

Pravits

DROSOPHILA

Information Service

35

July 1961

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Prepared at the

**DEPARTMENT OF BIOLOGY
UNIVERSITY OF OREGON
EUGENE, OREGON**

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D. melanogaster

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Ghana

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AUSTIN, TEXAS: UNIVERSITY OF TEXAS
Department of Zoology, Genetics Foundation

Wild Stocks

- 1 Austin, Texas
- 2 Canton-S
- 3 Espanola, New Mexico
- 4 Oregon-R

Chromosome 1

amx/In(1)dl-49, m² g⁴
 amx lz^g v/y f:=
 Bx³ ma-1
 1(1)7/dl-49, y Hw m² g⁴
 1(1)7e, 1(1)7/dl-49, y Hw
 1(1)55a B^g/In(1)sc^{S1}, S,
 lz³ w^a/B/sc⁸
 lz³/y f:=
 m
 ma²-1
 pn²
 Qd
 sc cho
 sc ec^{31d} cv ct⁶ v g f/FM3,
 y³ sc⁸ dm B 1
 sn³ lz^g v/y f:=
 sn² m ma²-1
 sy¹ -s sn²
 v¹ 58k
 v
 w m³
 w^a sn³
 w^a rb
 w^a spl ec
 w^{a2}
 w^a bf⁵
 w^a bf2^f
 w^a bl
 w^a bl spl sn³/y f:=
 w^a Bwx
 w^a Bwx w^a spl sn³/y f:=
 w^a cf
 w^a e spl sn³
 w^a

h sn³ m^N
 wⁱ f³ bb
 w^{sat}
 y cap/y f:=
 y ct ras² f
 y sc
 y sc m^f
 y sc w^{Bwx}
 y sc z w^a spl ec/y f:=
 y W ec f
 y w^e spl sn³
 y² w^e
 y² sc wⁱ
 y² sc wⁱ ec
 y² su-w^a w^a
 y² su-w^a w^a spl sn³
 y² su-w^a z sp³ (9a29)
 (sp³)-w=sp-w^a
 y² w^a
 z 11E4
 z w^a
 z w^a/y f:=
 z w^{Bwx} spl

Chromosome 2

al dp b pr blt bw/
 Cy, al² lt³ L⁴ sp²
 al dp b pr c px sp/Cy
 al S ast ho/Cy^{33k} E-S
 al S ho/Pm ds^{33k}
 ast dp cl
 b
 b pr c px sp
 b vg
 Bla/SM5, al² lt^v Cy sp²
 Ba/In² (2LR), dp
 Bl L²/SM5
 bri²
 bs²
 bw
 bw ba
 cn bw

ds dp
 ds S G b^{pr}/3 L⁴ sp²
 Cy al² lt³ L⁴ sp²
 ex
 ft^v
 G^r/Cy dp²
 ho ed cl
 ho ed dp cl^v
 pi/SM5, al² lt^v Cy sp²
 pr²/SM1, al² Cy sp²
 S^{56F}, Cy Pm/Gla
 Sp J L² Pin/Gla

Chromosome 3

cu kar
 Gl Sb/LVM
 H Pr/In(3R)C, e
 ma
 ma fl²
 ma ry²/TM1 Me
 mo/Ubx
 on ry²
 ri bod e^s/Me, In(3R)C,
 Sp e l(3)e
 ri p²
 R Ly/In(3L)P¹³⁸
 R Sb B⁶/Ubx¹³⁸
 R Sb H^{32y}/Ubx¹³⁰
 ry²
 Sb H/In(3R)c, cd¹⁰¹
 Sb/In(3LR), Ubx¹⁰¹
 st
 st c3G ca/TM1, Me^{ri}(sp²)
 tra/In(3LR), Ubx¹³⁰
 (FMA3/w^a v)
 Wine/C(3)X
 W Sb/In(3LR)ex

Chromosome 4

ci^D/ey^D
 pol

Attached X-Y

v f^L B X-Y/y² su-w^a w^a bb
 X^L·Y^L y^w(-8d)/y f/Y^w
 X Y^L·Y^S(108-9 Parker)y² su-w^a w^a
 (bb⁺)Y^L·Y^S/y v¹ bb/0
 X^L·Y^L y^w/y v f/Y^w
 y² su-w^a w^a bb/0/v f B XY; pol
 Y^S·Y^L In(1)EN -49, y v f car/
 y² w^a bb/0

Inversions

In(1)FM6, FM6, y^{31d} sc⁸ B dm
 y w^{m4} L·N^{264-84R} sn/Y
 dm sn

y Mul-5, In(1)sc^{S1}, S, y w^a B sc⁸
 y v Muller 5, In(1)sc^{S1}, S, y w^a v B sc⁸
 In(1)y, lz^{y4}/y v car

Modified Y

YB^S
 y^L/y/sc⁸·Y
 Y^L/f·Y^S/sc v f:=
 sg·Y^L/In(1)dl-49, y v f·Y^S
 Y^S/g B⁶Y^L/y f:=
 Y^S/y^Sct⁶ f·Y^L/y f:=
 Y^S·Y^S # 2/y v f·Y^L/y f:=

Multichromosomal

sq br w^aR m f; bw(Austin, Texas)(1;2)
v¹:bw(1;2)
w^{col}:bw(1;2)
y² w^a spl ec; SM1, al Cy sp²/In(2LR)102 ds^w
sp²; In(3LP, 3RC)Sb e^s/Ubx¹³⁰ e^s(1;2;3)
y f:=; al; st; pol(1;2;3;4)
ma-1; st(1;3)
w^a:st(1;3)
w^{Bwx}; st(1;3)
w^e; st(1;3)
y v car/Y; pol(1;4)
cn; ma(2;3)
Cy/S; D₃C III z(2;3)
dp; vo³(2;3)
pr; st(2;3)
SM1, al Cy sp²/In(2LR)102 ds^w sp²; In
(3LP, 3RC)Sb e^s/Ubx¹³⁰ e^s(2;3)
SM1/dp b Pm; C Sp₄/Ubx¹³⁰ e^s(2;3)
SML/dp b Pm; Ubx¹³⁰/C Sb (2;3)

Deficiencies

y w²⁵⁸⁻¹¹/y Hw dl-49 m²g⁴

Duplications

y w f/y w^{def} rst³; Dp w^{+51b7}
w^{def} rst³ car/y w f:=; Dp w^{+51C20}
y w²⁵⁸⁻⁴⁵; Dp(3)w^v, co/y f:=

Translocations

T(1;4)B^S264-12/W f:=; bw; e; ci ey^R
T(1;4)N⁵264-12/D/In(1) -49, w lz^S
T(1;4)w^{m5}ci^D18
T(1;4)w²⁵⁸⁻¹⁸ci^D18
T(1;4)w²⁵⁸⁻²¹ey^D
T(1;4)w^a lz B, y w/Ins(1) -49, sc⁸, y^{31d}
w lz B
T(Y;2)C/pr cn
T(Y;2)G/b H bw
T(2;3)Xa/1(3)Xa^R
T(3;4)c, Ubx/ci^D

Triploid

y² sc⁸w^a ec/FM₄, y^{31d} sc⁸ dm B; cn/cn/cn;
sc⁸·Y, y
v f/y Mul-5

BALTIMORE, MARYLAND: THE JOHNS HOPKINS UNIVERSITY
Department of Biology

Note: Additions and corrections to the list in DIS-33 (p. 12).

Additions:

c9 w m f
c9a w² m f (containing XXY ♀♀)
c9b y² cho
c31 In(1)X^{c2} w^{vc}/y w lz^S ♀♀ & y w lz^S/sc⁸·Y ♂♂
d7a cn Su-Pm/Cy cn vg Pm
d7b cn Su-Pm Tac/Pm (dp b c ?)
d9a l(2)m^g
d10a mi/Pm²
d11c px slt sp
e3a l(3)tr/M^e Sb
e6a red
g1e cn, bw; e
g1f ct^{45E} v; bw; e; (ey²)⁺
g3c Cy/tu bw; st su-tu
g9a v; bw; e
g9b y² v f; bw
g11a T(Y;2) J/px bw sp

Discarded:

c24 car l^{B3+1}/Ins(1)sc^{S1} sc⁸, B w^a
c25 car l^{B5+2}/Ins(1)sc^{S1} sc⁸, B w^a

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Zoology

Wild Stocks

106 In(1)dl-49, y Hw m²g⁴/y f:=
107 kz g²B/y
108 Df(1)N⁸/In(1) dl-49, m²g⁴
109 sd
110 sx vb² sy/In(1)AM
112 v car
114 w
115 wbl
116 wbl/FM₄
117 wch wy
118 wch wy/FM₄
119 wco sn²
120 wco sn²/FM₄
121 w^e bb¹/ClB (with extra Ys)
122 wvc
123 y
124 y ac/y
125 y ac sn³
126 y ac v
127 y ac w^a
128 y ac Dp w^a(w^a)₂/y² ac w⁵⁶¹ ec
129 y ac Dp w^a(w^a)₂/y f:=
130 y sc
131 y sc m f⁵
133 y w
134 In(1)y, In(1)w
135 y w spl sn³/y f:=
140 y² cv v f
141 y² sc w^a ec/y ac w^{bl}
150 Muller-5; y sc⁸·Y
160 X^{c2} f car/y f:=
Chromosome 2
200 a px sp

201 al b c sp²/In(2LR)Cy, al² lt³ L⁴ sp²
 202 al b pr cn vg c sp²/In(2LR)Cy, L⁴ sp²
 204 al dp b pr blt bw/Cy, al² lt³ L⁴ sp²
 205 al dp b pr c px sp/Cy pr
 206 al dp b pr cn vg c a px bw mr sp/S² Cy
 lt³ pr⁺ Bl cn² L⁴ sp²
 208 b
 212 bw
 214 c
 215 cg-c/U
 216 cl
 218 cn bw
 220 esc c sp/SM5, al² Cy lt^v sp²
 225 l(2) gl cn bw/Cy, al² lt³ L⁴ sp²
 226 L⁴
 228 pr cn ix/SM5, al² lt^v Cy sp²
 229 pr en
 232 vg
 233 vg^{no}

Chromosome 3

301 cu
 303 cv-c sbd²
 308 Gl Sb/LVM
 310 h
 312 Ly/D³
 314 p^p
 315 ru h st p^p ss e^s
 316 ru h th st cu sr e^s ca *mutica*
 319 se
 320 se h
 323 ss
 324 ssa
 325 ss^a-B
 340 In(3LR)TM1, Mé/In(3LR)Rbx¹³⁰ e^s
 350 Pc/T(2,3)M⁶

Chromosome 4

402 bt ey^R svⁿ
 403 bt^D/ci^D
 404 ci
 405 ci^W

408 ci ey^R 412 ey²
 420 M-4/ey^D

Multichromosomal

510 w; vg
 511 X^{c2} t/y f:=; en
 512 y ac sn³; stw³ en
 513 y ac sn³/Muller-5; en
 516 y f:=; bw; e; ci ey^R
 517 w^av/y v; tra/C(3)x
 520 b; p^p
 521 Cy/Pm; D/Sb
 522 vg; se
 530 se h; ci ey^R

Translocations

603 T(1;2)Bld/ClB
 606 T(1;2)sc¹⁹/y f:=; fes sc¹⁹ⁱ b pr/
 Cy, dpTh pr
 607 T(2;3)Xa/Sb Ubx

Multiple inversions (Weaver)

A sn³; $\frac{Cy}{cn\ bw}$; ri (Pasadena)
 B y; $\frac{Cy}{cn\ bw}$; ri (Pasadena)
 C sn³; $\frac{Cy}{cn\ bw}$; ri (Berkeley)
 D y; $\frac{Cy}{cn\ bw}$; ri (Berkeley)
 E sn³; $\frac{Me}{bw}$; ri
 F y; cn bw; $\frac{Ubx}{ri}$
 G sn³; cn bw; $\frac{Ubx}{ri}$
 H y; $\frac{Pm}{cn\ bw}$; ri (Pasadena)
 I sn³; $\frac{Pm}{cn\ bw}$; ri (Berkeley)
 J y; $\frac{Gla}{cn}$; ri
 K sn³; $\frac{Gla}{cn}$; ri
 L y; $\frac{In\ bw^D}{cn\ bw}$; ri
 M sn³; $\frac{In(2L)Cy}{cn}$; bw; ri

BUFFALO, NEW YORK: STATE UNIVERSITYCollege of Education

(George M. Lang, Assoc. Prof. of Science)

Wild StocksSex Linked

eosin eye
 white eye

Chromosome 2

black body
 brown eye
 curved wing
 vestigial wing

Chromosome 3

scarlet eye

Chromosome 4

eyeless

CHAPEL HILL, NORTH CAROLINA: UNIVERSITY OF NORTH CAROLINA

<u>Wild Stocks</u>	18 al Cy lt L sp/al dp c px sp	42 Sp Bl L bw ^D /Cy cn ² 43 vg: isogenic in 1959
1 Oregon-R: isogenics, mixed	19 al Cy pr stw c sp/al dp b pr lt stw c px sp	<u>Chromosome 3</u>
<u>Chromosome 1</u>	20 al Cy pr stw px/al sp pr lt stw c px sp	44 M(3)y Gl/Sb Ubx 45 M(3)y Gl Inv(3R)LVM/ Inv(3L)LVM Sb Ubx
2 f ⁵ su-f	21 al Cy stw L sp/al dp pr stw c sp	46 M(3)y Gl p ^P cu/p ^P cu Sb Ubx
3 v f BB	22 al sp Cy L sp/dp lt l sp	47 M(3)y Gl Sb Ubx/LVM
4 v f BBB/y v f car	23 al lt stw sp	48 M(3)y Sb/Gl Ubx
5 v f ++: reverted from v f BB	24 al sp	49 M(3)y Ubx/Gl Sb
6 w ⁵⁷	25 b pr lt stw	50 Me cu sr e ^S ca/ru h th st cu sr e ^S ca ("rucuca")
7 w ^e sn/C1B	26 b pr stw c	51 ru h th st cu su e ^S ca ("rucuca")
8 y ⁵⁸	27 b pr stw c sp	52 ru h th st sr e ^S Pr ca
9 <u>y v f car/y w</u>	28 dp b lt sp	<u>Multichromosomal</u>
<u>Chromosome 2</u>	29 sp b pr str px sp	53 ClB/w ^e sn; Sp Bl L ^{rm} /Cy
10 al	30 dp lt c px	54 f BB; th st cu sr
11 al b lt c sp	31 dp lt c sp	55 b pr stw sp; th st cu sr
12 al b pr stw c	32 dp lt px	56 Cy/Pm; H/Sb C sr
13 al b pr stw c px	33 dp lt sp	57 Sifter: Muller
14 al b pr stw c sp	34 dp Pm/Cy lt L ⁴	58 Sp Bl L/T (2:3) Mé
15 al b pr stw px	35 dp pr stw px	
16 al b pr stw sp	36 Pm/Cy pr stw L	
17 al Cy L sp/al dp b pr lt stw c px sp	37 S L ⁴ /Cy lt Pfd	
	38 S ^W Cy pr/Pfd L ²	
	39 S Pfd/Cy lt cn ² L ⁴	
	40 sp	
	41 Sp Bl L ² /lgl Cy cn ²	

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGODepartment of Zoology

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

<u>Wild-type</u>	17 In(2R)bw ^{v34} , Cy/al dp b Bl c px sp
	18 In(2LR)lt ^{m3}
1 Chicago wild-type	19 T(2;3)lt ^{m29}
	20 pr ltd
<u>Chromosome 1</u>	<u>Chromosome 3</u>
2 fu ^{57a} /FM-1	21 C ₃ G
3 l(1)J-1, sc ^{J-1} /Del (1)24	22 ru h th st cu sr e ^S ca
4 lix/y w	<u>Chromosome 4</u>
5 sc ² pn	23 spa ^{Cat} /ciD
6 sc ¹⁰⁻¹ /y Hw	<u>Inversions-X</u>
7 sc z ^m	24 In(1)EN, y/y f:=
8 w ^{ebb1} /y f:=	25 Ins(1)sc ⁴ EN, y sc ⁴ car y/y f:=
9 y a ^c z ec ct	26 Ins(1)sc ^{S1} sc ⁸ , y sc ^{S1} car n w ^a sc ⁸ /In(1)dl-49, y w lz ^S
10 y z ^Q	27 In(1)y ⁴ , y ⁴
<u>Chromosome 2</u>	<u>Deficiencies - Duplications - X Chromosome</u>
11 b pr lt stw ³	28 w-5gK13 spl; Dp(1;3)w ^{vco}
12 bw ^D	29 Df(1)w ²⁵⁸⁻⁴⁵ , y w- spl dm; Dp(1;3)w ^{vco} /y w f
13 bw ⁵⁹	
14 bw ⁷⁵	
15 bw ⁸¹	
16 bw ⁵⁻ /Cy cn ² L ⁴ sp ²	

30 Df(1)w²⁵⁸⁻⁴⁸, y w⁻; Dp(1;3)w^{vco}/y w f
 31 y w⁻ rst³; Dp(1;2R)w^{51b7}
 32 Dp(1)w^{vc} 6094b/Y^S wy·Y^Ly^{55f10}/y w
 33 Dp(1;3)w^{m264-58a}/+; y w f Y^L·Y^S/
 y w/sc⁸·Y

Ring-X

34 X^{c1}, y/sc⁸·Y/y v f car
 35 X^{c2}, y cv v f car

Reversed Ring

36 RR, In(1)EN, car f v y /In(1)sc⁸,
 y⁻ ac⁻ sc⁻ m/sc⁸·Y

Tandem Metacentric

37 TM(Hw f), originally y Hw v f·y⁺
 cv f y⁺

Reversed Acrocentric

38 RA, y ac sc pn -- In(1)sc⁸/In(1)sc⁸
 (C.O.J-3), y⁻ ac⁻ sc⁸ wa f/sc⁸·Y,y

X with Y Fragments Attached

39 FR1, Y^S y cv v f/y f:=
 40 y² su-wa wa Y^L·Y^L BS/Ins(1)sc^{S1}
 dl-49, v
 41 y wa Dp BS/Ins(1)sc^{S1} dl-49, v
 42 y w f Y^L·Y^S/sc⁸·Y/y w
 43 y Hw·Y^S y⁺/Y^L/y wa:=

Attached X-Y; no free Y

44 Y^S B f v y·Y^L y⁺/O/y v bb
 45 Y^S y B·Y^L/y/y v f/O with sc^{J4}
 46 Y^S w y·Y^L y⁺/O/y v bb
 47 y² su-wa wa Y^L·Y^S/y bb/O

48 y w f Y^L·Y^S/O/y w

Altered Complete Y's

49 Y:bw⁺/y v; bw
 50 sc⁸·Y:bw⁺/y v f/y f:=; bw
 51 sc⁸·Y, y^{54e} ac^{54e}/y v; bw

Y^S Fragments

52 Y^{cS};bw⁺ bb⁺/g² B·Y^L/y v bb; bw
 53 Y^S/g² B·Y^L/y f:=
 54 Y^S:y⁺ bb⁺-5/B·Y^L/y w
 55 Y^S:y⁺ bb⁺-6/g² B·Y^L/y v bb; bw
 56 Y^S:y⁺ bb⁺-7/g² B·Y^L/y w
 57 Y^S·Y^S/sc sc⁸·Y^L/y² su-wa wa bb
 58 Y^S·Y^S #2/y v f·Y^L/y f:=
 59 sc^{V1}·Y^S/y v f bb·Y^L/y f:=

Y^L Fragments

60 sc·Y^L/y ac wa ct⁶ f·Y^S/y f:=
 61 sc^{S1}·Y^L #2/y ct⁶ f·Y^S/y w
 62 Y^L/y ct⁶ f·Y^S/y wa:=
 63 Y^L-13/y ct⁶ f·Y^S/y v bb; bw
 64 Y^L-14/y ct⁶ f·Y^S/y v bb; bw
 65 Y^L-15/y ct⁶ f·Y^S/y v bb; bw

Multichromosomal

66 In(3LR)Ubx¹³⁰, Ubx¹³⁰ e^S/Xa
 67 SM1, al Cy sp²/In(2LR)102 ds^w sp²;
 In(3LP,3RC)Sb e^S/Ubx¹³⁰ e^S
 68 y/sc⁸·Y; ru h th st p^p cu sr e^S
 69 w; Cy/Pm; CxD/Sb, In(3R)Mo
 70 y; In53A, al² Cy sp²; Ubx¹³⁰ e^S/Xa
 71 y w/Y^S f y·Y^L y⁺; svⁿ
 72 Y^S:y⁺bb⁺-7/g² B·Y^L/±; Cy/Pm; CxD/Sb,
 In(3R)Mo
 73 Y^S:y⁺bb⁺-5/±/B·Y^L; Cy/Pm; CxD/Sb,
 In(3R)Mo

CLEVELAND, OHIO: WESTERN RESERVE UNIVERSITYChromosome 1

25 net
 26 pr
 27 vg
 1 ec dx
 2 sc cv v f
 3 v
 4 w
 5 y sc v₃g x y f:=
 6 y w sn³

Chromosome 2

41 cd
 42 cu
 43 e
 44 gl³
 45 h
 46 ru^g jv se by
 47 se
 48 st
 21 b
 22 dp
 23 ho
 24 ltd

Chromosome 4

76 cn; st
 77 Pm dp b/cy sp²;
 Sb/D,Cx F
 60 ci ey^R
 61 ey^D ci^D

Chromosome 1, 2

71 v; bw

Chromosomes 2, 3

72 bw; ss
 73 bw; st
 74 bw; ru h ri
 75 c; e

Chromosomes 2, 4

78 pr; Mal

Chromosomes 1, 2, 3

79 v; cn; st

Translocation

80 b pr tk
 T(Y;2)G

LAFAYETTE, INDIANA: PURDUE UNIVERSITY
Department of Biological Sciences

Note: Stock list is the same as given in DIS-33 except:

DeleteAdd

C-47 dp b Px⁴/Gla
H-59 y f:= and +; 1(2)551/Ins SM1,
al² Cy sp²; Sb/Ubx¹³⁰ es; pol

C-20 da/Ins SM1, al² Cy sp²

LAWRENCE, KANSAS: UNIVERSITY OF KANSAS
Department of Entomology

Note: Stock list unchanged. See DIS-30 p. 26.

LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY

Wild Stocks

Big Ridge, Tenn. (single female strain), 1948
Bikini Atoll (mass-inbred strain), 1947
Pine Ridge, Ky. (mass-inbred strain), 1954

MINNEAPOLIS, MINNESOTA: UNIVERSITY OF MINNESOTA
Departments of Zoology and Animal Husbandry

Note: Only unusual stocks are listed.

Equilibrium mutant segregating (EMS) populations: Non-inbred populations in which one or more mutants with visible effects are present and in which there is a near approach to linkage equilibrium with respect to each mutant locus relative to all other loci concerned. Each population was initiated by crossing mutant and wild type stocks and the derived populations have been reproduced by selecting 3:1 or 9:3:3:1 ratios so that the frequencies of the mutants are held at approximately .5. They were at generation 25 on November 25, 1960.

- | | |
|---------------------------|------------|
| 1. e x Wild Gilbert | 5. e x bw |
| 2. bw x Wild synthetic | 6. al x bw |
| 3. bw x Falcon Woods wild | 7. al x e |
| 4. al x Falcon Woods wild | 8. th x e |

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 Swedish-c a-5 Samarkand

Normal X Chromosome

b-1 B/y f:=	b-8 f BB/y f:=	b-16 fa ^{no}
b-2 car bb	b-9 f fu/CLB	b-17 fa ^{no} spl
b-3 Co/y w f	b-10 fa	b-18 fu ⁵⁹ /y f:=
b-4 cx ^t g t/FM1, y ^{31d} sc ⁸	b-11 fa fa ^{no} sn ³	b-19 l(1)J1 sc ^{J1} /Del(1)24
w ^a lz ^s B	b-12 fa N ^{22a} /In(1)d1-49,	b-20 kz/FM6, y ^{31d} sc ⁸ dm B
b-5 dow/FM6, y ^{31d} sc ⁸ dm B	y Hw m ²	b-21 m f car
b-6 ec dx	b-13 fa N ^{22c} sn ³ /In(1)d1-49,	b-22 N ²⁶⁴⁻⁴⁰ /In(1)d1-49,
b-7 f	y Hw m ²	y Hw m ² g ⁴
	b-14 fa rb	b-23 N ²⁶⁴⁻¹⁰⁹ /In(1)d1-49,
	b-15 fa spl sn ³	y Hw m ² g ⁴

b-24 N^{Co}/In(1)dl-49, y Hw m²
 b-25 nd
 b-26 nd rb
 b-27 ptg³ v m g² sd f/y f:=
 b-28 ras dy
 b-29 rst²/FM1, y^{31d} sc⁸ w^a lz^s B
 b-30 rux/FM6, y^{31d} sc⁸ dm B
 b-31 s
 b-32 sc cv v eq
 b-33 sc cv v f B/y f:=
 b-34 sc ec cv cy⁶ v g/In(1)dl-49,
 y Hw m² g⁴
 b-35 sc ec cv ptg³ v/y v f car
 b-36 sn⁴
 b-37 spl
 b-38 spl cho²
 b-39 spl dm/y f:=
 b-40 spl rb
 b-41 sw
 b-42 v
 b-43 v f su^W-f
 b-44 w
 b-45 w^a
 b-46 w^a fa
 b-47 w^a fa rb
 b-48 w^a fa spl
 b-49 w^a fa^{no} rb
 b-50 w^a fa^{no} spl
 b-51 w^a fa^{no} spl rb/y w f
 b-52 w^a nd rb
 b-53 w^a spl
 b-54 w^a spl rb
 b-55 w^{ch} rb/y w f
 b-56 w^e bb¹/y f:=
 b-57 w^e dy/y w f
 b-58 w^t fw
 b-59 y
 b-60 y inbred line A/sc⁸.Y
 b-61 y inbred line B/sc⁸.Y
 b-62 y ac sc pn/y f:=
 b-63 y ac sc pn w rb cm ct⁶ sn³ ras² v dy
 g² f car/Ins(1)sc^{S1},dl-49,sc^{S1} v f car
 b-64 y B/y f:=
 b-65 y bb^{13a}/y w/sc⁸.Y
 b-66 y bb¹⁷⁴/y w/sc⁸.Y
 b-67 y bb¹⁵⁸/y w/sc⁸.Y
 b-68 y bb¹⁴⁵²/y² su-w^a bb/sc⁸.Y
 b-69 y bb¹⁴⁵⁶/y² su-w^a w^a bb/sc⁸.Y
 b-70 y cv v f
 b-71 y cy v f car
 b-72 y f^{36a}
 b-73 y faⁿ sn³
 b-74 y 1259/sc⁸.Y/S-5
 b-75 y 1451/FM6, y^{31d} sc⁸ dm B
 b-76 y Hw/Ins(1) sc^{S1L},S,sc^{8R},sc^{S1} w^a B sc⁸
 b-77 y N²⁶⁴⁻⁴⁷/In(1)dl-49,y Hw m² g⁴
 b-78 y N²⁶⁴⁻¹⁰³/In(1)dl-49, y Hw m² g⁴
 b-79 y N²⁶⁴⁻¹⁰⁷/In(1)dl-49, y Hw m² g⁴
 g-15 y sc wcol spl f/In(1)rst³, rst³ f
 b-80 y v car/y f:=
 b-81 y w bb
 b-82 y w fa^{no}
 b-83 y w fa^{no} sn³

b-84 y w spl sn³
 b-85 y w^a
 b-86 y w^a m f car
 b-87 y w^a spl rb
 b-88 y² cho²
 b-89 y² cv v f
 b-90 y² spl
 b-91 y² w^a w/y f:=
 b-92 No. 1663 (Fahmy)
 b-93 No. 1920 (Fahmy)

Chromosome II

c-1 ab²/T(Y;2)E
 c-2 ab² tom bw sp²/Ins(2L+2R)Cy, Cy dpTh
 Bl L⁴ sp²
 c-3 al b c sp²
 c-4 al dp b pr c px sp
 c-5 al sp b pr Bl c px sp/SM1, al² Cy sp²
 c-6 b cn c bw
 c-7 b pr c px sp
 c-8 Bl L²/SM5, al² Cy lt^v sp²
 c-9 bw
 c10 bw^D
 c-11 cn bw
 c-12 fr² wt/Ins(2L+2R)Cy
 c-13 ho
 c-14 lt stw³
 c-15 M(2)1/In(2R)Cy
 c-16 M(2)S5/Ins(2L+2R)Cy, Cy (L⁴ sp²?)
 c-17 M(2)S10/Ins(2L+2R)Cy, Cy pr
 Dp(2:2)41²
 c-18 ms cn bw/dp^{txI} Cy pr Bl lt cn²
 L⁴ sp²
 c-19 net al ex ds S ast shv ho rub/SM1,
 al² Cy sp²
 c-20 Pin^{Yt}/Ins(2L+2R)Cy, Cy
 c-21 pr cn ix/SM5, al² Cy lt^v sp²
 c-22 sp² bs²
 c-23 Sp J L² Pin/SM5, al² Cy lt^v sp²
 c-24 stw³ c
 c-25 vg

Chromosome III

d-1 Bd^G/In(3R)C, 1(3)a
 d-2 bx^{34e}
 d-3 C(3)x/tra
 d-4 ca
 d-5 cand/In(3LR)Ubx¹³⁰, M(3)1 Ubx¹³⁰ e^s
 d-6 cu kar
 d-7 cy-c sbd²
 d-8 D³H/In(3L)P, Mé
 d-9 DL³/In(3R)C, e
 d-10 e
 d-11 e⁴ wo ro
 d-12 e^s
 d-13 e^s cand/In(3R)C, Sb e 1(3)e
 d-14 Gl Sb/LVM
 d-15 H²/In(3R)Vno, Vno
 d-16 jvl
 d-17 Ki red/TM1, Mé ri
 d-18 1(3)tra Sb/In(3LR)Ubx¹³⁰, Ubx¹³⁰ e^s

d-19 M(3)S34/T(2;3)Mé	d-30 ry ²	<u>Chromosome IV</u>
d-20 p ^P bx sr e ^S	d-31 se	e-1 bt
d-21 pb/In(3LR)Cx	d-32 se ss k e ^S ro	e-2 bt ^D /ci ^D
d-22 Pc/TM1, Mé ri	d-33 sr gl	e-3 Ce ² /spa ^{Cat}
d-23 Pr/In(3R)C, e	d-34 ss ^a	e-4 ci ey ^R
d-24 R Ly/In(3L)P, gm	d-35 st	e-5 ci gvl ey ^R sv ⁿ
d-25 red	d-36 st C ₂ G ca/In(3LR)Ubx ¹³⁰ , Ubx ¹³⁰ e ^S	e-6 ci ^D /ey ^D
d-26 ro Bd ca/In(3R)C, 1(3)a	d-37 st in ri p ^P	e-7 sv ⁿ
d-27 ru	d-38 p ^P Ki	e-8 spa
d-28 ru h th st cu sr e ^S ca	d-39 st sbd e ^S ro ca	e-9 pol
d-29 ru h th st cu sr e ^S Pr ca/TM1, Mé ri	d-40 st sr e ^S ro ca	
	d-41 ve h th	

Multichromosomal

f-1 v/y ^{bb} ; (1;Y)	
f-2 br ³ dx st ; ed Su ² -dx (1;2)	
f-3 In(1)w ^{m4} ; E-Var7/Ins(2L+2R)Cy, Cy (1;2)	
f-4 lz ^D /In(1)dl-49, w ^{a?} m; Ins(2L+2R)Cy, Cy/In(2LR)Pm, al ⁴ ds ^{33k} lt ⁻ bw ^{V1} (1;2)	
f-5 v; In(2R)bw ^{VDel} /Ins(2LR)SM1, al ² Cy sp ² (1;2)	
f-6 v f; In(2R)bw ^{VDel} /SM1, al ² Cy sp ² (1;2)	
f-7 Y ^{SX} .Y ^L , In(1)EN, Y ^S B y f.Y ^L /y v bb/0; bw (1;2)	
f-8 Y ^{SX} .Y ^L , In(1)EN, Y ^{Sy} .Y ^L +/y ² su-w ^a bb/Y; cn bw (1;2)	
f-9 In(1)AM, y ² /FM6, y ^{31d} dm B; SM1, al ² Cy sp ² /Bl; In(3R)Vno, Vno/In(3LR)Ubx ¹³⁰ Ubx ¹³⁰ e ^S (1;2;3)	
f-10 Ins(1)dl-49, B ^{M1} , sc v B ^{M1} , SM5, al ² Cy sp ² /Pm; R112 (1;2;3)	
f-11 spl rb; Ins(2LR)Sm1, al ² Cy sp ² /In(2LR)Pm, al ⁴ ds ^{33k} lt ⁻ bw ^{V1} ; C Sb/In(3LR) Ubx ¹³⁰ , Ubx ¹³⁰ e ^S (1;2;3)	
f-12 y; Ins(2L+2R)Cy, Cy/In(2LR)Pm, al ⁴ ds ^{33k} lt ⁻ bw ^{V1} ; Sb/In(3L)D, D (1;2;3)	
f-13 y f:=/T(3;1)O-5/Y; SM1, al ² Cy sp ² /In(2LR)Pm, al ⁴ ds ^{33k} lt ⁻ bw ^{V1} (1;2;3)	
f-14 Y ^{SX} .Y ^L , FR1 ^{LC-2R} , y; bw; st (1;2;3)	
f-15 v; In(2R)bw ^{VDel} /SM1, al ² Cy sp ² ; M-4/ey ^D (1;2;4)	
f-16 y ² v; bw; ci ^D /ey ^D (1;2;4)	
f-17 y ² v; bw; ey ^D /simulans-4 (1;2;4)	
f-18 y; bw; e; ci ey ^R (1;2;3;4)	
f-19 y f:=; bw; e; ci ey ^R (1;2;3;4)	
f-20 w; e (1;3)	
f-21 y; ru h th st p ^P gu sr e ^S (1;3)	
f-22 Y ^{SX} .Y ^L , In(1)EN, Y ^{Sw} y.Y ^L +/y ² su-w ^a w ^a bb/0; Sb/In(3LR)Ubx ¹³⁰ , Ubx ¹³⁰ e (1;3)	
f-23 XY, w/y ² w ^a bb/0; Ubx 130e/Sb (1;3)	
f-24 sc cv v f B; ci ey ^R (1;4)	
f-25 w cv v f; sv ⁿ /sv ⁿ /sv ⁿ (1;4)	
f-26 y; sv ⁿ (1;4)	
f-27 y f:=; ci ey ^R (1;4)	
f-28 Y ^{SX} .Y ^L , In(1)EN, y B/y ² su-w ^a w ^a bb/0; sv ⁿ (1;4)	
f-29 al; ru (1;3)	
f-30 b pr Bl/SM1, al ² Cy sp ² ; In(3R)Vno, Vno/In(3LR)Ubx ¹³⁰ , Ubx ¹³⁰ e (2;3)	
f-31 bw; st(2;3)	
f-32 Ins(2L+2R)Cy, Cy/In(2LR)Pm, al ⁴ ds ^{33k} lt ⁻ bw ^{V1} ; Sb/In(3LR)DcxF, D (2;3)	
f-33 SM1, al ² Cy sp ² /In(2LR)Pm, dp b ds ^{33k} ; Sb/Ins(3LR)Ubx ¹³⁰ , Ubx ¹³⁰ e ^S (2;3)	
f-34 stw ³ c; st (2;3)	
f-35 bw; ci sv ⁿ (2;4)	
f-36 bw; ci ^D /ey ^D (2;4)	
f-37 bw; ci ^D /simulans-4 (2;4)	

