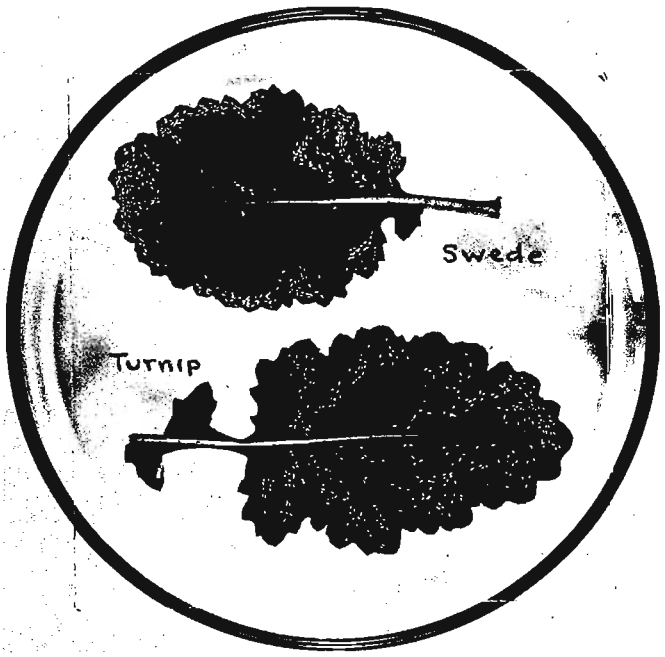
Basden, E.B. Institute of Animal Genetics, Edinburgh, Scotland.

Feeding by Scaptomyza flava.

Females of Scaptomyza flava (Fln) (= apicalis Hardy = flaveola auctt) make feeding punctures in the leaves of the cruciferous and other plants in which their larvae mine. The procedure is to press the truncate end of the toothed ovipositor-guide against the leaf surface, then strongly scrape sideways until the cells are ruptured. The puncture is then enlarged by the stronger spines along ventral edge of the guide. The immediate vicinity of the puncture becomes wet and sappy, and the female feeds at this.

Scraping takes from about 16 seconds to 1 minute, and feeding lasts 30 to 40 seconds. Eggs may or may not be inserted in the punctures.

These feeding punctures are shown in the photograph (x 0.9) of young leaves of turnip and swede from plants kept in cages with adult flava.



Adult Agromyzidae, whose larvae are also leaf-miners, perform the same feeding behaviour, and it reminds one of the feeding by adult parasitic Hymenoptera at oozing punctures in their hosts.

The other photograph (by G.R. Knight) shows larval mines of Scaptomyza flava in leaves of swedes growing as a crop at Dunglass, East Lothian, Scotland. Dunglass is the original locality of J. Hardy's new species, Sc. apicalis (1848, 1849), now considered to be the same species as Sc. flava, and it still persists there also in Cochlearia on the coast. John Curtis in his "Farm Insects" (1860:92) was the first to point out that the mine was always in the upper surface of the leaf, whereas that of Phytomyza (Dipt., Agromyzidae) was on the under side of the same host plant.

Rodinò, E. and A. Martini. University of Padua, Italy. Est 6 V: a new allele at the Est 6 locus in natural populations of D. melanogaster.

The existence in populations of D. melanogaster of a protein polymorphism involving two forms of a non-specific esterase was first established by Wright (1963). By means of electrophoresis it was possible to separate two forms of this enzyme called Est 6 Slow and Est 6 Fast for their different mobilities on starch gel. The inheritance of the two forms was found to be controlled by a pair of co-dominant alleles. A third allele of the same locus was then detected (Wright and MacIntyre, 1965). The third allele, Est 6 F2, has the same electrophoretic mobility as Est 6 F, but differs in producing a heat-stable form of enzyme, vs. a heat-labile form controlled by Est 6 F.

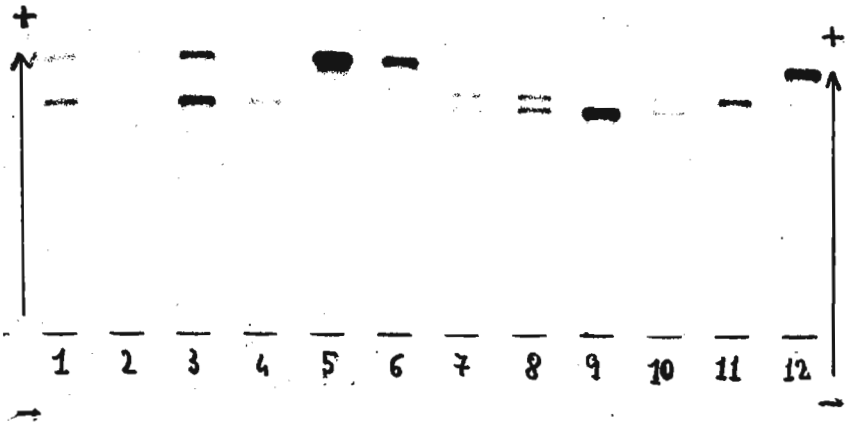


Figure 1. Vertical 5% polyacrylamide gel slab stained for Esterase (Tris, EDTA, borate buffer, pH 9.1)

Gel pockets	2, 5, 6, 12:	homozygotes	V/V
" "	4, 9	"	S/S
" "	7, 8, 10	heterozygotes	S/F
" "	1, 3	"	S/V
" "	11	homozygote	F/F

In this research note we want to report on another allelic form of Esterase 6 found in natural populations of D. melanogaster in North-East Italy. This new form has been called by us Est 6 V (V for Very fast), having a much greater electrophoretic mobility than Est 6 F (see Fig. 1).

In the course of our work we have made the assumption that the two esterase called by us S and F, are in fact homologous with those described by Wright.

The new allele was found in four populations, each founded with twelve females caught inseminated in the wild near Verona in the fall of 1969, and maintained in mass culture in our laboratory. The results obtained by sampling for Esterase 6 a total of 423 flies from the four cultures, are reported in Table I.

Table I. The expected values are calculated following the Hardy-Weinberg distribution.

Genotypes	S/S	F/F	V/V	S/F	S/V	F/V	Totals
Observed	287	13	1	94	27	1	423
Expected	285.5	8.5	0.5	99.4	24.6	4.3	423

Gene Frequencies (%): S 82.16; F 14.30; V 3.54

Controls were then made by crossing between flies of known genotype and screening the offspring for esterase 6 (Table II). Although the data obtained from any one cross are scanty the results follow closely the Mendelian rules of segregation confirming the expectations.

Table II. The expected data are reported in brackets.

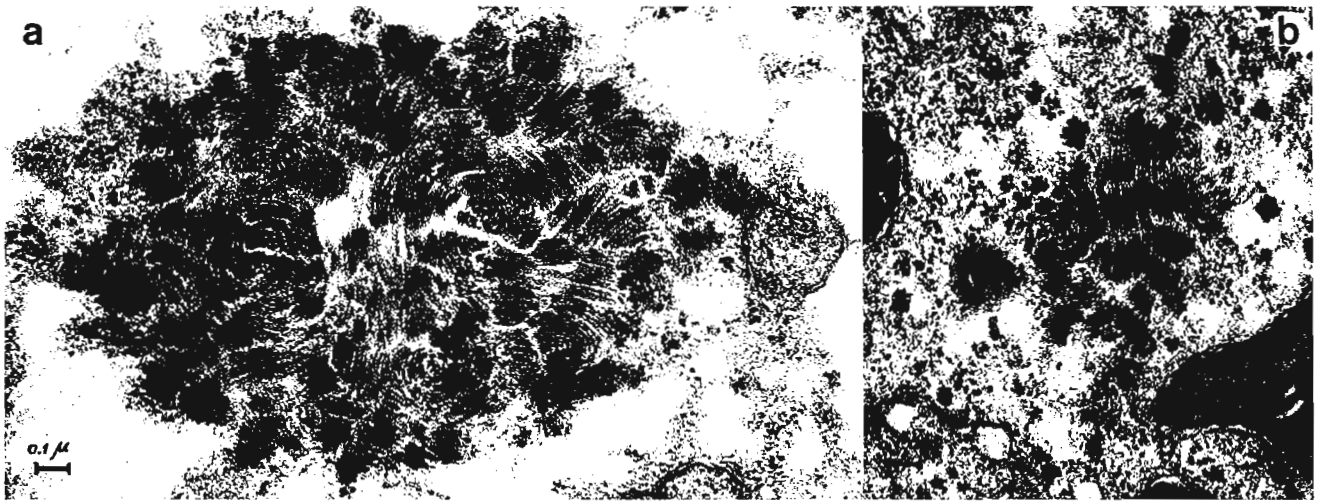
Parents genotype	S/S	F/F	V/V	S/F	S/V	F/V	Totals
F/F x S/V	-	-	-	7 (9)	-	11 (9)	18
S/S x S/V	18 (17)	-	-	-	16 (17)	-	34
S/V x S/V	9 (9.5)	-	7 (9.5)	-	22 (19)	-	38
F/V x F/V	-	2 (2.7)	3 (2.7)	-	-	6 (5.5)	11

During the summer of 1970 the new allele was detected also in other D. melanogaster populations of the Venetian region in N.E. Italy; these are being sampled for esterases. The gene frequency observed for Est 6 V is very low, being around 0.02 or 0.03. Work is also in progress to characterize the new esterase form with different substrates and inhibitors.

References: Wright, T.R.F, 1963 Genetics 48: 787; Wright, T.R.F. and R. MacIntyre, 1965 J.E. Mitchell Sci. Soc., 81: 1.

Van Breugel, F.M.A. and J. van Zuylen
Genetisch Laboratorium, Leiden, The Netherlands. Fibrillar spherulites in the Malpighian tubules of larvae of Drosophila hydei.

Electron microscopical observations on proximal cells after GDA and O_sO_4 fixation, of the anterior Malpighian tubules of late third instar larvae, revealed the presence of typical structures (Fig. a,b) resembling the fibrillar spherulites or 'stromacentre' of Avena chloroplasts (Gunning, 1965; Gunning et al. 1968; Steer et al. 1970). It has been suggested for the Avena structures that the fibrils have a proteinaceous nature and probably consist of linear aggregates of ribulose diphosphate carboxylase (Gunning et al. 1968). We found the spherulites in wildtype (Fig. 1a) as well as in



white (Fig. 1b) and white-mottled larvae. (Photographs were made with technical assistance of the division of Cell Biology).

References: Gunning, B.E.S., 1965 J. Cell Biol. 24: 79-91; Gunning, B.E.S., M.W. Steer and M.P. Cochrane, 1968 J. Cell Sci. 3: 445-456; Steer, M.W., J.H.W. Holden and B.E.S. Gunning, 1970 Canad. J. Gen. and Cy. 12: 21-27.

Ertha, D. and S.E. Moyer. Northeastern University, Boston, Massachusetts. A mosaic for two of three dominant markers in a male D. pseudoobscura.

A male was discovered with a spontaneous occurrance of Lobe and Delta on the right side but not on the left. Both wings were Blade but the phenotype for Bare was not clear. He was the result of a backcross mating of ♀♀ Ba/Δ;Bl/L x random ♂♂ having two of these markers, one for each of chromosomes II and III. He was able to sire progeny that indicated his genotype as $Ba/\Delta^{+};Bl/L$.

Hence, his somatic tissues expressed either ΔL or $\Delta^{+}L^{+}$, while the germinal tissue was $\Delta^{+}L$! We are puzzled for an explanation for this event. We would be grateful to hear from other Drosophila workers for interpretations and reports of similar mosaics.

Stocker, A.J. University of Texas (Southwestern) Medical School at Dallas. An apparent developmental anomaly in Drosophila pseudoobscura induced by injection.