Inverted X Chromosomes

g-1 In(1)AM/T(1;3)65 y	b-9 In(1)CI, sc l t ² v sl B (ClB)	g-7 In(1)dl-49, y v f car/y f:=
g-2 In(1)AB, sc/y f:=	g-6 In(1)dl-49, y fan	g-8 In(1)dl-49, vOf f
g-3 In(1)AB, sc cv	b-12 In(1)dl-49, y Hw m ²	g-9 In(1)dl-49, ty-1
g-4 In(1)AB, y f	b-22 In(1)dl-49, y Hw m ² g ⁴	bb ¹ /y v f car
g-5 In(1)B ^{M1} , cm/y f:=	i-8 In(1)dl-49, y Hw m ² g ⁴ f ⁵	g-10 In(1)dl-49, y w B

g-11 In(1)dl-49, y w lz^S/y f:=
 g-29 In(1)dl-49, y w lz^S bb/In(1)sc^{L8}, sc^{L8}
 car m wa
 g-12 In(1)EN, y/y f:=
 g-13 In(1)EN, y bb/sc⁸.Y
 g-14 In(1)rst³, rst³
 g-15 In(1)rst³, rst³ f/y sc w^{col} spl f
 g-16 In(1)rst³, w⁴⁸⁻³ rst³ v f/y f:=
 g-17 In(1)rst³, y rst³ car bb?/RA, y²
 wa M-5/Y
 g-18 In(1)sc⁴, y sc⁴
 g-19 In(1)sc⁴, y sc⁴ cv v f
 g-20 In(1)sc^{4L}, sc^{8R}, y sc⁴⁺⁸ cv v f/y f:=
 g-21 In(1)sc⁸, sc⁸
 g-22 In(1)sc⁸ v f car
 g-23 In(1)sc⁸, sc⁸ cv v f/In(1)dl-49,
 y Hw m²
 g-24 In(1)sc⁸, y^{31d} sc⁸ wa
 g-25 In(1)sc⁸, y^{31d} sc⁸ cv v f/y f:=
 g-26 In(1)sc⁸ (c.o.J-3L), y- ac- sc⁸ wa f/
 RA, y ac sc Pn---In(1)sc⁸.y sc⁸.Y
 g-27 Ins(1)sc⁸EN, y⁺ f y/y f:=
 g-28 Ins(1)sc⁸EN, y⁺ f y/sc⁸.Y/y w
 g-29 In(1)sc^{L8}, sc^{L8} wa m car/In(1)dl-49,
 y w lz^S bb
 g-30 In(1)sc^{L8L}, sc^{SLR}, sc^{L8} wa m car/
 In(1)dl-49, y w lz^S bb
 g-31 In(1)sc^{SLR}, sc^{8R}, sc^{SL} wa m car sc⁸
 g-32 In(1)sc^{SLL}, sc^{4R}, sc^{SL} cv v B/y w/
 sc⁸.Y
 g-33 In(1)sc^{SLL}, sc^{4R}, sc^{SL} wa m car
 y f:=
 n-4 In(1)w^{m4}, w^{m4}
 p-17 In(1)w^{m4L}, rst^{3R}, y w- rst³
 g-34 In(1)y^{3P}, y^{3P} B
 g-35 In(1)y⁴, y⁴
 g-36 In(1)sc^{V1} y⁺/Ins(1)sc⁸, dl-49,
 y^{31d} y f B
 g-37 In(1)sc^{V1}, y v.sc^{V1} y⁺/y² wa bb/sc⁸.Y
 g-38 Ins(1)dl-49, BM¹, sc v
 g-39 Ins(1)sc^{4L}, AB, sc^{8R}, y sc⁴⁺⁸/y f:=
 b-74 Ins(1)sc^{4L}, S, sc^{8R}, y sc⁴⁺⁸ wa B(S-5)
 /yl²⁵⁹/sc⁸.Y
 i-9 Ins(1)sc⁷, AM, sc⁷
 b-29 Ins(1)sc⁸, dl-49, y^{31d} sc⁸ wa lz^S
 B(FM1)
 g-40 Ins(1)sc⁸, dl-49, sc⁸ v f/y f:=
 g-36 Ins(1)sc⁸, dl-49, y^{31d} sc⁸ v f B
 h-37 Ins(1)sc⁸, dl-49, 3C-4EF, y^{31d} sc⁸
 dm B (FM4)
 g-57 Ins(1)sc⁸, dl-49, 3C-4EF, 15DE-20,
 y^{31d} sc⁸ dm B (FM6)
 g-41 Ins(1)sc⁸ (c.o.X J-3), S, y- ac- wa
 sc⁸/sc⁸.Y
 g-42 Ins(1)sc^{8L}, dl-49, sc^{SLR}, y sc- v B f/
 y¹²⁵⁹ w m f/sc⁸.Y
 g-43 Ins(1)sc^{8L}, S, y^{3P}, y^{SL} sc⁸ y^{3P}
 g-44 Ins(1)sc^{L8L}, sc^{8R}, sc^{L8+8} cv v car/
 y w/sc⁸.Y
 g-49 Ins(1)sc^{SL}, dl-49, sc^{SL} v/y f:=
 b-63 Ins(1)sc^{SL}, sl-49, sc^{SL} v f car
 g-46 Ins(1)sc^{SLL}, AB, sc^{4R}, sc^{SL} wa car/
 y f:=

g-47 Ins(1)sc^{SLL}, dl-49, sc^{8R}, sc^{SL+8} w m/
 w sn bb
 g-48 Ins(1)sc^{SLL}, dl-49, sc^{8R}, sc^{SL+8} v B
 car/ y f:=
 g-49 Ins(1)sc^{SLL}, dl-49, sc^{8R}, y sc^{SL+8} wa
 v B/y f:=
 g-50 Ins(1)sc^{SLL}, dl-49, sc^{8R}, y sc^{SL+8}
 wa v f/y f:=
 g-57 Ins(1)sc^{SLL}, S, sc^{8R}, sc^{SL+8} wa B
 (M-5)
 g-52 Ins(1)sc^{SLL}, S, sc^{8R}, sc^{SL+8} wa
 B bb¹/y f:=
 g-53 Ins(1)sc^{SLL}, S, sc^{8R}, y² sc^{SL+8}
 wa B/sc⁸.Y
 g-54 Ins(1)sc^{SLL}, S, sc^{8R}, y² sc^{SL+8} wa
 B
 g-55 Ins(1)sc^{SLL}, S, sc^{8R}, y^{54k7} ac^{54k7}
 sc^{SL+8} wa B/y f:=
 g-56 Ins(1)y^{3PL}, S, sc^{SLR}, y- ac- sc-/
 y f:=; Cy/sc¹⁹ⁱ
 g-57 In(1)481(12E-F;14B), y bb¹⁴⁸¹/
 FM6/sc⁸.Y
 g-58 In(1H)59(3-4), y 1⁵⁹/y w/sc⁸.Y
 g-59 In(1H)132(4E) y 1¹³²/y w/sc⁸.Y
 g-60 In(1H)146(4D), y 1¹⁴⁶/y w/sc⁸.Y
 g-61 In(1H)227(1F), y 1²²⁷/y w/sc⁸.Y
 g-62 In(1H)231(SC-D), y 1²³¹/y w/sc⁸.Y
 g-63 Imp(1)139(3C), y w^{m139} 1¹³⁹/FM6,
 y^{31d} sc⁸ dm B
 g-64 Imp(1)139, w^{m139}, rst^m 1¹³⁹/y w f/
 Y/Y
 g-65 In(2LR)1t^{m3}(60D)/SM5
 g-66 In(2LR)1t^{m12}(60D)/SM5
 g-67 In(3R)18/Xa
 g-68 In(3R)112

Deficiencies and Duplications

p-1 Df(1)N⁸/In(1)dl-49, y Hw m²
 b-29 Df(1)rst²/FM1, y^{31d} sc⁸ wa lz^S B
 g-42 Df(1)sc (see Ins(1)sc^{8L}, dl-49,
 sc^{SLR})
 p-2 Df(1)sc¹⁰⁻¹/y Hw
 p-17 Df(1)w (see In(1)w^{m4L}, rst^{3R})
 p-3 Df(1)w²⁵⁸⁻⁴⁵/FM4, y^{31d} sc⁸ dm B
 p-3a Df(1)w²⁵⁸⁻⁴⁵, y; Dp(1;3), w^{Vco}/y w f
 p-3b Df(1)w²⁵⁸⁻⁴⁵, y spl dm; Dp(1;3),
 w^{Vco}/y w f
 p-4 Df(1)w²⁵⁸⁻⁴⁸, y sc⁵ spl; Dp(1;3),
 w^{Vco}/y f:=
 p-5 Dp(1;f)3 = Del(1)3/y XY^L.Y^S, y 1²⁵⁹
 w Y^L.Y^S
 p-6 Dp(1;f)18 - Del(1)18/y v f/XY^L.Y^S, y
 1²⁵⁹ w Y^L.Y^S
 b-19 Dp(1;f)24 = Del(1)24
 p-7 Dp(1;f)52 = Del(1)52/y v f/y 1²⁵⁹
 w Y^L.Y^S
 p-8 Dp(1;f)112 - Del(1)112/y v f/y 1²⁵⁹
 w Y^L.Y^S
 p-9 Dp(1;f)122 - Del(1)122/y v f/y 1²⁵⁹
 w Y^L.Y^S
 p-10 Dp(1;f)164 - Del(1)164/y v f/y 1²⁵⁹
 w Y^L.Y^S

p-11 Dp(1;f)1492 = Del(1)1492/sc^{53k}
 p-12 Dp(1;f)1514 = Del(1)1514/sc^{53k}
 p-13 Dp(X^c;f)6 = Del(X^c)6/RA 1(1)J1.sc⁸/
 Y^SX.Y^L, In(1)EN, y
 p-14 Dp(1;1)B^S(RAG), B^S--In(1)sc⁸./Ins(1).
 sc⁷, AM
 p-15 Dp(1;1)B^S(TMG), In(1)sc⁴.B^S, y sc⁴ m
 f.B^S/Ins(1)sc⁷, AM
 p-16 Dp(1;1)B^S(TMG), In(1)sc⁸L, X^c2R.B^S,
 f.B^S/X^D/B^SY^L.Y^S
 h-3 Dp(1;2)sc¹⁹ⁱ
 p-17 Dp(1;2R)w^{51b7}/y w f/In(1)w^{m4L}, rst^{3R},
 y w⁻ rst³
 p-18 Dp(1;3)w^{49a7} (Spotter)
 p-19 Dp(1;3)51 - T(1;3)51/y v f/XY^L.Y^S, y
 1259 w Y^L.Y^S
 p-20 Dp(1;3)142 = T(1;3)142/y/XY^L.Y^S, y
 1259 w Y^L.Y^S
 p-21 Dp(1;3)sc^{J4}/y v f/0 ?/XY^L.Y^S, y
 1259 w Y^L.Y^S
 p-22 Dp(1;4)w^{51c20}/y w f:=/In(1)w^{m4L}, rst^{3R}
 car rst³
 p-23 Dp(1;4)174 = T(1;4)174/y v f/y
 1259 w Y^L.Y^S
 p-24 Df(4)M-4/ey^D

h-33 T(1;4)11(15A), y 1¹¹/y w/sc⁸.Y
 h-34 T(1;4)In(1)sc^{4L}, sc^{8R}, y sc⁴⁺⁸ wa m
 car/y f:=
 h-35 T(1;4)w²⁵⁸⁻¹⁸
 h-36 T(1;4)w^{m5}(3C3), w^{m5}
 h-37 Ts(1;4)w^{m5}.B^S(3C3,16A1), w^{m5} v B^S/
 FM4, y^{31d} w dm f
 h-38 T(1;4)(4C3)/y f:=
 h-39 T(1;4)(13B809)/y f:=
 h-40 T(XY^L.Y^S;4)B^S(16A1), X^D, B^SY^L.Y^S/
 y v bb:=/0
 h-41 T(Y;2)E(36D)/y² su-w^a w^a bb
 h-42 T(Y^S;4)
 h-43 T(2;1)223(41-50;14), y 1223/FM6
 h-44 T(2;3)bw^{V4}; bw^{V4}/Cy
 h-45 T(2;3)bw^{V5}; bw^{V5}/Cy
 h-46 T(2;3)lt^{m7}(98C), lt^{m7}/SM5
 h-47 T(2;3)S^M; S^M Cy/vgnw
 h-48 T(2;3)S^M, S^M Cy C₃G Sb Ubx/st
 C₂G ca
 h-49 T(2;3)Xa/In(3LR)Ubx¹³⁰, Ubx¹³⁰ e^S
 h-50 T(3;1)05, D
 h-51 T(3;4)86D, bx^{34e} e⁴
 h-52 T(3;4)88B, Ubx/ey^D
 h-53 T(3;4)89E, ss bx bxd/ey^D

Translocated Chromosomes

h-1 T(1;2)459, y 1⁴⁵⁹/FM6, y^{31d} sc⁸ dm B
 h-2 T(1;2)Bld/ClB
 h-3 T(1;2)sc¹⁹/y f:=; fes sc¹⁹ⁱ b pr/
 Cy dpTH pr
 h-4 T(1;1H)25(20), y 125.FM6
 h-5 T(1;1LH)150(16-17), y 1150/FM6
 h-6 T(1;2LH)219(10A), y 1219/FM6
 h-7 T(1;2RH)75(20), y 175/FM6
 h-8 T(1;2RH)135(18-19), y 1135/FM6
 h-9 T(1;2;3)220(14A;50A;75), y 1220/
 FM6
 h-10 T(1;2;3;4)454, y 1454/FM6
 h-11 T(1;2H)361(20), y 1361/FM6
 h-12 T(1;3H)453(12D), y 1453/FM6
 h-13 T(1;3H)463(20), y 1463/FM6
 h-14 T(1;2LH)163(17A-A), y 1163/FM6
 h-15 T(1;3LH)455(3C), y 1455/FM6
 h-16 T(s;3RH)3(3-4), y 13/FM6
 h-17 T(1;3RH)129(18B), y 1129/FM6
 h-18 T(1;4)A7, y w/y² su-w^a w^a bb
 h-19 T(1;4)A13(18C5)
 h-20 T(1;4)A17(8A2)/y f:=
 h-21 T(s;4)A17(8A2), y cv/y f:=
 h-22 T(1;4)A19
 h-23 T(1;4)A20/y f:=
 h-24 T(1;4)B^S(16A1), B^S/y f:=
 h-25 T(1;4)B^S(16A1), B^S car/y f:=
 h-26 T(1;4)B^S(16A1), y B^S/y f:=
 h-27 T(1;4)B^S(16A1), y cv v B^S/y f:=
 h-28 T(1;4)B^S(16A1), y² cv v B^S car/
 y f:=
 h-29 T(1;4)B^{SL}; 11^R, y/y w
 h-30 T(1;4)e15
 h-31 T(1;4)h4
 h-32 T(1;4)h6

Closed X Chromosomes

i-1 X^c, y/y f:=
 i-2 X^c2 (ET), +/M-5
 i-3 X^c3 (KOA), +/M-5
 i-4 X^c2, wcol 491/In(1)dl-49, y Hw m² g⁴
 i-5 X^c2, wspot v f/RM, Ins(1) sc^{SLL}, S,
 sc^{8R}, sc^{S1} wa sc⁸/Y
 i-6 X^c2, y⁴⁹
 i-7 X^c2, y f car
 i-8 X^c2, In(1)w^{vc} (stable), w^{vc}/In(1)
 dl-49, y Hw m² g⁴ f⁵
 i-9 X^c2, In(1)w^{vc} (stable), w^{vc}/Ins(1)
 sc⁷, AM
 i-10 X^c2, In(1)w^{vc} (stable), w^{vc} f/y f:=
 i-11 X^c2, In(1)AB, +/y f:=
 i-12 X^c56K-1 (from RRL-26), y cv v f/
 y f:=/sc⁸.Y
 i-13 X^c56k-4 (from RRL-26), y ? ?/In(1)
 dl-49, y w lz^S/Y ? ?

X Chromosomes with a Y Arm Attached

j-1 X.Y^L (A-2), y w.Y^L?y/Y^m
 j-2 X.Y^L (C-2), y cv v f car bb⁻.Y^L/RA,
 (ND-27) v f/Y^m
 j-3 X.Y^L (C-2), y w bb⁻.Y^L/Y/Y^m
 j-4 X.Y^L (U-8e), sc cv v f.Y^L/Y/Y^m
 j-5 X.Y^L (U-8e), y w.Y^L/Y/Y^m
 j-6 X.Y^L (Stern), g² B.Y^L/Y/Y^m
 j-7 X.Y^L, y cv v f car .Y^L/Y/Y^m
 j-8 X.Y^L, y v f bb(bb⁺).Y^L/y f:=/sc^{V1}.Y^S
 j-9 X.Y^L, In(1)sc^{8L}, EN^R, y⁺ car f v cv
 y.Y^L/Y/Y^m
 j-10 X.Y^L, In(1)sc^{8L}, EN^R, y^{31d} f v cv.Y^L/
 Y^m

- j-11 X^YL (K-7), In(1)sc^{8L,ENR}, y⁺ f y^YL/y/Y^W
 j-12 X^YL (K-7), In(1)sc^{8L,ENR}, y⁺ f v cv y^YL/y/Y^W
 j-13 X^YL (P-8b), In(1)sc^{8L,ENR}, y⁺ f y^YL/y/Y^W
 j-14 X^YS (A-3), y w^YS/y v f/Y^{Lc}
 j-15 X^YS (A-3), sc cv v^YS/y v f/Y^{Lc}
 j-16 X^YS (Muller), y w^YS/y v f/Y^{Lc}
 j-17 X^YS (U-8c), y w^YS/y v f/Y^{Lc}
 j-18 X^YS (U-8c), y cv v^YS/Y^{Lc}
 j-19 X^YS (PDP), y² cv v^YS/In(1)d1-49, y^{Hw} m² g⁴/Y^{Lc}
 j-20 X^YS (P-8b), In(1)sc^{8L,ENR}, y⁺ f y^YS/y v f/Y^{Lc}
 j-21 Y^SS., (FR-1), Y^S y cv v f/y f:=/Y
 j-22 Y^SX., (Fr-1^L,In(1)p^R), Y^S y cv v f.y⁺/Y^{Lc}
 j-23 Y^SX., (Fr-1), Y^S y m f car/y v f:=/Y^{Lc}
 j-24 Y^SX. (P-0),In(1)EN, Y^S y/y w/Y
 j-25 Y^SX. (P-7), In(1)EN, Y^S y f/y v f/Y

Attached XY Chromosomes

- m-1 XY^L.Y^S (2-10T13 Parker), y² su-wa wa Y^L.Y^S/y/Y
 m-2 XY^L.Y^S (2-10T15 Parker), y² su-wa wa Y^L.Y^S/y/Y
 m-3 XY^L.Y^S 108-9 Parker), y² su-wa wa Y^L.Y^S/y v bb/0
 m-4 XY^L.Y^S (112-17 Parker), y² su-wa wa Y^L.Y^S/y v bb/0
 m-5 XY^L.Y^S (127-29 Parker), y² su-wa wa Y^L.Y^S/y v bb/0
 m-6 XY^L.Y^S (129-11 Parker), y² su-wa wa Y^L.Y^S/y v bb/0
 m-7 XY^L.Y^S, y 1(1)259 w Y^L.Y^S/y/Dp(1;f)167
 m-8 XY^S.Y^L (110-8 Parker), y² su-wa wa Y^S.Y^Ly⁺/y v bb/0
 m-9 XY^S.Y^L (115-9 Parker), y² su-wa wa Y^S.Y^Ly⁺/y v bb/0
 m-10 XY^S.Y^L (129-16 Parker), y² su-wa wa Y^S.Y^Ly⁺/y v bb/0
 m-11 Y^SX.Y^L (FR 1^L,C-2R), Y^S y bb.Y^L/y² su-wa wa bb/0
 m-12 Y^SX.Y^L (FR-1^L,U-8d^R), Y^S y wa cv v f.Y^L/y² su-wa wa bb/0
 m-13 Y^SX.Y^L, In(1)EN, Y^S B f v y.Y^L y⁺/y v bb/0
 m-14 Y^SX.Y^L, In(1)EN, Y^S B f v w y.Y^L y⁺/y² su-wa wa bb/0
 m-15 Y^SX.Y^L, In(1)EN, Y^S B y.Y^L/y² su-wa wa bb/0
 m-16 Y^SX.Y^L, In(1)EN, Y^S y.Y^L/y² su-wa wa bb/0
 m-17 Y^SX.Y^L, In(1)EN, Y^S v cv y.Y^L y⁺/y² su-wa wa bb/0
 m-18 Y^SX.Y^L, In(1)EN, y⁺ Y^S y.Y^L y⁺/y² su-wa wa bb/0
 m-19 Y^SX.Y^L, In(1)EN, Y^S y.Y^L y⁺
 m-20 Y^SX.Y^L, Ins(1)EN,17, Y^S B f v y.Y^L y⁺/y f:=/Y
 m-21 Y^SX.Y^L, Ins(1)EN,18, Y^S B f v y.Y^L y⁺/y f:=/Y
 m-22 Y^SX.Y^L, Ins(1)EN,20, Y^S B f v y.Y^L y⁺/y f:=/Y
 m-23 Y^SX.Y^L, Ins(1)EN,24, Y^S B f v y.Y^L y⁺/y f:=/Y
 m-24 Y^SX.Y^L, Ins(1)EN,24^L,A-2^R, Y^S y v.Y^L/y² su-wa wa bb/0
 m-25 Y^SX.Y^L, Ins(1)EN,32, Y^S B f v y.Y^L y⁺/y f:=/Y
 m-26 Y^SX.Y^L, Ins(1)EN,39, Y^S B f v y.Y^L/y f:=/Y
 m-27 Y^SX.Y^L, Ins(1)EN,42, Y^S B f v y.Y^L y⁺/y f:=/Y
 m-28 Y^SX.Y^L, Ins(1)EN,44, Y^S B f v y.Y^L y⁺/y f:=/Y
 m-29 Y^SX.Y^L, Ins(1)EN,46 Y^S B f v y.Y^L y⁺/y² su-wa wa bb/0
 m-30 Y^SX.Y^L, Ins(1)EN,d1-49, Y^S car f v y.Y^L/y² su-wa wa bb/0
 m-31 Y^SX(Y^L) (FR-1^L,3-18^R), Y^S y.(Y^L.bb⁺) RA,y² su-wa wa---M-5/Y
 m-32 Y^SX(Y^L) (FR-1^L,118-2b^R#1), Y^S y.(Y^L.bb⁺)/y² su-wa wa bb/0
 m-33 Y^SX(Y^L) (FR-1^L,118-2b^R#20), Y^S y cv (Y^L.bb⁺)/y² su-wa wa bb/0
 m-34 Y^SX(Y^L) (FR-1^L,118-2b^R#?), Y^S y cv (Y^L.bb⁺)/y² su-wa wa bb/0
 m-35 Y^SX.Y^L (FR-1^L,174-13^R), Y^S y cv v bb Y^L/RA y² su-wa wa---M-5/Y
 m-36 Y^SXY^S.Y^L (FR-1^L,115-9^R), Y^S y cv Y^S.Y^L y⁺/y² su-wa wa bb/0

Compound X Chromosomes

- k-1 RA, 1(1)J1 sc^{J1}--In(1)sc⁸.XY^L.Y^S, y 1259^w Y^L.Y^S/y sc⁸.Y
 j-2 RA(ND-27), sc y f--In(1)sc⁸, f v sc⁸./X.Y^L(C-2), y cv v f car bb⁻.Y^L/Y^W
 n-12 RA, y--In(1)sc⁸./Y^SX.Y^L, In(1)EN, Y^SB y.Y^L/y⁺ ac⁺.Y^L
 g-26 RA, y ac sc pn--In(1)sc⁸.

- k-2 RA (ND 9-3), $sc^{8L}/Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} f y \cdot Y^L/y^+ ac^+ \cdot Y^L$
 k-3 RA, $y--In(1)sc^{8L}$, $EN^R \cdot Y^L/Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} y \cdot Y^L/y^+ ac^+ \cdot Y^L$
 k-4 RA, In(1)AB, $y--In(1)sc^{8L}/Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} y \cdot Y^L/y^+ ac^+ \cdot Y^L$
 n-14 RA (Muller), In(1)dl-49, $y w f--In(1)sc^{8L} f sc^{8L}/XY^S$, $y w \cdot Y^S/Y^L \cdot bb^+ ac^+ y^+$
 p-22 RA (Muller), In(1)dl-49, $y w f--In(1)sc^{8L} f w sc^{8L}/Dp(1;2R)w^{51b7}/Y/In(1)w^{m4L}$, rst^{3R}
 k-5 RA (ND-33), $y f car--In(1)sc^{8L}$, $car f sc^{8L}/Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} y \cdot Y^L/y^+ ac^+ \cdot Y^L$
 m-31 RA, $y^2 su-w^a wa--Ins(1)sc^{S1L}$, S, sc^{8R} , B $wa sc^{8L}/Y^S X(Y^L)$, $Y^S y(Y^L \cdot bb^+)/Y$
 n-6 RA, $y^{1259} w--In(1)sc^{8L}/y^{1259} w Y^L \cdot Y^S/y sc^{8L} \cdot Y$
 h-7 RA, Y^L , $+--In(1)sc^{8L}$, EN^R , $y \cdot Y^L y^+/Y^S X \cdot Y^L$, In(1)WN, $Y^{SB} y \cdot Y^L/y^+ ac^+ \cdot Y^L$
 j-1 RM, $y/X \cdot Y^L(A-2)$, $y w \cdot Y^L/Y^S$
 m-9 RM, $y v bb$
 n-13 RM, $y v f/X \cdot Y^S$, $y w \cdot Y^S/ac^+ y^+ \cdot Y^L$
 b-35 RM, $y v f car$
 n-16 RM, $y w/X \cdot Y^S$, $y w \cdot Y^S/Y^L bb^+ ac^+ y^+$
 n-17 RM, $y^2 su-w^a wa bb/X \cdot Y^S$, $y w \cdot Y^S/Y^L bb^+ ac^+ y^+$
 i-5 RM, $Ins(1)sc^{S1L}$, S, sc^{8R} , $sc^S wa sc^{8L}/Xc^2$, $wspont v f$
 k-8 RM (13-0-15=XYL.X), $y^2 su-w^a wa bb Y^L/y^2 su-w^a wa bb? bb^+/Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} y \cdot Y^L$
 k-9 RM (15-DRP=XYL.YLX), $y^2 su-w^a wa bb Y^L/y^2 su-w^a wa bb? Y^? bb^+/Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} f v y \cdot Y^L y^+$
 k-10 RM, In(1)EN, $Y^S y/O \cdot Y^S X \cdot Y^L$, Ins(1)EN, dl-49, $Y^S car f v y \cdot Y^L$
 k-11 RM(L-6), $Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} y \cdot Y^L$, originally $In(1)sc^{S1L}$, EN^R , $sc^{S1} car m y/In(1)sc^{8L} f v cv sc^{8L}$
 k-12 RM (L-26), $Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} y \cdot Y^L$, originally $In(1)sc^{S1L}$, EN^R , $sc^S car m y/In(1)sc^{8L} f v cv sc^{8L}$
 k-13 RM (TAX), In(1) y^4 , $wa y^4/y v f/Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} y \cdot Y^L/O$
 k-14 TM Hw f), originally $y Hw v f \cdot y^+ cv f y^+/X \cdot Y$, $y B$
 k-15 TM, In(1)dl-49, In(1)EN R , sc^{4L} , $y v f? \cdot y m sc^4 y$ (stabilized by a 1-2 crossover)
 B/Y

Altered Y Chromosomes

- n-1 $sc^{8L} \cdot Y (y^+ ac^+ Y^L \cdot bb^+ Y^S)/y w/y$
 n-2 $sc^{8L} \cdot Y:bw^+ (Y^L bw^+ \cdot bb^+ Y^S ac^+ y^+)/y f:=/y v f$
 n-3 $sc^{8L} \cdot Y (y ac^+ Y^L \cdot bb^+ Y^S)/Muller-5$
 n-4 $Y^{bb-}/In(1)w^{m4}$, w^{m4}
 n-5 $Y^{bb-}/y^2 eq$
 n-6 $Y^{BS} (BS Y^L \cdot bb^+ Y^S)/y^2 su-w^a wa bb$
 n-7 $Y^{BS} sc^{8L} (BS Y^L \cdot bb^+ Y^S y^+)/y v; bw$
 n-8 $Y^{Su-Var}/In(1)w^{m4}$, w^{m4}
 n-9 $Y:bw^+ (Y^L bw^+ \cdot bb^+ Y^S)/y v; bw$
 n-10 $Y y^{31d} BS/y w f/Y^S y cv v f$
 n-11 $Y^c:bw^+ (MYR)/y v; bw$
 n-12 $y^+ ac^+ Y^L (FR-2)/Y^S X \cdot Y^L$, In(1)EN, $Y^{SB} y \cdot Y^L/RA$, $y--In(1)sc^{8L}$
 n-13 $Y^L \cdot ac^+ y^+ (sc^{8L} EN c \cdot o \cdot Y B-2)/X \cdot Y^S$, $y w \cdot Y^S/y v f$
 n-14 $Y^L \cdot bb^+ ac^+ y^+ (sc^{8L} EN c \cdot o \cdot Y T-0)/X \cdot Y^S$, $y w \cdot Y^S/y f:=$
 n-15 $Y^L \cdot bb^+ ac^+ y^+ (sc^{8L} c \cdot o \cdot Y T_0)/X \cdot Y^S$, $y w \cdot Y^S/y f:=$
 n-16 $Y^L \cdot bb^+ ac^+ y^+ (sc^{8L} EN c \cdot o \cdot Y U-8)/X \cdot Y^S$, $y w \cdot Y^S/y w$
 n-17 $Y^L \cdot bb^+ sc^{S1} ac^+ y^+ (sc^S c \cdot o \cdot Y CY9)/X \cdot Y^S$, $y w \cdot Y^S/y^2 su-w^a wa bb$
 n-18 $Y^L \cdot bb^+ sc^{S1} ac^+ y^+ (sc^S c \cdot o \cdot Y EY80)/X \cdot Y^S$, $y w \cdot Y^S/y v f$
 j-15 $Y^L c/X \cdot Y^S(A-3) sc cv v \cdot Y^S/y v f$
 j-1 $Y^S \cdot Y^S (Y^S Stern)/y/X \cdot Y^L(A-2)$, $y w \cdot Y^L$
 j-8 $Y^S \cdot sc^{V1} ac^+ y^+ (sc^{V1} \cdot Y^S)/X \cdot Y^L$, $y v f bb(bb^+ \cdot Y^L)/y f:=$

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Department of Chemotherapy

Wild Stocks

a3 + Crimea	a8 + Seto, Japan
a4 + Florida-9	a9 + Swedish-b-6
a1 + Amherst 3 (homoz. Singh, 1939)	a5 + Lausanne-S
a2 + Canton-S, A (iso, 1952)	a6 + Oregon R
	a7 + Samarkand
	a10 + Urbana-S
	a11 + Wageningen

Chromosome 1 (X)

b1	ac ³ wa ^a .Dp(sc ^{V1} y ⁺) & y f:=	b13	br we ec rb t ⁴ /FM1, y ^{31d} sc ⁸ wa lz ^s B	b27	cv f
b2	amx/FM3, y ^{31d} sc ⁸ dm B ¹	b14	Bx	b28	cx
b3	amx ⁵⁵	b15	Bx ²	b29	cx ^{tg} t/FM1, y ^{31d} sc ⁸ wa lz ^s B
b4	Ax	b16	Bx ³	b30	Df(1)259-4/FM4, y ^{31d} sc ⁸ dm B
b5	B	b17	Bx ^J	b31	Df(1)260-1/FM4, y ^{31d} sc ⁸ sm B
b6	B Bx ^r car & y f:=	b18	Bx ^{r49k} & y f:=	b32	Df(1)g ¹ f B/InAM
b7	B car su ^W -f & y w f:=	b19	car	b33	Df(1)svr, Dp(1;f)101 (Dp. het. or hom.)
b8	BM2 f ^{B27} /CLB (mosaic in f/f ²⁷)	b20	car bb	b34	dor/CLB
b9	Bg B/InAM	b21	cm	b35	dow/y Hw In ⁴⁹ m ² g ⁴
b10	bi ci ⁶ g ²	b22	cm ct ⁶	b36	dm & y f:=
b11	bo	b23	cm ct ⁶ sn ³ & y w f:=		
b12	br	b24	cs ⁵³ & y w bb•=		
		b25	ct ⁶ v dy g f/InA99		
		b26	ct ⁿ oc/FM1, y ^{31d} sc ⁸ wa lz ^s B		
	b37	Dp(1;f)101	In(1)sc ⁸ , Df(O+ac)wa ^a sc ⁸ ; Dp(1;f)101		
	b38	Dp(1;f)107	In(1)sc ⁸ , Df(O+ac)wa ^a sc ⁸ ; Dp(1;f)101		
	b39	Dp(1;f)118	In(1)sc ⁸ , Df(O+ac)wa ^a sc ⁸ ; Dp(1;f)118		
	b40	Dp(1;f)135	In(1)sc ⁸ , Df(O+ac)wa ^a sc ⁸ ; Sp(1;f)135		
	b41	Dp(1;f)135, y ² ; y w bb			
	b42	Dp(1;f)Xc ²	Dp(s;f)Xc ² /y l(1)7/y l(1)7		
	b43	Dp(1;f)z ⁹	Dp(1;f)z ⁹ ; Df(1)sc ^{J4R} & y f:=		
	b44	Dp(1;1)112	y f, Dp(1;1)112 (homozygous stock)		
	b45	Dp(1;1)Co			
	b46	Dp(1;1)Co, Df(1)rst ² & y w bb•=			
	b47	Dp(1;Y ^L)sc ^{S1}	sc ^{S1} .Y ^L /y.Y ^S ; y f:=; cn bw; (e/+)		
	b48	Sp(1;3)126	v f; Dp(1;3)126/Payne, Dfd ca		
	b49	Dp(1;3)sc ^{J4}	Dp(1;2)sc ^{J4} /Df(1)sc ⁸ , wa		
	b50	dy			
b51	ec	b77	g ² pl.FM3, y ^{31d} sc ⁸ dm B ¹	b102	lz/FM3, y ^{31d} sc ⁸ dm B ¹
b52	ec ct ⁶ v g ³ /CLB	b78	g ² ty & y•=	b103	lz ³ & y f:=
b53	ec dx	b79	gim/y sc ^{S1} B InS	b104	lz ³ m & y w f:=
b54	ec dx/y su-Hw Hw ² In ⁴⁹ m ² g ⁴	b80	g ^x .Inh & y f:=	b105	lz ^{34k} & y f:=
b55	Ext/FM6, y ^{31d} sc ⁸ dm B	b81	gg ² /FM6, y ^{31d} sc ⁸ dm B	b106	lz ^{37h}
b56	f B car su ^W -f & y f:=	b82	gg ³	b107	lz ^{48f} & y f:=
b57	f B odsy car	b83	gt bb ¹¹ /CLB	b108	lz ^{BS} lz ^{46f} ras ⁴ v & y f:=
b58	f B odsy f ⁺ ih & y f:=	b84	gt v	b109	m
b59	f B ³ & y f:=	b85	gt wa	b110	m ^D /FM3, y ^{31d} sc ⁸ dm B ¹
b60	f B ¹ (Luce 436.1) & y f:=	b86	gt wa (Oregon-R)	b111	ma-l & y f:=
b61	f BiBi & y f:=	b87	Hw ^{49c} /FM1, y ^{31d} sc ⁸ wa lz ^s B	b112	M(1)o f/InAM
b62	f BB & y f:=	b88	if ³	b113	M(1)Sp/InAM
b63	f BB ^{36b} & y f:=	b89	InAB & y f:=	b114	na & y f:=
b64	f fu/CLB	b90	In49 B ^{M1}	b115	ny f/FM1, y ^{31d} sc ⁸ wa lz ^s B; (ri)
b65	f fu & y f:=	b91	In49 v Fl g & y w f:=	b116	N ⁸ /y Hw In49 m ² g ⁴
b66	f od car	b92	In49 lz ^s & y f:=	b117	N ²⁶⁴⁻³⁹ wch/FM4, y ^{31d} sc ⁸ dm B
b67	f ⁵ odsy f ⁺ ih & y w f:=	b93	In49 m v sn ^{x2} g/y CLB	b118	N ²⁶⁴⁻¹⁰⁵ (dm)/y Hw In49 m ² g ⁴
b68	f ⁵ su ^W -f	b94	In49 oc ptg & y f:=	b119	oc ptg ³ .Dp(sc ^{S1} y ⁺)/ CLB
b69	f ^{36a}	b95	In49 sn ^{x2} & y f:=	b120	oc ptg Tu/sc ^{S1} fu In49 sc ⁸
b70	f ^{36a} odsy f ⁺ ih & y f:=	b96	In49 v sn ^{x2} B & y f:=		
b71	f ^x car & y f:=	b97	In49 v ^{Of}		
b72	fa	b98	In(X ^{c2})w ^{vc} /y Hw In49 m ² g ⁴ f ⁵ (ring stabilized)		
b73	flp	b99	kz	b121	od
b74	(Triploid) FM4, y ^{31d} sc ⁸ dm B/y ² sc wa ec•=	b100	l(1)7/FM6, y ³⁴ sc ⁸ dm B	b122	od Dp(f ⁺ ih) & y f:=
b75	fo	b101	lh B car bb & y f:=	b123	pa/FM4, y ^{31d} sc ⁸ dm B
b76	g ²				

b124 p^{eb} v
 b125 ("bleached") pⁿ w r^b
 c^m c^t6 sⁿ3 r^as² v d^y
 g² f c^ar & y f:=
 b126 pⁿ, In^h 1/y Hw In⁴⁹
 m² g⁴
 b127 pⁿ2
 b128 p^tg²
 b129 r³⁹k f B/InAM
 b130 r⁹ & y f:=
 b131 r^as d^y
 b132 r^as²
 b133 r^as³ m
 b134 r^as⁴ m/CLB
 b135 r^b
 b136 r^b c^x
 b137 r^bS¹
 b138 r^g
 b139 rst2/FM1, y^{31d}
 s^c8 w^a l^zs B
 b140 rst3, In & y f:=
 b141 rst3, In m v c^t &
 y f:=
 b142 rst-(=rst2)/y Hw In⁴⁹
 m² g⁴
 b143 r^{ux}/FM6, y^{31d} s^c8 d^m B
 b144 r^{ux}2
 b145 s
 b146 s^{br} & y f:=
 b147 s^c
 b148 s^c c^ho
 b149 s^c c^t6 c^ar & y f:=
 b150 s^c c^v v d^wx/FM6,
 y^{31d} s^c8 d^m B
 b151 s^c c^v e^g
 b152 s^c c^v v f
 b153 s^c e^c c^v c^t6 f/
 FM3, y^{31d} s^c8 d^m B 1
 b154 s^c In⁴⁹ sⁿx² c^ar/s^c o^c
 p^tg s^d c^ar
 b155 s^c In⁴⁹ v B^{M1}
 b156 s^c o^c p^tg s^c c^ar/y In⁴⁹
 sⁿx².B^S(select B ♀)
 b157 s^c pⁿ3 g² Bx²=(g²
 reverted)
 b158 s^c t² v f Tu c^ar
 & y f:=
 b159 s^c w BBL, In.Y^S & y f:=
 b160 s^c z e^c c^t6
 b161 s^c z w^{17G2} e^c c^t6
 b162 Sc(Scotched eye)/y s^cS¹
 g In⁴⁹ m s^c8 (select
 Sc ♀)
 b163 s^cp t.
 b164 s^d f^{ex}/y s^c8 B f
 In⁴⁹ v
 b165 s^d; (se)
 b166 s^hf²
 b167 Sh2/FM1, y^{31d} s^c8 w^a
 l^zs B
 b168 sⁿ3
 b169 sⁿ3 l^z46f24 v & y f:=
 b170 sⁿ3 v B^{M1} & y w f:=
 b171 sⁿ4
 b172 sⁿ34e
 b173 sⁿ36a & y f:=
 b174 sⁿc/y In⁴⁹ m² g⁴
 b175 s^p-w
 b176 s^pl
 b177 s^pl r^b c^x & y f:=
 b178 s^pl r^bS²
 b179 s^ta & y f:=
 b180 s^ta/FM3, y^{31d} s^c8
 d^m B 1
 b181 s^u8-s v
 b182 s^u2-s w^a c^v t
 b183 s^u3-s c^v v f & y f:=
 b184 s^uS²-v-pr v & f B:=
 b185 s^uS²-v-pr v & y f:=
 b186 S^ux-dx dx
 b187 s^u-w^a w^a
 b188 s^vr
 b189 s^vr s^u-w^a w^a
 b190 s^vr w^a
 b191 s^vrPoi
 b192 s^w
 b193 s^x v^b2 s^y/InAM
 b194 s^y
 b195 t
 b196 t² v f
 b197 t³
 b198 Tu & y f:=
 b199 t^w/FM1, y^{31d} s^c8
 s^c8 w^a w^a l^zs B
 b200 uⁿ Bx² & y f:=
 b201 uⁿ4
 b202 v
 v203 v f s^uW-f
 b204 v f^{3N} c^ar
 b205 v r¹²
 b206 v² f^w
 b207 v^{36f}
 b208 v+(rev.v) B^{M2}
 b209 v+(rev.v) B^{M2}+(rev. B,
 rein.) f^{B15}(mosaic)
 b210 v+(rev.v) f^x Dp(f^{ih})
 b211 v^b
 b212 v^b213 w
 b214 w e^c
 b215 w Om⁴⁹ l^zs & y f:=
 b216 w, f
 b217 w^a=a^pr
 b218 w^a f³ o^ds^y f^{ih}
 & y f:=
 b219 w^a s^pl
 b220 w^a2
 b221 w^a3
 b222 w^a4
 b223 w^bf f⁵
 b224 w^bf²
 b225 w^bl
 b226 w^{Bwx}
 b227 w^ch w^y
 b228 w^co
 b229 w^co sⁿ2
 b230 w^co l
 b231 w^cp
 b232 w^e
 b233 w^e sⁿ/CLB
 b234 w^e2
 b235 w^ec³ (ecru)
 b236 w^h
 b237 wⁱ f³ nⁿN
 b238 wⁿ4(3C1-2&20)
 b239 wⁿ4 v w^mMc
 b240 wⁿ4w
 b241 w^mMc & y f:=
 b242 w^mMc f w^m4
 b243 w^mR7aH1
 b244 w^sat
 b245 w^t f^w
 b246 w^y
 b247 X^c1 y & y f:=
 b248 X^c2 c^v v f/CLB
 b249 X^c2 e^c f & y f:=
 (ring OK 1957)
 b250 X^c2 y B & y f:=
 (ring OK 1957)
 b251 X^c2 y f & y w f:=
 (ring OK 1957)
 b252 X^c2 y v & y f:=
 (ring OK 1957)
 b253 X^c3 (tm-ac)- s^c8 w^a
 In^S B & y f:=/s^c8.Y
 (ring from tandem X.X)
 b254 y
 b255 y ac d^vr(+) v b^b
 b256 (y ac)-⁵¹ f (from y f:=)
 & y f:=
 b257 y ac pⁿ In⁴⁹ v B^{M1}
 b258 ("tester-1") y ac pⁿ w
 r^b w^y2 g² & y f:=;
 s^c19ⁱ/Cy
 b259 y ac s^c pⁿ & y f:=
 b260 y ac s^c pⁿ w.Dp(s^cV1
 y⁺) & y f:=
 b261 y ac s^c pⁿ w s^pl r^b c^x
 & y f:=; (s^c19ⁱ(b pr)/)
 b262 y ac s^c v & y f:=
 b263 y ac v
 b264 y ac-⁵³ s^c/y Hw In⁴⁹ m²
 g⁴
 b265 y ac-⁵³ s^c B c^ar.Dp(ac⁺
 y⁺-tm)⁵³ & y f:=
 b266 y ac t².Dp(s^cS¹ y⁺ ac⁺)
 & y f:=
 b267 y Aa f:=♀ & B ♂
 b268 y B & y f:=
 b269 y c^t6 & y f:=
 b270 y c^t6 d^vr² v f & y f:=
 b271 y c^t6 f & ac³ w^a c^t
 f.+
 b272 y c^t6 f c^ar & y f:=
 b273 y c^t6 f.Dp(y⁺ s^cV1) & y
 f:=
 b274 y c^t6 t² v f c^ar
 b275 y c^t268-42/FM4, y^{31d}
 s^c8 d^m B
 b276 y c^tK

b277 y cv	b326 y w sn ³ f	b374 y ^{2s}
b278 y f B f ^{+ih} & y f:=	b327 y w spl	b375 y ^{2s} fw ^{34e}
b279 y Dp(y ⁺ sc ^{V1}) & y f:=	b328 y w t ² v f	b376 y ^{3d} & br ec.=
b280 y fa wy ² g ²	& y f:=	b377 y ^{3d} & y f:=
b281 y fa ⁿ In49	b329 y w-258-11 1/y Hw	b378 y ^{3P} , InB
b282 y fw ^{51g} & y f:=	In49 m ² g ⁴	b379 y ⁴ , In cv v f
b283 y Hw In49 B ^{M1} & y f:=	b330 y w-258-11 t ² v	b380 y ⁴ , In wa
b284 y Hw In49 m ² g ⁴ /	f/y sc ^{S1} B InS	b381 y ^{18c} H1
w ^e sn	b331 y w-258-45/y Hw	b382 y ^{34c}
b285 y In49 B ^{M1} & y f:=	In49 m ² g ⁴	b383 y ^{bg} ct ⁶ car
b286 y In49 f car & y f:=	b332 ("doubler") y wa (1?)	b384 y ^{td}
b287 y In49 Fl v g &	Dp(B ^S)/sc ^{S1} In49 v	b385 y ^{v2}
y w f:=	b333 y ²	b386 z w ^{11E4}
b288 y In49 sn ^{x2} B ^{M1}	b334 y ² cho ²	
& y f:=	b335 y ² cv v f	
b289 y In49 sn ^{x2} bb ¹ &	b336 y ² drv ² v	
y f:=	b337 y ² ec cv v f car	
b290 y In49 v f & y f:=	b338 y ² In49 lz ^s & y f:=	
b291 y In49 v f car	b339 y ² oc ptg B ^{M2} &	
& y f:=	y f:=	
b292 y In49 v ptg oc g ²	b340 y ² oc ptg g, Inh	
& y f:=	& y f:=	
b293 y lm1 w In49 f·Y ^S /sc ^{S1}	b341 y ² sc wa ec	
B InS	b342 y ² su-wa	
b294 y N ²⁶⁴⁻⁸⁴ /FM6, y ^{31d} sc ⁸	b343 y ² su-wa w	
dm B	b344 y ² su-wa wa	
b295 y oc & y f:=	b345 y ² su-wa wa spl	
b296 y pn	b346 y ² su-wa wa spl cv	
b297 y pn w cm ct ⁶ sn ³ oc	b347 y ² su-wa wa ²	
ras ² v dy g ² v dy g ²	b348 y ² su-wa wa ⁴	
f od car sw/y sc ^{S1} B	b349 y ² su-wa wbf	
In49 v	b350 y ² su-wa w ^{bl}	
b298 y pn ^{54c} spl	b351 y ² su-wa w ^{co}	
b299 y pn ^{54c} w spl	b352 y ² su-wa w ^{col}	
b300 y pn ^{54c} wa	b353 y ² su-wa wh	
b301 ("tester 3") y rb cn	b354 y ² su-wa wsat	
ras ² g ² & y f:=;	b355 y ² su ² -wa wa	
sc ¹⁹ⁱ /Cy	b356 y ² v	
b302 y rb cx B ^{M1} /y ClB	b357 y ² v f car	
b303 y rst ³ , In car bb	b358 y ² v f car su-f	
b304 y sc	& y f:=	
b305 y sc In49 v g·Dp	b359 y ² wa	
(sc ^{V1} y ⁺)	b360 ("tester-2") y ² wa	
b306 y sc In49 v g f	cm wy ² g ² car	
b307 y sc lz ^g v f & y f:=	& y f:=; sc ¹⁹ⁱ /Cy	
b308 y sc v g & y f:=	b361 y ² wa ct f·Dp(y ⁺	
b309 y sl ² bb ⁻ , In/InAM	sc ^{S1})/ClB	
b310 y sn oc & y f:=	b362 y ² wa ct mw f/y	
b311 y sn ³ bb	sc ^{S1} B InS	
b312 y t ² v f	b363 y ² wa ct ⁶	
b313 y v	b364 y ² wa ct ⁶ lz v f	
b314 y v & y f:=	& y f:=	
b315 y v car bb ⁻ , In/	b365 y ² wa cv m f ♂ &	
y InAM	y Aa f:= ♀	
b316 y v f ^x B & y w f:=	b366 y ² wa cv v f B	
b317 y v f ^x car	b367 y ² wa InS B	
b318 y v f ^x car su ^w -f	b368 y ² wa rb ^{S1}	
b319 y v f ^x Dp(f ^{+ih}) & y f:=	b369 y ² wa sn ⁵ B & y	
b320 y w	Aa f:=	
b321 y w Co & y f:=	b370 y ² wa spl	
b322 y w f	b371 y ² wa v	
b323 y w f·Dp(sc ^{S1} y ⁺)	b372 y ² wa w	
b324 y w In49 f	b373 y ² wy ² g ² (g ²	
b325 y w sn ³	Partly reverted?)	
		<u>Scute alleles</u>
		(listed alphabetically
		according to scutes
		regardless of position of
		scute in linear order:
		c1 sc ²
		c2 sc ² pn & y f:=
		c3 sc ³⁻¹ w & y f:=
		c4 sc ^{3B}
		c5 y sc ⁴
		c6 y sc ⁴ B f InS &
		y f:=
		c7 y sc ⁴ B InS & y f:=
		c8 y sc ⁴ B v ^{41b} /y w In49
		lz ^s
		c9 y sc ⁴ InS wa; S sc ¹⁹ⁱ
		B1/Cy L ⁴ sp
		c10 sc ⁵ bb ^{sc5}
		c11 y sc ⁵
		c12 y sc ⁵ w-258-48 spl;
		Dp(1;3)w ^{vco} ; y f:=
		c13 sc ⁶ car
		c14 sc ⁶ wa
		c15 sc ⁷
		c16 sc ⁷ InAM car/Df(1)
		B ²⁶³⁻²⁰
		c17 sc ⁷ oc ptg g, Inh &
		y f:=
		c18 sc ⁷ wa
		c19 sc ⁸
		c20 sc ⁸ B
		c21 sc ⁸ B f ^x v & y f:=
		c22 (w ^r -reddish) sc ⁸ B InS
		w ^r & y f:=
		c23 sc ⁸ bb wa
		c24 sc ⁸ car f In49 v &
		y f:=
		c25 sc ⁸ f In49 v & y f:=
		c26 sc ⁸ f v cv & y f:=
		c27 (w ^r -reddish) sc ⁸ InS w ^r
		c28 sc ⁸ Tu wa & y f:=
		c29 y ^{31d} sc ⁸ wa
		c30 (y ac)B ²⁷⁰ (dappled) sc ⁸
		B wa/w In49 lz ^s bb
		c31 y ^{S1} sc ⁸
		c32 y ^{S1} sc ⁸ B f In49 v
		c33 y ^{S1} sc ⁸ B f In49 v
		wa & y f:=

c34 y^{S1} sc⁸ B In49
 c35 y^{S1} sc⁸ f InS wa & y f:=
 c36 y^{S1} sc⁸ f³ sd & y w f:=
 c37 y^{S1} sc⁸ sn³ w
 c38 y^{OX} sc⁸ sn^{5.1} w & y f:=
 c39 sc⁹ Bx f t wa
 c40 sc¹⁰ wa
 c41 sc^{10-1/y} Hw
 c42 sc^{19-1/J1} sc^{J1}; fes sc¹⁹ⁱ b pr/Cy
 dptxl pr cn²
 c43 sc^{19-σ} & y f:=; fes sc¹⁹ⁱ b pr/
 Cy dptxl pr cn²
 c44 sc²⁸ wa
 c45 sc²⁹ wa
 c46 sc⁴⁵ l/y sc^{S1} B In49 v
 c47 sc²⁶⁰⁻¹⁴
 c48 sc²⁶⁰⁻²²
 c49 sc^{C/y} sc^{S1} B InS
 c50 y sc^{D1}
 c51 y sc^{D1}
 c52 sc^{H,TX4} & y f:=
 c53 LJ1 sc^{J1}/Del(sc7)2 & y f:=
 c54 LJ1 sc^{J1}/Del(X)24
 c55 LJ1 sc^{J1}/Del(X^C)Ag (Pontecorvo)
 c56 LJ1 sc^{S1} car·Dp(ac⁺ y⁺-tm)53 & y f:=
 c57 wm^{5L}; sc^{J4R} & y w f:= (wm^{5L/})♀
 c58 sc^{J6} B & y f:=
 c59 sc^{L3,TX4} (spoon-like)
 c60 sc^{L6}
 c61 sc^{Mc,TX3/y} Hw In49 m² g⁴
 c62 sc^{L8} car m wa^a/y w In49 lz^s
 c63 sc^{S1} B In49 oc ptg & y f:=
 c64 ("plex") sc^{S1} car f In49 v/y ac sc
 pn w rb cm ct⁶ sn³ ras² v dy g² f car
 c65 sc^{S1} f In49 v w & y f:=
 c66 y sc^{S1} B f In49 v & y f:=
 c67 y sc^{S1} B In49 sn^{x2} & y f:=
 c68 y sc^{S1} B InS & y f:=
 c69 sc^{S2,T(1;2)/Cy}
 c70 sc^{V1},Inp v/y^{S1} sc⁸ B f In49 v
 c71 sc^{V2}, Inh

sc^{S1}-sc⁸ (d14-d45)

d14 sc^{S1} At In49 sc⁸
 d15 sc^{S1} At In49 v wa sc⁸ & y f:=
 d16 sc^{S1} B g In49 m sc⁸ & y f:=
 d17 sc^{S1} B In49 lz^s sc⁸/y ac sc pn w v g f
 d18 ("Binsc") sc^{S1} B In49 sc⁸ & y f:=
 d19 ("Binsn ") sc^{S1} B In49 sn^{x2} sc⁸ &
 y f:=
 d20 ("Binsn") sc^{S1} B In49 sn^{x2} sc⁸/oc
 ptg Tu
 d21 (Basc") sc^{S1} B InS wa sc⁸
 d22 sc^{S1} car B In49 v sc⁸ & y f:=
 d23 sc^{S1} car m wa sc⁸/w In49 lz^s
 d24 sc^{S1} f In49 v wa sc⁸ & y f:=
 d25 sc^{S1} In49 m w sc⁸/y sn v
 d26 ("Insc") sc^{S1} In49 sc⁸
 d27 ("Insn") sc^{S1} In49 sn^{z2} sc⁸ & y f:=
 d28 sc^{S1} In49 v sc⁸ & sc v f.=
 d29 y sc^{S1} At In49 sc⁸/oc ptg
 d30 ("Binscty") y sc^{S1} B In49 ctns sc⁸
 d31 ("Binscy") y sc^{S1} B In49 sc⁸ & y f:=
 d32 ("new Binscy") y sc^{S1} B In49 sc⁸
 d33 y sc^{S1} B In49 sc⁸/oc ptg (H. Byers' 1
 in sc^{S1} chromosome, c-9-c4)
 d34 y sc^{S1} B In49 sn^{x2} v sc⁸ & y f:=
 d35 y sc^{S1} B In49 sn^{x2} w sc⁸ & y f:=
 d36 ("Bingscty-v") y sc^{S1} B In49 v ctns
 sc⁸
 d37 y sc^{S1} B In49 v sc⁸ & y w f:=
 d38 y sc^{S1} B In49 v wa sc⁸ & y f:=
 d39 ("winscyBx") y sc^{S1} Bx^M In49 w sc⁸
 d40 y sc^{S1} car odsy f sc⁸
 d41 y sc^{S1} f In49 v wa sc⁸ & y f:=
 d42 ("Inscy") y sc^{S1} In49 sc⁸
 d43 y sc^{S1} In49 v sc⁸
 d44 ("winscy") y sc^{S1} In49 w sc⁸
 d45 y sc^{S1} sc⁸
 d46 sc^{S1} f InS y^{3P} & y f:=
 d47 sc^{V2} B y^{3P}
 d48 (y ac)- y^{3P} sc⁸ (iso 2952)
 d49 (y ac sc)- y^{3P} InS sc^{S1} & y f:=;
 Cy/sc¹⁹ⁱ

Combination of scute or similar inversions

d1 y sc⁴ B InS wa sc⁸ & y f:=
 d2 y sc⁴ In49 sn^{x2} sc⁸ & y f:=
 d3 y sc⁴ B In49 lz^s v sc^{S1}/sc oc ptg
 sc car
 d4 y sc⁴ InS sc^{S1} (extra Y in ♀)
 d5 y sc⁴ InS sc^{S1}/CLB
 d6 y sc⁸ B InS wa sc⁴ & y f:=; sc¹⁹ⁱ/
 Cy cn²
 d7 y sc⁻(rein. sc⁸⁻⁴) wa InS B & y f:=;
 sc¹⁹ⁱ/Cy lt cn²
 d8 y sc⁻(rein. sc⁸⁻⁴) wa InS bb & y f:=
 sc¹⁹ⁱ/Cy lt cn²
 d9 y^{S1} sc⁸ B InS sc^{S1}/w sn^{5s} bb
 d10 y^{S1} sc⁸ InS y^{3P}; Cy/Scd
 d11 sc^{-17aH3} f car·sc^{V1}; Cy/sc¹⁹ⁱ ♂ &
 y f:=; Cy/sc¹⁹ⁱ ♀
 d12 sc^{L8} sc⁸ & y f:=
 d13 sc^{L8} g^s v lz^g sc⁸ & y f:=

Translocations of X and 4

e1 TX(1B3⁺)4 sc⁸ B wa
 e2 TX(3C2)4 wm⁵ & y f:=
 e3 TX(3C2)4 wm⁵ v f bb/wm⁵ CLB
 e4 TX(3C4)4 y wm^{258-18/y} Hw In49 m² g⁴
 e5 TX(3C5-6&7)4 N^{8a}/FM6, y^{31d} sc⁸ dm B
 e6 TX(3E5&6) w^{258-21/y} 31d sc⁸ B In49
 lz^s wa
 e7 TX(4c3)4 & y f:=
 e8 TX(9A1)4 & y f:=
 29 TX(9B&20)4 "W13"/CLB
 e10 TX(9B&20)4 "W13" car
 e11 TX(9B&20)4 "W13" sc v^m g/CLB
 e12 TX(9B&20)4 "W13" y w & y f:=
 e13 TX(11A7)4 & y f:=
 e14 TX(13B8-9)4 "Sidly a" & y f:=
 e15 TX(16A1)4 B^S & y w f:=
 e16 TX(16A1)4 B^S·Y^S/sc·Y^L & y w f:=

f54 sc⁸.Y/y sc w In49 v g f & y f:=
 f55 sc⁸.Y/y sc⁴ B f InS & y f:=
 f56 sc⁸.Y/y sc⁴ B f InS wa & y f:=
 f57 sc⁸.Y/y sc⁴ B InS & y f:=
 f58 sc⁸.Y/y sc⁴ B InS w^r sc⁸ & y f:=
 f59 sc⁸.Y/y sc⁴ f InS wa & y f:=
 f60 sc⁸.Y/y sc⁴ w sc⁸ (sc⁸.Y in ♂ & ♀)
 f61 ("Multipare D") sc⁸.Y/y sc^{S1} B InS/y Hw In49 m² g⁴; (ci gvl ey^R svⁿ)
 f62 sc⁸.Y/y sc⁻ (rein. sc⁸⁻⁴) B·Dp(sc^{S1} ac⁺ y⁺) & y f:=
 f63 sc⁸.Y/y v & y f:=
 f64 sc⁸.Y/y w^{m4}
 f65 sc⁸.Y/y² wa sn⁵ B & y N^M f:= (N with w)
 f66 sc⁸.Y/y^{S1} sc⁸ B f In49 v
 f67 sc⁸.Y·BS/lJ1 y & y ct⁶ f:=
 f68 sc⁸.Y·BS/sc⁸ B In49 w & y f:=; (ho ed cl/+)
 f69 sc⁸.Y·BS/y w^{m4} ras²
 f70 sc⁸.Y·BS/y w^{m4}; dp
 f71 sc⁸.Y·BS/y w^{m4}; (ho) ed cl
 f72 sc⁸.Y·BS/y² ct⁶ & y f:=
 f73 sc⁸.Y·BS/y² sd² & y f:=
 f74 sc⁸.Y·BS/y² w¹ & y f:=
 f75 sc⁸.Y·BS/y² w¹ ct⁶ & y f:=
 f76 sc⁸.Y·BS/y² w¹ ct⁶ f & y f:=
 f77 sc⁸.Y:bw⁺/ac³; bw
 f78 sc⁸.Y:bw⁺/ac³; cn bw
 f79 sc⁸.Y:bw⁺/sc⁸ B In49 w; bw
 f80 sc⁸.Y:w⁺/y wa
 f81 Y^L/f·Y^S & sc v f:=
 f82 Y^L/f·Y^S & y² wy² g² f:=
 f83 Y^Lc/InEN2·Y^S & y ct⁶ f:= (InEN2 from Xc² opened)
 f84 ("Y^Lc snocty") Y/oc ptg·Y^S & Y^Lc/y ctⁿ oc ptg car
 y ct¹, In In49 sn^{x2}).
 f85 Y^Lc/oc ptg·Y^S & y v f:=; S Sp ab² ltd/Cy, Ins cn²
 f86 ("Y^Lc snocty; bw") Y^Lc/oc ptg·Y^S & Y^Lc/y ctⁿ oc ptg car
 y ct¹, In In49 sn^{x2}). ;bw
 f87 Y^Lc/w oc ptg·Y^S & Y^Lc/y² X⁺·wa InS B sc⁸ (tandem X·X giving rings)

Sterilizer ("sz") stocks (f88-f97)

f88 ("sz w") Y^Lc/w·Y^S
 f89 ("sz +") Y^Lc/X·Y^S
 f90 ("sz bw") Y^Lc/X·Y^S; bw
 f91 ("sz bw e") Y^Lc/X·Y^S; bw; e
 f92 ("sz c") Y^Lc/X·Y^S & y v f:=; c
 f93 ("sz e") Y^Lc/X·Y^S & y v f:=; e
 f94 ("sz lz f") Y^Lc/lz³ f·Y^S & y v f:=
 f95 ("sz lz m f") Y^Lc/lz³ m f·Y^S & y v f:=
 f96 ("sz m f") Y^Lc/m f·Y^S & y v f:=
 f97 ("sz y w") Y^Lc/y w·Y^S & y ct⁶ f:=
 f98 Y^Lc/y v·Y^S; bw^{VA}/L² l
 f99 Y^Lc/y v B·Y^S & ac³ wa ct⁶ f:=
 f100 (new "facl", 1959) Y^Lc/y sn oc ptg·Y^Lc/y² oc ptg B^{M1}/sc^{S1} In49 sn^{x2} sc⁸
 f101 ("jynd") Y^Lc/y sn⁵ oc ptg v·Y^S & Y^Lc/sc^{J1} pn w rb cm ct⁶ oc ras² v dy g² f od car
 sw/y sc^{S1} B In49 sn^{x2} sc⁸ ♀
 f102 Y^Lc/y In49 v f·Y^S & y sc t² v f car·=
 f103 Y^Lc/y w sn⁵ oc ptg·Y^S & y v f:=
 f104 Y^Lc/y² oc ptg fu·Y^S & Y^Lc/y wa·=
 f105 sc·Y^L/oc ptg·Y^S & sc·Y^L/y f:=; Cy, Ins cn²/S Sp ab² ltd
 f106 sc·Y^L/sc w B·Y^S & y f:=; Cy, In/S Sp ab² ltd
 f107 sc·Y^L/sc w BBL, In·Y^S & y f:=
 f108 sc·Y^L/sc w ct⁶ f·Y^S & y f:=; Cy, In/S Sp ab² ltd
 f109 sc·Y^L/y ac sc ct⁶ f·Y^S
 f110 sc·Y^L/y In49 v f·Y^S

- f111 sc.Y^L/y In49 v f.Y^S;e
 f112 sc.Y^L/y sc⁻(rein.sc⁸⁻⁴).Y^S & y f:=
 f113 sc.Y^L/y.Y^S & y f:=; cn bw; (e)
 f114 sc.Y^L/y² v f.Y^S & y w f:=
 f115 sc.Y^L/y² wa ct⁶ f.Y^S ♂ & sc.Y^L/
 y² X⁺.sc⁸ wa InS B ♀ (tandem X-X
 giving rings)
 f116 y³.Y^L/sc w oc f.Y^S & y f:=
 f117 y³.Y^L/scV1- oc lz³.Y^S & y f:=
 f118 y³.Y^L/scV1- w.Y^S & y f:=
 f119 y³.Y^L/y ct⁶ oc lz.Y^S & y f:=
 f120 ("plond") y³.Y^L/y² oc lz.Y^S ♂ & y³.Y^L/
 y ac sc pn w rb cm ct⁶ sn³ oc ras
 v m g² f car/sc^{S1} B In49 lz^S sc⁸ ♀
 f121 Y^S/g² B.Y^L & y f:= (Stern) (dp^T)
 f122 Y^S/y ct⁶ f.Y^L & y f:=
 f123 Y^S.Y^S#2/y v f.Y^L & y f:=
 f124 sc^{V1}.Y^S/y In49 v.Y^L & y f:=
 f125 sc^{V1}.Y^S/y In49 v B.Y^L & y f:=
 f126 sc^{V1}.Y^S/y In49 v f B.Y^L & y f:=
 f127 sc^{V1}.Y^S/y In49 v ptg oc f B.Y^L &
 y f:=
 f128 sc^{V1}.Y^S/y v f bb⁺.Y^L & y f:=
 f129 TY2G/b pr (tk)
 f130 TH3(II4Aa³) l/ru h D InsCXF ca (TY3
 in and)
 f131 Tp⁴:Y (2 Y:4's in both ♂ & ♀)
 (Transpos. Edmondson)
 f132 Tp⁴:Y/Basc & y.=; ci ey^R
 f133 Tp⁴:Y/Cat/Cat ♂ & Cat/M⁴ ♀ (un-
 selected)
 f134 Tp⁴:Y/ci^D
 f135 Tp⁴:Y/ey^D or Cat ♂ & ey^D/Cat ♀
 f136 Tp⁴:Y/X^T & y f:=
- Chromosome 2*
- *S² and/or Cy are to be understood always to be accompanied by InCyL and cn² by InCyR even where not so designated. When cn is present InCyR is absent. Ins following S² or Cy after a comma refers to both of these inversions, but InL only to the left-arm one. If either of these inversions is designated in a chromosome without the other, the latter should be understood to be absent. InMis designates the long pericentric inversion of Mislove.
- g1 a px or
 g2 a px sp
 g3 ab
 g4 ab²/S² Ins(CyL,CyR) lc cn²
 g5 ab²/T(Y;2)E
 g6 ab² cn⁴ Pm¹/Cy pr Bl cn² L⁴ sp²
 g7 ab² ix² bw sp²/Cy, dpTh Bl L⁴ sp²
 g8 ab² InCyR L⁴ sp²/b InsNSL&R mr
 g9 ab² ms ta crs/Cy pr Bl cn² L⁴ sp²
 g10 abr/SM5, al² Cy lt^v sp²
 g11 ad
 g12 al
 g13 al b c sp
 g14 al b cn sp (iso)
- g15 al dp b bw l(2)ax/SM5, al² Cy lt^v sp²
 g16 al dp b pr
 g17 al dp b pr blt bw/SM5, al² Cy lt^v sp²
 g18 ("apl") al dp b pr c px sp
 g19 ("twelvepl") al dp b pr cn vg c a px bw
 mr sp/S² Cy lt³ pr⁺ Bl cn² L⁴ sp²
 g20 al S ast ho/Cy, En-S
 g21 al² Cy ab⁵¹g pr Bl cn² L⁴ sp²/S Sp
 cn bw sp
 g22 al² Cy, InL lt³/b pr Bl lt³ cn² InCyR
 L⁴ sp²
 g23 al² Cy pr Bl cn² InCyR c vg sp²/InsNS
 px sp
 g24 al² Cy pr Bl cn² L⁴ bw sp²/InsNS px sp
 g25 al² InMis dpt^{XI} Cy cn² L⁴ sp²/S Sp U,
 InLR bw
 g26 al² InMis dpt^{XI} Cy pr Bl cn² L⁴ sp²/
 S Sp U, InLR bw
 g27 Alu
 g28 an/SM5, al² Cy lt^v sp²
 g29 ang
 g30 ant; (ro)
 g31 ap⁴/Rvd, In2LR
 g32 ap⁴/SM5, al² Cy lt^v sp²
 g33 arch chl/SM5, al² Cy lt^v sp²
 g34 ast ho
 g35 ast ho ed dp cl
 g36 ast⁴ dp cl
 g37 ast⁴ dp cl sp
 g38 b
 g39 b cn bw
 g40 b el rd^S pr cn
 g41 b gp
 g42 b Go/Gla
 g43 b j
 g44 b l(2)Bld pr c px sp/SM5, al² Cy lt^v sp²
 g45 b lt bw
 g46 b lt l cn mi sp/b In(2)bwVDe1
 g47 b lt wxt bw
 g48 b nub pr
 g49 b pr
 g50 b pr Bl tk/S² Cy cn² L⁴ sp²
 g51 b pr c px sp
 g52 b pr tk
 g53 b pr tk/T(Y;2)G
 g54 b sf
 g55 b vg
 g56 Bl/esc
 g57 Bl/Cy, bw^{45a} sp² or^{45a}
 g58 Bl/In(2LR)dp
 g59 Bl bw^{VA}T(2;3)/Cy, InL L²
 g60 Bl L²/Cy, dp²
 g61 Bl stw³/In(2LR)dp
 g62 Bl stw⁴⁸ blt tuf/SM5, al² Cy lt^v sp²
 g63 Bla/SM5, al² Cy lt^v sp²
 g64 blo
 g65 blt
 g66 bran
 g67 br1
 g68 bs²
 g69 bw (iso 2, 1959)
 g70 bw ba
 g71 bw sp (iso 1954)

- g72 bw^D
 g73 bw^{2b}
 g74 bw⁴
 g75 bw⁵⁻/Cy cn² L⁴ sp²
 g76 bw⁵⁻/Cy, dp²
 g77 c
 g78 c bw
 g79 c px bw^D sp
 g80 c wt px
 g81 cg c/U, InLR
 g82 ch
 g83 chl
 g84 chl en/SM5, al² Cy lt^v sp²
 g85 chl 1(2)bw bw^{2b} mr²/SM5, al² Cy
 lt^v sp²
 g86 chy
 g87 ck/SM5, al² Cy lt^v sp²
 g88 cl
 g89 cl² px/T(Y;2)E
 g90 cn (iso 2)
 g91 cn bw
 g92 cn bw sp
 g93 cn px, InLR crs/S² dp^{tx1} Cy pr Bl
 cn² L⁴ sp²
 g94 cn sp (iso 1954)
 g95 cn² InCyR cg sp²/InsNS px sp
 g96 cn³ cg bw⁵⁻ mr/Cy cn² L⁴ sp²
 g97 cn³/T(Y;2)C
 g98 cn^{35k}
 g99 cg
 g100 cr-u/Cy; (we)
 g101 d/SM5, al² Cy lt^v sp²
 g102 da/SM1, al² Cy sp²
 g103 Df(2)42, en/Cy, al² lt³ L⁴ sp²
 g104 Df(2)al/Cy, En-S
 g105 Df(2)bwVDe2LCyR/Gla
 g106 Df(2)MB/SM1, al² Cy sp²
 g107 Df(2)MS4/SM1, al² Cy sp²
 g108 Df(2)MS8/Cy, al² lt³ Dp(2;2)41 L⁴ sp²
 g109 Df(2)MS8/SM1, al² Cy sp²
 g110 Df(2)MS10/Cy pr, Dp(2;2)41²
 g111 Df(2)MS10/SM1, al² Cy sp²
 g112 Df(2)Px/Df(2)P;Dp(2;3)P/In(3R)Mo,
 sr; we^e
 g113 Df(2)r1^{10a} lt cn/Cy
 g114 Df(2)r1^{10b} lt cn/Cy, al² lt³ L⁴ sp²
 g115 Df(2)S2/Cy, En-S
 g116 Df(2)S3;Dp(2;2)a, Cy, En-S
 g117 Dke c
 g118 dil² hv bw sp/SM5, al² Cy lt^v sp²
 g119 dp
 g120 dp b cn sp/al² Cy pr Bl cn² L⁴ sp²
 g121 dp b L⁴ Pm¹/IndpT23 b
 g122 dp bw⁵⁻ mr/al² InMis Cy cn² sp²
 g123 dp cn bw
 g124 dp⁰
 g125 dp⁰²
 g126 dp⁰³ ta sp/Cy pr Bl cn² L⁴ sp²
 (iso 2)
 g127 dp^{Nov}
 g128 dp^{Rf}/SM5, al² Cy lt^v sp²
 g129 dp^T ab² pr Bl rnT23 InNSR mr/al² Cy
 cn² L⁴ sp²
 g130 sp^T Sp ab² cn bw sp/S² ls Cy pr
 InsL&R cn² bw sp
 g131 dp^T Sp cn bw sp/S² (ls⁺) Cy, InL cn
 bw sp
 g132 dp^T Sp cn InRSR mr/S² ls Cy pr Bl
 cn² L⁴ bw sp²
 g133 dp^{tx} b/Cy, Ins cn²
 g134 dp^{tx} b/SM5, al² Cy lt^v sp²
 g135 dp^{tx} Sp ab²/S² ls Cy, InCyL
 g136 dp^{tx} Sp b/S² ls, InCyL
 g137 dp^{tx} Sp b/S² ls, InCyL b
 g138 dp^{tx} Sp cn/S² Cy, InCyL cn
 g139 dp^{tx} Sp cn bw/S² Cy, InCyL cn bw
 g140 dp^{tx} Sp cn²/S² Cy cn² (homoz. InCyR)
 g141 dp^{txI} Cy Bl cn² L⁴ sp²/InNSL
 InNSR px sp
 g142 dp^{txI} Cy, InsO⁴ pr cn²/InsNS px sp
 g143 dp^{txI} Cy, InsO⁶ pr cn²/InsNS px sp
 g144 dp^v; vo-3 (2;3)
 g145 dp^{v2}
 g146 dp^{v1}/SM5, al² Cy lt^v sp²
 g147 ds dp
 g148 ds ft dp^{v2} 1(2)M b pr/SM5, al² Cy
 lt^v sp²
 g149 ds S G b pr/Cy, al² lt³ L⁴ sp²
 g150 ds^{38k}/Cy(2L), dp² b pr
 g151 ds^W/In(2L)Cy-t, Su-S sp² pr
 g152 dsr
 g153 dw-24F cl/SM5, al² Cy lt^v sp²
 g154 ed dp cl
 g155 ed Su²-dx
 g156 el
 g157 ex
 g158 ex ds s^X ast^X/SM1, al² Cy sp²
 g159 fes Alu lt/SM5, al² Cy lt^v sp²
 g160 fes dp^{tx} Sp/al² Cy lt³ (L⁴) sp²
 g161 fes IndpT23 b sp/al² Cy cn³ L⁴ sp²
 g162 fes ms cn sp/net dp^{txI} Cy b pr Bl
 lt³ cn² L⁴ sp² (iso 1957)
 g163 fes pr rnT23/al² Cy b cn² L⁴ sp²
 g164 fj 1(2)Su-H/SM5, al² Cy lt^v sp²
 g165 fj wt/SM5, al² Cy lt^v sp²
 g166 fr/Cy, sp²
 g167 fr² wt/SM5, al² Cy lt^v sp²
 g168 ft
 g169 Grv/SM5, al² Cy lt^v sp²
 g170 Gla, InLR/S² Cy cn² bw sp
 g171 hk
 g172 hk pr
 g173 ho
 g174 hv/SM5, al² Cy lt^v sp²
 g175 Hx
 g176 hy/SM5, al² Cy lt^v sp²
 g177 In(2)bwVDe1/b lt i cn mi sp
 g178 In(2)bwDe2/Rev.1
 g179 In(2L)Cy, al² ast³ b pr (Cy not
 present)
 g180 In(2L)Cy, b pr cn² In(2R)Cy
 g181 In(2L)t, esc c sp/SM5, al² Cy lt^v sp²
 g182 In(2L)t, lt 1 L⁴ sp²/ds^{33k} Pm
 g183 Ins(2L+2R)Cy, al² En-S sp² (homo-
 zygous)
 g184 In(2LR)102, ds^W/SM1, al² Cy sp²