In an experiment to determine the effects of LSD and related compounds on Drosophila, third instar female larvae were injected with either a phosphate-buffered-saline LSD-25 solution or with phosphate-buffered-saline alone. A considerable number of the F-1 progeny from both the experimental and control animals exhibited abdominal segmentation anomalies in which the sternites and tergites were disrupted. These progeny were usually produced from eggs laid during the first few days after initiation of egg laying. An example of a fly bearing such a segmentation anomaly is shown in figure 1.

Figure 1. An example of a fly exhibiting the described segmentation anomaly.

These "segmentation defective" flies were usually fertile and produced normal progeny. The cause of this defect is not known. It is clear, however, that there is no increased incidence of such anomalies among the offspring of the LSD-25 (and related compounds) treated animals; neither were such anomalies observed in uninjected flies. The effect could not be attributed to the injection itself since the adults produced from the injected larvae completed their development without exhibiting any such abnormalities. It appears, therefore, that some agent transmitted during the injection affects the developing ovary in some unknown manner to produce a specific effect, which seems to be of a developmental rather than a genetic nature.

Work supported in part by Grant no. MH 15743-01 from the National Institutes of Mental Health, and a grant from the Moody Foundation.

Lefevre, G. and M.M. Green. San Fernando Valley State College, Northridge, California; University of California, Davis, California. Interactions of deficiencies in the 3C region.

Unexpected phenotypes appear among females heterozygous for different deficiencies involving the 3C region. Specifically, a short, male-viable deficiency for band 3C2-3 expresses only a white-eyed phenotype, but is delayed in emergence for 2 to 3 days (in uncrowded cultures) as compared with normal brothers. This deficiency, $w^{67c23(3)}$, when combined with N^{64i16} or N^{64j15}, both deficient from 3C3 through 3C7 and beyond, produces viable females that express the vertical phenotype, but are not rough-eyed. $Df\ w^{67k30}$, deficient for bands 3C2-6 inclusive, is male lethal, but when combined with N^{5419}, deficient from 3C6 to 3C11, again produces viable females that express vt, but not rst. Thus, homozygous deficiency for either 3C3 or 3C6 accompanied by heterozygous deficiency for the other, gives rise to the vt phenotype. This suggests the persistence of a genetic duplication associated with the 3C2-3 and 3C5-6 doublets. Furthermore, such a duplication would explain the anomalous lethality of the $w^{m4L}rst^{3R}$ chromosome, in which band 3C2-3 is deficient (which should not be lethal by itself), but band 3C5-6 is inactivated by position effect. When either one or the other of these two bands remains, the condition is not lethal and a vt phenotype is expressed, as in the rst^2 deficiency. When 3C5 is affected, the rst phenotype is observed, but its homozygous deficiency is not lethal. The only male-lethal mutants between w and spl are deficiencies that remove, at least, both 3C3 and 3C6, as in $Df\ w^{67k30}$. The effect of 3C5 on the viability of the 3C3, 3C6 double deficiency is being determined.

Harrod, M.J.E. and C.D. Kastritsis.
University of Texas (Southwestern) Medical School at Dallas. Nuclear inclusions in the salivary gland cells of D. pseuddobscura.

During an investigation of ultrastructural changes in the salivary glands of developing Drosophila pseudoobscura larvae we found certain nuclear inclusions. These occur in many of the cells examined both during the late third instar and the spiracle eversion stages.

The inclusions usually are found in contact with the nuclear envelope (Fig. 1 & 2) and frequently in conjunction with a bleb of that envelope (Fig. 1). They appear to be membrane-bound and to consist of from one to many discreet oval-shaped (also membrane-bound) bodies approximately 1600Å to 2500Å in length. The membrane surrounding the inclusion is frequently covered by an extension of the internal dense lamella of the inner membrane of the nuclear envelope (Fig. 1); this lamella is not seen to extend into the observed nuclear blebs. This

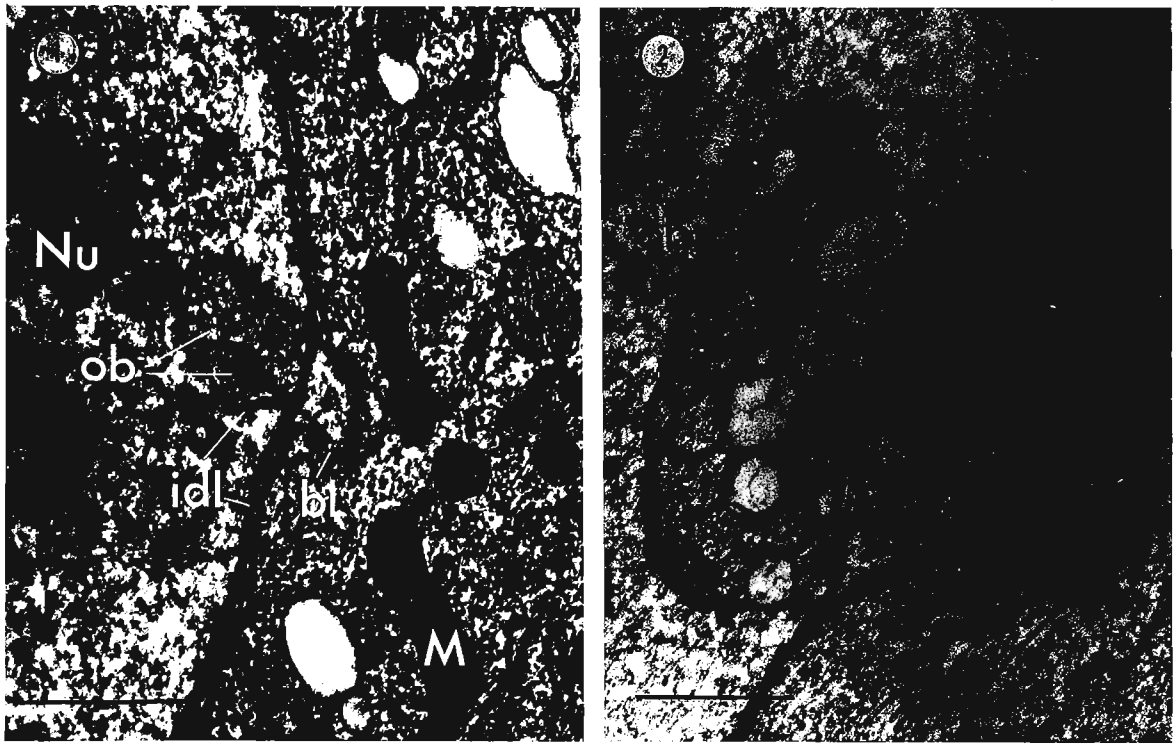


Fig. 1, 2. Electron micrographs of D. pseudoobscura salivary gland cells. Nucleus (Nu); internal dense lamella (idl); oval bodies (ob); nuclear bleb (bl); mitochondria (M). Bars represent 0.5μ in each case.

observation extends the list of species in which such a dense lamella is found to include D. pseudoobscura (Stevens and André, 1969). In a number of sections an oval body appears to be entering the nuclear bleb, possibly for extrusion into the cytoplasm; however, no unequivocal findings have been made of such bodies within the cytoplasm at any distance from the nuclear envelope.

Whether the nuclear inclusions observed are parasitic organisms or the result of some normal or abnormal cellular process in D. pseuddobscura is unknown at this time. Investigation will continue with earlier and later stages of larvae and an effort will be made to correlate these findings with the present data in an attempt to explain the origin and significance of the inclusion bodies.

Work supported in part by grant GM 16736-02 from the National Institutes of Health.

References: Stevens, B.J. and J. André, 1969 The Nuclear Envelope in The Handbook of Molecular Cytology. North-Holland Publishing Co., Amsterdam & London.

Wasserman, M. and H.R. Koepfer. Queens College, Flushing, New York. A new inversion found in a D. pseudoobscura balanced lethal stock.

We have been isolating D. pseudoobscura strains which are homozygous for their third chromosomes. These strains originated from males collected in Ruidoso, New Mexico and the Black Forest, Colorado. The technique used is a slightly modified version of that developed by Spassky et al. (1960). Wild males are crossed to the multiple marker stock: Blade, Scute, purple/orange, Lobe. This stock is a balanced lethal system with Blade, Scute, purple on a Standard chromosome and orange, Lobe marking a Santa Cruz chromosome. In our method, the cytological analysis is made on larvae which are heterozygous for the Blade, Scute, purple (Standard chromosome) and the wild chromosome. Between 15 and 20% of these larvae from both New Mexico and Colorado proved to be heterozygous for a new, small, subterminal inversion. This inversion is independent of, and distal to, both the Pikes Peak arrangement (Figure 1) and the Arrowhead arrangement (Figure 2). It was not found among the 244 chromosomes analyzed directly from the offspring of females taken in the same collections with our Ruidoso males (Dobzhansky, personal communication). It appears, then, that the new inversion is not a naturally occurring one, but arose and is present in the multiple marker stock. In our laboratory stock, many of the Blade, Scute, purple chromosomes carry this new stock inversion. The breakage points of the stock inversion overlap those of Santa Cruz and therefore these two inversions are mutually exclusive.

It is not known when or where the stock inversion arose. Perhaps it is not limited to our laboratory, but may be present in others as well. Therefore this report should serve to alert workers in other laboratories who may have occasion to use this balanced lethal stock or some other modification of the Blade, Scute, purple marker chromosome that their stocks may be heterozygous for this inversion.

Reference: Spassky, B., N. Spassky, O. Pavlovsky, M.G. Krimbas, C. Krimbas, and Th. Dobzhansky, 1960 Genetics 45: 705-722. This work is supported by grants from the City University of New York Faculty Research Award Program and from N.I.H. FR-07064.

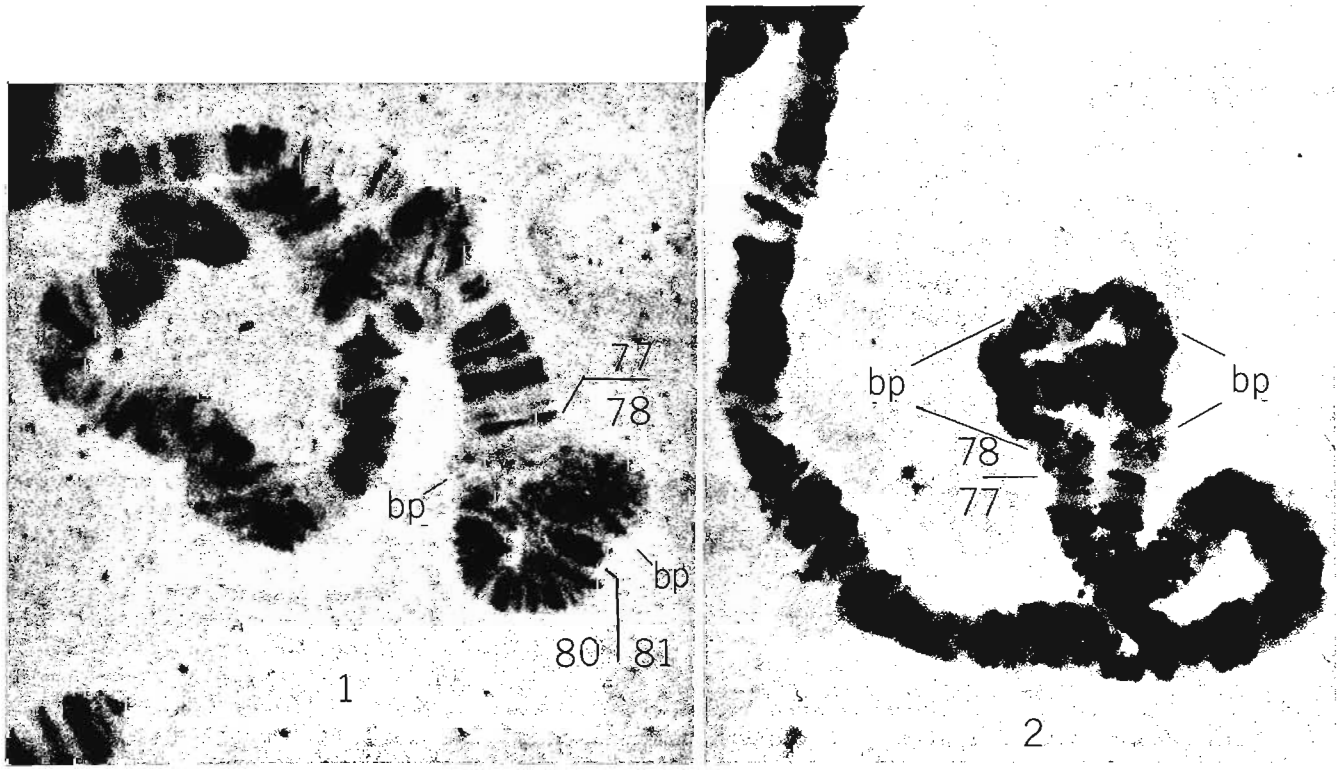


Fig. 1. Pikes Peak/Stock inversion heterozygote Fig. 2. Arrowhead/Stock inversion heterozygote