- g185 InNSL InNSR/al² Cy, InL lt³ L²
 g186 j
 g187 j-1 ab² InNSR mr/S² dptxi Cy cn²
 bw sp
 g188 J Bl/In(2L)NS
 g189 J^{34e}
 g190 kn
 g191 l(2)39a px slt sp/SM5, al² Cy
 lt^v sp²
 g192 l(2)a bs³, In(2L)t/ds^{33k} Pm
 g193 l(2)ay b c sp/SM5, al² Cy lt^v
 sp²
 g194 l(2)gl cn bs/SM5, al² Cy lt^v
 sp²
 g195 l(2)H L²/SM5, al² Cy lt^v sp²
 g196 l(2)mat/SM5, al² Cy lt^v sp²
 g197 l(2)me/SM1, al² Cy sp²
 g198 L
 g199 L²
 g200 L⁴
 g201 L⁵
 g202 LG
 g203 LK
 g204 Lr
 g205 Ls¹
 g206 ll²
 g207 lm/Cy, S² dp² En-S
 g208 ls dp¹/al² Cy cn² L⁴ sp²
 g209 ls dp¹ Sp ms ta cn crs/S² Cy lt³
 pr⁺ Bl cn² L⁴ sp²
 g210 lt/T(Y;2)A
 g211 lt bw
 g212 lt bwAmherst
 g213 lt std/SM1, al² Cy sp²
 g214 lt stw³
 g215 lt³ Dp(2;2)41 L⁴ sp² In(2R)Cy/
 ds^{33k} Pm
 g216 ltd
 g217 lw
 g218 lys rc; ss (2;3)
 g219 M(2)33a/al² InMis Cy cn² sp²
 g220 M(2)173/SM5, al² Cy lt^v sp²
 g221 M(2)B/In(2L)t, l(2)B
 g222 M(2)B/SM5, al² Cy lt^v sp²
 g223 M(2)l²/ds^{33k} Pm
 g224 M(2)l²/SM1, al² Cy sp²
 g225 M(2)p/Cy, al² lt³ L⁴ sp²
 g226 M(2)S3/SM1, al² Cy sp²
 g227 M(2)S6/SM5, al² Cy lt^v sp²
 g228 M(2)S7/SM5, al² Cy lt^v sp²
 g229 M(2)S9/SM5, al² Cy lt^v sp²
 g230 M(2)S11/Cy, bw^{v34}
 g231 M(2)S11/SM5, al² Cy lt^v sp²
 g232 M(2)z/SM5, al² Cy lt^v sp²
 g233 M(2)z Sk b/Cy(2L)dp² b pr
 g234 mi/Pm²
 g235 mn/Cy cn² L⁴ sp²
 g236 mr bs²/Cy, sp²
 g237 mr bs²/ds^{33k} Pm
 g238 mr²/Bld, In(2R)Cy
 g239 ms bw/Cy pr Bl cn² L⁴ sp² (iso
 1955)
- g240 ms cn bw/dp^{txI} Cy pr Bl lt³ cn² L⁴
 sp²
 g241 ms cn rm/Cy cn² L⁴ sp²
 g242 ms cn sp/dp^{txI} Cy pr Bl lt³ cn² L⁴
 sp²
 g243 msf/SM5, al² Cy lt^v sp²
 g244 net
 g245 net al ex ds S ast shv ho rub/SM1,
 al² Cy sp²
 g246 net b cn crs/dp^{txI} Cy pr Bl lt³
 cn² L⁴ sp² (iso 1955)
 g247 net bw crs/dp^{txI} Cy pr Bl lt³ cn²
 L⁴ sp² (iso 1955)
 g248 net bw mr crs/al², InMis di^{txI} Cy
 Bl cn² L⁴ sp² (iso 1956)
 g249 net bw sp
 g250 net dp b pr cn/dp^{txI} Cy pr Bl cn²
 L⁴ sp²
 g251 net ed Su²-dx
 g252 net ta sp/al² ly pr Bl cn² L⁴
 vg sp²
 g253 net ta vg^{S2} sp/dp^{txI} Cy pr Bl cn²
 L⁴ sp
 g254 nub²
 g255 nw²/Cy-RNS
 g256 pd
 g257 pd ll² sp
 g258 Pfd/SM5, al² Cy lt^v sp²
 g259 pi/SM5, al² Cy lt^v sp²
 g260 pi l(2)301/SM5, al² Cy lt^v sp²
 g261 Pin
 g262 pk cn
 g263 Pm¹/T(Y;2)G
 g264 Pm²/mi sp²
 g265 po vg
 g266 po²
 g267 pr
 g268 pr cn/T(Y;2)C
 g269 pr cn ix/SM5, al² Cy lt^v sp²
 g270 pr^{bw}
 g271 pu
 g272 puf
 g273 pw-c/SM5, al² Cy lt^v sp²
 g274 px
 g275 px bl (old Berlin stock of Goldschmidt)
 bl=bs?
 g276 px bw mr sp/ds^{33k} Pm
 g277 px bw sp/T(Y;2)J
 g278 px slt sp
 g279 Px²-/Cy cn² L⁴ sp²
 g280 Px²-, bw sp/SM1, al² Cy sp²
 g281 pys
 g282 Q
 g283 rd/SM5, al² Cy lt^v sp²
 g284 rdo
 g285 rdo² pr
 g286 rh
 g287 rk cn bw (iso 2)
 g288 rl
 g289 rnT23/Cy Bl cn² L⁴ sp²
 g290 rub
 g291 Ruf/ds^{33k} Pm

g292 S/Cy, En-S
 g293 S dp¹/al² Cy cn² L⁴ sp²
 g294 S fes Alu lt/al² Cy cn² L⁴ sp²
 g295 S fes Sp b/Cy b lt³ cn² L⁴ sp²
 g296 S fes Sp ms ta cn mr crs/al²
 InMis dptxI Cy pr Bl cn² L⁴ sp²
 g297 S Sp ab² ap⁴ InNSR px sp/al² Cy
 Bl cn² L⁴ sp²
 g298 S Sp Bl bw^D/Cy cn² lc
 g299 S Sp Bl L² Px⁻/dp^{txI} Cy, Ins0 pr cn²
 g300 S Sp Bl L^{rm} bw^D/dptxI Cy, Ins0 pr cn²
 g301 S Sp Bl Pfd Bw^D/dptxI Cy, Ins0 pr cn²
 g302 S Sp cn bw/dp^{txI} Cy cn bw
 g303 S Sp crs/al² Cy pr Bl L⁴ sp²
 g304 S Sp InNSR mr/dp^{txI} Cy pr Bl cn²
 L⁴ sp²
 g305 S Sp (ls?) cn/dp^{txI} Cy cn
 g306 S Sp (ls⁺?) cn bw sp/dp^{txI} Cy, InL
 cn bw sp
 g307 S Sp ms ta cn crs/al² InMis dptxI Cy
 pr Bl cn² L⁴ sp²
 g308 S Sp ms ta cn crs/dp^{txI} Cy, Ins0 pr
 cn²
 g309 S Sp pr Bl rnt23 InNSR mr/dp^{txI} Cy
 pr cn²
 g310 S Sp pr cn² InCyR/dp^{txI} Cy pr cn²
 g311 S Sp rnt23/dp^{txI} Cy pr Bl cn² L⁴
 sp²
 g312 S² ab^{51b} InCyL/dp b L⁴ Pm¹
 g313 S² Cy lt³ pr⁺ Bl cn² L⁴ sp²/InNSL
 InNSR px sp
 g314 S² dp^{txI}, InCyL/l^s Sp b
 g315 S^R/ds^{33k} Pm
 g316 sca
 g317 sca 1(2)C/SM5, al² Cy lt^v sp²
 g318 sf²
 g319 shr bw^{2b} abb sp/SM5, al² Cy lt^v sp²
 g320 shv
 g321 shv ho
 g322 sn px/SM5, al² Cy lt^v sp²
 g323 sm px pd/SM5, al² Cy lt^v sp²
 g324 so
 g325 so² b cn
 g326 sp² bs²
 g327 Sp/In(2L)t, 1(2)R
 g328 Sp/Sm5, al² Cy lt^v sp²
 g329 Sp pur cn InNSR px sp/Cy pr Bl cn²
 L⁴ bw sp
 g330 Sp J/In(2L)Cy-t, Su-S dp² pr
 g331 Sp J/SM5, al² Cy lt^v sp²
 g332 Sp J L² Pin/SM5, al² Cy lt^v sp²
 g333 Sp ms cn mr crs/Cy pr Bl cn² L⁴ sp²
 g334 spd gt-4/Gla, InLR
 g335 sple
 g336 spt
 g337 std/SM5, al² Cy lt^v sp²
 g338 stw
 g339 stw²
 g340 stw³
 g341 stw³/T(Y;2)B
 g342 stw⁵
 g343 stw⁴⁸ blt tuf
 g344 ta cn bw/al² Cy pr Bl cn² L⁴ sp²
 g345 ta cn bw sp/Cy pr Bl cn² L⁴ sp²
 g346 Tft/Cy
 g347 tkd/SM5, al² Cy lt^v sp²
 g348 tkv
 g349 tri vg^{No2}/SM5, al² Cy lt^v sp²
 g350 tuf ltd
 g351 U/cg c
 g352 Uf
 g353 vg (iso 2,3)
 g354 vg bw
 g355 vg^B/SM5, al² Cy lt^v sp²
 g356 vg^C/Rvd, In2LR
 g357 vg^C/SM5, al² Cy lt^v sp²
 g358 vg^{-D}/SM5, al² Cy lt^v sp²
 g359 vg^{-D} sp²/Cy cn² L⁴ sp²
 g360 vgni
 g361 vgnp
 g362 vg^{nw} Hia/SM5, al² Cy lt^v sp²
 g363 vg^{nw} Hia/T(2;3)SM Cy
 g364 vg^U/Roi, bw sp or
 g365 vst/SM5, al² Cy lt^v sp²
 g366 whd
 g367 wt

Chromosome 3

(containing genes of 2 in a few cases)

h1 a-3	h17 ca K-pn	h32 D/Gl
h2 aa h	h18 ca ²	h33 D InsCXF/Tri
h3 abd	h19 ca ^{572jIIIa3} /Me, Ins ri	h34 D tra/InLP Dfd InRP ca
h4 app	Sb ¹	h35 D ³ H/InsP
h5 as ^{hg}	h20 Cbx	h36 D ³ Sb ca ² /Payne
h6 bar-3(Ives)	h21 cd	h37 det
h7 Bd ^G /In(3R)C, 1(3)a	h22 cmp ca/In(3R)C, e	h38 Df(3)MS31/T(2;3)Me
h8 bf/In(3R)C, Sb e 1(3)e	h23 Cor/ru h D InsCXF	h39 Df(3)sbd ¹⁰⁵ /Xa
h9 bp/TM1, Me ri	h24 cp in ri p ^p	h40 Dfd/In(3LR)Cx
h10 bul	h25 cp	h41 Dfd ^r
h11 bv	h26 cu	h42 DL H e ^s cd/In(3R)spr,
h12 bx ³ Cbx Ubx bxd pbx/Xa	h27 cu kar	spr
h13 bx ^{34e}	h28 cur	h43 DL ³ In(3R)C, e
h14 bx ^D =Ubx	h29 cv-c	h44 DL ⁵ /In(3R)C, 1(3)a
h15 ca	h30 cv-c sbd ²	h45 DL ¹⁴ /In(3R)Cyd, Cyd
h16 ca bv	h31 cv-d	h46 DL ^x /Payne

h47 drb	h104 M(3)w/In(3R)C, e 1(3)e	h152 ("rupes") ru h th st
h48 dwh/Payne, Dfd ca	h105 M(3)y/Me	pP cu sr e ^s
h49 e ^{P1} /ru h D InsCXF e	h106 ma	h153 ru h th st pP H e ^s ro/
h50 e ⁴ wo ro	h107 ma fl	C(3)x, M(3)x e ^s
h51 e ¹¹	h108 mah	C(3)x = In(3L+R)P
h52 e ^s	h109 Mc/Xa	h154 ru st C3G e ^s (iso 3)
h53 eg/In(3LR)Cx	h110 Me, InL bx ^D /ru h D	(b sp)
h54 eg ² /In(3LR)Cx	InsCXF Sb	h155 ru st C3G sr e ^s
h55 eyg	h111 Me, InL InRC e 13e/ru h	h156 ru tra p/ru h D
h56 fz	D InsCXF Sb e ^s	InsCXF e
h57 gl	h112 Me, InL Sb/ru h D	h157 ru ^s jv se by
h58 gl ² e ⁴	InsCXF	h158 ry
h59 gl ³	h113 Me, Ins ri Sb ¹ /ru h D	h159 ry ²
h60 Gl bx ^D /InsLVM	InsCXF ca	h160 Sb/In(3LR)Ubx ¹⁰¹
h61 Gl Sb H/Payne	h114 Me, Ins ri Sb ¹ /D ³ st ri	h161 Sb bx ^D /Xa, T23
h62 gs	InRC e 13e	h162 Sb H/In(3R)C, cd
h63 h	h115 Mio	h163 Sb Ubx/Xa
h64 h ri	h116 N-X/Xa	h164 Sb ^{Sp1} /In(3LR)Cx
h65 h ri ca (iso 1953)	h117 obt	h165 sbd ² bx ³
h66 h ri e ^s (iso 1957)	h118 p	h166 se
h67 h ²	h119 pP	h167 se h
h68 H/In(3R)hp, hp	h120 pP bx sr e ^s	h168 se ss
h69 H Pr/In(3R)C, e	h121 pP cu	h169 se ss k e ^s ro
h70 H ² /Xa	h122 pb/In(3LR)Cx	h170 se ^{51j}
h71 H ³ /In(3R)C, Sb e 1(3)e	h123 pbx/Xa	h171 se rt ² th/Me, InL
h72 Hn ^r h ri/ru h D Sb	h124 Pc/TM1, Me ri	h172 ("separated arms of 3"
InsCXF	h125 Pr/In(3R)C, e	DubinIn) T3L.41;4R.
h73 Hn ^{r3} sr	h126 Pr Dr/TM3, y ⁺ ac ⁺ ri	3R/1 InLP Dfd InRP 1
h74 in	pP sep bx ^{3/4e} e ^s	h173 sep, InLR ri pP
h75 In(3L)p ^{mot-36e} /R	h127 Pr ^K Dp/InPL InPR	h174 sep, InLR ri pP Sb/Me,
h76 In(3L)P, In(3R)P18, Me	(Krivshenko)	InL Dfd InRC e 13e
Ubx e ⁴ /In(3LR)Cx	h128 Pt/Xa, ca	h175 sep, InLR ri pP Sb/Me,
h77 Ins(3)Ubx ¹³⁰ /T(2;3)Xa	h129 pyd	InL InRC e 13e
h78 In(3R)Antp ^B /TM1, Me ri	h130 R Ly/In(3L)P, gm	176 Ser/In(3R)C, e 1(3)e
h79 In(3R)Dl ^B , st Dl ^B /In(3R)	h131 ra	h177 snb
P ^W , st 1(3) W ca	h132 red (Malpighians)	h178 sr
h80 In(3R)Hu, Hu Sb ^{Sp1} /Payne	h133 red (Malpighians) e	h179 sr gl
h81 In(3R)MO, sr/Xa, ca	h134 ri	h180 ss
h82 In(3R)P ^{FLA} (homozygous)	h135 ri bad e ^s /Me, In(3R)C,	h181 ss bx
h83 jv	Sb e 1(3)e	h182 ss bx Su ² -ss
h84 jv Hn ^r h	h136 ri e	h183 ss bxd k e ^s /Xa
h85 jv1	h137 ri pP	h184 ss ca (iso 1953)
h86 kar ²	h138 ri pP Ina (/ru h D	h185 ss e ^s (iso 1953)
h87 Ki	InsCXF ca)	h186 ss ^a
h88 1(3)ac e ^s M(3)w/LVM	h139 ri pP Inc 1/ru h D	h187 ss ^{a-40a}
h89 1(3)36d10/In(3LR)Cx, D	InsCXF ca	h188 ss ^{a-B}
h90 1(3)tr Sb/In(3LR)Ubx ¹³⁰ ,	h140 ri sbd e ²	h189 ss ^A , In3/Sb bx ^D
Ubx ¹³⁰ e ^s	h141 ro	h190 st
h91 1(3)tr Ubx/TM1, Me	h142 ro Bd ca/In(3R),	h191 st c 3G ca/TM1, Me ri
ri Sb ¹	1(3)a	Sb ¹ (sp ²)
h92 ld	h143 rs ²	h192 st in ri pP
h93 Ly/D3	h144 rsd	h193 st Ki pP
h94 Ly Sb/LVM	h145 ru	h194 st Sb ^r e ^s rv ca
h95 M(3)1/In(3R)C, 1(3)e	h146 ru h e ^s	h195 st sr H ² ca/In(3R)P ^W ,
h96 M(3)36e/In(3R)C, 1(3)a	h147 ru h ri	st 1(3)W ca
h97 M(3)40130/Payne, Dfd ca	h148 ru h ri pP Inb	h196 st ^{sp}
h98 M(3)B/In(3R)C, e 1(3)e	h149 ("threepl") ru h st	h197 st ⁵⁴ⁱ ri pP
h99 M(3)B ² /In(3R)C, Sb e	pP ss e ^s	h198 su ve ru ve h th
1(3)e	h150 ("rucuca") ru h th	h199 su-ve ru ve bv
h100 M(3)S32/T(2;3)Me	st cu sr e ^s ca	(h? th?)
h101 M(3)S34/T(2;3)Me	h151 ("ruPrica") ru h th st	h200 su-ve ru ve h th
h102 M(3)S36/T(2;3)Me	cu sr e ^s Pr ca/Me, T23	h201 (sp ² ;) su ² -Hw bx bxd/
h103 M(3)S37/Me		Me, Ins ri Sb ¹

h202 th	h219 ve st (iso 3)	111 ci gvl ey ^R sv ⁿ
h203 th cu sr e ^s ro ca	h220 ve st sbd	112 ci ³⁶¹
h204 th st cp	h221 W	113 ci ^W
h205 th st pb p ^P /In(3LR)Cx	h222 W Sb/InsCXF	114 ey
h206 th st pb p ^P kar su ² -Hw	h223 wk/Payne, Dfd ca	115 ey ²
jvl ss bx sr gl/TM1,	h224 wo	116 ey ⁴
Me ri Sb ¹	h225 Xa,T23 ca/e ^s cd ro	117 ey ^D /ci ^D
h207 tra/Me,T23	cmp ca	118 gvl
h208 Tri/ru h D InsCXF		119 4-sim/ci ^D
h209 tt wo	<u>Chromosome 4</u>	120 gvl ey ^R
h210 tx		121 gvl ey ^R sv ⁿ
h211 Ubx e ⁴ /Payne, Dfd ca	i1 ar/ey ^D	122 gvl sv ⁿ
(Ubx=bx ^D)	i2 bt	123 M(4)/ey ^D
h212 ve	i3 bt ey ^R sv ⁿ	124 pol
h213 ve bv (iso 1957)	i4 bt ^D /ci ^D	125 spa
h214 ve ca (iso 1953)	i5 Cat/ci ^D	126 sv ^{35a}
h215 ve h th	i6 Ce ² /spaCat	127 sv ^{de} /ey ^D
h216 ve R/In(3L)P, gm	i7 ci ^D /ey ^D	128 sv ⁿ
h217 ve R D ³ bx ^D (e ^s ?) Pr	i8 ci ey ^R	129 Tp ⁴ :Y (Edmondson)/
ca/InLP Dfd InRP ca	i9 ci ey ^R sv ⁿ	4-sim ♂ & 4-sim ♀
218 ve R D ³ SbSp ⁱ Bd ^G /InsP	i10 ci gvl bt	

Multiple ChromosomesX,2 (j1-j15)

- j1 Bld,T12 InCyR/sc² pn; II⁺
j2 ("scute,twelvel") y sc⁵; al dp sc¹⁹ⁱ b pr cn vg c a px bw mr sp/al² Cy pr Bl
 cn² L⁴ sp²
j3 sc⁸ f In⁴⁹ v; bw^{VA}/L² l (iso Y,X,2)
j4 X·Y InEN v y; S dp Sp cn/dp^{tx} Cy cn (no free Y)
j5 y; S Sp cn/dp^{tx} Cy cn
j6 y ac; sc¹⁹ⁱ/S² Cy
j7 y f:=; bw^{VA}/L² l
j8 f^{56e} & y f:=; cn bw
j9 y f:=; Cy,Ins cn²/Gla,InLR
j10 y f:=; dp^{tx} Sp cn bw/S² Cy cn bw
j11 y f:=; net bw sp
j12 y Hw In⁴⁹ m g/y sc^{S1} B InS; net bw sp
j13 y sc^{S1} In⁴⁹ v sc⁸; dph b bw/dp^{tx1} Cy pr Bl cn² L⁴ sp²
j14 y v f^x:fth; bw^{VA}/L² l
j15 y² t²; cn bw

X,3 (j16-j24)

- j16 sc w B^{S3}.Y^S & y f:= (B^{S3} Del.-Inser, into 3)
j17 sn³; Mw/l InLP InRP l
j18 ("Tam tester 1") y f:=; D³ Sb/InLP Dfd InRP ca
j19 ("Tam tester 2") y sn oc ♂ & Y⁺/y sc^{S1} B In⁴⁹ l sn^{x2} sc⁸/lJ1 sc^{J1} oc ptg ♀; ru h
 D InsCXF/Me,Ins ri Sb¹
j20 ("Tam X3") TX3,red ♂ & y f:= ♀
j21 ("Tam X3sn") sn TX3,red ♂ & y f:= ♀
j22 wa ♂ & y v f:= ♀; tra/D InsCXF
j23 y Hw In⁴⁹ m g/y sc^{S1} B InS; ru bw
j24 y f:=; su-ve ru ve bv (h? th?)

X,4 (j25-j27)

- j25 y sc^{S1} InS sc⁸ ♂ & y:=♀; ci ey^R
j26 y f:=; Cat/ci^D
j27 y f:=; spa

2,3 (j28-j104)

- j28 al b cn sp/al² Cy Bl cn² L⁴ sp²; ru
 j29 "apl"/Cy sp; ru h InsCXF ca/Sb InRMO
 j30 b; pP
 j31 bw; e
 j32 bw; ru h st D³ ri InRC e l3e/Me,Ins ri Sb¹
 j33 bw; ru h ri
 j34 bw; ss
 j35 bw sp; ru h D¹ ri InRC e l3e/Me,Ins ri Sb¹
 j36 bw; st
 j37 c; e
 j38 cn bw; ri ej39 cn bw; ru h th ri e^s
 j39 cn bw; ru h th ri e^s
 j40 cn crs/al² Cy lt³ pr Bl cn² L⁴ sp²; e^s
 j41 cn crs/Cy pr Bl cn² L⁴ sp²; ve (iso)
 j42 Cy/Pm; ru h D Ins CXF ca/InLP Dfd InRP ca
 j43 Cy/Pm; st (iso X,2,3)
 j44 De bw; ro
 j45 dp cu bw/Cy Fl cu² L⁴ sp²; h ri e^s
 j46 dp^{o3} cn bw; ru h D³ ri In3RC e l3e/Me,Ins ri Sb¹
 j47 dp^T Sp cn bw/S² Cy cn bw; ri e
 j48 dp^T Sp cn/S² Cy cn; ri e
 j49 dp^T Sp cn/S² Cy cn; ru h D³ ri InC e l3e/Me Ins ri Sb¹
 j50 dp^{tx} Sp cn/S² Cy cn; ru h D InsCXF/Me Ins ri Sb¹
 j51 dp^{tx} Sp cn/S² Cy cn; ru h D InsCXF Sb/Me,InL InC e l3e
 j52 dp^{tx} Sp cn/S² Cy cn; sep ri pP Sb/Me InL InC e l3e
 j53 dp^{tx} Sp ms ta cn crs/S² Cy pr Bl cn² L⁴ sp²; e^s
 j54 dp^{tx} Sp pr cn²/S² Cy cn²; Me.InL InC e l3e/ru h CXF Sb
 j55 dp^V; vo³
 j56 dp^{V2} cn bw; h ri e^s (iso 7/57)
 j57 fes ms cn sp/dp^{txI} Cyc05 pr cn²; h ri e^s/Me Ins ri Sb¹ (iso 7/57)
 j58 InNSL InNSR mr/al² Cy pr Bl lt³ cn² L⁴ sp²; ri^{53j}
 j59 ("iser 1") S Sp (crs)/Cy InL lt³; Me Ins ri Sb¹/BdG
 j60 ("iser 2a") ms cn rm sp/al² Cy lt³ pr Bl cn² L⁴ sp²; ru h D InsCXF/ve th l
 j61 ("iser 2b") dp b cn c P-/al² Cy lt³ pr Bl cn² L⁴ sp²; ru h D Ins CXF/D; J e pⁱ
 j62 M33a/al² Cy pr Bl cn² L⁴ sp²; ru
 j63 ms sp/Cy pr Bl cn² L⁴ sp²; ri^M (iso)
 j64 net bw mr crs; Dl H e P¹/ru h D InsCXF (low iso 7.57)
 j65 net bw mr crs/dp^{txI} Cy O pr cn²; ve bv/Me,Ins ri Sb¹
 j66 net dp sp/dp^{txI} Cy, O pr cn²; Me,Ins ri Sb¹/ve bv
 j67 ("Pale e") dp b cn c P-/Cy cn²; e pⁱ/e p¹
 j68 ("Pale H") dp b cn c P-/Cy cn²; p⁵⁶ Dl H e Pⁱ/p⁵⁶ In3R l
 j69 ("Pale Indp") IndpT23 b P-·Dl H e Pⁱ/dp b Pm¹; Sb In3R
 j70 S fes Sp T23B D³ ri Sb/Cy cn² sp²; InsCXF
 j71 S fes Sp T2301 ms cn mr crs D³ st ri InC e l3e/al²,InMis Cy pr Bl cn² L⁴ sp²;
 Me Ins ri Sb¹
 j72 S fes Sp T2301 ms cn mr crs D³ st ri InC e l3e/dp^{txI} Cy,Ins05 pr cn²; Me,Ins
 ri Sb¹
 j73 ("sifter O") S Sp P- T23,InsCXF/dp^{txI} Cy,Ins05 pr cn²; Dl H e Pⁱ
 j74 S Sp cn/dp^{txI} Cy cn; h ri e^s
 j75 S Sp cn/dp^{txI} Cy cn; Me,InL InRC e l3e/ru h D Sb InsCXF
 j76 S Sp cn/dp^{txI} Cy cn; ru h D³ ri InRC e l3e/Me,Ins ri Sb¹
 j77 S Sp cn/dp^{txI} Cy cn; ru h e¹¹
 j78 S Sp cn/dp^{txI} Cy,Ins05 pr cn²; h ri D³ InC e l3e/Me,Ins ri Sb¹
 j79 S Sp cn/dp^{txI} Cy,Ins 05 pr cn²; ru h D InsCXF/Me,Ins ri Sb¹
 j80 S Sp cn bw/dp^{txI} Cy cn bw; h ri e^s
 j81 S Sp cn bw/dp^{txI} Cy cn bw; ru h D³ ri InRC e l3e/Me,Ins ri Sb¹
 j82 S Sp ms ta cn crs/dp^{txI} Cy pr Bl cn² L⁴ sp²; e²
 j83 Sp T23M1/dp^{txI} InsCy,05 pr cn²; Me,Ins ri Sb¹
 j84 sp; ru h D InsCXF/Me,InL InRC e l3e
 j85 S Sp T23B/dp^{txI} Cy,Ins05 pr cn²; Me,Ins ri Sb¹
 j86 T23B cn bw InC e l3e/al² In,Mis Cy cn² L⁴ sp²; Me,Ins ri Sb¹

- j87 T23B cn bw InRC e l3e/Cy,Ins05; ru h D InsCXF (InAM?)
 j88 T23B cn bw D³ ri/dp^{txI} Cy,Ins05 pr cn²; Me ri Ins Sb¹
 j89 ta/Cy Bl cn² L⁴ sp²; ru ri (iso)
 j90 ta sp/Cy Bl cn² L⁴ sp²; jv (iso)
 j91 ta sp/Cy cn² L⁴ sp²; ru (iso)
 j92 ("TIn") dp^{txI} Cy,Ins05 pr cn² T23 Me,Ins ri Sb¹/S Sp cn; ru h D³ st InRC e l3e
 j93 (2;4) "apl" l/Cy cn² sp²; IV-sim/ci ey
 j94 (2,4) bw; ci^D/IV-sim
 j95 (3,4) bv; Cat/ci^D
 j96 (X,Y,2) Y:bw⁺/y v & sc ctⁿ oc ptg car·y ct¹, In In49 sn^{x2}; bw
 j97 (X,Y,3) ("multi-") X·Y InEN y; st (no free Y)
 j98 (X,Y,3) X·Y y; st (no free Y, no In)
 j99 (X,Y,3) sc⁸·Y/X·Y InEN y; ru h D InsCXF/ru tra p
 j100 (X,Y,3) X·Y InEN In49 y; st (no free Y)
 j101 (X,Y,4) sc⁸·Y/X⁺ & y f:=; ci gvl ey^R svⁿ
 j102 (X,Y,4) Tp⁴:Y/X·Y InEN In49 v y; ci gvl ey^R svⁿ
 j103 (X,Y,4) Y^S·InEN y·Y^L: 4 & y:=; ci ey^R (no free Y)
 j104 (X,Y,4) Y^S·InEN y·Y^L sc⁸ y⁺; ci ey^R (no free Y)

X2,3 (j105-j112)

- j105 ("MI") y^{Si} sc⁸ InS y^{3P}; al² Cy lt³ cn² sp²/dp b Pm¹; ru h D InsCXF ca/Sb In3R
 j106 ("Pale") w^e; P-/Cy; P¹/P¹
 j107 y^{Si} sc⁸ B f In49 v; T23B D³ st ri InC e l3e/dp^{txI} Cy,Ins05 pr cn²; Me,Ins ri Sb¹ (select AT)
 j108 y f:=; cn bw; e
 j109 y Inr⁹ v; bw; e
 j110 y sc^{S1} f In49 v sc⁸; bw; e
 j111 y^{Si} sc⁸ B f In49 v; bw; e
 j112 y sc^{S1} In49 sc⁸; bw; st pP (to cross by sc⁸·Y/y B for losses, l's & T's)
 j113 (X,2,4) ("gcar") sc t² v f car; Cy/bw; ey
 j114 (Y,2,3) sc⁸·Y:bw⁺; dp^{v2} cn bw; h ri e^S (iso 7/57) (Cy Bl cn² L⁴ sp²)
 j115 (Y2,3) sc⁸·Y:bw⁺; fes ms cn sp/dp^{txI} Cy,Ins05 pr cn²; h ri e^S/Me,Ins ri Sb¹ (iso 7/57)
 j116 (Y2,3) sc⁸·Y:bw⁺; net bw mr crs/dp^{txI} Cy,Ins05 pr cn²; Me,Ins ri Sb¹ (iso 7/57) (ve bv)
 j117 (Y,2,3) sc⁸·Y:bw⁺; net dp sp/dp^{txI} Cy,Ins05 pr cn²; ve bv/Me,Ins ri Sb¹
 j118 (Y,2,3) Y:bw⁺; Me,T23/dp^{txI} Cy cn² bw sp
 j119 (2,3,4) Cy/bw; e; ci^D/IV-sim
 j120 (2,3,4) bw; e; ci ey^R

X,Y,2,3 (j121-j131)

- j121 ("Multipare") sc⁸·Y/y sc In49 B^{M1}; twl bw; st⁵⁴ⁱ
 j122 ("Multipare R") sc⁸·Y/X^{c2} y & y f:=; twl bw; st⁵⁴ⁱ
 j123 ("Taxy") sc⁸·Y/y sn oc & sc⁸·Y/y In49 sn^{x2} B^{M1}/y oc lz·Y^S; twl bw; st⁵⁴ⁱ
 j124 ("y s cn bw e") Y^{Lc}/y s·Y^S/y sc^{S1} B f In49 v sc⁸; cn bw; e
 j125 ("y cn bw e") Y^{Lc}/y·Y^S; cn bw; e
 j126 sc^{VI}·Y^S/y In49 v f·Y^L; bw; e
 j127 X·Y InEN In49 y; cn bw; e (no free Y)
 j128 X·Y InEN In49 y; cn bw; ro (no free Y)
 j129 (X,2,3,4) y f:=; bw; e; ci ey^R
 j130 (X,Y,2,3,4) Y^S·InEN In49 y·Y^L; cn bw; e; ci ey (no free Y)
 j131 (X,Y,2,3,4) Y^S·InEN In49 y·Y^L ♂ & "snocty" ♀; cn bw; e; ci ey^R (no free Y)

non-lethal "tumorous" stocks

- m1 bw tu
 m2 tu^{50j}
 m3 tu^{51m}
 m4 tu^h
 m5 vg tu

SALT LAKE CITY, UTAH: UNIVERSITY OF UTAH
Department of Genetics

Note: Stock list unchanged. See DIS 33, p.53

SYRACUSE, NEW YORK: SYRACUSE UNIVERSITY
Department of Zoology

Several wild strains, each derived from a single inseminated female.
Several polygenic crossveinless (cve) strains.

TUSCON, ARIZONA: UNIVERSITY OF ARIZONA
Department of Zoology

w
y
y; w; ec; f
b

dp
vg
ss

bw; e; ey
T(3,4)A96,ca²
S/Cy; D/c3X

ARGENTINA

Buenos Aires: Atomic Energy Commission, Section of Genetics

Note: This list was copied from DIS 31, pg. 51 and DIS 32, pg. 40.