Wakahama, K.[1], O. Kitagawa and O. Yamaguchi[2]. 1. Shimane University, Matsue, Japan; 2. Tokyo Metropolitan University, Japan. Evolutionary and genetical studies of the D. nasuta subgroup. I. Chromosomal polymorphism found in the natural populations of D. albomicans.

Drosophila albomicans is one of very polymorphic species chromosomally. From the analyses of the salivary gland chromosomes of this species in two strains of the Okinawan Islands and eighteen strains in Formosa, we have found 4 heterozygotic inversions in the X chromosome, 3 in chromosome 2R, 11 in chromosome 2L and 2 in chromosome 3. Among them, an inversion named 2L A is the most common in all but two strains.

In the previous note (DIS 44), we reported on the karyotype variations found in this species. Generally, the karyotype of this species consists of 1V (chromosome 2), 2 Rods (the sex chromosome and chromosome 3 which are seen in V shape chromosome as a result of fusion of both chromosomes) and one short rod (chromosome 4 which is made from dot plus heterochromatin). However, some individuals showed 1V+3 Rods+1 short rod configuration in their larval ganglion cells. Added to these larval ganglion cells, six arm cells were seen in the salivary gland chromosomes of some individuals. And also there were two cases in the six arm cells. In one case, the basal region of the X was inverted to make one short arm. In the other case, double length chromosome (3) was divided into two chromosomes as a result of the inversion that occurred in the middle portion. In the first case, the X chromosome changes to a J type from its original Rod type and in the second case, chromosome 3 alters to V type from its original long Rod shape.

Fig. 1

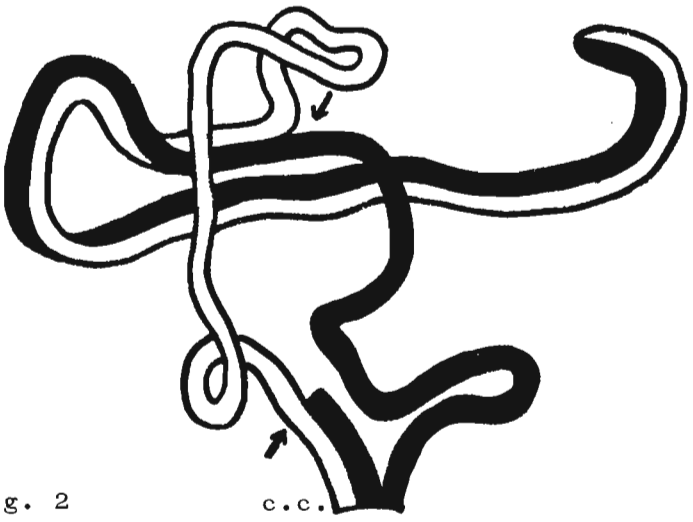


Fig. 2

Further, an individual which proves the above mentioned phenomena was found in the salivary chromosomes of the Okinawan strain (Fig. 1). As seen in Figures 1 and 2, one chromatid of the X chromosome was cut near the chromocenter. And disjoined chromatid was attached to the opposite side of chromocenter and at the median region, it was paired again with the unmutated chromatid of this chromosome. If an individual has this altered chromatid homogeneously, and the X or the third chromosomes will be divided into two chromosomes, this individual will have 1V+3Rods+1 short rod karyotype. And it seems very significant to study the evolutionary process of this species and this subgroup.

Band, H.T. Michigan State University, East Lansing, Michigan. Environments, disruptive selection and lethals.

Disruptive selection may operate when two or more optima are favored in the population. In the South Amherst (Mass.) D. melanogaster population the frequency of second chromosome lethals and semilethals recovered in a sample has been found to be significantly negatively correlated with temperature range of the week prior to collection (Band and Ives, 1961, 1968). In agreement with hypotheses that can be deduced, from this negative relationship, heterozygotes carrying these drastic variants have been found to have significantly higher viability in narrow temperature range conditions (Band, 1963; Oshima, 1968, 1969) while heterozygotes free of drastics have the significantly higher viability in wide range conditions (Band, 1963, 1969).

Maintenance of two major classes in the population with contrasting viability properties suggests that disruptive selection is operating. Band (1969) also suggested that only a genetic system maintained by disruptive selection could track the recent environment. The numerous samples during summer and fall from the breeding site population in 1967 have provided good evidence of le + sle frequency changes in relation to the recent temperature range conditions. If disruptive selection is involved, then with respect to temperature range the environment must be sufficiently heterogeneous to promote different major classes in the population.

To investigate this possibility, the number of days of narrow, intermediate and wide ranges have been computed for the summer months. We concentrate on this season because it is a standardized period of the year (a May-Oct. interval includes months from spring and fall quarters also) and because most collections since 1938 have been made in the fall, usually September. Narrow range has been defined to include ranges from 3 to 20 degrees between daily minimum and maximum temperatures, intermediate ranges are from 21 to 25 degrees and wide temperature ranges are 26^{o} and over. Viabilities of the different heterozygous classes have not yet been investigated in the intermediate ranges. The three groups can arbitrarily be condensed into two by defining ranges 23^{o} and below as narrow and 24^{o} and above as wide. Data presented are from summers 1938 through 1946, the period when le + sle frequency was highest and rainfall averaged over 12 inches per summer. Data for summer 1967 is also given.

Most summers in this initial period of genetic investigations on the S. Amherst D. melanogaster population tend toward similar numbers of days in the various categories or toward

Table 1. Average daily temperature range per summer

1938	1939	1940	1941	1942	1943	1944	1945	1946	$\bar{X}$	1967
21.1	22.7	23.6	23.6	21.8	22.4	24.4	21.9	23.8	22.7	23.4

Table 2. No. of days with narrow (N), intermediate (I), or wide (W) temperature ranges. No. of days with ranges above or below 23^{o} also indicated.

	1938	1939	1940	1941	1942	1943	1944	1945	1946	1967
N	41	30	26	26	37	30	24	35	28	30
I	25	30	30	27	28	30	22	27	18	28
W	26	32	36	39	27	32	46*	30	46*	34
$\leq 23^{o}$	57	51	45	42	55	51	32	52	34	48
$\geq 24^{o}$	35	41	47	50	37	41	60*	40	58*	44

* means $P<.05$ that significantly more days are in the wide range category

more days with ranges 23^{o} and below, as would be expected from the average daily temperature ranges shown in Table 1. Summer 1967, during which data demonstrating the ability of le + sle frequencies to track the recent environment were obtained, is similar. Summers 1944 and 1946 have significantly more days above 25^{o} but le + sle frequency was not measured in 1944, and in 1946 June is the month with less than a third of the days with temperature range 23^{o} or below. Any reduction in le + sle frequency in June would have gone undetected by fall when le + sle frequency among second chromosomes was estimated to be 49.3% (Ives, 1954).

One consequence of the maintenance of both heterozygous classes by disruptive selection is that the level of drastics can fluctuate in relation to the recent environment while the

frequency of any one lethal need not necessarily reflect either present or past environments. Indeed, if many such lethals can participate in the genotype-environmental interaction, the genetic load is minimized. In fact, one possible way to distinguish between lethals perhaps deleterious in all environments (the classical genetical load) and those conditionally heterotic (heterotic in narrow range environments) would be to compare viabilities for heterozygotes carrying different lethals in the narrow fluctuating temperature range conditions.

A further point to emerge from the data is that temperature ranges appear not to be the exclusive environmental variable influencing the frequency of lethals and semilethals in the population; the average frequency of these genetic variants in samples collected during the 1938-1946 period is 48.8% while the average in summer 1967 samples is 24.2%. But this is not unexpected from the significantly positive relationship also between le + sle frequency and summer rainfall (Band and Ives, 1968) and the finding of parallel environmental changes in rainfall and temperature range in the area from 1930 through 1969.

References: Band, H.T., 1963 Evolution 17: 307-319; ____, 1969a Japan. J. Genet. 44, Suppl. 1: 200-208; ____, 1969b, unpublished manuscript; ____ and P.T. Ives, 1961 P.N.A.S. 47: 180-185; ____ and ____, 1968 Evolution 22: 633-641; Ives, P.T., 1954 P.N.A.S. 40: 87-92; Oshima, C., 1968 Proc. XIIth Intern. Cong. Genetics 2: 170-171; ____, 1969 Japan. J. Genetics 44, Suppl. 1: 209-216.

Mazar Barnett, B. Comisión Nacional de Energía Atómica, Buesos Aires, Argentina.

Lack of effect of DMSO on the fertility of irradiated males exposed to low temperature.

We have previously reported (1970) some data on the viability of sperm in inseminated females exposed to 0°C during different periods of time.

While working on the action of radioprotectors at the genetic level, some experiments were done to study the combined effect of X-radiation and dimethyl sulfoxide at a low temperature, on sperm treated in adult males. Six day old Canton S males, pre-treated with a 10% solution of DMSO, were irradiated with 1000 R and submitted to 0°C during the irradiation and before and/or after the irradiation in three different treatments. Immediately after treatment the males were allowed to mate for 3.30 hours and then provided with new virgins until completion of 24 hours (a procedure followed to study the effect on immotile and on fully mature sperm). The Basc females were transferred twice, every 4 days, so the broods covered a period of 12 days. Although the proportion of F_1 males and females seemed to be normal, there was a sharp decrease in the number of offspring. It was hoped that the problem of diminished progeny, which is known to be the joint effect of radiation and low temperature, would be overcome by the presence of DMSO, a cryoprotective agent, Ashwood-Smith (1967). However, this was not the case, the number of offspring of the so-treated males did not differ from the ones submitted to irradiation and low temperature only. Males exposed to 0°C for 10 to 20 minutes produced a normal number of offspring. Only the F_1 females, which were collected for standard sex-linked recessive lethal tests are shown in the tables.

From the results shown in Table I, it is interesting to note that the males submitted to 0°C for a total period of 20 minutes, of which 10 minutes were previous to the irradiation, produced more offspring than those submitted to 0°C for a total period of 10 minutes during and after irradiation only.

In another experiment the mating procedure was changed. Of 270 males, irradiated and kept for a total period of 10 minutes at 0°C, half were mated immediately and for 24 hours, then provided with new virgins for another day. The other half were witheld from mating for one day, after which the same procedure was repeated. The two groups yielded a similar number of F_1 females, all from the first mating period (See Table II). No progeny was obtained from the second mating period. The amount of F_1 females per treated male was higher than in the previous series of experiments.

The results obtained with the second group of treated males could perhaps be explained by a process of recovery of the damaged sperm or a higher resistance of spermatids, plus a higher sensitivity of less mature germinal cells. To account for the similar results obtained with the first group of males is rather difficult; a test in dominant lethality is in progress.

References: Ashwood-Smith, M.J., 1967 Radioprotective and cryoprotective properties of DMSO in cellular systems. Ann. N.Y. Acad. Sci. 141: 45 Mazar Barnett, B. and E.R. Muñoz, 1970 Effect of low temperature on inseminated females. DIS 45: 123.

TABLE I

Treatment	No. treated males	1st mating period: 3.30 hrs. No. F_1 females 1st brood	2nd brood	3rd brood	No F_1 ♀♀/treat ♂♂	2nd mating period: up to 24 hrs. No. F_1 females 1st brood	2nd brood	3rd brood	No F_1 ♀♀/treat ♂♂
1) Irradiation	250	434	673	483	6.36	805	752	686	8.97
2) DMSO $\xrightarrow[\text{min}]{30}$ Irr	150	629	397	346	9.14	577	545	444	11.10
3) Irr+0 °C: 30 min	250	---	---	---	----	---	---	---	----
4) DMSO $\xrightarrow[\text{hr}]{20}$ Irr+0 °C: 30min	250	---	---	---	----	---	---	---	----
5) DMSO $\xrightarrow[\text{min}]{30}$ Irr+0 °C: 30min	250	---	---	---	----	---	---	---	----
6) 0 °C $\xrightarrow[\text{min}]{10}$ Irr+0 °C: 20min	234	207	29	---	0.92	230	143	79	1.93
7) DMSO $\xrightarrow[\text{hr}\ 10\text{min}]{20\ \ 0\,^{\circ}\text{C}}$ Irr+0 °C: 20min	234	103	112	65	1.19	223	126	88	1.86
8) DMSO $\xrightarrow[\text{hr}]{20}$ Irr+0 °C: 10min	275	63	20	59	0.51	92	53	27	0.62

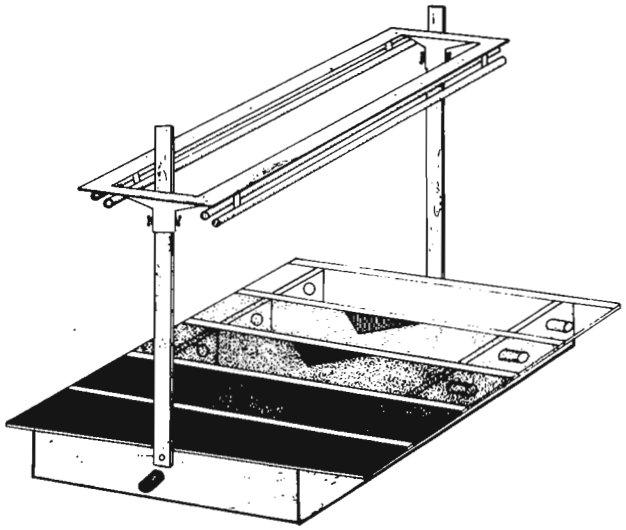
Except for experiments 1 and 2, all irradiations were done at 0 °C.
The underlined number of minutes indicates total time submitted to 0 °C.

TABLE II

Treatment: Irradiation + 0 °C = 10 minutes

135 males, mated immediately after treatment (Mating period: 24 hrs) No. F_1 females 1st brood	2nd brood	3rd brood	No. F_1 ♀♀/treat. ♂♂	135 males, witheld from mating for 24 hrs. (Mating period: 24 hrs) No. F_1 females 1st brood	2nd brood	3rd brood	No. F_1 ♀♀/treat. ♂♂
272	65	23	2.66	174	138	19	2.45

Kekić, V., D. Marinković, N. Tucić and M. Andjelković. Institute for Biological Research, Belgrade, Yugoslavia. Apparatus for a measurement of phototactic behavior in Drosophila at different light intensities.

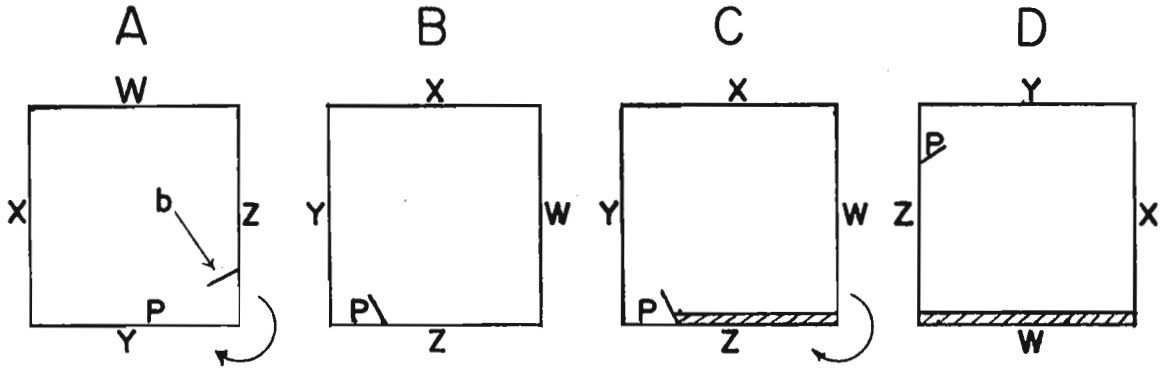


The apparatus contains a few rectangular cells where different light intensities in very broad limits may be simultaneously obtained. Dimensions of each cell are 10 x 40 x 10 cm, and they are connected through a triangular space 12 cm wide. At both sides of each rectangular cell a bottle with culture medium is placed, where the flies can be collected. Every rectangular cell is covered by a glass plate, on which a thin layer of gelatin with a different concentration of ink is resolved, giving in each space a determined intensity of light (this can also be made by using different numbers of thin white paper sheets). As the light source, four neon tubes (40 w each) were used giving the light-spectrum in a range of 400-7000 Angströms. By changing the filters and the distance of the light source, gradual differences in the amount of light in the rectangular spaces from 0 to 5000 lux can be obtained.

Williamson, R. University of British Columbia, Vancouver, B.C., Canada. A screening device for separation of immobilized adults from normal flies.

In order to screen for temperature-sensitive paralytic adult flies from a large population of offspring of mutagenized males, a plexiglass screening box was devised. The following diagrams are end-on views of the box whose only internal feature is a diagonal barrier (b) which runs the length of the box.

Flies which have been raised at 22°C are shaken into the screening box which has been preheated to 29°C. The box is then maintained at this temperature for 1/2 to 4 hours. Any



fly which is immobilized (P) will have fallen to the y surface (A). The box is then rotated 90° so that P falls onto the z surface (B) to the left of the diagonal barrier. A small quantity of vineger and detergent is poured onto the z surface to the right of the barrier (C). The box is then rotated 90° and the active flies which are not behind the barrier are shaken into the solution (D).

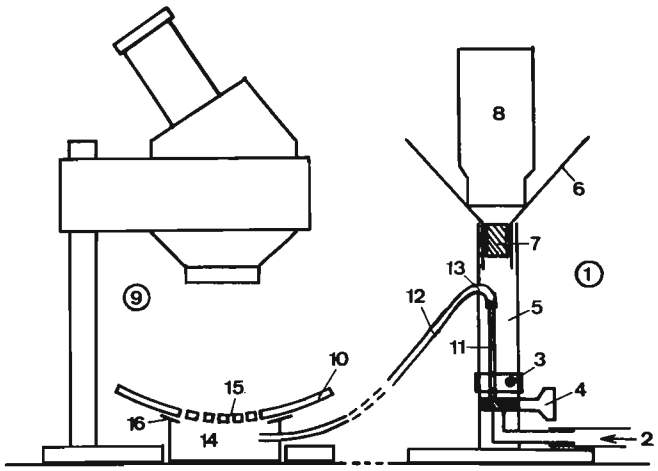
Provision must be made for good ventilation.

The design minimizes the deleterious effects of crowding as well as the number of physical obstacles which tend to inhibit free movement. A 6" x 6" x 18" box will easily screen six thousand flies at a time. The apparatus has been successfully used for isolating a temperature-sensitive paralytic mutant.

(This work is supported by research grants to Dr. David T. Suzuki from N.R.C. of Canada and the National Cancer Institute of Canada.)

Würgler, F.E., M. Lezzi and U. Graf. Swiss Federal Institute of Technology, Zürich, Switzerland. A device for easily anaesthetising large numbers of flies.

The figure shows a scheme of the device. A bunsen burner equiped for a pilot flame (1) is connected by plastic tubing to a CO_2-cylinder (2). The air inlet (3) of the burner is sealed with Araldit. If the valve of the burner (4) is in position "flame", the carbon dioxide flows through the large tube of the burner (5) and enters a plastic funnel (6) fixed to the top of the burner. A foam rubber stopper (7), through which the gas can penetrate, is placed in the neck of the funnel. Bottles or vials (8) containing flies are put upside down into the funnel. Upon gentle shaking the flies fall into the funnel and are immediately immobilized by the CO_2 and accumulate on the foam rubber stopper. For inspection of the flies under a microscope (9) they are - by removing the funnel - transferred to a concave plastic dish (10). In order to keep the flies continuously in a CO_2-atmosphere, the valve (4) is switched to position "pilot". Now the gas stream passes through the thin tube (11) of the burner. On top of the shortened thin tube a plastic tubing (12) is fixed. This plastic tube passes through a hole (13) in the wall of the large tube (5) and is connected to a cylindrical box (14). From this box the gas stream reaches the flies through a large number of very small holes (15) in the plastic dish. This dish is made by cutting out a circular piece from a conventional plastic bowl. The edge of the box (14) is covered with a strip of rubber (16) to avoid electrostatic loading of the plastic dish resulting from its movements. The use of a heat shielded microscope lamp is recommended. With this device large numbers of flies can be inspected over a very long period without the interruption of work for reanaesthetising and without danger of killing the animals.



Work supported by Schweizerischer Nationalfonds zur Förderung der wissenschaftlichen Forschung.

Shorocks, B. University of Leeds, England. A culture medium for rearing Drosophila species.

The following medium is quickly prepared and members of the quinaria group of Drosophila often difficult to breed on standard laboratory media can be reared quite successfully on it.

The following ingredients are required: 100 cc water, 5 g Instant Breakfast Cereal, 5 g brown sugar, 12 g yeast (dried), 3 g agar, 0.3 g nipagin. The Instant Breakfast Cereal, brown sugar and dried yeast are added to about 2/3 of the cold water. After bringing the mixture to a boil and cooking for a few minutes, the agar and nipagin, dissolved in the remaining 1/3 cold water, are added. The whole mixture is cooked for about one minute before being poured into culture bottles.

Bennett, J. and G.B. Stanton. Northern Illinois University, DeKalb, Illinois.

A "Y" maze for Drosophila.