Wild Stocks

W1 Leningrad
W2 Buenos Aires
W3 Oregon-R

Chromosome 1 (attached-X)

Chromosome 1 (homozygous)

Xa1 In⁴⁹ B^{M1}
Xa2 y fa wy² g²
Xa3 y t² v f
Xa4 y v
Xa5 y w f
Xa6 y w sn³ f
Xa7 y² v f car
Xa8 y³P, In B
Xa9 sc⁶ car
Xa10 sc⁸ B
Xa11 sc⁸ bb wa
Xa12 y^{S1} sc⁸
Xa13 sc^{V2}, Inh
Xa14 sc^{S1} B InS wa sc⁸
Xa15 sc^{S1} In⁴⁹ sc⁸
Xa16 y sc^{S1} In⁴⁹ w sc⁸
Xa17 X·Y In EN v y (no free Y)
Xa24 B
Xa25 g^W
Xa26 w^{mR7}aH1
Xa27 y w sn³
Xa28 y sc^{S1} sc⁸
Xa29 X·Y y In⁴⁹ v f car (no free Y)
Xa30 sc⁸·Y/y sc⁴ w sc⁸ (sc⁸·Y in ♂ & ♀)
Xa31 y^{Lc}/X·Y^S

Xb1 lz & y f:=
Xb2 sc ct⁶ car & y f:=
Xb3 sc t² v f & y f:=
Xb4 y ac sc v & y f:=
Xb5 y ac t²·Dp(y⁺ ac⁺ sc^{S1}) & y f:=
Xb6 y ct⁶ f & ac³ wa ct f:=
Xb7 y ct⁶ t² v f car & y f:=
Xb8 y·Dp(y⁺ sc^{V1}) & y f:=
Xb9 y In⁴⁹ sn^{x2} bb¹ & y f:=
Xb10 y sc lz^g v f & y f:=
Xb11 y w^{m258-18} T² v f & y f:=
Xb12 sc⁸ B In⁴⁹ & y f:=
Xb13 sc⁸ B In⁴⁹ m & y f:=
Xb14 sc⁸ car f In⁴⁹ v & y f:=
Xb15 sc⁸ Tu wa & y f:=
Xb16 y^{S1} sc⁸ B f In⁴⁹ v wa & y f:=
Xb17 y^{S1} sc⁸ f Ins wa & y f:=
Xb18 sc^{S1} f In⁴⁹ v w & y f:=
Xb19 y sc^{S1} B f In⁴⁹ v & y f:=
Xb20 sc^{L8} sc⁸ & y f:=
Xb21 sc^{S1} B In⁴⁹ sc⁸ & y f:=
Xb22 y sc^{S1} B In⁴⁹ sc⁸ & y f:=
Xb23 sc^{S1} At In⁴⁹ v wa sc⁸ & y f:=
Xb24 sc^{S1} f In⁴⁹ y wa sc⁸ & y f:=
Xb25 sc^{S1} f Ins y³P & y f:=
Xb26 y^L/f·Y^S & sc v f:=
Xb27 y³·y^L/y·Y^S & y f:=
Xb28 Y^S/g² B·Y^L & y f:=
Xb29 Y^S·Y^S 2/y v f·Y^L & y f:=
Xb30 sc^{V1}·Y^S/y In⁴⁹ v B·Y^L & y f:=
Xb36 sn³ v B^{M1}, In & y w f:=

Xb37 spl rb cx & y f:=
 Xb38 Tu & y f:=
 Xb39 y ac⁶ sc pn & y f:=
 Xb40 y ct⁶ f & ac³ wa⁸ g⁸ f:=
 Xb41 y sc⁴ B InS wa⁸ sc⁸ & y f:=
 Xb42 ("snoc") X·Y InEN ptg oc sn⁵ & sc ctⁿ
 oc ptg car y In49 sn^{x2} · ♀ (no free
 Y)
 Xb43 ("X·Y ") Y^S·X InEN y·Y^L sc⁸ y⁺ & y²
 su-wa^a wa^a bb⁺ (no free Y)
 Xb44 ("X·Y By") Y^S·X InEN B y·Y^L & y²
 su-wa^a wa^a bb⁼ (no free Y)
 Xb45 sc⁸·Y/y B ♂ & y f:= ♀
 Xb46 Y^{Lc}/w⁸ gc ptg·Y^S ♂ & Y^{Lc}/y² X⁺·wa^a InS
 B sc⁸ ♀ (tandem X·X giving rings)
 Xb47 oc ptg Tu & y f:-
 Xb48 we^e sn & y f:=
 Xb49 we^e sn B & y f:=
 Xb50 sc^{S1} In49 w sc⁸ & y f:=

Chromosome 1 (balanced)

Xc1 N⁸/y Hw In49 m g
 Xc2 oc ptg³·Dp(y⁺ sc^{S1})/CLB
 Xc3 oc ptg Tu/sc^{S1} fu In49 sc⁸
 Xc4 ras⁴ m/CLB
 Xc5 sc ctⁿ oc car/y In49 sn^{x2}·B^S
 Xc6 sd fex/y sc⁸ B f In49 v
 Xc7 y sc^{S1} B In49 ct-1 lz^s/w sn^{5s} bb
 Xc8 CLB/we sn
 Xc9 y sl² bb⁻, In/InAM
 Xc10 ct⁶ v dy g f/InA99 sn^{33f}
 Xc11 f fu/CLB
 Xc12 un Bx/InAM ptg⁴

Chromosome 1 (multiple loci)

Xd1 y ac sc pn w rb cm ct⁶ ras² v g² f
 car & y f:=; sc¹⁹ⁱ/Cy, InL lt
 Xd2 y pn w cm ct⁶ sn³ oc ras² v dv g²
 f od car sw/y sc^{S1} B In49 v
 Xd3 sc^{S1} car f In49 v/y ac sc pn w rb
 cm ct⁶ sn³ ras² v dy g² f car
 Xd4 Y^{Lc}/y sn⁵ oc v·Y^S ♂ & Y^{Lc}/sc^{J1} pn
 w rb cm ct⁶ oc ras² v dy g² f
 od car sw/y sc^{S1} B In49 sn^{x2} sc⁸ ♀
 Xd5 y³·Y^L/y² oc lz·Y^S ♂ & y³·Y^L/y ac sc
 pn w rb cm ct⁶ sn³ oc ras v m g² f
 car/sc^{S1} B In49 lz^s sc⁸ ♀
 Xd6 lJ1·Y/lJ1 sc^{J1} In49 B^{M1}/y ac pn w ec
 rb cm ct⁶ sn³ oc ras² v dy g² f
 Tu car
 Xd7 lJ1⁺·Y/lJ1 sc^{J1(+)} In49 v f^{S1} B^{M1}/y
 sc^{S1} car odsy f g² dy v ras² sc³
 ct⁶ cm rb ec w pn l sc⁸

Chromosome 2

II-1 ab² ms ta crs/Cy pr Bl cn² L⁴ sp²
 II-2 al b cn sp (iso, 1954)
 II-3 cn bw sp
 II-4 net b cn crs/dp^{txI} Cy pr Bl lt³
 cn² L⁴ sp² (iso, 1955)

II-5 al dp b pr cn vg c, a px bw sp/S² Cy
 lt³ pr Bl cn² L⁴ sp²
 II-6 Y:bw⁺/X⁺; cn bw
 II-7 Y^c:bw⁺/X⁺; bw
 II-8 c (iso 2,3)
 II-10 ast ho
 II-11 b pr
 II-12 bw^D

Chromosome 3

III-1 e
 III-2 ru h th st p^P cu sr es

Chromosome 4

IV-1 Cat/gvl ey^R
 IV-2 ci ey^R
 IV-3 spa
 IV-4 svⁿ

Multichromosomal

M1 wa^a ♂ & y v f:= ♀; tra/D Ins CXF
 M2 y sc⁴ InS wa^a; S sc¹⁹ⁱ Bl/Cy L⁴ sp
 M3 sc¹⁹⁻/lJ1 sc^{J1}; fes sc¹⁹ⁱ b pr/Cy
 dp^{txI} pr cn²
 M4 Y^{Lc}/X·Y^S & y v f:=; e
 M5 Y^{Lc}/X·Y^S; bw; e
 M6 y sc⁵; al dp sc¹⁹ⁱ b pr cn vg c a
 px bw mr sp/al² Cy pr Bl cn² L⁴
 sp²
 M7 y² t²; cn bw
 M8 bw; e
 M9 y^{S1} sc⁸ InS y^{3P}; al² Cy lt³ cn² sp²/
 dp b Pm¹; ru h D InsCXF ca/Sb
 In3R
 M10 we; P-/Cy; pi/pi
 M11 y f:=; cn bw; e
 M12 sc t² v f car; Cy/bw; ey
 M13 Y bb⁻/v; bw^{VA}/Bl L²
 M14 ("tester 1") y ac pn w rb wy² g² & y
 f:=; sc¹⁹ⁱ/Cy
 M15 ("tester 2") y² wa^a cm wy² g² car & y
 f:=; sc¹⁹ⁱ/Cy
 M16 ("tester 3") y rb cm ras² g² & y f:=;
 sc¹⁹ⁱ/Cy
 M17 y ac sc pn w spl rb cx. & y f:=;
 (sc¹⁹ⁱ(b pr)/)
 M18 sc⁸ B InS wa^a sc⁴ & y f:=; sc¹⁹ⁱ/Cy cn²
 M19 y sc⁻ (rein. sc⁸⁻⁴) wa^a InS bb & y f:=;
 sc¹⁹ⁱ/Cy lt cn²
 M20 Y^c:bw⁺/X^{c2} y f; bw (ring OK '57)
 M21 sc⁸·Y:bw⁺/ac³; b bw
 M22 Y^{Lc}/X·Y^S; bw

AUSTRALIABrisbane, Queensland: University of Queensland

Note: This list was copied from DIS 33, pg. 55

<u>Wild Stocks</u>	<u>Chromosome 1</u>	<u>Multichromosomal</u>
1 Oregon R-C	2 sc cv v 3 w 4 \underline{y}/B	5 e ¹¹ dp 6 \underline{y} ; Cy/Pm, ds ^{33k} , H/Sb

AUSTRIAVienna: Institut für allgemeine Biologie

Note: This list was copied from DIS 28, pg 51

<u>Wild Stocks</u>			
	30 y		55 ru h th st cu sr e ^s ca
	31 y w		56 Sb H/Payne
1 Oregon-S	32 y ec ct ⁶ v wy ² car		57 se e ¹¹
2 Oregon-R-c (Df(2)Ore)	33 y v f		58 Ser/In(3R)C, e l(3)e
3 Oregon-R-c P	34 \underline{y}/f		59 ss ^{a-F}
4 8 strains from different places in Austria	35 bo		60 st
	<u>Chromosome 2</u>		<u>Chromosome 4</u>
<u>Chromosome 1</u>	36 b pr vg a sp		61 ey ²
12 Ax ^{Oslo}	37 bs (4 different alleles)		<u>Multichromosomal</u>
13 B	41 bw		62 Bx; J
14 BB	42 cn		63 v; cn bw
15 B w ^{bf}	43 al dp		64 $\underline{y v f}/+$; bw; e wo ro; ey ²
16 ClB/dl-49 m ² g ⁴	44 ex		<u>Aberrations</u>
17 car	45 j		65 Df(1)N ⁸ /y Hw dl-49 m ² g ⁴
18 cv	46 L ² /Cy		66 Df(2)vg ^S , cn/Cy, al ² lt ³ L ⁴ sp ²
19 fa ⁿ	47 vg		67 Dp(1;f)135 y ² ; In(1)sc ⁸ , Df(0+ac) wa sc ⁸
20 lz w ^{bf}			68 In(2LR)Gla/Cy
21 pn ²	<u>Chromosome 3</u>		69 T(2,3)bw/Cy
22 spl ²	48 ca		
23 v	49 c3G		
24 w	50 e		
25 w ^{bf}	51 e cu		
26 w ^{bl}	52 gl		
27 w ^{ch} wy	53 jv se		
28 w ^e	54 ru se h st bv		
29 w ^h			

BRAZILCuritiba, Paraná: Universidade do Paraná, Faculdade de Filosofia, Ciências e Letras, Laboratório de Genética

Note: This list was copied from DIS 32, pg 43.

<u>Wild Stocks</u>	Curitiba, Paraná	Petrolina, Pernambuco
	Florianópolis, Santa Catarina	Ponta Grossa, Paraná
Boa Esperança, Minas Gerais	Gaspar, Santa Catarina	Salvador, Bahia
Buenos Aires, Argentina	Goiânia, Goiás	Santa Felicidade, Paraná
Campina Grande, Paraíba	Gruta, Argentina	São Paulo, São Paulo
Cosmópolis, São Paulo	Iratí, Santa Catarina	Teixeira Soares, Paraná
Cuiabá, Mato Grosso	Lins, São Paulo	Uberlândia, Minas Gerais
	Paranaguá, Paraná	Xapecó, Santa Catarina

CANADAToronto: University of Toronto

Note: Inbred stocks of Marvin Barr Seiger (see DIS 32, pg. 156)

Oregon-R: 291 generations of inbreeding as of 60k28
 Ives Oregon-R: 311 generations
 M Oregon R: 284 generations
 P₁I Oregon R: 332 generations
 2b Oregon R-C: Lost generation 256
 f Oregon R: 221 generations
 y Oregon R: 220 generations
 Canton-S: 97 generations

Ives Oregon R: mass culture, extracted from inbred stock at generation 300
 f Oregon-R: mass culture, extracted from inbred stock at generation 200
 y Oregon-R: mass culture, extracted from inbred stock at generation 200

Multichromosomal

- 1 yf:=; bw; e; pol (1;2;3;4)
- 2 $\frac{yv}{FM6}$; Deb^D; $\frac{HnHu}{TM3}$ (1;2;3)
- 3 $\frac{yv}{v}$; bw^D; $\frac{Hu}{Ubx}$ (1;2;3)
- 4 $\frac{yv}{yv}$; $\frac{sp\ bw^D}{SM5}$; $\frac{Sb}{TM3}$; (1;2;3)
- 5 bw; st (2;3)

FRANCELyon: Faculté des Sciences, Laboratoire de Zoologie

Note: This stock list copied from DIS 29, pg. 51.

Wild Stocks Champetières (inbred) Lyon

GERMANYGöttingen: Max-Planck-Institut für Tierzucht und Tierernährung

Note: This stock list copied from DIS 33, pg.60

<u>Wild Stocks</u>	<u>Chromosome 2</u>	<u>Chromosome 4</u>
1 normal (Berlin wild)	12 fes lt L ⁴ /Cy al ² lt ³	17 ci ^D /+ 18 ey ^D /+
<u>Chromosome 1</u>	13 fj px sp 14 lgl cn bw/Cy cn bw L ⁴ sp ²	<u>Multichromosomal</u>
2 br ec rb	<u>Chromosome 3</u>	19 w; j; e ¹¹ ; ey ²
3 ClB/+	15 pb/C Mé SB C	20 Cy/Pm.ds ³ 3k; H/C Sb
4 sc ⁸ Y/y fx sc ⁸ y/Xc ² y v	16 ss ^a	21 L Cy/+; C Mé Sb C/+
5 svr		
6 w ^e		
7 w ^{ch} wy		
8 w ^{co} v f		
9 w ^{co} v f		
10 w sn ³ B		
11 y w bb		

Hamburg: Zoologisches Staatsinstitut und Zoologisches Museum

Note: This stock list copied from DIS 32:49.

<u>Wild Stocks</u>	<u>Chromosome 3</u>	13 bw; cu; ey ²
		14 cu; ey ²
1 Oregon-S	7 cu	15 <u>y</u> ; ey ²
	8 e cu	
<u>Chromosome 1</u>	9 Sb/H Payne	<u>Triploid</u>
2 w ¹	<u>Chromosome 4</u>	16 y ² sc w ^a ec/FM4, y ^{31d}
3 <u>y/f</u>		sc ⁸ dm B
	10 ey ²	17 y ² sc w ^a ec/FM4, y ^{31d}
<u>Chromosome 2</u>		sc ⁸ dm B; bw; cu; ey ²
	<u>Multichromosomal</u>	
4 bw		
5 dp b	11 bw; cu	
6 Pm L Cy	12 bw; ey ²	

Hamburg-Eppendorf: Universitäts-Frauenklinik, Strahlenbiologische Abteilung

Note: This stock list copied from DIS 28:56

<u>Wild Stocks</u>	5 w
	6 X ^c /ClB
1 normal (Berlin wild)	
	<u>Multichromosomal</u>
<u>Chromosome 1 (X)</u>	
	7 cn; ss
2 ClB/+	
3 sc ^{S1} B.InS w ^a sc ⁸	<u>Attached-X</u>
4 sc ⁸ Y/ <u>y f</u> x sc ⁸ Y/X ^{c2} y v	
	8 <u>y</u>

GREAT BRITAINBayfordbury, Hertford, Herts, England: John Innes Horticultural Institution

Note: This stock list copied from DIS 33:62.

<u>Wild Stocks</u>	<u>Inbred Lines</u>	<u>Chromosome 1</u>	<u>Chromosome 2</u>
1 Bayfordbury	5 Bayfordbury (A)	9 v	12 b pr vg
2 Hampton Hill	6 Bayfordbury (B)	10 w	
3 Samarkand	7 Oregon	11 y w	<u>Multichromosomal</u>
4 Teddington	8 Samarkand		13 Cy L ⁴ /Pm; H/Sb

Glasgow, Scotland: University of Glasgow, Department of Genetics

Note: This stock list copied from DIS 32:51.

<u>Wild Stocks</u>	<u>Chromosome 1</u>	7 sn ³	13 w ^{co}	19 w ^t
		8 v	14 w ^e	20 y w ^e ec
1 Florida-4 (inbred)	3 B	9 w	15 w ^h	21 w ^{1P} z(zeste)
2 Oregon-K (inbred)	4 gt w ^e	10 w ^{FF33}	16 w ^{sat}	22 w ^{14G2} z(zeste)
	5 sc w ^{ec} ev	11 w ^a	17 w sn ³	
	6 sc w ^{bl} ec cv	12 w ^{bf2}	18 w sn ³ B	

July 1961

Melanogaster - Stocks - Great Britain

35:37

<u>Attached-X</u>	<u>Chromosome 3</u>	29 bw; v
23 <u>y v f</u>	26 E	30 sn ³ ; cu sr e ^s p ^D
<u>Chromosome 2</u>	<u>Multichromosomal</u>	<u>Inversions</u>
24 b cn vg	27 y v f/v; bw ^{VA} /Bl L ²	31 Muller-5
25 bw	28 bw; e	

Harwell, Berks., England: Medical Research Council,
Radiobiological Research Unit

Note: This stock list copied from DIS 33:63.

<u>Wild Stocks</u>	<u>Chromosome 1</u>	18 Cy/Bl L ²
1 Oregon-K	10 Muller-5	19 ds dp
<u>Inbred lines</u>	<u>Chromosome 2</u>	20 el
2 Crianlarich (186 gens.)	11 a px	21 hk pr
3 Kaduna (90)	12 al dp b pr c px sp/Cy	22 ho ed cl
4 lt (18)	13 b	23 lt
5 Nettlebed (268)	14 b el	24 ltd
6 Oregon-R (336)	15 bw	25 ltd cn
7 Oregon-S (229)	16 bw pd	26 net
8 stw (18)	17 cn	27 pd
9 Wild Edinburgh (260)		28 pr
		29 stw
		30 stw lt

INDIA

Calcutta: Indian Statistical Institute

Note: This list copied from DIS 34:37-38.

<u>Wild Stock</u>	13 wbf	<u>Chromosome 3</u>
1 Canton-S	14 wbl	28 cu
<u>Chromosome 1</u>	15 w ^{co}	29 e ^s
2B	16 w ^e	30 Ly/D ³
3 Bx ³	17 w ^h	31 ry
4 cv	18 w ⁱ	32 se
5 Df(1)N ⁸ /dl-49 y Hw m ² g ⁴	19 y	33 ss ^a
6 ec	20 y ²	<u>Chromosome 4</u>
7 f		34 ci ^W
8 In(1)Cl sc [?] t [?] v ⁺ sl [?]	21 bw	35 ey ²
B ^{36d} /dl-49 y Hw v m ² f	22 cn	
9 m	23 dp	<u>Multichromosomal</u>
10 v	24 ho	36 Cy/Fm D/Sb
11 w	25 px	
12 w ^a	26 vg	
	27 vg bw	

Calcutta: University of Calcutta, Department of Zoology

Note: This stock list was copied from DIS 32:53.

<u>Wild Stocks</u>	<u>Chromosome 2</u>	<u>Chromosome 3</u>	<u>Chromosome 4</u>
a1 Canton-S	c2 L ⁴	d1 se h	d3 Ly/D ³
a2 Oregon	c3 vg	d2 ss ^a	d4 e ^s
	c4 ho		
<u>Chromosome 1</u>			
b1 y Hw 49 v ^o m ² , f/ClB ^{36d}			e1 ey ²
b3 x ^{c-2} y/y w f			e2 ci ^w
b6 y			
			<u>Multichromosomal</u>
			f1 Cy/Pm; D/Sb

Hiroshima: Hiroshima University, Zoological Laboratory

Note: This stock list was copied from DIS 33:68.

<u>Wild Stocks</u>	<u>Chromosome 1</u>	<u>Chromosome 3</u>
1 Kandy, Ceylon	10 BB	14 e
2 Hiroshima	11 w	
3 Matsuyama		<u>Multichromosomal</u>
4 Miyakonojio	<u>Chromosome 2</u>	
5 Naze		15 Muller-5; Cy/Pm; Sb,
6 Oregon-R	12 S/Cy E-S	e/Ubx ¹³⁰ e
7 Samarkand	13 vg	16 y; bw; e; ci, ey ^R
8 Sapporo		
9 Taichu, Formosa		

Mitaka, Tokyo: International Christian University

Note: This stock list was copied from DIS 33:70.

<u>Wild Stocks</u>	<u>Mutants</u>	
Oregon-S	Muller-5	w ^e
Tokyo	w	y
	w ^a	y w m f
		cu
		e

NETHERLANDSLeiden: Rijksuniversiteit, Laboratorium voor Stralengenetica

Note: This stock list was copied from DIS 33:74-75.

<u>Wild Stocks</u>	6 f B odsy car
	7 fu ^{ff} /ClB
1 Oregon-K	8 g ²
	9 pn
<u>Chromosome 1</u>	10 pn ²⁷⁻⁹ cv v f ^{3N}
	11 ras dy
2 car ²⁶⁻⁴⁸ f ^{3N} 7 y f:=	12 rb
3 cm ct ⁶ sp ³ & y w f:=	13 rb ²⁷⁻⁴ v f ^{3N} & y f:=
4 cv cm ²⁸⁻⁴ f ^{3N} & y f:=	14 sc ^{S1} B In-S w ^a sc ⁸ (Muller-5)
5 cn f ^{3N} & y f:=	15 sw

16 "tester 1-y" y ac pn w rb wy ² g ² & y f:=	<u>Altered Y's</u>
17 "tester 2-y" y ² wa cm wy ² g ² car & y f:=	27 X·Y In-EN v ptg oc sn ⁵ & y sc t ² v f car·= (no free Y)
18 "tester 3-y" y rb cm ras ² g ² & y f:=	28 X·Y In-EN y; st (no free Y) ("Multi♀")
19 un ⁴ Bx ² & y f:=	29 1 J1 ⁺ ·Y/l J1 sc ¹ (+) In-49 ptg oc B ^{M1} / y ^{S1} sc ^{S1} In car odsy f g ² dy v ras ² sn ³ ct ⁶ cm rb ec w l pn sc ⁸ ("Maxy- new")
20 w sn B	
21 y ac sc pn w rb cm ct ⁶ ras ² v g ² f car & y f:=; sc ¹⁹ⁱ /Cy, In-L lt ("Maple")	30 1 J1 ⁺ ·Y/y sc ⁴ B v ^{41b} & y sc ⁴ B v ^{41b} /y w In-49 lz ^S
22 y sc ^{S1} B In-49 sn ^{x2} sc ⁸ /oc ptg	31 1 J1 ⁺ ·Y/y In-49 f ^{S1} B ^{M1}
23 y sc ^{S1} In-49 v sc ⁸	32 sc ⁸ ·Y/y sc ^{S1} B f In-49 v & y f:=/ sc ⁸ ·Y
24 y w In-49 f	33 sc ⁸ ·Y/y Hw In-49 v ptg oc f ^{S1} B ^{M1} / y ^{S1} sc ^{S1} , In car odsy f g ² dy v ras ² sn ³ ct ⁶ cm rb ec w l pn sc ⁸
25 y w ^a cv v f	34 sc ⁸ ·Y/y In-49 B; bw ^D ♂ & y f:=; bw ^D ("Multi♂")
26 X ^{c2} y B & y f:=	35 Y ^{Lc} /X·Y ^S ("Sterilizer +")
	36 Y ^{Lc} /X·Y ^S ; cn bw; e ("Sterilizer cn bw e")

Chromosome 2

37 b pr vg
38 dp
39 dp b cn bw
40 dp stw ³ bw
41 dp Th Cy, In-L pr cn ² In-Cy R-0/Ins-NSL Ins-NSR px sp ("Cy Oster")
42 dp Th Cy cn bw/S Sp cn bw
43 fes ms cn sp/net dp ^{txI} Cy b pr Bl lt ³ cn ² L ⁴ sp ²
44 ls dp ^T Sp ms ta cn crs/S ² Cy lt ³ pr ⁺ Bl cn ² L ⁴ sp ²
45 net bw mr crs/al ² , In-Mis dp ^{txI} Cy Bl cn ² L ⁴ sp ²
46 S fes Sp ms cn mr crs/al ² In, Mis dp ^{txI} Cy pr Bl cn ² L ⁴ sp ²

Chromosome 447 Ci^D/spaCatMultichromosomal

48 y sc ^{S1} In-49 sc ⁸ ; dp b cn bw
49 cn bw; e
50 Cy/Pm; Cx, D/In(3R)Sb
51 y sc ^{S1} In-49 sc ⁸ ; bw; st pP

Leiden: Rijksuniversiteit, Genetisch Laboratorium

Note: This stock list was copied from DIS 32:62
Only some unusual stocks are listed.

Chromosome 2

cn bw Dr/Pm
cn px bw Kr/Cy
crc cn/Pm

Chromosome 3

gs
h gs th

Triploid♀ 3N:W/FM₄ & Y/FM₄ ♂Utrecht: Rijksuniversiteit, Genetisch Instituut

Note: This stock list was copied from DIS 33:75-76.

Wild Stocks

1 Oregon-K

Chromosome 1

2 cm ct ⁶ sn ³ & y w f:=	5 fu/CLB
3 cv car ²⁶⁻⁴⁸ f ^{3N} & y f:=	6 g ²
4 cv f ^{3N} & y f:=	7 pn
	8 ras dy
	9 rb
	10 rb ²⁷⁻⁴ cv v f ^{3N} & y f:=
	11 sc ^{cv} v f
	12 sc ^{S1} B In-S wa sc ⁸
	13 ("tester 1") y ac pn w rb wy ² g ² & y f:=; sc ¹⁹ⁱ /Cy

- 14 ("tester 2") $y^2 w^a cm wy^2 g^2 car & y f:=;$ 34 $dp^{Th} Cy, In-L pr cn^2 In(2)Cy R-)/$
 sc^{19i}/Cy Ins-NSL Ins-NSR px sp
 15 ("tester 3") $y rb cm ras^2 g^2 & y f:=;$ 35 $dp^{Th} Cy cn bw/S Sp cn bw$
 sc^{19i}/Cy 36 $J/In(2)LoT, 1(2)B$
 16 w sn B
 17 $y sc^{S1} B In-49 sn^{x2} sc^8/oc ptg$
 18 $y^{S1} sc^8 B f In-49 v$
 19 y w m B
 20 $y w^a cv v f$

Altered Y's

- 21 X·Y·In-EN y; st (no free Y) ("Multi ♀")
 22 $lJ1^+·Y/lJ1 sc^{J1(+)} In-49 ptg oc B^{M1}/y^{S1}$
 $sc^{S1}, In car odsy f g^2 dy v ras^2 sn^3$
 $ct^6 cm rb ec w l pn sc^8$ ("Maxy-new")
 23 $sc^8·Y/y Hw In-49 v ptg oc f^{S1} B^{M1}/y^{S1}$
 $sc^{S1}, In car odsy f g^2 dy v ras^2 sn^3$
 $ct^6 cm rb ec w l pn sc^8$
 24 $sc^8·Y/y In(1)49 B; bw^D ♂ & y f:=; bw^D$
 ("Multi ♂")
 25 $Y^{Lc}/X·Y^S$ ("Sterilizer +")
 26 $Y^{Lc}/X·Y^S; bw$ ("Sterilizer bw")
 27 $Y^{Lc}/X·Y^S; dp$ ("Sterilizer dp")
 28 $Y^{Lc}/X·Y^S; cn bw; e$ ("Sterilizer cn bw;
 e")

Chromosome 2

- 29 b pr vg
 30 bw
 31 Bl L/Cy
 32 dp
 33 dp b cn bw

Chromosome 3

- 37 e
 38 h ri
 39 l tr/e In(3R) In(3L)
 40 Mio/In(3R)Sb
 41 St

Chromosome 4

- 43 ci
 44 $ci^D/spaCat$

Multichromosomal

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

Stocks selected for abnormal abdomen

- 49 (AA) $DcxF/Mé Sb$
 50 (AA) $Cx, D/In(3R)Sb$

Deficiencies

- 53 $Df(1)N^8/dl-49, y Hw m^2 g^4$
 54 $Df(1)N^{264-105} (dm)/dl-49, y Hw m^2 g^4$
 55 $Df(1)N^{264-39} wch/FM4, y^{31d} sc^8 dm B$

SOUTH AFRICA

Johannesburg: University of the Witwatersrand, Department of Zoology

Note: This stock list copied from DIS 33:78

<u>Wild Stocks</u>	<u>Chromosome 1</u>	
1 Bethulie	17 bi $ct^6 g^2$	35 g^3
2 Bloemfontein	18 bo	36 m
3 Canton-S	19 B	37 pn^2
4 Cape Town	20 car	38 ras dy
5 Cedara	21 car^2	39 ras^2
6 Florida	22 cm	40 $ras^3 m$
7 Graaff-Reinet	23 cm car	41 rb
8 Inhaca Island	24 cm $g^3 car$	42 rb car
9 Johannesburg	25 ct v	43 rb cm g^3
10 Limpopo	26 $ct^v dy g$	44 rb cm car
11 Nelspruit	27 ct^6	45 rb cx
12 Oregon-R	28 ec	46 rb g^3
13 Stanford Lake	29 ec $ct^6 v g^3$	47 rb $g^3 car$
14 Stellenbosch	30 f_B	48 sc ec cv $ct^6 v g^2 f$
15 West Rand	31 $f^5 m$	49 sc ec cv $ct^6 v g^2 f/FM3$
16 Zoutpansberg	32 $f^5 v$	$y^{31d} sc^8 dm B l$
	33 g	50 svr w^a
	34 g^2	51 v
		52 v^{36f}
		53 v g^3

54 w	98 cn	<u>Multichromosomal</u>
55 w m	99 cn ^{35k}	
56 w m f	100 cn vg	138 bw; e
57 wa	101 cl	139 bw; e; ci ey
58 wa ³	102 cl ^{50a}	140 bw; ci ey
59 wa ⁴	103 dke c	141 bw; st
60 wa rb	104 dp	142 Cy/Pm, ds ^{33k} ; H/ In(3R)Mo; sr
61 wbl	105 lt std/cy sp ²	143 g ³ ; bw
62 wch	106 lt stw ³	144 g ³ ; st
63 w ^{co} sn ²	107 ltd	145 g ³ ; st pP
64 wcol	108 pd	146 ras ² ; st
65 we	109 pr	147 rb; bw
66 we ²	110 pr ^{42d}	148 rb; ry
67 we ³	111 sf ²	149 rb; se
68 we car	112 Su-H/Cy, pr	150 rb; st
69 we cm	113 tk sf ² abb	151 car; ry
70 we g ³	114 vg	152 car; se
71 we rb	115 vg ^{dn}	153 vg; se
72 we rb car		154 we rb; se
73 wsat	<u>Chromosome 3</u>	155 w ^w ; cd
74 w ^t fw	116 ca	156 y; bw; e; ci ey
75 w ^w f ⁵	117 cd	
76 w ^w rb 77 y	118 cu Kar	<u>Attached-X</u>
77 y	119 D/Gl	157 f B/su ^{S2} -v-pr v
78 y g ⁴	120 e	158 y/+
79 y m	121 e ^s	159 y ² su-wa wa bb/y sc ^{4L} sc ^{8R}
80 y pn	122 e ^s cd ro cmp ca/Xa, ca	
81 y rb	123 ma fl	
82 y w	124 mah	
83 y w m	125 p	<u>Inversions</u>
84 y ² su-wa wa bb	126 pP cu	160 In(1)A99b
85 y ² wa w	127 pP cu sr e ^s	161 In(1)dl-49, y fan
	128 res	162 In(1)rst ³ , y rst ³ car bb
<u>Chromosome 2</u>	129 ru	163 In(1)rst ³ , y rst ³ g ³ car
86 albasp/Cy L ⁴ sp ²	130 ry	164 In(1)w ^{m4}
87 al dp b pr Bl c px sp/ SM1 al ² Cy sp ²	131 se	165 In(1)w ^{m4} ; bw
88 a sp ²	132 sr	166 In(1)w ^{m4} ; st
89 b	133 st ^{sp}	167 Ins(1)sc ^{S1} , S, sc ^{S1} wa B sc ⁸
90 b pr	134 su ^B -pr/In(3R)C, e; pr	
91 b pr cn	135 th st	<u>Translocations</u>
92 b pr cn a	136 th st pP	168 T(1;3)04, D/ClB
93 bw	<u>Chromosome 4</u>	169 T(1;4)w ^{m5}
94 bw ^{2b}	137 ci ey	
95 bw ⁴		
96 bw ^D		
97 c px		

SPAIN

Madrid: Centro de Investigaciones Biológicas, Laboratorio de Genética

Note: This stock list was copied from DIS 33:79

Wild Stocks

Madrid	Ribadeo	Ronda 10
Mallorca	Rocafort	Ronda 30

SWEDENStockholm: University of Stockholm, Institute of GeneticsWild Stocks

- 1 Algeria
2 Canton-S
3 Djursholm 55
4 Florida
5 Karsnäs
6 Oregon
7 Stäket
8 Tunnelgatan
9 Örebro
10 Skaftö

Chromosome 1

- 11 B
12 bb
13 BB; sc⁸ Y ♂ y f:=; sc⁸ Y ♀
14 B car; sc⁸ Y ♂ y f:=; sc⁸ Y ♀
15 BB car; sc⁸ Y ♂ y f:= sc⁸ Y ♀
16 Bx²
17 car
18 cm ct⁶ sn³ ♂ y f:= ♀
19 cv
20 cv sn
21 cv v B ♂ y f:= ♀
22 ct⁶
23 ec ct v f
24 f
25 f B
26 f BB; sc⁸ Y ♂ y f:=; sc⁸ Y ♀
27 fa
28 fu; sc⁸ Y ♂ y f:= sc⁸ Y ♀
29 g² B
30 g f car ♂ y f:= ♀
31 lw^{29a} H₂/y Hw g In-49 m
32 lw^{47b} H₂/y sc⁸ f In-49 V wa
33 lz ♂ y f:= ♀
34 m
35 m f
36 Df(1)N⁸/wa
37 od car
38 pn
39 rb
40 sc
41 sc⁸
42 sc cv
43 sc cv v f
44 sc cv v car
45 sc ec cv ct⁶ v g f/FM3, y^{31d} sc⁸ dm B l
46 sc t² v f Tu car. ♂ y f:= ♀
47 sc^{S1} B In-S wa sc⁸
48 sc^{S1} B In-S wa sc⁸; y sc⁸ Y
49 sn³
50 v
51 w
52 w cv
53 w cv sn³
54 w sn³

(From DIS 33:79-81)

- 55 wa
56 wbf2
57 wbl
58 wch2
59 wco
60 wcol
61 wec2
62 wh
63 wr sc⁸ In-S
64 wt
65 Xc2 (closed-X)
66 y
67 y² eq; Df(Y)Y-bb
68 y³²⁹
69 y^{3P}
70 y⁴
71 y ac sc pn sn; sc⁸ Y
72 y ac sc pn w rb cm ct⁶ sn³ ras⁴ v m g f
73 car/sc^{S1} B In-S wa sc⁸
74 y B²⁶⁷⁻⁴⁷
75 y ec ct⁶ v f
76 y f car
77 y f Eb/sc^{S1} B In-S wa sc⁸ ♀ sc^{S1} B In-S
wa sc⁸ ♂
78 y Hw m f g car; sc⁸ Y ♂ y f:=;
sc⁸ Y ♀
79 y rb ct⁶
80 y sc
81 y sc w^e ec rb
82 y sc⁸ B f In-49 v ♂ y f:= ♀
83 y sc^{S1} f In-49 m sc⁸ ♂ y f:= ♀
84 y sc^{S1} In-49 v sc⁸ ♂ y f:= ♀
85 y sc^{S1} In-S wa sc⁸
86 y v f.= ♀ y w ♂
87 y v f car.= ♀ sc v f car ♂
88 y v g f
89 y w sn³
90 y w sn; sc⁸Y
91 y w f Bx²
92 y w spl sn
93 y wa sn

Chromosome 2

- 94 a px sp
95 a px or
96 al b c sp
97 al sp b pr Bl c px sp/SM1, al² Cy sp²
98 al dp b pr c px sp/al² Cy lt³ L⁴ sp²
99 al dp b pr cn vg c a px bw mr sp/ S² Cy
lt³ pr⁺ Bl cn² L⁴ sp²
100 al² Cy lt³ L⁴ sp²/Pm
101 al S ast ho/Cy, E-S
102 al sp
103 b
104 b cn vg
105 b pr vg

106 Bl/In(2LR)dp	134 ru se h st p ^D ss e ^S
107 bw	135 ru h th st cu sr e ^S Pr ca/Mé,
108 cn bw	T(2;3)
109 cn vg bw	136 se ss k e ^S ro
110 Cy/Pm	137 ss
111 dp b	138 st
112 dp b pr c px sp	139 st ss e ¹¹
113 ed Su ² -dx	140 ve h th
114 ft	141 ss e ¹¹
115 L ² /Cy	
116 L ⁴	<u>Chromosome 4</u>
117 pr	142 ey
118 px bw mr sp/ds ^{33k} Pm	143 ci ey
119 S ² Cy pr Bl cn ² L ⁴ bw sp/In-NSL	
In *NSR px sp	
120 sca	<u>Multichromosomal</u>
121 sp	144 cn bw; e ¹¹
122 stw ³	145 bw; st
123 vg	146 rb cm ras ² g ² ; sc ¹⁹ⁱ /Cy ♂ f:=
<u>Chromosome 3</u>	sc ¹⁹ⁱ /Cy ♀
125 ca	147 sc cv v; ri
126 D ³ /In-P	148 sp; th
127 e ^S	149 T(1;2)B ^{bd} /Cy ♀ M(2)e/Cy ♂
128 gl	150 T(1;2)B ^{ld} /Cy
129 Hnr ²	151 TX(16A1)4 B ^S y w f:=
130 ri ²	152 T(2;3)Met/Cy
131 ro	153 lt/T(Y;2)A
132 ru Hnr ²	154 y v; bw ^{VA} /L ² l
133 ru h st p ^D ss e ^S	155 y; pr; ss
	156 +=; sv ⁿ ♀ +; sv ⁿ ♂

Uppsala: Botanik-Genetiska Institutionen, Lantbrukshogskolan

Note: This stock list was copied from DIS 32:65-66.

Wild StocksChromosome 1

1 Algeria	21 B
2 Amherst-3	22 B/Y
3 Bayforbury	23 BB car; sc ⁸ Y ♂ y f:=; sc ⁸ Y ♀
4 Boa Esperanca, Minas Gerais, Brazil	24 ct
5 Canton-S	25 cv
6 Crimea	26 cv sn ³
7 Curitiba, Paraná, Brazil	27 Dp(1)w ^a ♂ y w f:= ♀
8 Florida	28 ec
9 Gruta, Argentina	29 ec ct v f
10 Hikone-R (resistant to BHC, DDT, parathione, nicotine)	30 f
11 Karsnäs	31 f B od sy car
12 Kochi-R (resistant to parathione)	32 f BB; sc ⁸ Y ♂ y f:=; sc ⁸ Y ♀
13 Oregon	33 f od car
14 Salvador, Bahia, Brazil	34 In(1)w ^{m4}
15 Samarkand	35 is
16 San Miguel, Buenos Aires, Argentina	36 lz/CLB
17 Stäket	37 ma-l y f:=
18 Tunnelgatan	38 m f
19 Örebro	39 sc ^{S1} B InS w ^a sc ⁸
20 Örebro-R (resistant to parathione)	40 sc ^{S1} InS w ^a sc ⁸
	41 sc z w ^{17G2} ec ct
	42 sn ³
	43 sp-w
	44 su-w ^a w ^a

45 w
 46 w ct
 47 w cv
 48 w cv sn³
 49 w sn³
 50 w^a
 51 w^aE
 52 w^a su-f ♂ y f:=♀
 53 wbf²
 54 wbf f⁵
 55 wbl
 56 wch wy
 57 wco
 58 wco sn²
 59 we
 60 we²
 61 we² en-we ♂ y f:=♀
 62 wh
 63 wh ct
 64 wi yb
 65 w^{sat}
 66 y
 67 y ac sc pn w rb cm ct⁶ sn³ ras⁴ v m g
 f car/sc^{S1} B InS wa sc⁸
 68 y ec ct v f
 69 y f Eb/sc^{S1} B InS wa sc⁸
 70 y² sc w^a wch fa ♂ y f:=♀
 71 y² sc wⁱ
 72 y² sc wⁱ wch ♂ y f:=♀
 73 y² su-wa w^a we ♂ y f:=♀
 74 y² w^a
 75 y² w^a w
 76 y z
 77 z ec
 78 z we ec
 79 z w¹¹ E⁴

Chromosome 2

80 bw

81 cn vg bw
 82 Cy/Pm 83
 83 Cy/S
 84 fes lt³/cy al² lt³ L⁴ sp²
 85 net
 86 pr
 87 S/NS, px sp
 88 vg

Chromosome 3

89 D³/InP
 90 ri²
 91 ro
 92 ru h st p^P ss e^S
 93 se
 94 ss
 95 st ss e¹¹

Chromosome 4

96 ey
 97 svⁿ

Multichromosomal

98 cr-u/Cy; (we)
 99 Cy/S; D/InP
 100 In(1)w^{m4} y⁵¹¹; E-Var 4/Cy
 101 In(1)w^{m4}; E-Var 5/Cy
 102 In(1)w^{m4}; E-Var 8/Cy
 103 L²/+, sp; th
 104 sp; th
 105 T(2;3)bw VDe⁴/Cy
 106 T(1;4)w^{m5}
 107 wch; Su-wch/Cy
 108 y^{S1} sc⁸ InS y^{3P}; al² Cy lt³ sp²/dp b Pm¹;
 ru h D³ InCXF ca/Sb In(3R)

NEW MUTANTS

Report of A. B. Burdick

dy^{58k}: dusky-58k M.E.Krawinkel, 1958k. Spontaneous in an isogenic pol stock, W-160 pol. A bona fide new mutant since dy, the only other dy mutant known at the time dy^{58k} was discovered, was not present in stocks at this institution at that time, and the pol marker of the stock in which it occurred was present in the single mutant male observed. Recombination and complementation tests show that dy^{58k} is the right-most known element of the m-dy complex. It recombines with Df(1)259-4, with all m's except m^D, and not with dy. It appears to be the right-most element because it consistently shows higher recombination and more complementation with m-type mutants than does either m^D or dy. Wing length is shorter than dy, about the same length as the longer m-type mutants. Fertile in both sexes. RK1.

m^{59a}: miniature-59a M.E.Krawinkel, 1959a. Probably spontaneous in an isogenic wild-type background (W-126) which several generations before had been treated with about 50r of X-ray. Shows low complementation with m-mutants and high with dy-mutants. Recombines with Df(1)259-4 on its left and m on its right. No m-mutant (except Df(1)259-4) has been shown to be left of m^{59a}. Female fertility very low; male fertile. RK2.

dy^{60k}: dusky-60k A.B.Burdick, 1960k. Spontaneous in a SM5, al² lt^V Cy sp²/da stock; isolated in uniform stock background so that we know that it could not have arisen as a contamination. Allele tested with m⁶⁰¹ and dy^{61a} (see DIS-35, new mutant report of P. T. Ives); shows high complementation with m⁶⁰¹ and low with dy^{61a}. Similar to other dy's; fully fertile in both sexes. RK1.

rk⁴: rickets-4 R.C.Jackson, 1954. Originally called cq (creeper) in DIS-28, 1954. H. U. Meyer in DIS-32 reports cq to be an allele of Edmondson's rk. Therefore, cq is now rk⁴.

r^{58a}: rudimentary-58a M.Burdick, 1958. Induced in mature wild-type sperm by 4000r X-ray. Resembles r (Morgan, 10f) and r⁹ (Bridges, 20b3), wings truncated, blistered, wing-veins, particularly L4, frequently interrupted, marginal bristles sparse, somewhat longer, and disarrayed. Expression variable but does not overlap wild-type. Female entirely sterile. Recombination tested in 2894 flies to car and 974 to f giving confidence intervals that include 1-54.5, Morgan's locus for r. Functionally allelic with r⁹. RK2.

Report of W. W. Doane

(This report supersedes that in DIS-34 inadvertently attributed to S. Counce.)

fs(2)adp: female sterile(2)adipose Counce, 1956. 2-83±. Pub. Doane, 1959, Genetics 44:506; developmental and physiological studies, Doane, 1960, Ph.D. Thesis, Yale Univ. Spontaneous in Kaduna wild stock maintained in Edinburgh. Adult fat body hypertrophies as cells become distorted by enormous oil globules. Abnormal fat bodies visible through body wall of 6-day old and older adults when submerged in 95% alcohol, then water. Adult corpus allatum of mated females hypertrophies. Females completely sterile, sterility autonomous. Eggs laid by homozygotes show meiotic and/or mitotic abnormalities and never develop beyond early cleavage stages. Males 78% fertile. Heterozygotes fertile, but females become sterile with age. Viability generally good, but longevity reduced; homozygotes with selective advantage under starvation; heterozygotes superior under desiccation. Average water content of well fed adults reduced, while percentage of lipids, as a function of dry body weight, is almost double that of wild type. Iodine numbers show greater degree of saturation of mutant lipid extracts than of wild type. RK2.

The mutant flag (fg), reported as a new mutant in DIS-34 and located approximately at 2-20±, has been more accurately located by linking it with a dumpy marker. The new cross-over data indicate it to be at 2-22±, and so it has been checked for allelism with the mutant spade (spd), located at 2-22.3±. The latter was obtained from the spd gt-4/SM5, al² Cy lt^V sp² stock at Pasadena, California. Spd is given

the rank of RK₅ in Bridges and Brehme because of its poor penetrance; fg is fully penetrant with an RK₁. None of the spd/sdp flies examined from stock bottles has shown wing effects comparable to those described for fg, and most of them resemble wild type. However, when spd gt-4/Cy females were crossed to fg/fg males (or the reciprocal cross made), all of the non-Cy offspring showed a wing effect which ranged from a slight shortening to a shape mid-way between the phenotypes of the two different mutants. It appears, therefore, that fg and spd are allelic and that the former should henceforth be referred to as spd^{fg} (spade^{flag}).

Report of J. L. Hubby

Recovery of another rosy allele. Ins(3RC;3LP) Sb e^s/ry² was shown to have a rosy phenotype and greatly reduced xanthine dehydrogenase activity (Hubby and Forrest, 1960). A double crossover between st and Sb was recovered from this inversion which proved to have a rosy phenotype when homozygous or when in combination with rosy or rosy². No crossovers have been recovered among approximately 5000 progeny from females carrying rosy² and this mutant. This mutant has therefore been designated rosy³.

Homozygous ry³ males show traces of isoxanthopterin and uric acid in their testes, hence ry³ is a "leaky" mutant with respect to the products of xanthine dehydrogenase. Thus far no satisfactory procedure has revealed convincing evidence of xanthine dehydrogenase activity in extracts of this mutant.

Report of P. T. Ives.

dy^{61a}: dusky^{61a}. Ives, 61a24. Like dy. Induced by 1 kr γ radiation in an Oregon-R/rucuca sperm which was deposited on day 5 of an exhaustive mating schedule. Functional allelism to dy established by A. B. Burdick who found normal recombination (with g) and good fertility and fecundity in both sexes. RK1

m⁶⁰¹: miniature⁶⁰¹. Ives, 60126. Like m. Induced by 1 kr γ radiation in an Oregon-R/rucuca sperm which was deposited on day 6 of an exhaustive mating schedule. Functional allelism to m established by A. B. Burdick who found recombination with g somewhat reduced and fertility and fecundity good in both sexes. RK1

sd^{58d}: scalloped^{58d}. Ives, 58d14. Strongest sd allele, with vg-like wings and weak vg-like effects on halteres and bristles. Induced by 1 kr γ radiation in an Oregon-R sperm which was deposited on day 7 of an exhaustive mating schedule. Functional allelism to sd indicated by strap shaped wing in sd/sd^{58d}. Recombination normal in y ct⁶ ras² regions but reduced by 80% between ras² and f, suggesting a small In associated with sd^{58d}. Not studied cytologically. Genetic tests show no T. Relative frequency of sd^{58d} is sometimes low when competing with non-sd flies but a pure line breeds well. Combines readily with vg alleles and vg deficiencies. RK-2.

Report of James F. Kidwell

Dfdr^{60J} - Spontaneous recurrence in sixth generation of full sib mating derived from Princeton wild stock. Expression varies from both eyes absent to wild type. Expression may be asymmetrical. Penetrance varies from 75 to 100 per cent. Penetrance increased by selection for reduced eye. About 5 per cent of flies Dfdr^{60J}/+; ey/+ exhibit deformed phenotype.

Report of H. W. Lewis

alpha⁻¹. The report of this mutant was erroneously included in DIS-34 under the report of E. B. Lewis.

Report of A. Schalet

ma-l²: maroon-like² Schalet, 1961. From X-rayed y ct⁶f. Dp(y⁺sc^{v1}) ♂ mated to ma-l¹ females. Hemizygous male and homozygous female are viable and fertile. Brownish-red eye color is like ma-l¹ and ma-l^{bz}. ma-l² does not complement ma-l¹

ma-1¹, ma-1^{bz} or ma-1³ (see below).

ma-1³: maroon-like³ Schalet, 1961. From X-rayed y ct⁶f. Dp(y⁺sc^{vi}) ♂♂ mated to ma-1¹ females. Hemizygous males inviable. In a small-scale test the absence of crossovers between v and ma-1³ indicate possible association with a gross rearrangement. Heterozygotes of ma-1³ with ma-1¹ or ma-1² are mutant in appearance and similar in color to the homozygotes of the latter alleles. A chromosome carrying v and ma-1³ heterozygous with y v f ma-1^{bz} chromosome also shows a mutant appearance with respect to maroon-like.

Report of A. H. Sturtevant

Correction:

spa^{pol}: sparkling-poliert. The mutant poliart of Hadorn (Rickenbacher, DIS 27:59) is an allele of spa, giving when crossed to spa, heterozygotes with eyes slightly more extreme than spa. spa^{pol} is probably the most useful recessive in the fourth chromosome.

Report of V. Tinderholt

Cyg: The Curly gene Tinderholt, 58f 2-6.1±.9. The Curly gene is generally inseparable from the inversion, In (2L) Cy, but has been crossed out by increasing the frequency of double crossovers. This was accomplished by using females carrying the complex inversions FM6; SM5 (which includes In (2L) Cy); TM3 Ser, as the three major chromosomes. The dominant character associated with this gene when free of the inversion is identical to the one resulting from its presence within the inversion. The symbol Cyg was chosen because Cy generally is used to designate the gene-inversion combination.

The Curly gene was first localized between heldout (ho, 2-4.0) and echinoid (ed-11.0). Out of 2,489 flies, there were 1,127 Cyg ed and 1,241 wild type non-crossovers. The crossovers consisted of 64 Cyg and 57 ed giving a value of 4.9±.9 crossover units to the left of ed. RK1.

TM3 Sb Ser: Third Multiple 3 with Stubble and Serrate See DIS 34:51, Report of E. B. Lewis, and DIS 34:53-54, Report of V. Tinderholt. The third chromosomal multiple rearrangement, TM3 Sb Ser carries the genes bx^{34c} Sb ri p^D sep and e^S. The Stubble gene apparently arose from a new mutation and not from a rare double crossover as was suggested in the previous DIS note. The gene bx^{34c}, 0.6 crossover units from Sb still remains in the chromosome. A double crossover would have removed bx^{34c} as Stubble was inserted.

LINKAGE DATA

Report of D. J. Nash and E. C. Keller, Jr.

The dominant sex-linked miniature wing mutant reported in DIS-34 appears to be identical to m^D. The mutant is allelic to both m and dy, and in its interactions with m and dy cannot be distinguished phenotypically from the interactions involving m^D. No crossovers have been found among over 5,000 progeny which might have shown a crossover between this allele and m^D.

STOCK LISTS

PITTSBURGH, PENNSYLVANIA: UNIVERSITY OF PITTSBURGHD. persimilis

(From DIS 33:106)

Wild StocksChromosome 2

Yosemite National Park, el. 8000' (White Wolf)
 Whitney (4 strains)
 Klamath (4)
 Mendocino (4)

Dl cd
 cd

Multichromosomal

Yosemite, el. 10,000' (Timberline)j
 Whitney (4)
 Klamath (4)
 Mendocino (4)

Dl or Cy

Mather, California: 15 strains incl. Wh, Kl, and St

ROCHESTER, NEW YORK: THE UNIVERSITY OF ROCHESTERD. busckii

(From DIS 33:106)

There are more than 300 stocks of *D. busckii* in the collection of J. Krivshenko. They include X-chromosomal and autosomal mutants (with visible effects), in various combinations, as well as a number of special stocks in which lethals are associated with chromosomal aberrations. Dominant and recessive markers, associated with inversions or other types of chromosomal aberrations, are available for each chromosome. There are also 17 strains from geographically remote populations.

D. persimilisWild Stocks: 12 strains from various localities in Western North America.Chromosome 1Chromosome 2Chromosome 3

se

ssa

Delta or Cy

D. pseudoobscuraChromosome 1Chromosome 3Multichromosomal

ct se ll sp tt

or

Ba Cy sparky

Chromosome 2Chromosome 4

up bx Ba gl (in)/lethal

in hk j

Wild strains homozygous for gene arrangements on the third chromosomes, as follows:

Standard
 Mather (6 strains)
 Pinion (6)

Ferron (13)
 Gunnison (16)
 Leman Cave (13)
 Mono Lake (10)
 Pinion (6)

Chiricahua
 Mather (6)
 Pinion (6)

Arrowhead
 Bryce (8)

114 inbred lines of the above Arrowhead stocks in F₁₉-F₂₈ of sib mating
 33 strains from various geographical localities in Western North America
 12 strains each from: Helena, Calif. Lone Pine, Calif.
 Sebastopol, Calif. Wild Rose, Calif.
 Hopeland, Calif. Placerville, Calif.

Other Species

D. fuliginea: Rochester, N.Y. (1 strain)
 D. funebris: Rochester, N.Y. (1)
 D. hydei: 1 strain heterozygous for an inversion on the second chromosome including sections 2-D1 through 2-G2 (collected at Raleigh, N.C., August 1954). 1 wild strain from Rochester, N.Y.
 D. immigrans: Rochester, N.Y. (1)
 D. miranda: Big Basin (1)
 D. repleta: Rochester, N.Y. (2)
 D. robusta: Rochester, N.Y. (1)
 Megacelia scalaris: Princeton, N.J. (1)

FRANCE

Gif sur Yvette (S et O): Centre National de la Recherche Scientifique,
Laboratoire de Génétique Evolutive (From DIS 27:70)

D. funebris

Wild type from Challuz
 Wild type from Chatenay-Malabry
 Wild type from St Mandé

D. simulans

Wild type from Dr. Haldane Mutant types: Net Pm
 Wild type from Dr. Sturtevant se (?)
 Wild type from South Africa

ITALY

Pavia: University, Institute of Genetics
 (Type Culture Collection of Drosophila Species) (From DIS 33:113)

D. acanthoptera (1 strain)	D. duncani (1)	w sn np
D. affinis (1)	D. funebris	D. gibberosa (1)
D. algonquin (1)	Wild Stocks (3)	D. guttifera (1)
D. ambigua (2)	Mutants: Bb ^v	D. helvetica (1)
D. athabasca (1)	b w s; st	D. hydei (1)
D. azteca (1)	cn	D. immigrans (1)
D. bifasciata	co np; st	D. kuntzei (1)
Wild Stocks (3)	co no; st, cu	D. latifasciaeformis (3)
Mutants: a	co np/Stub ^v	D. lativittata (1)
f	cu; st	D. lebanonensis (1)
g	ev	D. littoralis (1)
ob	miniature-vermillion	D. miranda (1)
or	np	D. montium (1)
y	Pch	D. narragansett (1)
sex-ratio (2)	sn ² ; st	D. nitens
D. busckii (1)	sn ² w y np	Wild Stocks (3)
D. buzzatii (1)	st 45h	Mutants: or
D. cameraria (1)	Va	y
D. cardini (1)	w	D. obscura (2)
D. dscabibi Burla (1)	w N np	D. persimilis (1)

D. Phalerata (1)	D. spinofemora (1)	D. tripunctata (1)
D. prosaltans (1)	D. subbadia (1)	D. tristis (2)
D. pseudoobscura (2)	D. subobscura	D. victoria (1)
D. putrida (1)	Wild Stocks (1)	D. virilis (1)
D. repleta (1)	Homozygous standard:	D. willistoni (1)
D. robusta (1)	Esperöd	D. yakuba Burla (1)
D. setifemur (1)	Kussnächt	Zaprionus vittiger (1)
D. simulans (2)	D. transversa (1)	Z. tuberculatus (1)

KOREAKwangju: National Chunnam University, Laboratory of GeneticsD. virilis

(From DIS 31:104)

Wild StocksInversion

Japan (2 strains)
Korea (5 strains)

In(X)Spd

Other Species

D. alboralis (1 strain)	D. cheda (3)	D. lutea (3)
D. angularis (1)	D. coracina (3)	D. mirim (1)
D. arizonensis (1)	D. duncani (1)	D. mulleri (1)
D. auraria (5)	D. hamatofila (1)	D. nigromaculata (3)
D. bifasciata (2)	D. hayashii (2)	D. repleta (1)
D. bizonata (3)	D. histrio (2)	D. suzukii (2)
D. busckii (3)	D. immigrans (3)	D. testacea (1)
D. buzzatii (1)	D. lacertosa (2)	D. unispina

SPAINBarcelona: Centro de Genética Animal y Humana del Consejo Superior de Investigaciones Cientificas

(From DIS 33:117)

D. ambigua: several Spanish stocks	D. mercatorum pararepleta: Jijuca (Brazil)
D. bifasciata: Pavia (Italy)	D. phalerata: several Spanish stocks
D. busckii: Barcelona	D. repleta: New Haven (Conn.); Berlin
D. cameraria: Cantonigrós (Spain)	D. simulans: Barcelona
D. funebris: several Spanish stocks	D. subobscura: several Spanish stocks; mutant stocks
D. immigrans: Barcelona	D. transversa: Montnegre (Spain)
D. kuntzei: Cantonigrós (Spain)	Parascaptomyza disticha: Barcelona
D. mercatorum mercatorum: Barcelona	

Irwin H. Herskowitz, Editor

D. = Drosophila; D.m. = Drosophila melanogaster

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Angus, D. *Drosophila* collection from the Territory of Papua-New Guinea.

The following are the results and ecological notes of flies of the *immigrans* sp. group caught during February 1961.

Results:

	rubida (%)	pararubida (%)	setifemur (%)	Total
Port Moresby	479 (60)	169 (21)	153 (19)	801
Bulolo	245 (45)	199 (36)	105 (19)	549
Lae	14 (3)	247 (58)	169 (39)	430
Kavieng	17 (20)	40 (50)	24 (30)	81
Rabaul	428 (18)	632 (26)	1348 (56)	2408
	1183 (28)	1287 (30)	1799 (42)	4269

It was noted that *D. pararubida* was the dominant species over fermenting cocoa pods and citrus and that *D. setifemur* was particularly associated with rotting five corners (*Averrhoa carambola*).

Barbour, Evelyn and S. Zimmering
Preliminary analysis of a Y chromosome from nature carrying a mutant allele of bobbed.

In routine experiments to introduce Y chromosomes derived from males caught in nature into a y f double-X female, it was found that in the presence of one of these Y chromosomes, symbolized Y-+17, the double-X females hatched very late and showed an extreme bobbed effect, but proved to be fertile. Similar results were obtained from $sc^4-sc^8/Y-+17$ males. To test the dosage effect of one vs. two Y-+17 chromosomes, y In49 f car/y $sc^4 w sc^8/Y-+17$ females were crossed by $w^{ec3}/Y-+17$ males. Of 194 y $sc^4 w sc^8$ males recovered, 121 (62%) appeared non-bobbed and 73 (38%) appeared bobbed. It is inferred that the non-bobbed males carried two Y-+17 chromosomes and the bobbed males only one. On this interpretation, the bobbed allele in the Y-+17 chromosome acts as a typical hypomorph, as described for bb by Stern (1929). Experiments were carried out to determine the effect of this modified Y chromosome on secondary non-disjunction. Females of the constitutions y In49 f car/y² $sc w^a ec/Y-+17$ and y In49 f car/y² $sc w^a ec/Y-Oregon R$, and having approximately the same autosomal background, were crossed by Oregon R males. The frequencies of XX-Y segregations, calculated from F₁ female offspring only, were found to be as follows: 40.2% (2577 F₁ ♀♀) from the former, and 64.9% (1794 F₁ ♀♀) from the latter. The results suggest a possible impaired pairing site in the Y-+17 chromosome. No information is as yet available on disjunction of X and Y in males carrying the Y-+17 chromosome.

Bateman, Angus J. X-ray induced "crossing-over".

Analysis is continuing of "cross-overs" recovered from matings of b pr vg/ + + + ♂ to b pr vg ♀♀, over the period 5 to 11

days from irradiation of the ♂. It had earlier been supposed that r₁ recombinants (b, pr or vg) could be point mutations or deletions as well as true cross-overs, but that r₂ recombinants (b pr or pr vg) must represent true cross-overs. The latter assumption is now found to be untrue for two reasons: some r₂ recombinants are lethal when homozygous; and in some samples the r₂ class is larger than the r₁ class. It is concluded that there are at least 4 modes of origin of "cross-overs" during the period under study (which we presume to consist largely of irradiated spermatocytes)

1. Point mutations (r₁)
2. Deletions (r₁)
3. Illegitimate crossing over (r₁ = deletion;
r₂ = duplication)
4. True crossing over (r₁ = r₂)

Each illegitimate cross-over will yield one deletion and one duplication. But the duplication would be expected to be more viable in the zygote than the deletion, so that the observed yield of r₂ from this source would be more than that of r₁. We have found that on days 5, 6 and 7 r₁ exceeds r₂
on days 8 and 9 r₂ exceeds r₁
on days 10 onwards r₁ = r₂

This is interpreted to mean that the commonest modes of formation of "cross-overs" on days 5, 6 and 7 are point mutations and simple deletions, on days 8 and 9 illegitimate crossing over and on later days true crossing over.

Beatty, R. A. and N. S. Sidhu. A note on the occurrence of bulbous testes ends in Crianlarich strain of Drosophila melanogaster.

The testes of the Crianlarich strain of Drosophila melanogaster show two differences from other strains: (1) the ends are bulbous to a greater or lesser extent, (2) the colour varies from normal yellow to nearly white. The former condition is here named bulbous testes and apparently has not been reported before in this species.

Stern (1941) demonstrated the importance of the vas in determining the shape of the testes. He showed that the vasa deferentia from species with non-coiling testes fail to induce coiling in testes which normally have coils, and vice-versa, that the vasa deferentia of "coiling-species" will cause normally uncoiled testes to coil. In coiling species, the testes are uncoiled and ball shaped without any attachment up to 30 hours after puparium formation (Stern 1941). The coiling of testes is a differential growth function due to a growth promoting substance. The interaction between testes and vas gives rise finally to the imaginal form of testes. This sort of development of testes in males is common in the "coiling species" of Drosophila. However, the high frequency of bulbous testes ends in Crianlarich strain, and a low frequency of cases of uncoiled testes (mostly cases of failure of one testis to coil, and remaining ball-like) show that the differential growth function, of the growth promoting substance, fails to complete the growth in the first case and leaves the testes uncoiled in the second case.

It has been noted that there are cases of failure of completion of growth in testes in D. subobscura also. In one male belonging to a natural large strain from Aberdeen, the testis of one side was found to be ball-shaped, instead of having the elongated and tubular shape, characteristically found in the species.

The uncoiled testis lobe sends no spermatozoa into the vesicula seminalis of its side, and the latter is smaller in size than the normal one of the other side, and empty. This vesicula seminalis is seen to be translucent, (being empty), in a permanent stained preparation.

It is believed that the fertility of the males must be affected, in case of occurrence of ball-shaped testes which yield no mature spermatozoa and their seminal vesicles remaining empty. In the case of bulbous testes, probably the fertility remains unaffected. It is still a matter of speculation that the Crianlarich strain of Drosophila melanogaster does not have a low fertility because of the character of bulbous testes ends and some though very low frequency of uncoiled testes. The overall fertility of the strain probably remains unaffected in spite of the occurrence of some half-sterile males (i.e. males with one testis uncoiled).

The colour of testes in Crianlarich strain varies from yellow to creamy white, the colour of the eyes of the flies always being red. The previous literature on the colour of testes shows that the colour of eyes and pigmentation of the tunica externa of testes go together, i.e. if the eyes are dark coloured as in wild type, the testes are yellow in colour, and flies with light eye colour have light colour of testes also, ranging from yellow to nearly white. In the Crianlarich strain, it has been found that the colour of testes varies from yellow to creamy white, in spite of the colour of eyes always remaining dark, i.e. red.

The bulbous testes ends character is inherited as a recessive. The crossing of Renfrew stock and Crianlarich gives all normals in F_1 , and nearly $\frac{1}{4}$ bulbous testes ends, in F_2 .

We are grateful to Dr. F. W. Robertson for very kindly providing us the stocks of flies used for these studies.

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Table to show the higher incidence of bulbous testes ends in Crianlarich strain compared with Oregon.k.

Strain	No. of flies investigated	No. of flies with bulbous testes ends	No. of flies with bulbous testis end on one side only	No. of flies with Uncoiled testis on one side	No. of flies with normal testis in the population
Crianlarich	46	25	10	5	6
F ₁ generation of Renfrew X Crianlarich (C x R) flies	51	-	1	-	50
F ₂ (same)	25	7	2	-	16
Or.K. flies	60	6	4	-	50

Brousseau, George E., Jr.

The effect of M(2)S10 on the fertility of some compound XY chromosomes.

On two separate occasions an attempt to establish a stock of the constitution: $y^2 su-w^a w^a bb: = /y^+Y; Cy/M(2)S10 X Y^{SX} \cdot Y^L, In(1)EN In(1)d1-49, y v f car/y^+Y; Cy/M(2)S10$ failed owing to the infertility

of the M(2)S10 males, while their non-Minute brothers were normally fertile. A survey of the effect of M(2)S10 on the fertility of several compound XY's of diverse origin and structure was then undertaken. In these tests, the fertility of males of the constitution $X \cdot Y/y^+Y; M(2)S10/+$ was compared to their $X \cdot Y/y^+Y; Cy/+$ brothers. These males were crossed individually with 2 $yv/yv; bw/bw$ females from a stock that consistently yields over 95% fertile matings. Twenty males of each genotype were tested. Over 90% of the non-Minute males were fertile in every case. In contrast, all but one of the compounds showed a lowered fertility when M(2)S10 was present. The exception was $Y^{SXY^L} \cdot Y^S$ which showed 100% fertility. Only 60-65% of the males that were $X \cdot Y^L, X \cdot Y^S$ or Y^{SXY^L} were fertile. For $Y^{SXY^S} \cdot Y^L$ and $Y^{SX} \cdot Y^L, In(1)EN In(1)d1-49$ the value was 35-40% and for $Y^{SX} \cdot Y^L, In(1)EN$ it was only 15%. In all cases of a lowered number of fertile males, the fertility of the fertile males was drastically reduced, single males often yielding only a very few progeny. No generalization concerning the structure of a compound and the effect of M(2)S10 on its fertility is possible. Nor does the amount of Y chromosome material, as expressed by the number of fertility factors sets present, seem to be important. This latter point also argues against a position effect explanation (M(2)S10 is a strong enhancer of variegation). It is likely that M(2)S10 interacts with some, as yet undefined, property of the sensitive compounds. These results suggest that this factor may not be the same in each of the compounds.

Burdette, W. J. Effect of penicillin on mutation rate following irradiation in different concentrations of oxygen.

Previous work indicated diminution in lethal mutation rate in Drosophila melanogaster following irradiation when antibiotics were administered. These studies have been extended to an inquiry into the

effect of penicillin on the alteration in pattern of induced mutations in an atmosphere of oxygen. Males of the $st sr e^S ro ca; tu 36a$ strain were irradiated (3000 r) and lethals detected on the X chromosome by appropriate crosses with the $sc^{S1} B InS w^a sc^8$ stock. The first group was not treated, the second was irradiated, the third was raised on medium containing 20,000 units of penicillin per ml. of medium and irradiated, the fourth group was maintained one minute before and ten minutes during irradiation in an atmosphere of 100 per cent oxygen, and the fifth group was similar to the fourth except it was raised on medium containing penicillin in the same concentration as group three. After irradiation at 20 hours of age, males were mated successively to different virgin females at intervals of two days. Lethals in the progeny of these respective matings representing successive stages of spermatogenesis during irradiation are indicated by the letters A - G and their number and distribution are recorded in table 1 and figure 1.

Striking reduction in mutation rate was found when penicillin was added to the medium both in the groups irradiated in air and in oxygen. In the latter, the effect is apparent in stages B and C, but in the former it is evident throughout spermatogenesis.

Table 1

Stage of Spermato-genesis	Control			3000 r			3000 r Penicillin			3000 r O ₂			3000 r O ₂ Penicillin		
	Total	Lethals	Per Cent	Total	Lethals	Per Cent	Total	Lethals	Per Cent	Total	Lethals	Per Cent	Total	Lethals	Per Cent
A	795	1	<u>0.12</u>	600	30	<u>5.00</u>	570	17	<u>2.98</u>	229	2	<u>0.87</u>	216	1	<u>0.46</u>
B	654	1	<u>0.15</u>	609	40	<u>6.56</u>	560	26	<u>4.64</u>	287	23	<u>8.01</u>	294	12	<u>4.08</u>
C	649	2	<u>0.30</u>	354	23	<u>6.49</u>	446	16	<u>3.58</u>	175	24	<u>13.71</u>	235	20	<u>8.51</u>
D	647	0	<u>0</u>	278	17	<u>6.11</u>	298	11	<u>3.69</u>	209	6	<u>2.87</u>	178	5	<u>2.80</u>
E	551	0	<u>0</u>	405	10	<u>2.63</u>	400	7	<u>1.75</u>	222	2	<u>0.90</u>	185	1	<u>0.54</u>
F	512	0	<u>0</u>	452	11	<u>2.65</u>	365	3	<u>0.92</u>	211	3	<u>1.74</u>	225	2	<u>1.05</u>
G	440	0	<u>0</u>	365	4	<u>1.24</u>	370	2	<u>0.54</u>	161	1	<u>0.75</u>	213	2	<u>1.14</u>
Total	4248	4	<u>0.09</u>	3063	135	<u>4.41</u>	3009	82	<u>2.73</u>	1494	61	<u>4.08</u>	1548	43	<u>2.78</u>

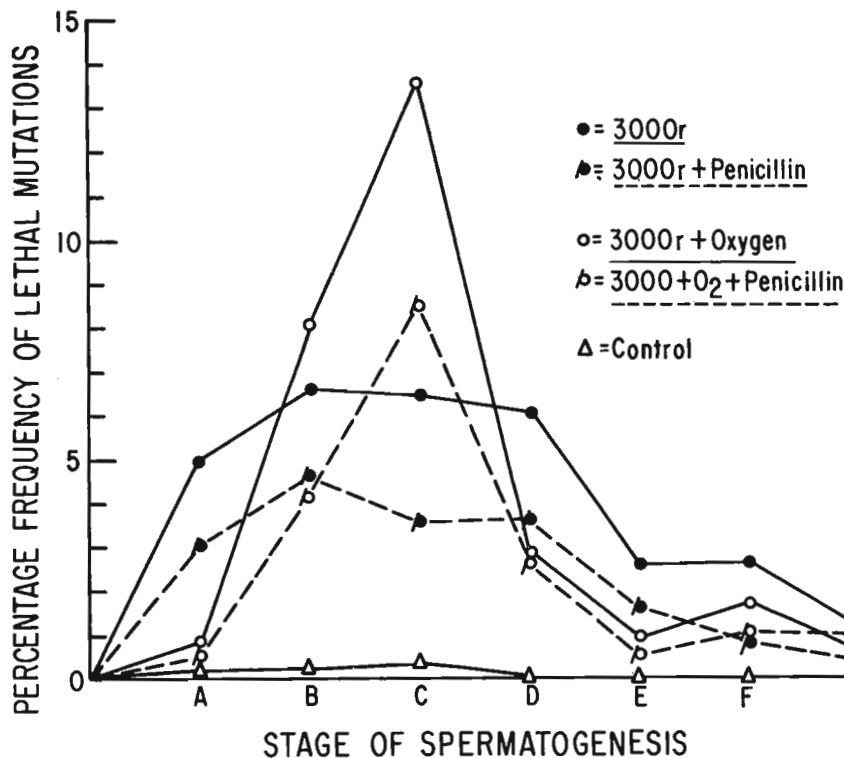


Figure 1: Frequency of Lethal Mutations at Different Stages of Spermatogenesis

genesis. The increase in frequency of lethal mutations when the content of oxygen prior and during irradiation is raised from that in the ambient atmosphere to 100 per cent is greatest in stage C, but this is at the expense of such a change in other stages. When the total frequency of lethal mutations at all stages for the groups irradiated in air is compared to the frequency for those irradiated in 100 per cent oxygen, no increase is found in the latter group. Apparently an increase in percentage of oxygen has resulted in a redistribution of the mutations without increasing total number, whereas penicillin has reduced the number when appropriate groups are compared. The reduction with antibiotic treatment is rather uniform throughout spermatogenesis when irradiation is carried out in air.

Burdick, A. B. 1(2)55i at Erie, Pennsylvania.

laboratory. Various reports by T. Mukai, S. M. Schnick, and myself have dealt with heterozygote viability of this lethal. We have found that this heterozygote is super-viable and have not been able to implicate anything but the single locus itself as a cause of the apparent heterosis. All through these studies we have wondered whether the lethal came from the wild population, or was a mutation that had occurred in the laboratory after the stock had been brought in. Certain considerations have led us to think that even if the lethal had come from the wild population it would not necessarily still be in the population -- six years later. I went back to the Erie area this fall and collected flies again. Our tests show that 1(2)55i is still in the Erie wild population.

Carlson, E. A. and R. Sederoff. A selective scheme for recovering pseudoallelic recombinants,

1(2)55i was found in our so-called "Erie" wild stock (W-1) about a year after the stock had been brought into the laboratory. The principle first suggested by Whittinghill (Science 111:377) for the recovery of selected recombinant types has been

"conversion" phenomena, and reverse mutations.

successfully applied in the construction of a lethal selector system for complex loci by Schalet and Chovnick (DIS 34:104).

However, this has the inherent disadvantage that four lethals must be used, with each pair of lethals on either side of the allelic region very close to one another. This makes such a system difficult to construct without considerable effort. Furthermore, two sets of such "tester" stocks must be employed so that the wild type crossovers between the alleles can survive in the "sifter" stock used to kill the undesired chromosomes. Finally, this system is valid almost exclusively for single wild type crossovers and it cannot be used to detect double crossovers, reverse mutations, or "conversion" phenomena in the complex locus.

It is possible, however, to use a modification of this principle with certain loci and obtain any wild type event picked up by the "sifter." This proposed system is also simpler to construct and involves only one initial stock for the allelic tests.

In the system illustrated here, the dumpy region is used for the allelic series in question. The stocks are $S dp^x Sp / InCyL,Cy$ and $Cy dp^y / InCyL$. The "tester" heterozygotes are thus: $S dp^x Sp / Cy dp^y \text{♀}$. In this example the Cy is a dominant visible with recessive lethal manifestation which has been obtained free of its former location near the left end breakpoint of the $InCyL,Cy$ chromosome (Tinderholt, unpublished). S and Sp are also dominant visible with recessive lethal effects. The alleles dp^x and dp^y represent any two members of the dp series used for analysis. The "sifter" stock has the composition $Df(ed^+ dp^+)2MB / S^2 InCyL,Cy$. Hence all non-recombinant chromosomes from the "tester" heterozygote are killed by the $S^2 InCyL,Cy$ chromosomes. The distance between S and Cy is about six map units. Hence half of these, carrying neither S nor Cy may be passed on to the "sifter" stock, with a total of 1.5% surviving as a $S^+Cy^+dp Sp / S^2 InCyL,Cy$ combination. The other chromosome in the "sifter" stock, bearing the dumpy deficiency renders the allelic combinations semi-lethal, with a survival to the adult stage of about 5% at 27°C. The total surviving progeny would thus not exceed 7%. Any change occurring in the dumpy region resulting in a non-dumpy phenotype, would complement the deficient area and hence its chances of survival would be nearly unity. Phenotypically there are only two classes of wing mutation in this system -- those with oblique, curly wings and those with very reduced truncated wings. The exceptions would appear as non-dumpy flies. The rate of recombination can be determined by the same procedures outlined by Schalet and Chovnick.

If males are used in this tester stock, and mature sperm are irradiated, then reverse mutations can be selected appearing with an apparent frequency 20 times greater than would be obtained without selective techniques. The tester stock in these reversion studies could use the same scheme as outlined before, but the allele tested would be homozygous. By further increasing the temperature to 29°C, the viability of the heterozygotes is diminished to less than 1% and the selective technique can thus determine reversion frequencies of 1×10^{-7} with the same amount of labor as is presently used for frequencies of 1×10^{-5} .

This system should work for any allelic series which expresses a decrease in viability in compound with a deficiency for its entire region. Other types of selective techniques can be devised using other viability characteristics (such as prune-killer as a "sifter" for pseudoallelism or reverse mutation among prune alleles). This study is supported by Grant G 14222 from the National Science Foundation.

Chandley, A.C. Mutations induced in presumed spermatocytes

Mating of F_1 males of Drosophila melanogaster following 1000r X-rays has shown maximum sensitivity to the induction of

sex-linked lethals and translocations on the 5th day from irradiation followed by a period of low fertility on the 8th. By analogy with the mouse, the 8th day could be expected to represent irradiated spermatogonia and the 5th day irradiated spermatids.

The intervening days 6 and 7 could therefore be expected to represent spermatocytes. These have been investigated for the incidence of dominant lethals, sex-linked recessive lethals, translocations and deleted X's, with the following results:

DAY	5	6	7	8
Dominant Lethal %	38.4	63.0	62.0	59.6
Sex-linked Lethal %	4.08	4.09	4.00	2.62
Translocation %	6.22	5.71	2.92	0.70
Deleted X's %	0.17	0.35	1.24	2.54

Sex-linked lethals and translocations show a rough parallelism with the peak on days 5 and 6. In the case of translocations the level drops sharply through day 7 to the lowest level on day 8.

For sex-linked lethals the high level on days 5 and 6 is maintained through day 7 and then drops sharply to day 8.

Deleted X's (as in previous studies) show a continuous and steep rise from day 5 to a peak on day 8.

Dominant lethals maintain a high level over days 6, 7 and 8. Previously it had been thought that the large percentage of unhatched eggs on day 8 might include some which were unfertilized. However, recent studies on eggs collected within $\frac{1}{2}$ - 1 hr. of laying from matings with day 8 irradiated males have shown almost 100% fertilization - indicated by the presence of polar bodies and early mitotic cleavages.

Amongst the eggs examined were some showing micronuclei, chromosome fragments and stickiness, effects which would lead to breakdown of the mitotic cleavages at an early age and so cause death of the egg.

In order to study directly the effect of X-rays upon spermatocytes, cytological examination was made on testes of irradiated late pre-pupae when only spermatocytes and spermatogonia were present. Twenty-four hours after doses of 1000r and 2000r many of the dividing spermatocytes showed chromosome aberrations including sticky anaphases, chromosome breaks and fragments.