The apparatus was devised in this laboratory to investigate some behavioral preferences of D.m. It was constructed from three micro-sample containers sold by the Aloe Scientific Co., St. Louis, Missouri. The containers, Analocups™, (#V9327), measured approximately 25 mm in height and 11 mm in diameter with the base outside diameter slightly less than the inside diameter of the mouth. Thus the base of one cup could be fitted firmly into the mouth of another. The cups have conical bottoms,

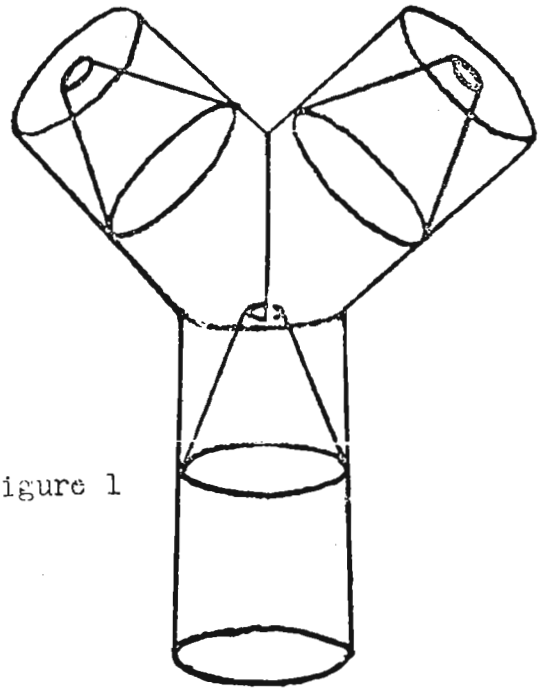


Figure 1

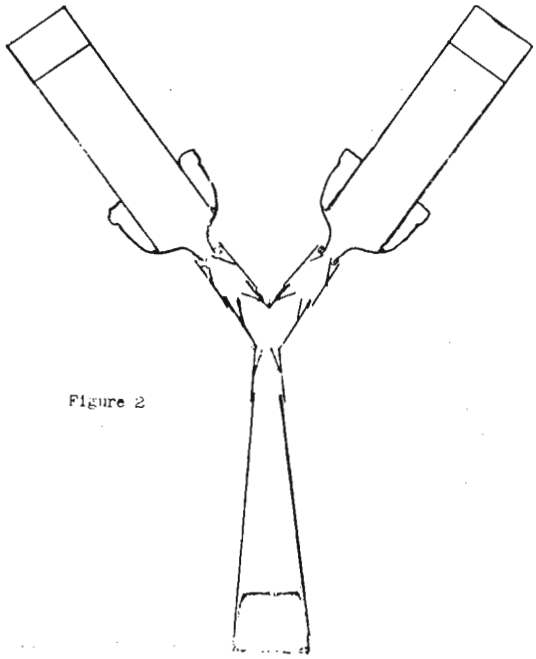


Figure 2

which when drilled with 2 mm openings formed one way traps. One cup with the base pointing upwards formed the stem of the maze, while two other cups formed the arms. The mouths of these latter cups were cut on one side at 45° angles to the axis and were glued together with acetate cement at their respective 45° angles to set the arms of the maze 90° apart. The other side of the mouth of each cup was sanded to allow proper seating on the base rim of the stem cup. A glass grinding wheel was used for the cutting because its fast, water cooled blade allowed a straight cut of the plastic cups without splintering, and the movable platform could be adjusted to cut the cups uniformly. Y mazes assembled in the above manner were consistently symetrical (Figure 1).

Flies were placed in a start chamber which was fitted into the stem of the Y maze and collected in glass shell vials (25 x 95 mm) connected to each arm of the maze. The start chamber was a section of a polypropylene cone (Poly Cone™, Cole-Parmer #36432) cut so that the small end of the cone would fit firmly into the stem of the Y maze. The floor of the large end of the cone was a rubber stopper. The conical nature of the chamber funneled the ascending flies into the maze. The collection vials, with 6 ml of food, were connected to the arms of the maze with an assembly of Analocups™, polyethylene thistle tube tops (Bel-Art, #H-14750) and polyurethane foam gaskets (Figure 2). The base of an Analocup™ was cemented on the the stem of a thistle tube top so that the assembly (vial, gasket, top) could be firmly connected to the arm of the maze. The assembled maze, start chamber, and collecting vials were supported by a test tube clamp inside a carboard box which, with fan and light, provided constant temperature and light.

Mazes with two color choices each were made by placing Contak™ self-sticking cellophane sleeves around the cylinder of each arm of the maze. The color choices were yellow-blue, green-blue, purple-red and orange-red. The mixed colors were made by combinations of the primary colors, yellow, red and blue. The color pairings were matched as closely as possible for light transmittance using a Science and Mechanics model A-3 light meter with Clairex™ CL-505L photo-conductive cell. The mazes could be oriented vertically or horizontally by

adjusting the test tube clamp.

The basic Y maze was also modified to provide a temperature choice apparatus by attaching 7 x 3.5 x 3 cm water compartments around each arm of the Y. A temperature difference in the two compartments was created by pumping water directly from constant temperature baths to each compartment.

Holmquist, G. University of Illinois, Urbana, Illinois. Removal of RNA from polytene chromosomes by lacto-aceto-orcein.

The lactic-acetic orcein (LAO)[1] squash method, as described by Nicoletti (DIS 33: 181), gives excellent band resolution of polytene chromosomes. After analyzing ^{3}H-RNA autoradiographs of salivary squashes which had been prepared by the Nicoletti method, it was found that LAO removes RNA from chromosomes. Since some workers have used LAO to stain RNA labelled material before autoradiography, the effects of this stain preparation on RNA should be understood.

Salivary glands were dissected into insect ringers solution containing ^{3}H-uridine (0.8 mC/ml, 24 C/mM) and incubated for ten min. before fixing in cold 45% acetic acid for 5 min. After fixation the glands were placed in LAO for 15 min. and squashed. The siliconized coverslip was removed by the dry ice method at specific times after squashing, the frozen slide with adhering material was immersed in 95% ethanol, rehydrated and autoradiographs were prepared and allowed to expose for two weeks. If the squashed preparation was allowed to differentiate in the stain for 2 days before coverslip removal, most of the radioactivity from ^{3}H-RNA was removed. After one day in stain, some RNA was removed and the remaining radioactive material was evident as a diffuse halo of silver grains surrounding each chromosome set. If the coverslip was removed immediately after squashing, most of the resulting silver grains appeared over the chromosomes. Thus, some component or combination of components in LAO appears responsible for the removal of RNA from chromosomes.

The following experiments were done in order to analyze the characteristics of the loss of chromosomal RNA. First the effect of 45% acetic acid on the molecular weight of newly formed RNA was analyzed, and this was followed by an examination of the effects of LAO and its components. To determine the effect of 45% acetic acid on chromosomal RNA, five gland pairs were incubated in ^{3}H-uridine as described above. One gland from each pair was fixed in alcoholic formalin and the RNA was extracted according to the SDS[2]-pronase and phenol method of Edstrom and Danholt (J. Mol. Biol. 28: 331-343, 1967). The remaining glands were fixed for 5 min. in cold 45% acetic acid, squashed, rinsed 5 min. in acetic acid and dehydrated in ethanol. The gland material on these slides was fixed in ethanolic formalin and digested from the slide in SDS-pronase solution. This digested material was extracted with phenol along with the digested material from the unsquashed glands and the RNA prepared by both methods was spun in a 5-20% sucrose gradient at 25,000 rpm in a SW 25.1 rotor for 6 hr. according to the conditions of Edstrom and Danholt. Recovery of labelled RNA was similar for both methods of extraction and radioactivity profiles from both extractions were indistinguishable with a peak at about 38s and material sedimenting as fast as 80s.

Since chromosomal RNA is not degraded or selectively removed by short treatments with cold 45% acetic acid, squashes prepared by this method were used as controls to test the effects of LAO components on squashed material. Salivary glands labelled with ^{3}H-uridine were squashed in cold 45% acetic acid, the coverslips were removed and different slides were subjected to the following conditions:
(1) 45% acetic acid at 3^{o}C for 5 min., (2) 45% acetic acid at 3^{o}C for 18 days, (3) 45% acetic acid plus 1% orcein at 3^{o}C for 18 days and (4) a 1:1 mixture of 85% lactic acid and glacial acetic acid for 3 days. The ^{3}H-RNA was retained as indicated by excellent autoradiographs with the first three conditions, but the fourth condition completely removed the chromosomal RNA. Apparently exposure of chromosomes to cold acetic or acetic-orcein solutions has no effect on RNA removal, while mixtures of lactic and glacial acetic acid do. Mixtures of lactic and glacial acetic acid should be avoided in studies of chromosomal RNA.

[1]LAO 2% by weight of powered orcein in equal parts of 85% lactic acid and glacial acetic acid. (Orcein Natural, G.T. Gurr Ltd., London, England.)

[2]SDS, sodium dodecyl sulfate.

Bischoff, W.L. and J.C. Lucchesi.
University of North Carolina, Chapel Hill, North Carolina. The preparative mass isolation of melanotic pseudotumors from larvae of Drosophila melanogaster.

Prior to initiating a series of experiments designed to investigate patterns of protein synthesis in Drosophila melanotic pseudotumors, it was necessary to devise a technique for the mass isolation of experimental material. The procedure to be described was based on that of Fristrom and Mitchell (1965) and Fristrom and Heinze (1968) who have succeeded in isolating 90-93% pure preparations of Drosophila imaginal discs. Although the present technique was utilized for the collection of tumors from deep orange-lethal (dor^1 I-0.3±) larvae, it should be useful for any strain in which tumors appear in late larval stages and are free-floating in the larval hemocoel rather than being associated with various larval organs.

(1) Culture techniques and collection of larvae: 50 to 75 grams of larvae are required for this operation. Since the dor^1 melanotic masses are larger and more numerous in female larvae than in males, and because this phene is associated with a recessive third instar lethal, a special cross is required. Males of the genotype dor^1/Dp(1:Y)67g24-2 (Dp(1:Y)67g24-2 representing a special Y chromosome to which a short segment of X carrying the wildtype allele of dor has been translocated) are crossed to dor^1/FM3 females. Any method, suitable for the collection of large numbers of larvae (50 to 75 gram batches) can be used. In our laboratory the above cross is made in 1/2 pint milk bottles on standard cornmeal-agar medium which has been lightly seeded with dry yeast. Ten pairs of parents are used per bottle and permitted to lay eggs for four days before clearing. Larvae are collected by washing the walls of the culture bottles and surface of the food with distilled water; dor^1 larvae characteristically crawl out of the food onto the sides of the bottles as their normal sibs are pupating. Third instar larvae are cleaned of contaminating younger larvae, pupae, dead imagos and food debris by the following method. The larval suspension is poured into a glass column, 40cm x 6.5cm OD. The column is filled with 12-15% w/v sucrose solution in which the larvae float while food and other debris and sediment can be removed from the bottom of the column. Pupal and adult contaminants are removed by adding distilled water to the layer of larvae causing adults and pupae to float to the surface where they may be aspirated off. Subsequent operations are performed in the cold (0-6°C).

(2) Preparation of homogenates: Larvae are ground in batches of 50-75 grams per 200 to 250 ml of cold Ephrussi-Beadle ringer's solution in a semi-micro Waring blender at full speed for 2 minutes. After grinding, the brei is filtered through a metal screen with 0.5mm openings. The filtrate is saved and the material remaining on the screen is reground in 150ml of ringer's solution and refiltered.

(3) Washing of material: The combined filtrates of step #2 are placed in a 600ml beaker, brought to a total volume of about 500ml with additional ringer's solution, and stirred vigorously for about 30 seconds. After stirring, tumors and mouthparts settle quickly to the bottom and can be easily followed visually because of their black color. The various non-tumorous debris is aspirated from the top of the suspension after 2-3 minutes. Aspiration is continued until about 200ml of suspension remains. This process of resuspension, stirring, and settling followed by aspiration is repeated three times.