Chandley, Ann C. Timing spermatogenesis in Drosophila melanogaster with tritiated thymidine.

In view of the spate of research on mutation in immature male germ cells it was felt that there was a great need for direct timing of spermatogenesis using tritiated thymidine to label the germ cells. We have injected the abdomen of newly emerged ♂♂ with 0.08 c.mm. of tritiated thymidine (activity 25 μ C/ml).

On each successive day from injection, the testes of mated and unmated ♂♂ were fixed in 3 : 1 alcohol-acetic, cut at 8 μ and stained with Feulgen. The exposure time for autoradiographs was 3 weeks. Labelling of young spermatocytes was detectable on the second day but in the later auxocyte stage the degree of Feulgen staining is so slight and the dispersion of the label so great that it was difficult to recognize label in these cells. We have since found that a much clearer picture of this stage can be obtained using eosin as a counterstain. By day 5, however, groups of very young labelled spermatids were visible, all the labelled nuclei being apparently in a single cyst. With succeeding condensation of the nuclei and agglomeration into sperm bundles, the label became increasingly obvious. To date we have not observed testes more than 8 days from injection. By this time, heavily labelled sperm bundles are present in the testis close to the exit into the seminal vesicle. Assuming that the last stage to incorporate tritiated thymidine is the early spermatocyte the complete life span of a spermatocyte would appear to be 4 days. No differences were apparent between mated and unmated ♂♂ in the rate of spermatogenesis. This study is now being repeated using a heavier dose of tritium, eosin as a counterstain, making autoradiographs over a longer period than 8 days, and also looking for labelled sperm in ejaculates.

Divelbiss, J. E. A sterility factor affecting both males and females in Drosophila melanogaster.

An attempt to make a stock of the constitution In(2L)t, Roi·In(2R)Cy, bw^{45a} sp² or^{45a}/Ins(2L+2R)Cy, Cy bw^{45a} sp² or^{45a} (abbreviated Roi and Cy respectively)

failed due to sterility. Outcrosses of Roi/Cy males and females to Oregon-R showed the sterility to be present in both sexes. Since mutant females produced no eggs

and only very few eggs were produced by Oregon-R females mated to mutant males, the observed results are most likely due to sterility rather than zygote lethality. Roi probably arose as the consequence of crossing-over between In(2L)t and In(2L)Cy in In(2L)t, Roi/Ins(2L+2R)Cy, bw^{45a} sp² or^{45a}, hence it would carry the right hand portion of In(2L)Cy. Ives, DIS-25:70, reported the presence of a lethal near each end of In(2L)Cy in Ins(2L+2R)Cy, Cy bw^{45a} sp² or^{45a}. Roi/Cy would be homozygous for the right hand portion of In(2L)Cy and, hence, also for the right hand lethal. However, Roi would carry a duplication for salivary bands 22D2-3; the duplicated piece originating from In(2L)t and carrying 1^+ . This would suggest that the right hand lethal is associated with bands 22D2-3. Roi must also be 1^+ for the left hand lethal. Since the right arms of the two homologues are derived from the same source, they are probably genetically identical. The sterility could be explained by the presence of an undetected mutant in In(2R)Cy which arose previous to the time that Roi was derived, and which would become homozygous in the Roi/Cy heterozygote.

Doane, W. W. Persistence of fs(2)adp in the Kaduna population after four years.

in the Kaduna wild stock, maintained at the Institute of Animal Genetics in Edinburgh, from which it was originally screened in 1956 (Doane, 1960, J. Exp. Zool., 145: 23-42). In the summer of 1960, 18 sample vials of this stock were received from Dr. A. Robertson of that laboratory and all emerging males were mated individually to Cy/fs(2)adp females. Five non-Cy female progeny from each of these matings were tested for fertility and, where sterility occurred, the lines were perpetuated by crossing their Cy brothers to H-40 females (stock #114 in DIS-34, with dominant markers and cross-over suppressors on chromosomes I, II and III). There followed a breeding program for these lines by means of which the individual Kaduna chromosomes were isolated in the H-40 background so that their effects on the adult fat body and on fertility might be tested. Through this procedure, the factor fs(2)adp has been screened from the descendants of at least 13 of the original 18 samples, suggesting that it persists in the Kaduna wild stock at a fairly high frequency. In addition, other factors affecting the ovaries and fat body have been screened out. Certain second chromosome lethals picked up this way are able, in the heterozygous condition, to mask the effect of fs(2)adp on the fat body. A second chromosome factor, apparently allelic with fs(2)adp but which causes hypertrophy of the fat body without accompanying sterility, is very prevalent in the Kaduna wild stock. This latter mutant is especially well-suited for histochemical studies on fatty tissues. (This work was supported by a Postdoctoral Fellowship under the Yale University NIH Training Grant in Genetics.)

Various traits associated with female sterile(2)adipose and relating to reproductive physiology and fat metabolism suggested that the mutant might persist

Dorn, G. L. and A. B. Burdick. Recombination between Df(1)259-4 and various mutants of the miniature-dusky complex in D. melanogaster.

Df(1)259-4 produces a hemizygous effect with the mutants in the miniature but not the dusky cistron. This would seem to indicate that the deficiency extends through the miniature cistron but stops

short of the dusky cistron.

Transheterozygotes of Df(1)259-4 with each of three miniature mutants (m, m⁵⁹, and m^D) and two dusky mutants (dy and dy⁵⁸) were formed. These transheterozygotes have been examined for recombination. All five combinations have been found to yield recombinants.

Below is constructed a genetic map which indicates the relative distances between Df(1)259-4 and each of the five mutants.

Df(1)	0.080	dy ⁵⁸
	0.040	dy
0.019	m ^D	
0.015	m	
0.011	m ⁵⁹	

If it is assumed that Df(1)259-4 lies to the extreme left, then the gene order agrees with that which we have previously determined (see DIS-33, 1959). Vermilion and garnet markers were also employed in these recombinational experiments. At present, it seems that recombination between Df(1)259-4 and any one of the miniature mutants is also associated with a high negative interference for the outside markers. This does not seem to be the case with the dusky mutants.

Ehrman, Lee. Mutant genes in the Transitional subspecies of D. paulistorum.

Young males from the Bucaramanga, Columbia strain of D. paulistorum were exposed to approximately 4000 r-units of X-rays, and then crossed to untreated virgin females

of the same strain. The F₁, F₂, and F₃ progenies were examined for autosomal and sex-linked, dominant and recessive mutations. Although this work is still in progress, and more males will be irradiated, the initial results permit the reporting, for the first time, of mutant genes in this important subspecies:

Delta-Autosomal dominant, lethal when homozygous. This mutation occurred frequently in X-rayed cultures. Wing veins thickened at the margins and the cross-veins. Eyes very rough. This mutation already exists in the Amazonian and the Andean-South Brazilian subspecies of D. paulistorum.

Minute-Autosomal dominant, lethal when homozygous. Bristles reduced in size, especially the scutellar bristles. The developmental period is lengthened, and the viability of both sexes is poor. A Minute exists in the Centro-American subspecies.

Star-Autosomal dominant, lethal when homozygous. This mutation occurred several times in X-rayed cultures. Eyes roughened because of irregularly arranged facets. Star has also been induced in the Amazonian, Andean-South Brazilian, and Centro-American subspecies; thus, it has been acquired in every D. paulistorum subspecies irradiated.

veinless-Sex-linked recessive. This mutation occurred frequently in X-rayed cultures. Many of the wing veins are absent or shortened or interrupted. The wings themselves are warped distally where there are virtually no veins. This mutation was previously induced in the Centro-American subspecies.

The mutations listed above, and others which may be induced and firmly established in stocks, will be employed as genetic markers in the study of reproductive isolating mechanisms (hybrid male sterility and sexual isolation). Because Drosophila paulistorum is now known to represent, at our time level, a number of forms in a state of transition between race and full species, the response of its "bridging" subspecies to a metagenic agent is a necessary prerequisite for further genetic analysis.

(This investigation was supported by a postdoctoral fellowship, GF-9033, from the Division of General Medical Sciences, U.S. Public Health Service.)

Fabergé, A. C., and B. H. Judd. Chromosome breaks by alpha particles.

A fairly high proportion of the chromosome breaks produced by alpha particles in Tradescantia do not rejoin, and by an

indirect method, it can be shown that the same is true of the chromosomes of maize endosperm, Fabergé, 1959. For this reason, a preliminary trial was made on treating Drosophila with alpha particles. The penetration of unaccelerated alpha particles is much too low to permit the use of an external source, and the treatments were made by exposing flies to an atmosphere containing Radon, in our case 6.1 microcurie per ml of air. The Radon is believed to equilibrate with the tissues of the fly very rapidly, and since, moreover, the time to gain Radon activity is about the same as the time to lose activity, quite short exposures are practical. The exact distribution, on a microscopic scale, of the ionization does in the tissues of the fly is difficult to assess, since Radon is, in round figures, about 50 times more soluble in lipids than in water. A discussion of these problems will be found in Gray and Read (1942). About 5% of the ionization is also attributable to β -particles, and a negligible amount to gamma radiation.

A solution of Radium bromide in a closed vessel was allowed to equilibrate with its Radon for several weeks. The Radon was then swept over into a one liter spherical flask which has previously been evacuated by admitting air into it which was bubbled through the Ra Br₂ solution. The spherical flask now contained air at atmospheric pressure, mixed with 6.1 μ c of Radon per ml. This spherical flask had a small finger-shaped reentrant consisting of an outer sleeve and an inner cylinder,

ground-fitted to each other. The inner cylinder could be put into communication with the atmosphere in the flask by rotating it in the outer sleeve, and aligning several large holes bored through the walls of both cylinder and sleeve. Flies were placed in a lusteroid plastic centrifuge tube, 12.5 mm in diameter, 40 mm long, in which numerous holes had been punched; the stopper of the tube had a cavity filled with mashed banana. To expose the flies this plastic cage was placed inside the re-entrant cylinder, which was then closed to the outside atmosphere, and rotated in its sleeve to align the holes. The Radon atmosphere then diffused among the flies. Since the volume of the re-entrant cylinder was about 10 ml, each exposure resulted in a dilution of the remaining Radon by about 1%. Five exposures of 3.3, 10, 30, 90 and 270 minutes were made, at 25°C, treating about 200 wild type Oregon R males at each exposure. After exposure, the flies were left in fresh food vials in a chemical hood for 30 hours. After this time very little radioactivity remained, and they could be safely handled without special precautions. The males were mated between 48 and 50 hours after exposure to y v f XX ♀♀, using 10 ♂♂ and 10 ♀♀ per bottle. Four transfers were made to fresh bottles every 2 days.

The first dose of 3.3 minutes was discarded as having had too little exposure. From the other four doses, a total of 40 duplications were found, among 22922 ♀♀ examined. The relative frequencies of different classes of duplications are about the same as have been observed from X-ray treatment, in so far as can be judged from so small a sample.

X Duplications covering the markers	Radon treatment (all doses) (22922 ♀♀)	X-ray Treatment 1000 r, + 2000 r, + 4000 r (58081 ♀♀) (Bishop 1941)
y	31	298
v	2	0
f	2	1
y v	3	28
y f	2	48
v f	0	4
y v f	0	4

Thus there is no suggestion from this limited information that alpha particles produce duplications of a different nature in *Drosophila*.

A very small estimate of dominant lethals was also made on a sample of the treated males. The dosage effect curve for dominant lethals from X-rays is of a complex character, and our very small counts do not permit the establishment of the equivalent function for alpha particles. It is thus difficult to make quantitative comparisons of alpha particle and X-ray doses. However, from the dominant lethal counts, as well as the deficiencies, we would hazard the estimate that 1 microcurie per ml of atmosphere for 1 minute is roughly equivalent to 2 r of X-rays, in the region of 1000 r. This estimate may easily be wrong by a factor of 2. Despite the fact that F₁ males were not carefully examined for mutations, several X-linked visibles and autosomal dominants were found. A large fraction were Minutes or Rough-eye mutants. Only 11 of more than 45 mutants were saved for mating. Among the X-linked visibles were y, spl, ec, pn-like, lz-like, sn. Among the autosomal dominants were a furrowed-thorax, a Delta, and several good Minutes.

The method for exposing flies and the apparatus was devised by Dr. D. G. Ott, who, with Dr. W. H. Langham, made preliminary estimates of a reasonable dosage range. We are very much indebted to them for their assistance in carrying out the treatment at the Biomedical laboratory, Los Alamos Scientific Laboratory.

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Thesis, M. A., University of Texas.

Gray, L. H. and Read, J., 1942 The lethal effect of alpha radiations. IV. The effect of ionizing radiations on the Broad Bean Root. Brit. J. Radiol. 15L 320-336.

Fabergé, A. C., 1959 Production by alpha particles of functionally stable broken chromosome ends in maize. Genetics 44: 279-285.

Fox, Allen S., and Eileen A. Sweeney*.
Chemical structure and time of appearance of the sex peptide of males in Drosophila melanogaster.

The presence of a peptide in adult males, but not in females, has been reported previously (Fox, 1956, *Physiol. Zool.* 24:288; Fox et al., 1959, *Science* 129:1489). This object, called the "sex peptide",

has now been purified by elution from paper chromatograms and subjected to partial chemical characterization.

Acid hydrolysis of the sex peptide yields ten ninhydrin-positive products. These have been identified by paper chromatography of the free products themselves and of their dinitrophenol derivatives, prepared by reaction with 1-fluoro-2,4-dinitrobenzene according to the method of Levy (1954). Eight are conventional amino acids: aspartic acid, glutamic acid, serine, glycine, α -alanine, leucine, valine, and methionine. The ninth is ethanolamine. The tenth remains unidentified.

Since ethanolamine lacks a carboxyl group, it must occupy what would otherwise be the C-terminal position of the peptide chain, or constitute a side branch through peptide linkage with one of the dicarboxylic amino acids or phosphate linkage with the serine. Initial attempts to identify the N-terminal residue by preparation of the dinitrophenol derivative of the intact peptide according to the method of Sanger and Thompson (1953), suggest that this position in the peptide is occupied by the unidentified residue. Molar ratios have not yet been determined.

The sex peptide is not present in detectable amounts in male third instar larvae, nor in pupae, nor in males during the first two hours after emergence. In male third instar larvae and pupae, but not in females, there is present instead a substance tentatively identified as phosphoethanolamine. This disappears in newly emerged males just prior to the appearance of the sex peptide. (Supported by grant C-2440, National Institutes of Health, U. S. Public Health Service, and by a grant from the Rackham Foundation. *National Science Foundation Undergraduate Research Participant in the Department of Agricultural Chemistry and the Honors College of Michigan State University.)

Frost, J. N. Double fertilization mosaics.

In experiments involving triploid females and in which approximately 109,000 offspring were examined eight double fertilization mosaics occurred. In all these experiments the parental origins of the sex chromosomes could be determined. Four of the mosaics were diploid female-intersexes, one was a diploid male-intersex, one was a diploid male-triploid female, one was an intersex-triploid female, and the last was intersexual in both portions of the mosaic. In four of the mosaics the two original egg nuclei had carried complementary chromosome sets, in another the two egg nuclei had carried identical chromosome sets. In three of the mosaics the chromosome sets of the two egg nuclei had been neither complementary nor identical, a fact suggesting that at least some, and perhaps most of the double nuclei in the eggs had arisen from independent meiotic divisions of the two nuclei in a binucleate oonium.

The distribution and proportions of the mosaic parts were quite variable, only two mosaics being bilateral. Each part of the sex mosaics showed complete autonomy in development.

Frost, J. N. Two mosaics of unusual origin.

A diploid female-intersex mosaic occurred in the following cross: y w 3N (free X) females by y:CY/Gla;D/Sb males. The diploid female portion was yellow, Curly, Glazed, Dichaete, and Stubble and thus (with the possible exception of one X chromosome) obtained all its chromosomes from the male. The intersex portion was yellow, Glazed, and Stubble and arose from a normal zygotic nucleus while the diploid female portion apparently originated from the independent development of a diploid sperm nucleus. The latter could have been produced by a tetraploid spermatogonium.

Another unusual mosaic occurred in a cross of y w (attached-X) 3N females by y, sc⁸.Y;L, sp/L, sp;Sb, e/e^s males. The entire fly was a diploid female and both the left and right sides were yellow, Lobe, Stubble, and ebony, indicating that both of the third chromosomes on each side had come from the male. In addition the left side of the fly was speck and the left eye was completely absent indicating that the left side had received both of its second chromosomes as well as both of its third chromosomes from the male. A satisfactory explanation for this mosaic has not yet been devised.

Frye, Sara H. Evidence that achaete may not be to the right of yellow.

Out of 547 transmissible X-ray-induced "yellow" mutants in scute-8 chromosomes the frequencies of different combinations of affected loci in order of decreasing frequency were as follows: (using the same symbolism as before, Frye, 1960, DIS 34) - - - + (398), - - - - (76), + - - + (45), + - + + (23), + - - - (5). No "yellows" were recovered of the other 3 possible classes (+ - + -, - - + + or - - + -). This obviously means that ac is closer to y than lJl and that ac is closer to lJl than to bb. None of the foregoing tabulations yet allows for the decision as to whether ac is to the left or to the right of y. However, other evidence suggests that ac may not be to the right of y since genetic analysis of 10 cases of dark yellows, included in the above tabulation, showed that 4 were not deficient or affected at the loci of lJl, ac or bb (+ \pm + + where \pm represents the dark yellow and the order is assumed to be lJl y ac bb), 2 were deficient or affected at the locus of ac, but not lJl or bb (+ \pm - +), and 4 were deficient or affected at the loci of lJl, ac, but not bb (- \pm - +). This can be seen by comparing the two possible orders (ac to the right and ac to the left of y) of the 4 loci concerned with the 3 recoverable classes of dark yellows.

lJl	y	ac	bb		lJl	ac	y	bb
+	\pm	+	+	(4)	+	+	\pm	+
+	\pm	-	+	(2)	+	-	\pm	+
-	\pm	-	+	(4)	-	-	\pm	+

The latter order (ac to the left of y) is the only one that allows for one of the breaks (in this case, the right one) to fall in a common region. One must also allow for the difference in the size of the regions -- on the basis of breakage the third region is about 6X the first region (Frye, unpublished) regardless of the order of the loci. Another advantage of the suggested order (that of the right hand column) is that it allows for a plus allele of bobbed to border the y locus since those cases of dark yellows that have a normal allele bordering on either the left or the right side of the yellow locus can be explained by position effect, sub-gene deficiency of "point" mutation, whereas those cases where the dark yellows occur between 2 affected loci (as in the last row on the former order) are less easily explained (except as skipping effects on intermediate loci in cases of minute inversions).

Traditional evidence that places ac to the right of y is based chiefly on the mutant sc3, found by Dubinin to affect both achaete and scute strongly, whereas here are 4 cases in which lJl and ac were strongly affected but y (which on Dubinin's view would be between them) was only slightly affected.

(Work supported by a grant to H. J. Muller and associates from the U. S. Atomic Energy Commission (Contract AT (11-1)-195).)

Frye, Sara H. Frequency of "transmissible mutation" at the w and f locus in the scute-8 chromosome in relation to X-ray dose in *Drosophila*.

Males of composition sc⁸ B (0-24 hrs. old) were given 500r, 1000r, 3000r or 4000r and these and simultaneous controls were mated to y w In49 f virgin-females (48-72 hrs. old). The offspring recovered

were from sperm ejaculated from 1 to 4 days after irradiation. The number of female and male parents introduced per bottle, representing different X-ray doses, were respectively: 500r - 6 prs., 1000r - 8 prs., 3000r - 14 prs., 4000r - 20 prs. Controls were run with 2, 3, 4, 5, or 6 prs. in order to test for crowding. Usually 4 prs. were used. The frequencies of exceptional phenotypes that were transmissible after progeny testing among the F₁ Bar females were for the control, 500r, 1000r, 3000r, and 4000r treatments, respectively: "white" 0.0002% (1/412,439), 0.0050% (14/277,667), 0.0060% (11/180,942), 0.0173% (8/46,067), 0.0217% (9/41,310); "forked" 0.0% (0/263,694), 0.0028% (8/277,667), 0.0033% (6/180,942), 0.0195% (9/46,067), 0.0121% (5/41,310).

Since the number of transmissible mutants per each X-ray dose is small and a genetic analysis was not conducted to determine their qualitative composition no conclusions are drawn (even though most of the w and f mutants were male-viable it does not follow that these are gene mutations since presumptive point mutations,

inversions, or extremely small deletions could survive in the male) concerning their relation to X-ray dose but only their frequencies are reported.

(Work supported by a grant to H. J. Muller and associates from the U. S. Atomic Energy Commission (Contract AT (11-1)-195).)

Frye, Sara H. Persistence of qualitatively diverse "yellow" mutants in scute-8 chromosomes in the absence of selection for one year.

Many "yellow" mutants were induced by exposing young ♂'s carrying the scute-8 chromosomes to various X-ray doses, mating them to y w In49 f virgin-females, and recovering the yellow mutants among the

F₁ Bar ♀♀'s. After an extensive genetic analysis (Frye, 1958) to determine their qualitative composition each yellow mutant was kept for several years in an unbalanced stock (y⁻ sc⁸ B/y w In49 f♀ x y w In49 f♂) which meant that frequent selection was necessary in order to maintain the y⁻ sc⁸ B chromosome and to prevent the stock from becoming homozygous for y s In49 f♀♀'s. However, the last year before these yellow mutant stocks (each stock was kept in a group of 4 vials) were discarded no selection was performed. At the time of discarding (total no. of yellow mutant stocks remaining was roughly 150) I randomly sampled 52 of these stocks to see what qualitative types had persisted and which had not in spite of the absence of selection. All one had to do was to etherize and check for y B ♀♀'s (where they were present no counts were taken to compare the no. of y B ♀♀'s with the no. of y w In49 f ♀♀'s). Out of 30 stocks, selected randomly, the following qualitative types had persisted (using the same symbolism as before, Frye, 1960, DIS 34) - - - + (9), + - - + (7), + - + + (5), - - - - (4), - - - + 1 (3), - - - - 1 (2). Out of 22 stocks the following had not persisted - - - + (3), + - - + (3), - - - - (6), - - - + 1 (2), - - - - 1 (5), + - - + 1 (2), IV* - - - - (1).

It can be seen that several of the same qualitative types are common to those that did and those that did not persist, and that there is no simple correlation between sheer number of loci absent or affected and the ability to persist, therefore persistence must be a very complex phenomenon. (This is not to be taken as meaning that persistence is independent of qualitative composition of these mutants). It would be of interest to see if one could correlate the no. of generations that is required for different genetically analyzed mutants to become eliminated in a population with their qualitative structure.

Frye, Sara H. Spontaneous "yellows" as gross rearrangements in *Drosophila*.

Frye (1958) reported 4 yellows, recovered in the female, that arose spontaneously and singly in separate control series in

crosses of scute-8 B males to y w In49 f virgin-females, to be attached-X's with one break having occurred in the paternal-X and the other in the maternal-X. Their resemblance to Sidky-like rearrangements (X-ray induced break in one chromosome, spontaneous break in another non-homologous chromosome) is only superficial since in my cases both breaks are spontaneous. Genetic analysis showed that all 4 were deficient or affected at the loci of lJ1, y, and ac in the paternal scute-8 chromosome.

In order to see if these 4 yellows acted as if they were structurally the same (as implied by the genetic analysis) 5 virgin-females of each of the 4 yellow stocks were crossed with 5 y w In49 f males in half-pint bottles and a count of the sex ratio was made to see if the proportion of F₁ males to females varied among the 4 yellow stocks; the idea being that if the break in the paternal-X had resulted in more of the X-chromosome being lost or affected in some cases than in others this differential viability would shift the sex ratio in favor of the males. The 4 yellow stocks were designated y⁰⁰⁰¹, y⁰⁰⁰², y⁰⁰⁰³ and y⁰⁰⁰⁴, and the results (giving the count of the males first) were for each of the above, respectively: 1652 - 730 appx. 2:1; 838 - 684 appx. 1:1; 993 - 727 appx. 1:1; 1146 - 399 appx. 3:1. Thus y⁰⁰⁰¹ and y⁰⁰⁰⁴ are of lower viability than y⁰⁰⁰² and y⁰⁰⁰³ and perhaps involve a greater loss of distal X-chromosome material (especially the bb* locus which could not be tested for directly since the attached-X "yellows" carried a Y-chromosome).

The most unexpected fact is that no yellows with attached-X's occurred in the treated series even though all 607 X-ray induced yellows (in scute-8 chromosomes) were progeny tested and analyzed in exactly the same way as the yellows of spontaneous origin. The total number of F₁ females in the treated series here was 583,248 and that in the above-mentioned controls was 412,439.

Also, there is no reason to conclude that "yellows" occurring spontaneously in the scute-8 chromosome are exclusively or even highly likely to always be exchanges with Y^S , but may be due to breaks the results of which may be classifiable as minute chromosome changes, crossovers, or gross rearrangements. (None of the above 4 involved exchanges with Y^S , and out of a total of 10 "yellows" arising spontaneously in the scute-8 chromosome of males two were found to involve an exchange with the Y^S . Tests for the exchange with Y^L were not made.)

Other spontaneous gross rearrangements involving the tip of the X-chromosome are known (Burkart, 1930 - Blond, Muller, 1943 - "double-X") and the tip of the autosomes (Bridges, 1919 - Pale). Chromosome tips may be more likely to enter into gross rearrangements spontaneously (and possibly when X-rayed) than is ordinarily thought.

Fuscaldo, Kathryn E., and Allen S. Fox. Immunogenetic studies of white-variegated position effects.

Agar-diffusion techniques were employed to investigate the antigenic specificities of proteins extracted from the following stocks of *D. melanogaster*: $In(1)w^{m4}$; a derivative, $In(1)w^{m4w}$, in which the rearrangement is unaltered but a change has occurred in the white pseudoallelic segment (Schultz, 1943, D.I.S. 17:64); the translocation, $T(1;4)w^{m5}$; the mutants w , w^e , and w^{a2} ; the double mutant, $bw\ cn$; and the isogenic wild stock, Oregon-R-I.

In all cases an alteration of the relationship of heterochromatin to the white pseudoallelic segment resulted in a change in the immunochemical properties of an antigen, designated $H(w)-1$. The protein $H(w)-1$ exhibits a higher antibody combining power in the inversion and translocation stocks than in the wild stock. The difference most probably is associated with a difference in the number of combining sites on the antigen molecule, along with a small difference in the configuration of the antigenic site. The effect is reminiscent of the effect of the Y chromosome on the antigen Y-1 (Fox, 1959, J. Nat. Cancer Inst. 23:1297).

The properties of $H(w)-1$ extracted from the mutants w , w^e , and $bw\ cn$ are the same as that extracted from the wild stock. $H(w)-1$ extracted from w^{a2} , on the other hand, behaves like that extracted from the inversion and translocation stocks. The mutants w and w^e occupy a locus to the right of that occupied by w^{a2} (Lewis, Green). It thus appears that some, but not all, alterations of the white pseudoallelic segment affect the structure of $H(w)-1$. Furthermore, the effect is not directly associated with eye pigmentation (vide $bw\ cn$).

The results may be rationalized by the hypothesis that the euchromatic white pseudoallelic segment determines the primary structure (amino acid sequence) of the protein $H(w)-1$, but that the tertiary structure of the protein depends on the relationship of this euchromatic segment to heterochromatin. The participation of heterochromatin in the determination of tertiary structure has been postulated previously in connection with the effects of the Y chromosome on the protein Y-1, and the respective roles of euchromatin and heterochromatin in protein synthesis have also been discussed (Fox, 1959, Science 130:1417).

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Ghini, Clara. Effect of nebularine and EOC (8-etoxy caffeine) on selection response for sternopleural hairs in *D. melanogaster*.

The effects of two mutagens: EOC(8-etoxy caffeine) and nebularine on the induction of genetic variability as shown by progress under selection, has been studied. An isogenic stock has been used; the character selected for was high number of sternopleural hairs. A family selection method was applied every two generations. The mutagens were given by intra-abdominal injections to adult flies of both sexes, aged 18-24 hours. Three concentrations (.050%; .025%; .010%) were tested for nebularine and two for EOC (.30%; .10%)

With nebularine a positive response to selection was obtained from the first generation on, but the response continued only in the .025% lines. From average number of hairs of $20.31 \pm .099$, after 8 selection cycles corresponding to 16 generations, one obtained values of $26.01 \pm .379$ and $29.52 \pm .302$ in two different replications. In the same period the two untreated lines from an initial average value of $20.74 \pm .098$ reached a level of $21.29 \pm .128$ and $20.96 \pm .106$. The response to selection was found associated with an increase in variability, expressed both in terms of standard deviations and of coefficients of variation. With EOC the average

values of sternopleural hairs reached, after 8 selection cycles, were 21.22±.115 and 21.19±.109, not significantly different from the values reached by untreated control lines. From experiments with plants one knows the mechanism of action of the two mutagens as being very different, EOC causing mainly chromosome breakage while nebularine (Ehrenberg and Gustafson, 1954) is supposed to produce mainly point mutations. Experiments designed to compare the mutagenic action of these substances in D. melanogaster are in progress.

Grell, R. F. The penetrance of sparkling-Cataract.

A research note in DIS 33: 150 (1959) by H. J. Muller states that a single fourth chromosome carrying sparkling-Cataract

(spa^{Cat}) in combination with two normal fours produces a wild-type eye. Flies have been synthesized in this laboratory that carry three free fourth chromosomes, each marked with a single dominant mutant (spa^{Cat}/ci^D/ey^D), in an otherwise diploid background. The penetrance of spa^{Cat} in these triplo-four flies is complete, although its expression is less extreme than is one dose of spa^{Cat} in the diplo-four condition.

The mutant, spa^{Cat}, has also been used to mark the free fourth chromosome of triplo-four females that are homozygous for T(3;4)86D in order to follow the assortment of the free four and an extra Y chromosome. In this situation one dose of spa^{Cat} was found to be fully penetrant and classifiable both in the mother and in her triplo-four progeny. Mothers of this genotype (T(3;4)86D/T(3;4)86D/spa^{Cat}) mated to diplo-four males carrying two normal fours produced 332 spa^{Cat} and 329 non-spa^{Cat} offspring [Genetics 44: 421 (1959)]. Mothers of the same genotype mated to M-4/ey^D males produced non-eyeless-Dominant flies that were either M-4 or spa^{Cat}, clearly demonstrating that the mutant phenotype is always classifiable when present in one dose in triplo-four flies.

It is of interest that one dose of spa^{Cat} in the F₁ diplo-four hybrid between D. melanogaster and D. simulans (synthesized by E. H. Grell) shows an extreme mutant phenotype, whereas spa^{Cat}/simulans-four in an otherwise completely melanogaster background, as observed by Muller, is wild type.

(This work was done at the Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee, operated by Union Carbide Corporation for the United States Atomic Energy Commission.)

Hildreth, P. E. and J. C. Lucchesi. Fertilization in D. melanogaster and D. virilis.

Early findings by Huettnner (J. Morph. 39:249, 1924) concerning the occurrence of polyspermy in D. melanogaster have been supported by Counce (DIS 33:127, 1959) and

extended to eight additional species, including D. virilis; this author found an average of 5 to 6 spermatozoa in D. melanogaster and 50 to 100 spermatozoa in D. virilis eggs.

Using the Feulgen whole-mount procedure of von Borstel and Lindsley (Stain Technol. 34:23, 1959), preliminary work on D. melanogaster eggs failed to show polyspermy (Hildreth, unpublished); therefore further cytological examination of D. melanogaster and also D. virilis eggs was conducted. In D. melanogaster 96 eggs were found in meiotic stages; among these 91 had a single sperm, 2 had two sperms, and 3 had no visible sperm. This is consistent with the observations of Hinton and Lucchesi (Genetics 45:87, 1960). Among 127 meiotic eggs of D. virilis, 87 eggs had a single sperm, no sperm was visible in 40 eggs, and no case of polyspermy was observed.

The reason for the differences between our results and those of Huettnner and Counce are not known. Autoradiographic studies of fertilization in D. virilis are now being conducted in an attempt to obtain further information on the question of polyspermy.

(This work was carried out under the auspices of the U.S. Atomic Energy Commission.)

Hochman, B. On the viability of the brown-Variegated¹/brown-Variegated^{57e} heterozygote.

More than 20 brown-Variegated (bw^V) alleles are listed in Bridges and Brehme (1944). Their viabilities in the homozygous state are described variously as, generally

lethal, nearly always lethal, lethal in 95% of the cases, etc. The first one found,

bw^{V1} (usually referred to as Plum (Pm)), is reported to be, "generally lethal when homozygous, and also lethal with all other brown-Variegateds."

During the course of an experiment involving the Notch locus, data were obtained on the viability of the Pm/bw^{V57e} genotype which demonstrate that the statement in the preceding sentence does not apply to this combination of bw^V alleles. (Dr. E. H. Grell produced bw^{B57e} by irradiating the SMI chromosome. The presence of Cy in SMI (see DIS 27: 57-58) precludes an examination of the bw^{V57e} homozygote. When heterozygous with wild type, bw^{V57e} causes the same eye mottling as $Pm/+$. Under certain genetical and environmental conditions, it was observed that 0.2-0.4 of expected Pm/bw^{V57e} individuals reached the adult stage. The findings also indicate that a slight temperature rise markedly increases the number of surviving Pm/bw^{V57e} flies.

From the cross, $w^a fa^{no} spl rb/fa^{no} spl; SMI, Cy/Pm \times y w^a N^{40}/Y; SMI, Cy, Dp(1;2)w^{51b7} bw^{V57e}/+$, three classes of male offspring are expected if one disregards the X chromosomal genes. These three phenotypic categories may be expressed simply as: $Cy/+$, $Pm/+$, and Pm/bw^{V57e} . If one assumes equal viabilities, each class should comprise one-third of the total male progeny. The expected numbers and observed results are presented in the following table:

Temperature	Combined $Cy/+$ and $Pm/+$		Pm/bw^{V57e}		
	obs.	exp.	obs.	exp.	obs./exp.
26 + 1°C	5,219	4,028	823	2,014	0.41
23.5 + 0.5°C	10,888	7,788	794	3,894	0.20

The Pm/bw^{V57e} genotype, while considerably below the other two classes in viability, survives too frequently to warrant the lethal designation. A more appropriate description would be semi-lethal. It is possible that the higher than expected rate of survival of this particular heterozygote is due in part to a reduction in the number of its competitors by factors associated with the cross. All female zygotes (except rare cases of nondisjunction) are N^{40}/fa^{no} , a lethal combination permitting less than 0.01 imagoes. The $Dp(1;2)w^{51b7}$, which had been placed in the SMI, Cy, bw^{V57e} chromosome by Dr. W. J. Welshons, also carries N^+ . Since the presence of N^+ cancels the N^{40}/fa^{no} lethal interaction, there emerges a single group of female offspring namely, Pm/bw^{V57e} . Male progeny of this same genotype must compete only with other males and genetically similar females. The effect that additional classes and larger numbers of females will have on the relative viability of Pm/bw^{V57e} males is currently under investigation.

The following unpublished observations by Dr. W. J. Welshons on the phenotype of Pm/bw^{V57e} have been confirmed:

- (1) Bodily dimensions range from clearly smaller than normal to approximately normal in size. The larger individuals are often characterized by a chubby (or bloated) appearance.
- (2) Wings either fail to expand completely or, if unfolded, they tend to diverge and curl to an extent greater than that of Curly alone.
- (3) Occasional patches of unpigmented microchaetae are another feature of the syndrome.

A limited number of tests show that these flies can be fertile.

The emergence of twice as many Pm/bw^{V57e} adults at the higher of the two temperatures employed provides one more example of the strong influence of environmental factors on the viability of a given genotype. It is interesting to note that Gowen and Gay (1933) found that the extent of variegation is diminished by increased temperature. From the results reported here it appears that the degree of semi-lethality of this particular brown-Variegated heterozygote manifests a like tendency.

(This work was done at the Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee, operated by Union Carbide Corporation for the United States Atomic Energy Commission.)

Hunter, Alice S. and Sara
Newball. *Drosophila* of old
Providence Island.

Old Providence Island is in the Caribbean
Sea at a latitude of 13°19' to 13°24' North
and a longitude of 81°21' to 81°23' West.
It is about 150 miles from the coast of
Nicaragua and 350 miles from Cartagena on the coast of Colombia. The temperature

varies from 18-30°C. depending on the sun and wind, but averages 24°C. The terrain varies from the swamps and bays to wooded hills and mountains up to 1,152 feet above sea level. The wind is generally northeast from December to February, and the weather is calm from then until September. During October and November there may be hurricanes with strong winds mostly from the North.

The island is about 15 square miles, and there are about 3,000 human inhabitants. There are many fruits which might be natural breeding sites of *Drosophila*, such as the semi-cultivated mangos, papayas, coconuts, bananas, and the wild grapes, pine-apples and guavas. However, despite the abundance of fruits, a total of only six different species of *Drosophila* were encountered in our collections made during the months of January and February.

Collections were made by sweeping over a bait composed of cut oranges, bananas and squash pulp. Whenever possible bait was placed in shaded protected areas, but in some places there were no trees, and it was impossible to avoid the wind. In fifteen sites the various species encountered in at least one of the sweepings were sorted and counted. The totals of the six different species found in the counted collections are as follows: *D. melanogaster-simulans*- 1,140, *D. ananassae*- 1,017, *D. hydei* (?) - 922, *D. latifasciaeformis*- 493, and *D. willistoni*- 236. No attempt was made to determine the relative proportions of *melanogaster* and *simulans*. The identity of *hydei* is being checked in test-crosses with a University of Texas stock. Because of the lack of electricity on the island, all of the *willistoni* group flies collected were taken to Bogotá for identification. A sample of 50 males was checked by studying the genitalia. In addition 50 females were isolated in separate culture jars and the offspring were checked. In all the 100 cases the male genitalia were those of *D. willistoni*.