(4) Centrifugation: After washing is complete, the pseudotumor suspension (about 200ml total volume) is again stirred vigorously, divided among eight 30ml glass centrifuge tubes and spun at 10,000 x g in a refrigerated centrifuge for 15 minutes. After centrifugation all but 1-1.5ml of the supernatant from each of the tubes is discarded. The pellets are resuspended by vigorous mixing on a vortex mixer, and each is layered on the top of a discontinuous sucrose density gradient tube (2.5ml each of 50, 60, 70, and 80% w/v reagent grade sucrose per 15ml centrifuge tube) and spun for 3 minutes (or as necessary) at 1350 rpm in an International Clinical centrifuge with model #213 head. Larval debris bands on the top of the gradient tubes or at the interfaces between the 50-60% and 60-70% steps. The pseudotumors band at the interface between the 70-80% steps. They may be gently removed with a Pasteur pipette, washed in buffer and frozen under liquid nitrogen in a number of protective solutions until needed. The preparations are estimated to be about 90% pure, the major source of contamination being mouthparts. Starting from 50 grams of larvae 50-60 mg of pseudotumors (wet weight) are routinely obtained.

References: Fristrom, J.W. and H.K. Mitchell 1965 The preparative isolation of imaginal discs from larvae of Drosophila melanogaster. J. Cell Biol. 27: 445-448; Fristrom, J.W. and W. Heinze 1968 The preparative isolation of imaginal discs. DIS 43: 186.

(Aided by an Institutional Grant of the American Cancer Society.)

Anderson, B.A.S. University of Oregon, Eugene, Oregon*. Mass isolation of fat body tissue from Drosophila larvae.

A method has been developed (drawing on the work of Boyd, Berendes and Boyd, 1968) for isolation of fat body tissue from D. melanogaster and D. hydei. Mid to late third instar larvae are placed on a watch glass or glass plate over ice. A rotary circular blade (a pizza cutter or noodle cutter) is used to cut the larvae quickly into two or three sections each. Larvae are then washed into a beaker with Drosophila Ringer's solution, pH 7.2 (0.01 M Tris-HCl), containing 1 to 1 1/2% Ficoll. After stirring for fifteen minutes the suspension is allowed to stand for five to ten minutes. Large fragments of fat body rise to the top and can be removed by pipetting or decanting. repetition of the stirring and settling is sometimes necessary.

All steps are carried out at 2-5°C. All glassware is siliconized.

Fragments of fat body attached to testes or salivary glands sink in the settling step.

Use of higher concentrations of Ficoll led to contamination of fat body tissue with Malpighean tubules and tracheae. Omission of Ficoll reduced the extent to which fat body was concentrated in the surface layer.

Fat body tissue thus isolated was almost entirely free of other tissue. It was compared by acrylamide gel electrophoresis to fat body tissue isolated by dissection. All major bands were present, though two bands, D and E (see Research Note, this issue), were somewhat diminished in relative intensity. Preliminary work indicates that this fat body tissue is active in incorporation of ^{14}C amino acids into protein.

Reference: Boyd, Berendes, and J. Boyd, 1968 J. of Cell Biol. 38: 369-376.

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Posch, N.A. University of California, Los Angeles, California*. Effective means of eliminating bacterial contamination in Drosophila culture media.

One of the studies conducted in this laboratory required the raising of Drosophila melanogaster cultures with no live yeast added to the media, one a cornmeal-molasses-agar medium and the other a banana-corn syrup-agar medium (General Biological Supply House, Chicago, Illinois). Both media contained mold inhibitors. The usual procedure of using sterile media and sterile glass bottles was followed. Within three generations, bacterial contamination was present in 40% of the cultures. The organism was identified simply as a gram-negative bacillus and produced a dark brown color in both types of media, although the contamination, as judged by color, appeared more severe in the banana medium (pH 4.8), than in the cornmeal medium (pH 3.4). Many larvae failed to pupate. Adult viability was slightly reduced.

Several measures were taken to eliminate the contamination. Enough concentrated water solution of merthiolate (sodium ethylmercurithiosalicylate) was added to each medium immediately after boiling so that each medium contained 0.01% (W/V) merthiolate. This eliminated the bacteria, but also the flies.

Benzalkonium chloride (Winthrop: Zephiran chloride), a germicidal solution commonly used in hospitals, was added to hot media so that the media contained 0.08% (W/V) of the chemical. The bacterial contamination was not alleviated by the presence of Zephiran chloride.

A combination of antibiotics was added to the hot (~70°C) media prior to dispensing, specifically, Squibb potassium Penicillin G, 100 units/ml medium, and Squibb Streptomycin sulfate, 0.2 mg/ml medium. Adult flies from contaminated cultures were placed in the bottles containing the antibiotic supplemented media. No bacterial growth appeared. Larval viability was not affected and adult fecundity was good. From these cultures, using sterile technique, it was possible to raise several subsequent contamination-free generations in media with no antibiotic content.

It is important to point out, however, that we also observed that addition of live yeast to the surface of the media just prior to the introduction of contaminated flies resulted in clean cultures. No form of bacterial contamination ever occurred in our cultures when live yeast had been added to the surface of the medium. It is therefore advisable to use live yeast in all cultures, unless the experiment demands otherwise, from a bactericidal point of view as well as a nutritional one.

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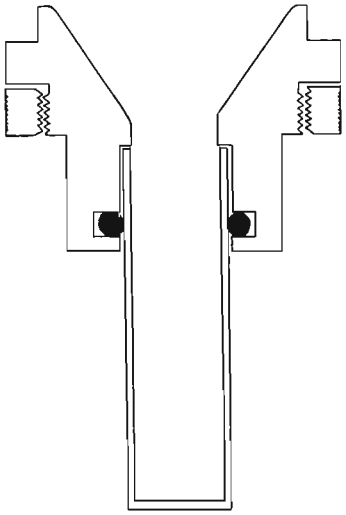
Seecof, R.L., W. Kaplan, P. Wong, W. Trout III and J. Donady. City of Hope Medical Center, Duarte, California. A versatile etherizer.

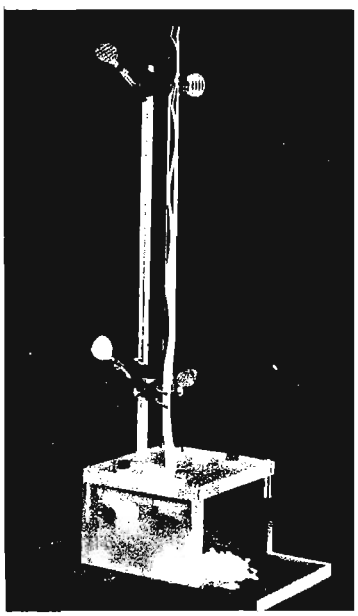
The actual size of the etherizer shown in scale drawing is 50 mm. in widest diameter. A polyethylene tube, with holes that have been poked with a hot needle, is inserted into a hard aluminum alloy head, as shown, with an O ring seal at the junction. The etherizer can be used with a half-pint milk bottle containing cotton and ether. It can also be fitted to a jar lid in which a hole about 40 mm. in diameter has been cut. The removable metal ring is used to clamp the etherizer in the lid. Cotton and ether are added to the jar and the lid attached so that the tube is within the jar. If contaninated, the plastic tube can be withdrawn for disposal and another one pushed in as a substitute. Plastic tubes are polyvials from Van Waters and Rogers, 1363 Bonnie Beach Place, Los Angeles, Calif., Cat. No. 66017-026 (with cap removed). Metal head can be obtained from Nordin and Ericksson Tool and Die Co., Inc., 490 E. Duarte Road, Monrovia, Calif., about $25.00.



Supported by NIH Grant No. AI05038 and NS09330 to R. Seecof.

Gordon, J.W. Indiana University, Bloomington, Indiana. A simple device for the preparation of small filter paper rectangles for use in starch gel electrophoresis.

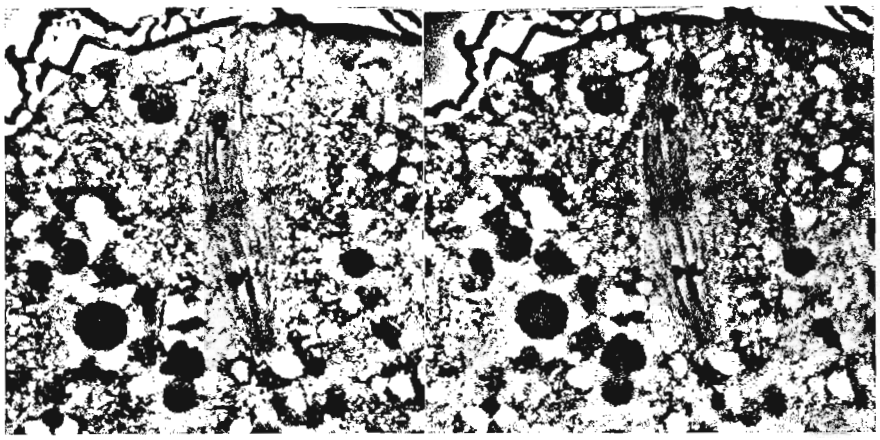
The technique of starch gel electrophoresis of homogenized Drosophila as a means of assaying for genetic variability in this organism has become a widely practiced procedure. Many investigators use small rectangles of filter paper as a means of inserting samples into a starch gel. Preparation of these small rectangles (approx. 4 x 9 mm) is tedious unless the operation is mechanized. I have constructed a simple machine which produces large numbers of filter paper pieces of varying width and length (see figure). The construction of this machine requires a drill press and surface grinder in addition to a set of standard hard tools. The cutting blades, pressure plate and blade guides are made of die-steel and the remainder of mild steel. The pressure plate is automatic and squeezes the paper at the point of cutting providing accurate square pieces. In operation, strips of filter paper cut to width are inserted six at a time into the glass tube and a brass slug put in the top to push the strips down against the stop. Strips up to 1" wide can be accomodated and the stop is adjustable to give square lengths from 1/8" to 1". A single operator can produce up to 15,000 pieces per hour.



Inquires about the device should be addressed to Gordon Instruments Co., 1908 Viva Sr., Bloomington, Indiana, 47401. Cut squares made to order can also be obtained at the rate of $1.00 per 1000.

Evans, W.H. and J. Erickson. Western Washington State College, Bellingham, Washington. Application of E. M. techniques to light microscopy of meiotic stages in Drosophila eggs.

In order to investigate meiotic stages in Drosophila eggs, we have used plastic embedding and ultra-microtome techniques. Eggs are collected within 15 min. after laying by newly-mated females. The eggs are fixed in Kahle's fixative, Fuelgen-stained (von Borstel and Lindsley 1959), carried thru alcohol series to propylene oxide and embedded in Epoxy resin (Luft, 1961). Eggs are cut posteriorly to admit the various solutions but are not dechorionated. Thus, the chorionic filaments provide a reference for orientation of the meiotic spindle during the embedding. This is readily accomplished if the embedding is done in a silicone rubber flat mold (available from Ladd Research Industries, Inc., P.O. Box 901, Burlington, Vermont).



Two levels of focus, 2 micron section of first anaphase in an attached-X/Y egg. Phase contrast 1700x.

The blocks are trimmed with a file, cut at 2 μ and sections mounted in Permount. This technique may be applicable to investigation of bridges, fragments, nondisjunction and gross irregularities of meiosis in meiotic mutants.

References: Luft, J.H., 1961 Improvements in epoxy resin embedding methods. J. Biophys. Biochem., Cytol. 9: 409-414; von Borstel, R.C. and D.L. Lindsley, 1959 Insect embryo chromosome techniques. Stain Technology 34: 23-26.

Gordon, J.W. and R.C. Richmond. Indiana University, Bloomington, Indiana. A pressurized Drosophila media dispenser.

One of the more tedious aspects of the preparation of fly media is dispensing it into bottles or other culture containers once it has been prepared. We have designed and one of us (J.G.) has built a pressurized media dispenser modeled on a similar but more complex apparatus in use at the Rockefeller University. This device consists of a large pressure cooker (21 qt. cap.), a short length of washing machine drain hose (3/4" dia.) and a lever-type lawn hose gun (see figure). A hole is bored in the side of the cooker and a composite of plumbing fittings is obtained to fit through the hole and connect to the drain hose. A length of flexible copper tubing (1/4" dia.) runs from the fitting to the bottom of the cooker. The hose gun's valve rod is removed and the spray pattern removed by turning on a lathe. The gun is reassembled and the dispenser is ready for use. Media is prepared in the cooker with the lid removed. When the preparation is complete, the lid is put in place and the apparatus is pressurized from the lab air line (10 psi). Media is dispensed simply by pressing the lever on the hose gun. The apparatus can be used to fill 1/2 and 1/4 pt. milk bottles as well as vials and creamers. We routinely prepare 100, 1/2 pt. milk bottles of Spassky media (DIS 17: 67) in less than an hour.

Borack, L.I.* and W.H. Sofer. The Johns Hopkins University, Baltimore, Maryland.
Pyrazole suppression of alcohol dehydrogenase activity after electrophoresis.

We have experienced difficulty in visualizing the weakly active enzymes beta hydroxybutyrate dehydrogenase (BDH) and beta hydroxypropionate dehydrogenase (PDH) after agar gel electrophoresis. Both of these enzymes (Borack, Water and Sofer, DIS, this issue) migrate close to alcohol dehydrogenase (ADH) (Ursprung and Leone, 1965) which masks their appearance because ADH exhibits formazan deposition even in the absence of added substrate. This "nothing dehydrogenase" activity (Fig. 1a) may be due to the presence of an alcohol contaminating one of the components of the reaction mix. Rather than attempt to remove the alcohol, which might be difficult under some circumstances, we found a means of selectively inhibiting ADH.

A typical staining pattern for ADH is shown in Figure 1c. Pyrazole is a potent inhibitor of horse liver ADH (Theorell and Yonetani, 1963) and also of Drosophila ADH

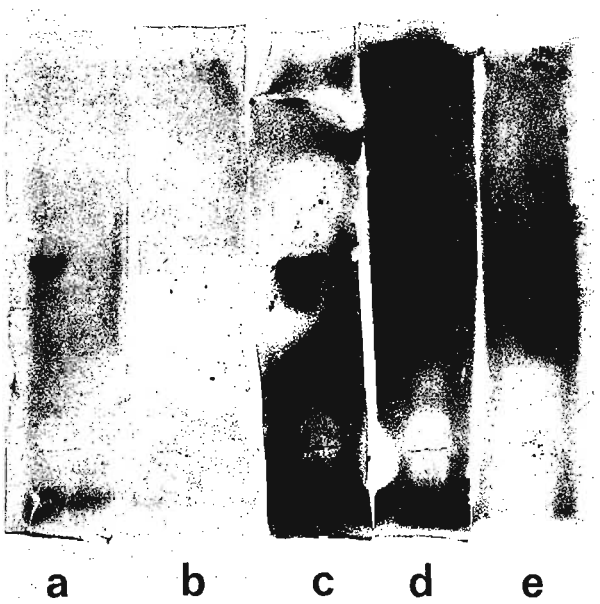


Figure:
a) No substrate
b) No substrate + pyrazole
c) 2-butanol
d) beta-hydroxy propionate + beta-hydroxy butyrate
e) same as d, but with pyrazole added

(Sofer, unpublished observation). The addition of pyrazole (final concentration, 0.05M) to the staining mix for PDH or BDH effectively suppresses the appearance of ADH bands (Figure 1b, e) but does not effect the intensity of staining for PDH or BDH (Figure 1d, e).

References: Ursprung and Leone, 1965 J. Exp. Zool. 160: 147; Theorell and Yonetani, 1963 Biochem. Z. 338: 537.

Supported by N.I.H. Training Grant GM-57 to L.I.B. and N.S.F. Grant GB 7803 to W.H.S.

*Current address and address to which requests for reprints should be sent: Dept. of Zoology and Physiology, Rutgers University, Newark New Jersey 07102.

Van Dyke, D.L. and J. Bennett. Northern Illinois University, DeKalb, Illinois.
Mite elimination from stock cultures.

In December 1969 many stocks were successfully treated for mite infestation by using strips of paper toweling (1 x 4 cm) that had been soaked with 10% Benzyl Benzoate in 95% Ethanol, then air dried, after the method of Barker (1967, Evolution 21: 606). One drop of distilled water was added to each strip to make it stick to the glass surface when it was slipped into the 25 x 95 mm shell vials. This was sufficient to hold the strip in place even when the vial was shaken. The method has proved entirely successful in eliminating mites from the cultures and is apparently not deleterious to the highly inbred stocks treated.

Moyer, S.E. Northeastern University, Boston, Massachusetts.
Disposable "vials".

Moyer and Yarbrough (1969) Am. Biol. Teach. 31: 593-596 described disposable containers for culturing Drosophila, including a container for small cultures, such as single pair matings. It is made of sturdy clear plastic with a pliable plastic cover (Van Brode Milling Co., Clinton, Mass.) Its cost is less than one cent. An improved method of providing air is to punch two or three holes in the cover with a hand paper punch and cover with porous tape (Johnson and Johnson Zonas Porous #5104). Carbon dioxide injected through the tape immobilizes the flies.

Stocker, A.J. and J. Jackson. University of Texas (Southwestern) Medical School at Dallas, Texas. A technique for the synchronization of Drosophila for developmental studies.

During our studies on different aspects of the development of Drosophila pseudoobscura we have formulated a synchronization technique which provides uniform results as evidenced by puffing and electrophoretic patterns. To implement this technique we have used a type of population cage which was designed and built by us and is shown in figure 1. This cage consists of a 7" diameter cylindrical plexiglass chamber which is mounted upon a 8" by 15" base containing two 3 3/4" diameter holes for exact insertion of 95 mm Petri dishes. The Petri dishes are supported from beneath by three pieces of spring steel. The fly-containing upper chamber was constructed so that it could be easily rotated to fresh food and the used food removed. Thus, the flies can be changed to fresh food with a minimum of work and disturbance.

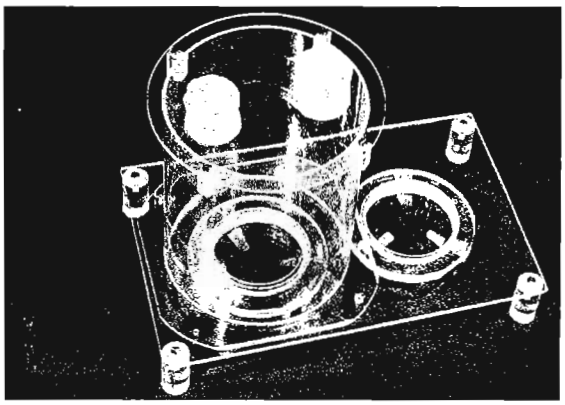


Figure 1. Population chamber designed to facilitate mass egg collections.

The egg collection and synchronization techniques were modified from those of Mitchell and Mitchell (1964), Berendes (1965) and Ashburner (1967). Several hundred newly emerged parent flies are put into the chamber and aged for one week on cream of wheat-molasses media. (These parents go through their larval stage under the same conditions as described for the larvae used in post-spiracle eversion studies). Egg collections are then carried out during the following two weeks on Ohba media (Ohba, 1961) blackened with charcoal and brushed with fermented yeast. This medium is provided for the flies when eggs are to be collected. It is a rich medium and larvae grown on it reach a uniformly large size. The Petri dishes containing the eggs are capped with 5 1/2" high plastic food containers and are incubated at 25°C for the required time.

Under the above conditions, many spiracle eversions take place at about 165 hours after egg laying. Therefore, for work done at and post spiracle eversion, newly everted larvae are easily recognized and either used or aged from that point.

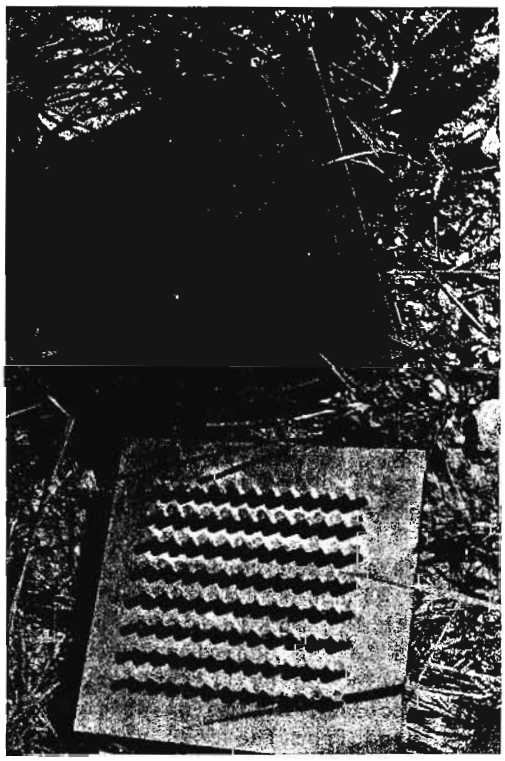
For studies during the third instar, the flies are allowed to lay for a three hour period. The eggs produced during this period are incubated for 25 hours. At this time all newly hatched larvae are removed from the food by rinsing with Drosophila Ringer's and subsequent removal by hand. The remaining eggs are then incubated for an additional hour and the larvae hatching during this period rinsed from the food and collected by filtration. These larvae are placed on fresh food and aged to 72 hours after hatching, a time at which the majority have just entered the third instar. They are then rinsed from the food again and those of approximately the same size are retained on fresh food for experimentation. (Approximately 100 larvae are placed in each petri dish of food.) For mass experiments, the second sorting out is probably not necessary as most of these early third instar larvae are of the same size. The majority of spiracle eversions among larvae collected in this manner take place between 138 and 146 hours from the midpoint of the hatching period. All studies done during the third instar use the midpoint of the hatching period as a base.

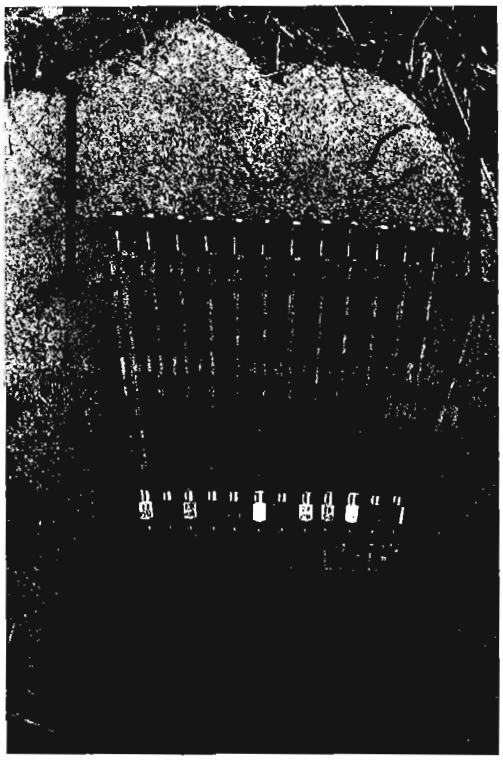
References: Ashburner, M., 1967 Chromosoma (Berl.) 21: 398-428; Berendes, H.D., 1965 Chromosoma (Berl.) 17: 35-77; Mitchell, H.K. and A. Mitchell, 1964 DIS 39: 135-137; Ohba, S, 1961 Okayama Univ. Biol. J. 7: 87-125.

Work supported in part by Grant no. GM 16736-02 from the National Institutes of Health.

Roberts, R.M. University of Chicago, Illinois. A multiple sample homogenizer and multiple microsyringe applicator for acrylamide gel electrophoresis.

At present, flies to be individually electrophoresed on acrylamide gels are usually placed singly in centrifuge tubes and ground with a hand grinder in a precise volume of sucrose-buffer solution added to each tube with a single microsyringe. After centrifugation, the supernatant from each is transferred to the gel pockets with the microsyringe. The following devices, a multiple sample homogenizer inspired by Adamkewicz and Milkman (DIS 45: 192) and a multiple microsyringe holder, enable the worker to homogenize 96 flies similtaneously and to load 12 gel pockets at once in approximately the same time previously necessary to crush and load a single fly. A plate of plexiglass or brass drilled with 1/4-inch flat-bottomed holes, the same distance apart as the pockets of the gel, arranged in eight rows of twelve, receives the individual flies, which are mashed with a corresponding plate of 1/8-inch flat-bottom stainless-steel prongs. The desired volume of homogenizing buffer is placed in the holes 12 at a time with a plexiglass holder for 12 microsyringes; it consists of twelve 20-gauge syringe needles placed the same distance apart as the gel pockets, glued in a plexiglass sandwich, and a frame to support the syringe barrels, which can be removed for cleaning.





A bar under the plungers lifts them simultaneously to a height pre-set on two guide rods, which are simply long metal screws with a nut on each. The desired volume is set by adjusting the height of the nuts. In use, the flies are ground on a bed of ice; the homogenizer should not be allowed to stand on the ice bed for sufficient time for condensation to dilute the sample. The plate is tilted nearly upright so that the homogenate collects in the lower portion of the wells; the multiple holder then either loads the desired volume of homogenate directly into the gel pockets twelve at a time, or transfers the total volume of homogenate to conventional centrifuge tubes held in a row of twelve by a simple holder, should centrifugation be necessary for the desired assay. After centrifugation, the tubes are replaced in the holder for removal of the supernatant for loading. (I have found that centrifugation is not necessary for good separation of D. montana, D. virilis, and D. pseudoobscura esterases; it is likely that other enzyme systems similarly do not require centrifugation prior to electrophoresis.) If an eight-pocket gel is desired, the plate should be oriented to display twelve rows of eight wells, each the same distance apart as the eight pockets of the gel.

Burdette, W.J. and J.E. Carver. University of Texas M.D. Anderson Hospital and Tumor Institute, Houston, Texas. A procedure for quantitative analysis of RNA synthesis at puffing sites in salivary gland chromosomes of D. melanogaster.

Visual determination of concentration of DNA and RNA in the salivary chromosomes of Drosophila is troublesome because of difficulty in obtaining reproducible preparations in larvae at a comparable stage (Burdette and Kobayashi, 1969) in the life cycle. Experience in labeling RNA with tritiated uridine and DNA with tritiated thymidine has been acquired in the course of determining effects of oncogenic viruses on chromosomes, and the method used should be applicable to those studies in which localized quantification of nucleic acid is desired. Therefore, the details of the procedure are offered as a workable methology for those who may

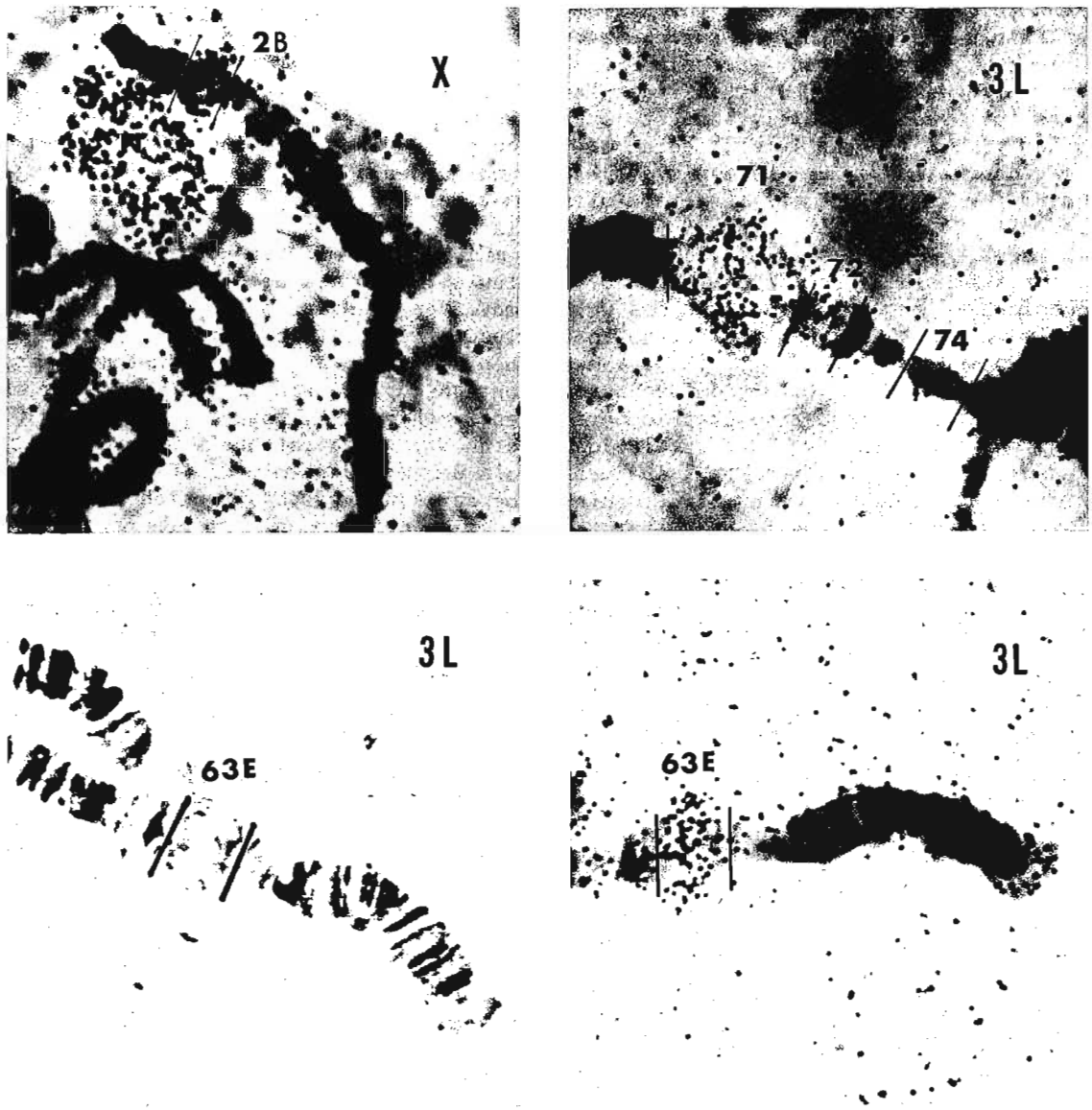


Figure 1. Drosophila melanogaster salivary-gland chromosomes labeled with ^{3}H-uridine. Prominently large puffs and other regions synthesizing RNA are labeled differentially. Upper left: X" distal, including puff region 2B (note heavily labeled nucleolus "N"). Upper right: 3L: divisions 71-74, with conspicuous labeling of puff 71. Lower photographs: Before (left) and after (right) autoradiographic development of 3L: distal, including differentially labeled puff 63E.

have similar requirements.

Adults from an inbred Oregon-R strain were isolated without etherization immediately after eclosion from stock bottles and placed in vials containing cream-of-wheat, molasses medium. After aging together for four days, they were transferred to fresh medium and eggs were collected after 30 minutes. First-instar larvae hatching over the following 23 hours were discarded, and only those hatching during the subsequent period of 1 hour were used. Salivary glands were excised in Ringer's solution precisely at respective intervals determined by the stage of maturation required. Cultures were maintained after hatching at 18°C.

As a valuable aid in reducing the variation in usual puffing pattern, two prepupal stages were recognized by specific changes in larval appearance and behavior and designated white and brown respectively. The onset of the transition of 3rd-instar larvae to the white prepupal stage was determined by the following characteristics: (1) the larval body becomes noticeably shorter and thicker, (2) the color turns from translucent to opaque white, (3) the cuticle remains flexible and soft but the surface becomes sticky, (4) locomotion and inversion-eversion movements of the anterior spiracles cease, and the spiracles remain everted. The period during which larvae remained in this stage varied from forty to sixty minutes. The brown prepupal stage followed immediately, and its onset was marked by the appearance of a faint brown pigmentation visible on the lateral edges of the cuticle. The following period of 60 (±10) minutes was characterized by the rapid darkening and hardening of the cuticle, the beginning of operculum formation, and the cessation of all movement involving the cuticle.

After dissection, salivary glands were immediately submerged in tritiated uridine (Schwartz/Mann, specific activity: 8.0 Ci/mM, 0.5 mCi, in 1.0 ml., aqueous solution) for 10 minutes, stained with aceto-lacto-orcein for 20 minutes, then prepared for analysis by the usual technique of pressure. Cover slips were removed by freezing over dry ice, autoradiographic film (Kodak AR-10) was applied to the stained slides (stripping method), the preparations were exposed for a period of three weeks, then developed and read. Photomicrographs of representative regions were taken before autoradiographic processing for comparisons with the identical regions after autoradiographic development. (Figure 1, bottom). Bridges' (1941) revised map was used to define the cytological distances. Preparations were examined at 1000X magnification, grain counts recorded, and mean grain density determined. Grain counts were taken as the mean ratio of the number of autoradiographic decay spots counted within a given puff to the mean count in a different region of the same chromosome. In general, terminal areas of the salivary chromosomes are consistently more suitable for accurate readings between bands. Thus the area between the distal end of the X-chromosome and bands 2A1-2, and between the distal end of chromosome 3L and bands 63E2-3 were used as ratio standards for counts of X- and 3L-chromosomal puffs, respectively.

Figure 1 shows several regions of labeled salivary-gland chromosomes containing puffs suitable for the procedure of counting described. Despite the higher concentration of decay spots clearly seen over the puffed regions of the chromosomes in these photographs, variation in the amount of labeling with tritiated uridine from preparation to preparation requires that grain counts of the background should be made and subtracted from counts in the region of puffs and between bands.

References: Bridges, C., 1941 (Feb.) Suppl. to J. of Hered. Vol 32, No. 2; Burdette, W. J. and M. Kobayashi, 1969 Proc. Soc. Exptl. Biol. and Med., Vol. 131.

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Improved food medium.

Our standard food medium (Mittler & Bennett, 1962 DIS 36: 131) became unsatisfactory for maintenance of a developing series of inbred and mutant lines. An experiment to modify the medium led to a replacement of the sugar content with molasses and a doubling of the yeast content. The resulting recipe: water, 1000 ml agar (powdered), 20.5 gm; brewer's yeast (powdered), 72 gm; molasses (Grandma's™ or Brer Rabbit Gold™, unsulfured), 125 ml; cook 30 minutes in autoclave at 1,055 gm/cm^2; propionic acid, 5 ml; and 1.5% benzyl bensoate in 95% ethanol, 28.5 ml, added after autoclaving. This medium is delivered to 25 x 95 mm shell vials by automatic pipette (6 ml each) and each tray is immediately cooled in cold water to prevent yeast settling. It has been used fresh and after several weeks frozen and seems equally effective in either case.

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3. New mutants, melanogaster
4. Linkage data, melanogaster
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6. New mutants, other species
7. Linkage data, other species
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