Comparing one collection site with another, considerable variation in the relative frequencies of the different species was encountered. For example:

	Free Town	Bush-pen	Camp
<i>D. melanogaster-simulans</i>	38	38	131
<i>D. ananassae</i>	349	20	68
<i>D. hydei</i>	54	1	250
<i>D. latifasciaeformis</i>	1	1	163
<i>D. willistoni</i>	0	32	0

The lack of variety of species is notable, but may be related to the isolation of this island and the winds.

(Biology Department, University of the Andes, Bogotá, Colombia)

Imaizumi, T. XXY strain derived from the wild Miyazu stock of *D. melanogaster* and its lethality.

Of the lethal strains in the preceding report (DIS 33 : 140), it is ascertained genetically that a strain derived from an X-rayed male is XXY. Perhaps primary non-

disjunction occurred in a sperm of the X-rayed Miyazu male. The crossing tests are as follows:

Crosses	♀		♂		Totals
(1) Y/+/Basc ♀ x Basc ♂	B/+	B	B	+	
	235	172	318	0	725
(2) Y/+/Basc ♀ x w m ♂	red eye	orange eye	B	w m	+
	212	156	134	191	0
					850
(3) Y/+/w m ♀ x w m ♂	+	w m	w m	w m	+
	572	244	15	9	0
			523	0	8
(4) Y/+/y w m f ♀ x y w m f ♂	+	y w m f	y w m f	m f	+
	449	231	1	32	0
			413	9	0
	m	(rb)*			
	7	8			

*A new character found in cross (4); it represents reduced bristles, not forked, phenotypically and its chromosomal constitution is XXY.

It is noticed firstly from the crosses (3) and (4) that the crossing-over between two X-chromosomes is very suppressed in this XXY strain. Next, at meiosis, three kinds of segregation are expected in the XXY females: (a), (Miyazu-X, introduced-X) and (Y); (b), (Miyazu-X, Y) and (introduced-X); and (c), (introduced-X, Y) and (Miyazu-X). But we can prove that the last segregation (c) never occur in this strain by the tests in F₁ flies of the cross (2). In F₁ of this cross the chromosomal constitution of the females with hetero Bar (orange eye) phenotype is all XX and that of the females with wild phenotype all XXY. Further, it is clear that the wild male never appears in all cases. The XY zygotes (wild genotype) are not formed from the segregation (c) and the XYY males (wild genotype) derived from the segregation (b) would be lethal in this strain, though one knows that the XYY males are viable in general.

The percent mortality in F₁ of several crosses is shown in the following table. The lethality of the XXY strain is given in the top cross (1) and that of the attached-X strain in the next cross (2); and the other three are controls. Percentages per the first total eggs tested are indicated in each stage.

Crosses	Total eggs tested	Mortality in egg				Mortality in larva	Mortality in pupa	Total mortality rate	
		Total	Items of stages*						
			U	C-B	Bk	T			
(1) Y ⁺ /Basc ♀ X (XXY) Oregon-RS	757	66.4	1.3	19.5	29.2	16.4	15.0	1.7	83.1
(2) g ² ty/y (Attached-X) ♀ X Oregon-RS	745	37.9	2.8	27.1	4.6	3.4	18.4	12.9	69.2
(3) Basc/Basc ♀ X (XX) Oregon-RS	727	10.3	4.7	0.4	1.0	4.3	15.5	0.3	26.1
(4) Oregon-RS ♀ X Oregon-RS	542	1.3	0.9	0	0.4	0	19.9	0.4	21.6
(5) Miyazu ♀ X Miyazu	673	3.3	2.5	0	0.2	0.6	11.3	0.9	15.5

*U; unfertilized eggs; C-B, Bk and T indicate primary, middle and late stage in the embryonic development respectively.

Regarding triple-X females, it is revealed that they mostly die in the pupal stage, but some in the late larvae as indicated in the progeny of attached-X strain. But in this XXY strain the mortality rate in the pupal stage is evidently low as compared to the case of the attached-X. On the contrary the mortality in middle or late embryonic stages is higher. This fact suggests that the time of death of the triple-X females in this XXY strain is not in pupal stage, but it would be in embryonic stage, perhaps in the middle of the stage. Finally, the YY individuals die in the primary stage of embryonic development both in XXY and attached-X strains.

Ives, P. T. More data from ras²/ras⁴ and y/y² recombination tests.

In 1951 I reported a count of 66,907 flies from ras²/ras⁴ which showed no ras⁺. In 1961 I have added 69,385 flies to that total with the same result. No evidence

of pseudo-allelism or conversion has appeared in these 136,292 flies. Both series of tests were done with outside markers, with free X-chromosomes and without autosomal inversions. During the summer of 1961 Dr. Hexter and I plan to test ras/ras⁴ with outside markers in free X's and with Cy and Ubx¹³⁰ present. The 69,385 flies recorded here also tested y/y². No y⁺ flies appeared.

Jacobs, M. E. Influence of ebony and + alleles on oxygen consumption and egg production in D. melanogaster.

increase significantly the rate of pupal oxygen consumption and adult egg production.

Studies of flies homozygous and heterozygous for the ebony gene found in a wild population, with randomization of other gene loci, showed heterozygosity to

Kato, M. Influences of essential fatty acids on the growth and egg productivity of D. melanogaster.

yeast with absolute alcohol and ether. In this medium the emergence rate of the adult fly was $8.0 \pm 4.0\%$ on an average while in the standard medium it was $86.33 \pm 1.57\%$. When whole EFA were added to the medium it increased to $44.16 \pm 0.93\%$. When only EFA with double bonds at 6,7- and 9,10- position were added, the rate approached the level obtained with whole EFA, namely, $36 \pm 0.55\%$. In the EFA-deficient medium, most of the larvae died before pupation and most of those that pupated did not survive to adulthood. Neither abnormality in morphology nor that in sex-ratio were found. Daily ovulation of females emerged in the EFA-deficient medium is very low, being $9.34 \pm 1.13\%$ as compared to that of 28.55 ± 2.09 produced by the female reared in the EFA added medium.

Influences were tested by addition of the so-called essential fatty acids or vitamin F to the culture medium which was prepared by extracting lipids from the homogenized

Khishin, Aziz F. Induction of mutations in D. melanogaster by "immersion" in solutions.

gave definite positive results (Khishin, published and unpublished). However, it should be questioned whether in these cases the formalin itself or even a derived product is the sole mutagenic agent, or whether the mere immersing has an effect and thus should be held responsible for all or some of the observed changes.

The experiments designed to test the possibility of inducing lethal mutations in Drosophila melanogaster by "immersing" larvae or pupae in formaldehyde solutions

To test the second possibility a set of experiments were started in which tap water, saline solution prepared with distilled water, and formaldehyde solutions were used as media for immersion. The same procedure was followed for the three agents used, and larvae were treated for 30 minutes and 60 minutes. In all cases dominant lethality (D.L.) was calculated over certain period. Preliminary results indicate the following:

1. Tap water induces more D.L. than either saline (0.05% Na Cl) or 10% formaldehyde solutions.
2. Saline solution induces about the same percentage of D.L. as 10% formaldehyde solution when either is used for 30 minutes.
3. Saline induces more D.L. than formaldehyde when either is tested for 60 minutes.
4. 10% Formaldehyde used for 30 minutes gives about 2-3 times as much D.L. as when applied for 60 minutes.
5. Saline solution used for 30 or 60 minutes gives about the same result.

Mead, Charles G.*, and Allen S. Fox. The characterization of the deoxyribonucleic acids of Drosophila melanogaster.

per gram dry weight of flies. The isolated product had an E(P) of 6930, exhibited a 20% increase in O.D. at 260 m μ upon alkaline denaturation, and was unusually low in viscosity. Upon precipitation of the isolated product with cold ethanol, two types of DNA were observed. One of these was fibrous, typical of most DNA's, and the other was of a flocculent nature. After exhaustive deproteinization the two types of DNA retained their differences.

DNA was isolated from lyophilized Oregon R flies by a modification of the Kay, Simmons and Dounce procedure (1952).

Approximately 2 mg. of DNA were recovered. Perchloric acid and formic acid hydrolysates of the isolated DNA, when subjected to paper chromatography, exhibited an exceptional UV-absorbing spot. This unusual compound was identified as 5-methylcytosine by chromatographic and spectrophotometric means. The two types of ethanol precipitable DNA's, one fibrous and the other flocculent, were analyzed for their 5-methylcytosine contents. A preparation of DNA was precipitated with cold ethanol and the fibrous DNA collected by winding the fibers on a glass rod. The fraction which could not be collected in this manner was considered flocculent DNA. Each fraction was hydrolyzed with perchloric acid, the hydrolysates chromatographed on paper, the UV-absorbing spots eluted, and the molar concentration

of 5-methylcytosine and thymine measured spectrophotometrically. The molar ratio (5MC/T) of the fibrous fraction was 0.165 ± 0.008 ($n=4$), and that of the flocculent fraction was 0.234 ± 0.007 ($n=6$). Thus, the pyrimidine composition of these two fractions is definitely different.

Ion exchange chromatography of a phosphodiesterase digest of whole DNA yielded five UV-absorbing peaks. The molar concentrations of these peaks, as calculated from their respective molecular extinction coefficients at $260 \text{ m}\mu$, are as follows:

Peak No.	Nucleotide	μM
1	Deoxy-5-methylcytidilic	0.79
2	Deoxycytidilic	1.63
3	Deoxythymidilic	2.78
4	Deoxyadenylic	2.76
5	Deoxyguanylic	2.47

The existence of 5-methylcytosine in the DNA of *Drosophila* makes possible a variety of experiments which might lead toward the elucidation of the relationship between the genetic units and the chemistry of DNA. We now have a non-randomly distributed label in a DNA which can be defined genetically with great accuracy and manipulated with ease. An attempt to identify this label with specific genetic units could yield valuable information.

(Supported by grant C-2440 from the National Institutes of Health, U.S. Public Health Service. *Predoctoral Fellow, National Institutes of Health, U.S. Public Health Service).

Meyer, Helen U. and Michael L. Criswell. Crossover analysis of sex-linked mutations induced in oögonial cells by repeated treatments with 4000r of X-rays.

When heavy doses of X-rays are divided into instalments and given at 4-day intervals, only the potential chromosomal breaks from the same irradiation can collaborate with one another. The proportion of chromosomal rearrangements is therefore determined independently by each of these particular instalments. This method of fractionated treatment lowers the mortality of the treated cells considerably in comparison with that with that occurring when the same total dose is given as a continuous, "acute" treatment. Such a method has been used in many of our experiments in which mutations induced in gonial cells were studied, where a rather heavy X-ray dose is needed to counteract the relatively low X-ray sensitivity of these cell stages. Not many gross changes in chromosomal structure were expected when irradiating in this manner; however it was desirable to get more information on this question. From an experiment in which a total dose of 24,000r, given in 6 instalments, resulted in 15.2% sex-linked lethals, we selected nine different cases of sex-linked mutations at random, derived from eggs laid 8-12 days after the last irradiation, for analysis of the crossover pattern. The mutation-carrying X-chromosomes were originally of isogenic origin, had the normal gene sequence and were marked by *sn oc*. One was a visible mutation, one a detrimental (about 15% of expected survivors), four near-lethals (less than 10% of expected survivors), and three fully lethal. In only one out of these nine cases had some rearrangement of the gene sequence occurred; this had resulted in a small inversion which was connected with a lethal. In the remaining 8 cases the crossover values were found to be in reasonable agreement with the expected map frequencies. Similar, more recent studies on mutations recovered from gonial cells by only one treatment with 4000r seem to agree with these findings that intrachromosomal rearrangements may be recovered from gonial cells, but in a much lower proportion than when a similar dose is given to mature spermatozoa.

(This work was supported by a grant to H. J. Muller and associates from the Public Health Service, Contract RG-5286 (3), and a grant from the National Science Foundation Summer Programs for Secondary School Students.)

Meyer, Helen U. and Evelyn R. Meyer. Sperm utilization from successive copulations in females of *Drosophila melanogaster*.

To learn how sperm from successive copulations is utilized, young virgin females homozygous for the second chromosome markers *cn bw sp* were mated first with

homozygous cn sp males and then remated to homozygous bw sp males. Only one copulation with each of these two types of males was allowed, and the females isolated immediately afterwards.

An interval of 4-5 days was unfortunately necessary between the two inseminations, since the females refused to mate for a second time before a sizable number of fertilized eggs had been deposited. It was found that the length of this interval varied with temperature and with the type of culture medium used. That various strains behave differently in this respect has been pointed out by Ehrlich in a similar study (D.I.S. 33:129-130, 1959).

39 females, which had been observed to have copulated once with each type of male and had been immediately separated from them, were bred individually and transferred to fresh culture vials every 24 hours for 12 successive days. After this time only one of them still laid fertilized eggs. All offspring from these daily broods were classified for phenotype, cn sp progeny indicating fertilization by the first male, and bw sp by sperm from the second one.

Two parent-females gave only one type of offspring, cn sp in one instance and bw sp in the other one, even though they had been observed to have copulated with both types of males. This could be explained by either non-functional sperm (as in XO males) or by copulation without insemination.

The results from the remaining 37 doubly inseminated females are summarized in the following table:

EGGLAYING PERIOD	AV. NO. FERTILE P-♀♀	AV. NO. F ₁ PER P-♀♀ PER DAY	PHENOTYPE OF F ₁		% F ₁ , FIRST INSEMIN.
			cn sp	bw sp	
BEFORE 2nd Insemination (4-5 days)	31*	19.5	2936	-	100 %
AFTER 2nd Insemination					
days 1-2	36.5	35.4	64	2521	2.48
3-4	33.0	24.3	27	1578	1.68
5-6	27.0	13.6	13	719	1.78
7-8	19.5	5.1	4	196	2.00
9-10	11	3.5	1	76	1.30
11-12	6	3.8	1	45	2.17
Totals, days 1-12			110	5135	2.1

*No record kept on the no. of F₁ from 6 other P-♀♀ during this period, which are included in the second part of this table.

Thus an average of only 2.1% of the total offspring obtained after the second mating was derived from sperm retained from the first insemination. However, this frequency did not vary significantly from brood to brood except for a slight drop after the first two days. These findings therefore do not support the view that spermatozoa are stored in the order in which they are received, but imply that they are mixed in the storage organs of the females and used at random to fertilize the eggs, in agreement with similar results obtained by Ehrlich as described in the report quoted above.

At the end of the observation period only 6 of the initial 37 females were still reproducing. Since the females were not yet very old, this was undoubtedly due to sperm exhaustion.

Fewer offspring resulted from the first insemination than from the second one. This might have been due to a variety of factors: the lower egg production of very young females, possibly a not yet fully developed storage capacity for sperm, or discharge of stored sperm before or during the second copulation. It also might have been due to the different genotype of the first male.

On the basis of our data it is possible to make a tentative estimate of the number of eggs which can be successfully fertilized by the amount of sperm deposited during one copulation. By adjusting the number of progeny from the first male by addition of an estimated 568 (for those 6 females for which no record was kept during the first period), a total of 3614 offspring were obtained from the first male, and 5135 from the second male. Dividing their average by 37, we obtain an estimate of

118 offspring from one combination; or, if one considers the result of the second mating to be more representative for the middle period of reproductive life, the number would be 140 successfully fertilized eggs.

(This work has been supported by a grant to Dr. H. J. Muller and associates from the U.S. Public Health Service, Contract RG-5286(3)).

Novitski, E. Post-treatment of irradiated sperm by low temperature.

In a talk given before the Conference on Problems of General and Cellular Physiology in 1949, I made the statement that post-

treatment of irradiated sperm by low temperature caused an increased recovery of sex-linked lethals. The transcript of this talk was subsequently published (*American Naturalist*, LXXXIII, 185-193) without the inclusion of any supporting data. Since that time I have been asked several times about these experiments.

In these runs, *Basc* ♀♀, previously inseminated by Canton-S ♂♂, were irradiated in order to remove the ambiguity of possible differential sensitivity of the stages of spermatogenesis. A control series, irradiated with 3600r at 25°C, gave 128/1246 (9.7%) lethals; a parallel series, held at 0.5°C during the irradiation, gave 34/138 or 24.6% lethals.

In the more extensive sets described below the dose was decreased to 1800r. The controls at 25°C gave 54/1166 (4.6%) lethals. An unirradiated control exposed to -1°C for 14 hrs. produced no lethals in a total of 877 tests. When the cold treatment (6 hrs.) preceded the irradiation (but with an hour and a half separating the two) there was no appreciable change in the lethal frequency (50/1172 = 4.3%). In two runs the females were kept at 0°C during the treatment and the lethal rate jumped appreciably (78/1113 = 7.0%; 99/1121 = 8.8%). Finally, in a run in which the females were exposed to the low temperature immediately after X-raying, the lethal rate was 11.7% (11/94). Although statistically significant, the low numbers in this last case, which was the only one involving bona fide post-treatment only, suggested repetition. Unfortunately, at this stage, deseminatation (which was undoubtedly responsible for the low numbers in this last experiment) became a serious problem and attempts at repetition failed dismally. This line of experimentation was then abandoned, although the effect of cold temperature in deseminating females was duly investigated and published.

Parker, D. An apparent incompatibility among seemingly normal members of the species *D. simulans*.

Peculiar results were observed in experiments with *D. simulans* whenever a cross involved the mutant vermilion. Difficulties with *D. simulans* crosses cropped up

continuously when vermilion was mated with the mutants yellow-white, black, sepia, scarlet peach, peach-hairless, plum, and with various wild type stocks. These difficulties were due to rather unpredictable abnormalities in the development of the offspring.

The crosses using vermilion males were moderately successful with the only deviation from normal being in the reduced amount of progeny from each cross. But the results when using vermilion females were more noteworthy. In roughly 70% of all the vermilion female crosses, development went no further than the egg stage. In approximately 15% of all crosses involving vermilion females, the development progressed until death occurred in the larva stage. The time of death established no predictable time pattern. Death occurred any time between the first instar and pupation. In the remaining 15% of the crosses, adult offspring emerged only to die sometime within the first five days. Even when such adult progeny did appear, they were much reduced in number from what is normally found.

Roberts, Paul A. Bristle differentiation in genetic mosaics of *D. melanogaster*.

It was demonstrated by Stern (*Proc. 9th Intern. Congr. Genetics. Part I:355, 1954*), using gynandromorphs, that achaete (ac) tissue is autonomous in mosaics.

That is, in flies in which the site of a posterior dorsocentral bristle is ac tissue, a bristle will usually not differentiate even when most of the surrounding tissue is ac⁺. Conversely, when the site of the bristle is ac⁺, differentiation is always initiated regardless of the amount and distribution of surrounding ac tissue.

Out of 1600 duplication carrying male offspring of the cross of females, $Dp(w^{VC})6094b/y w f:=/Y$, by males, $y ac w^a ct^{6f} \cdot Y^S / Y^L$, 110 had some loss of the duplication (carrying non-yellow, y^+ , and non-achaete, ac^+) and were mosaics involving the dorsocentrals. In this genotype, 99% of the male progeny not carrying the duplication (patroclinous males) had both anterior and posterior dorsocentrals missing, so both of these sites were scored. Of these 110 mosaics, 91 exhibited autonomy as described above, and 19 exhibited non-autonomy in which dorsocentral bristles differentiated in sites in ac tissue close to ac^+ tissue. Stern interpreted this as due to spread of ac^+ material into the ac tissue patch. However, 12 of the 91 autonomous cases had similar proximity of ac^+ tissue to the bristle site with maintenance of ac autonomy. Seven cases were observed in which a dorsocentral bristle differentiated at an abnormal site near a bristle site occupied by achaete tissue, but apparently within a potential area of differentiation.

Results are consistent with Stern's observations and his interpretation that ac and ac^+ are not establishing a regional singularity but responding to a "prepattern" present in both genotypes.

Sandler, L. and C. W. Cotterman.
A possible interpretation of the conversion of X chromosome by SD.

In the presence of SD (which is located on chromosome II and conditions abnormal segregation of this chromosome pair in males), some X chromosomes may be specifically converted

into suppressors of SD action (Sandler and Hiraizumi, 1961).

It is not known by what mechanisms either (1) the conversion of the X chromosome takes place, or (2) the converted X chromosome suppresses SD action. One possible supposition that can account for both of these effects is as follows. It may be imagined that the conversion results from the X chromosome physically acquiring a part of the SD locus. This modified X chromosome, in subsequent generations, can pair with the SD region of chromosome II in SD heterozygotes and thus prevent SD from pairing properly with SD^+ . This should indeed suppress the phenomenon of segregation-distortion because synapsis of SD and SD^+ is known to be necessary for distortion (Sandler, Hiraizumi and Sandler, 1959).

The only test of this notion that immediately suggests itself is to see whether there are any differences in the segregation of the X and chromosome II in SD heterozygotes, according to whether or not the X chromosome has been converted.

Accordingly, males heterozygous for SD and $In(2LR)Cy$ (Cy , itself, suppresses SD action by failing to pair properly with SD and is used here so as to maximize the probability of X-II synapsis) with either a modified X or an unmodified X, were crossed to $cn bw$ females. The results were as follows:

Type of X	Progeny			
	$Cy \sigma\sigma$	$Cy \text{♀♀}$	$Cy^+ \sigma\sigma$	$Cy^+ \text{♀♀}$
Modified	229 (.23)	259 (.26)	265 (.27)	246 (.25)
Normal	315 (.26)	288 (.24)	315 (.26)	277 (.23)

It is clear that X-II segregation is the same irrespective of whether or not the X has been modified. Thus we must suppose that either the suggested hypothesis is incorrect or that pairing between the X and SD, while sufficient to suppress SD action, is not of such a kind as to affect the pattern of segregation.

Sederoff, R. and E. A. Carlson.
The relation between allelic phenotype and allelic localization within the dumpy region.

A specific sublocus has been established in the dumpy gene for the ov mutants. These ov mutants exhibit two phenotypic effects of the dumpy gene, the oblique wing, written as o , and the bristle disturbances and protuberances of the thorax called vortex, written as v .

A series of six independently arising ov mutants (ov^1 , ov^n , ov^x , ov^{51f} , ov^{52b} , ov^h) were localized within the genetic map of the dumpy region. A modified "four-point" test using the outside markers -- echinoid (ed) at 11.0 and clot (cl) at 16.5 was used in the mapping procedure. The position of these alleles was determined with respect to two other alleles of the dumpy region, the thoraxate (lv) allele on

on the left of ov^1 and the vortex (v) allele on its right. In all instances the other ov alleles were mapped between lv and v. These localizations establish a sublocus within the dumpy gene which appears to be specific for the ov expression. No other alleles have been localized at this site. Therefore, the region may be referred to as the ov sublocus. (See Table 1 for the summarized results.)

One of the ov mutants, called ov^h (dumpy-humpy-like) shows a more extreme effect than the other ov alleles, and it is a facultative lethal in the homozygote. It is not lethal, however, in the heterozygous compound with any of the alleles of the dumpy region containing the lethal factor (e.g. l, ol, lv, olv). The localization of this allele between thoraxate, lv, and vortex, v, suggests that the ov^h mutant might be a minute deletion of the ov sublocus, or that it might occupy a separable site within the region.

These localizations indicate that mutation at the dumpy locus will usually be specific in expression for that portion of the map which is affected. A possible exception to this may be found for the olv alleles. These mutants are characterized by a loss of the total function of the dumpy gene. In phenotype they resemble deficiencies for the dumpy region. These extreme mutants could possibly be located anywhere on the pseudoallelic map. Localization of a series of olv alleles is now in progress (Southin and Carlson, unpublished).

The localization of the ov alleles to a specific sublocus makes possible an investigation of the fine structure of this portion of the dumpy region. A selective technique has been designed (see p. ____, this issue) which will be used to investigate this possibility of fine structure. Southin (1961, unpublished) has obtained recombination between two similar oblique alleles, o^2 and o^{bm} . Resolving power of this test was approximately 1×10^{-5} and we anticipate possible resolution with the selective technique to reach 1×10^{-7} .

Table 1

Trans Alleles	Localization of the ov Alleles		Total Count
	Verified "Conversions"	Verified Single C.O.'s	
lv^1/ov^1	1	6	66,009
lv^1/ov^h	0	1	3,548
lv^1/ov^x	0	2	22,668
lv^1/ov^n	1	2	22,766
lv^1/ov^{51f}	0	1	34,544
lv^1/ov^{52b}	0	1	21,477
v^2/ov^1	0	7	35,200
v^2/ov^h	0	1	2,955
v^2/ov^x	0	2	7,894
v^2/ov^{52b}	0	1	20,820
v^2/ov^{51f}	0	1	7,298
v^2/ov^n	0	2	4,539
TOTALS	2	27	249,718

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Seto, F. The relative constancy of phase specific action of recessive lethal factors in D. melanogaster.

In a recent paper Hadorn (1959 Arch. Jul. Klaus-Stiftg., 34:234-239) reported that the phase specificity of 19 recessive lethals had remained unchanged over the

past 7-8 years. Similar observations made in this laboratory tend to confirm Hadorn's general conclusions. Several strains of second chromosome recessive lethals, which manifest developmental effects in the larval-pupal and pupal stages, have been maintained in a balanced condition (C_y/le) for several years. During this interval the period of action of various lethals had been determined on several occasions in the course of various experiments, either by counts or by direct observations of their visible effects. In most of the lethals the phase specific action and characteristic phenotypes remained unchanged but a few had lost their larval-pupal or pupal effects and were later discarded. A summary of the observations on the various lethals are given below:

Period of action	Lethals	1951/52	1953/54	1956	1958	1960
Larval-pupal	N-59,N-1,X-11	*	--- D			
	X-3	*	*	*	---	(*)
Early pupal	N-61,N-51	*	*	*	*	*
	N-50	*	*	*	---	D
Pupal	N-32	*	*	*	*	*
	Co-7,Co-3A		*	*	*	*
Late pupal	N-42(N42A)	*	(*)	(*)	---	*
	N-13			*	---	---
Late pupal, adult	N-1A		*	*	*	*
	N-45		*	*	*	*
	N-55			*	*	*

* phenotype typical (*) phenotype altered --- phenotype lost D culture discarded

Lethal X-3 lost its L/P effect by 1958 but by outcrossing and re-isolating new lines it regained its L/P phase specificity but with an altered phenotype. N-42 initially manifested a late pupal effect which was lost or replaced soon after by a larval-pupal phenotype. By 1958 the L/P effect weakened and was lost but one of the outcrossed lines had regained the original pupal effect which it still maintains.

In another study 37 second chromosome recessive lethals obtained from wild populations were tested for their period of action initially toward the end of 1959 and a second time at the beginning of 1961. The ontogenetic distributions of lethality of the 37 lethals in the two counts were:

year	(phase of action)	E	E/L	L	L/P	P	Total
1959		1	5	22	5	4	37
1961		4	5	20	6	2	37

A X^2 test of heterogeneity indicates that there is no significant difference in the distributions. A comparison of the individual lethals showed that 13 of the 37 lethals displayed a change in the stage of lethal action. However, only two of the thirteen showed marked changes (from the P or L/P to the E stage) whereas the others showed only minor shifts to an adjacent stage. The results of the observations on these 11 lethals are summarized below:

Shift to an earlier stage		Shift to a later stage	
E/L to E	B-55	E/L to L	B-6
L to E/L	B-19,B-20,B-39	L to L/P	B-14,B-27
L/P to L	B-33	E/L/P to P	B-23
P to L	B-58		
P to L/P	B-52		
	(7)		(4)

24 lethals did not show any change in phase specificity. Of the changes observed, most of them were minor changes and are not significant. It is known from earlier studies that cultural conditions and differences in genetic background can alter the time of expression but in general the phase specificity tends to be relatively constant.

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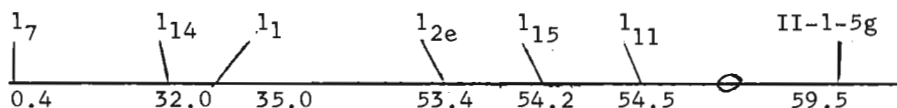
Spieß, E. B. and Helling, R. B.
Linkage of chromosome II lethals in
D. melanogaster.

A series of lethals on chromosome II were extracted from a population cage of D. melanogaster descended from the control #3 population of Bruce Wallace, which was

initiated with Oregon-R in 1949 and had been maintained for at least 170 generations at 25°C in a plastic population box before this laboratory obtained subsamples from it. Lethals were extracted in conjunction with an attempt to obtain "good viability" chromosomes (done by Mr. Archie Allen using the Cy L/Pm technique), and they were maintained in balanced condition with Cy L. Salivary analysis revealed no aberrations. The frequency of lethal chromosomes in the cage was estimated at 0.175, but linkage tests showed that the lethals were all in the left arm of chromosome II. Mapping of lethals was done in two sets of crosses: the first set utilizing markers *al pr c sp* and the second set using markers in either right or left arms depending on where the

first set of crosses placed the lethal: $al\ dp\ b\ pr$ and $pr\ c\ px\ sp$. Females heterozygous for lethal/recessive markers were backcrossed to males with these markers homozygous plus a dominant such as Cy or $B1$. All crossover classes containing the dominant were backcrossed to the lethal/ $Cy\ L$. By counting 100 flies in the progeny and finding no wild type, lethality was classified.

The following linkage map was obtained from classifying ten lethal chromosomes; a single lethal obtained as a spontaneous mutant in another experiment (Allen) (II-1-5g) was placed in the right arm:



By testing lethals for allelism, 17 occurred four times, twice with another lethal locus (for example 12 was lethal at the 17 locus plus $12e$ at 53.4); 11 occurred twice; and in addition two other multilocus lethals were obtained which have not yet been located exactly. With such high frequency of certain lethals and high frequency of multilocus lethals, it might be inferred that high fitness must be conferred upon lethal heterozygotes in this population. Relative viability tests are now being made by the senior author. Certainly the non-random distribution of these lethals would imply possibly blocks of genes in the left arm of chromosome II which might be heterotic.

Lethals obtained by similar tests on chromosome III are currently being carried out also. Allelism is very high but linkage analysis has not been completed.

Stern, C. and E. Sherwood.
A search for maternally influenced sex-ratio in *Drosophila melanogaster*.

In order to find out whether females of *Drosophila melanogaster* or *Drosophila simulans* caught in nature would produce offspring of unusual sex-ratios, traps were

set out at various localities, far enough away from experimental laboratories to exclude the possibility of trapping laboratory-bred flies. Each female was put singly into a culture bottle and allowed to lay eggs for seven days.

Of a total 626 females 606 produced offspring in a $1\text{♀} : 1\text{♂}$ ratio, 14 females gave an F_1 in the ratio of $3\text{♀} : 1\text{♂}$, and 6 females had only female progeny. Each one of the all-female progeny was first mated to *D. melanogaster*, then to *D. simulans* males, and none were fertile. One of the mothers was mated to *D. melanogaster* males and gave offspring in the normal sex-ratio.

The results fit well with Sturtevant's findings of 1929, where he reports 10-40% all female hybrid offspring from *D. melanogaster* ♀ x *D. simulans* ♂♂ in crosses done in the laboratory. In the reciprocal crosses, i.e. *D. melanogaster* ♂♂ x *D. simulans* ♀♀, he obtained 2% all-male hybrids. None was found in our experiments.

Whether the hybridizations occurred in nature or within the trapping bottles is unknown. In any case, no maternally determined "sex-ratio" condition was found in the sample of 606 tested females.

Strangio, V. A. Radiosensitivity to certain breakage aberrations during spermatogenesis in *D. melanogaster*.

A re-investigation of radiosensitivity during the spermatogenesis of *D. melanogaster* has been made utilising a doubly-marked Y-chromosome, y^+YB^S (see Brosseau

et al, 1961. Genetics 46:339-346 for full description). Recently emerged Canton-S males carrying this Y-chromosome were irradiated with 1000r and then mated daily for twelve days afterwards either to three $y\ apr\ ec$ females (Series I) or to four attached-X $y\ v\ f\ car$ females (Series II). In Series I, the regular offspring were wild type females and $apr\ ec$ - Bar males; in Series II, $v\ f\ car$ - Bar females and wild type males. The improved technique described here allows the following irradiation-induced aberrations to be detected simultaneously:

(a) sex-chromosome losses as:

XO males in Series I, phenotypically "y apr ec".

XXO females in Series II, phenotypically "y v f car".

(b) individual Y-chromosome marker deletions as:

XY^- males in Series I, where loss of y^+ yields $y\ apr\ ec$ - Bar ("yellow") males or loss of B^S results in $apr\ ec$ ("non-Bar") males.

XXY^- females in Series II, loss of y^+ produces $y\ v\ f\ car$ - Bar ("yellow")

Day	1	2	3	4	5	6	7	8	9	10	11	12
<u>SERIES I</u>												
Total Offspring	2390	2207	2637	2125	1625	845	420	409	1862	1858	1481	1747
% "y apr ec" ♂♂	0.04	0.09	0.15	0.56	0.98	1.07	1.43	-	0.21	-	-	-
% "yellow" ♂♂	-	0.05	-	0.09	0.06	0.83	0.95	0.24	0.05	0.16	-	-
% "non Bar" ♂♂	0.04	0.09	0.04	0.19	0.25	0.71	0.48	1.71	0.05	0.11	0.07	0.06
% "Bar" ♀♀	0.04	-	-	-	-	0.24	0.95	0.24	-	-	0.14	-
<u>SERIES II</u>												
Total Offspring	1278	2235	2330	2287	1245	952	380	194	997	1709	1582	1685
% "y v f car" ♀♀	0.31	0.22	0.26	0.48	1.37	1.05	2.63	1.55	0.10	0.12	-	-
% "yellow" ♀♀	-	-	-	-	0.16	0.32	2.63	1.03	0.20	0.23	0.13	-
% "non Bar" ♀♀	-	0.04	0.26	0.57	0.64	1.47	3.42	4.12	0.20	0.12	-	-
% "non yellow" ♀♀	-	-	0.04	0.17	0.08	0.11	0.79	0.52	-	-	-	-
% "Bar" ♂♂	-	-	-	-	-	0.11	0.26	-	-	-	-	-

- females or loss of B^S yields v f car ("non-Bar") females.
- (c) non-disjunction of X and Y as:
 XXY females in Series I, phenotypically "Bar".
 XYY males in Series II, also "Bar".
- (d) large interstitial X-deletions as:
 XXX^- hyperploid females in Series II, phenotypically "non-yellow" and usually also showing one or two of the v f car markers.

The results are summarized in the accompanying table and are similar to those published as separate experiments by Ives (1960), S \ddot{u} vhagen (1960) and Chandley & Bateman (1960). The egg-laying period of the females is kept constant for each brood so that the relatively low adult numbers on the seventh and eight days are probably a reflection of maximal dominant lethal induction. This has been confirmed by direct egg mortality counts which incidentally also reveal a remarkable consistency in the radiosensitivity patterns for individual males. The aberration frequency versus brood curves for sex-chromosome loss, Y-deletions and X-deletions are all essentially similar and reach peak level on the seventh or eight day after irradiation. When these are compared with the onset of induced non-disjunction of X and Y chromosomes which must occur before their separation during the reductional meiotic division, it appears that the chromosomes exhibit their greatest radiosensitivity to the induction of at least these types of aberration during the early meiotic stages i.e. in the spermatocytes. Further investigations are in progress with ring and inverted X-chromosomes.

Tates, A. D., and F. H. Sobels.

The genetic effects of post-radiation treatment with cyanide in pupal spermatids.

In earlier experiments it was observed that treatment with cyanide following high-intensity X-irradiation (2200r/min.) resulted in a significant increase of the frequency of sex linked lethals and trans-

locations in stages with greatest sensitivity to X-rays (Sobels 1960). These stages, presumably corresponding to spermatids, were sampled by means of the "brooding technique" after treatment of adult males. The extent to which cyanide enhanced the mutation rate showed, however, a considerable variation from experiment to experiment. This was felt as a serious handicap at a further analysis of the post-treatment effect. Assuming that susceptibility to the action of cyanide is restricted to one particular stage only, imperfections of the brooding technique in specific sampling could be responsible for the variation mentioned above. Since according to observations of Khishin (1955) and Oster (1955) radiated spermatids can be obtained in a more selective manner by treating 48-hour pupae, we investigated whether post-treatment of 48-hour pupae would yield more uniform results. Also, this method would be less time consuming than that of sampling spermatids by the brood technique from treated adults.

Male pupae of the genetic composition $In(1)d1-49, y B/sc^8 \cdot Y; bw^D$ were irradiated with either 1200 or 2000r at a dose rate of 3000r/min., 100 KVP, 3.9 mA, without additional filtration. Ninety seconds after completing the radiation, part of the pupae were exposed to hydrocyanic acid, equivalent to 37.5 mg KCN, at a rate of flow of 100 ml/min. during 5 minutes. Preliminary tests had shown that with this procedure sterility was approximately 25% and mortality less than 10%. After hatching the males were mated individually to three females of the $Y^S In(1)EN \cdot Y^L, y; st$ stock. Their progeny was tested for the incidence of sex-linked lethals and translocations of the II-III, Y-II, Y-III and Y-II-III types, according to Muller's (1954) multipurpose method. The results are presented in tables 1 and 2.

Table 1

Experiment	Dose (r)	Sex-linked lethals		
		n. chrom.	leth.	% leth.
R ₁	1200	968	129	13.3
R ₁ -CN		914	139	15.2
R ₂	2000	436	95	12.8
R ₂ -CN		293	69	23.5

Table 2

Experiment	Dose (r)	Translocations (total number)			Translocations II-III	
		n gametes	transl.	%transl.	transl.	%transl.
R ₁	1200	424	71	16.7	45	10.6
R ₁ -CN		483	96	19.9	54	11.1
R ₂ -	2000	260	57	21.9	32	12.3
R ₂ -CN		199	59	29.6	34	17.1

The data show that post-treatment enhanced the frequency of both lethals and translocations, though not in a significant manner. Compared to the observations on treated flies, the modification is less pronounced. The method of treating pupae does not offer, therefore, technical advantages for a quantitative study of the cyanide effect.

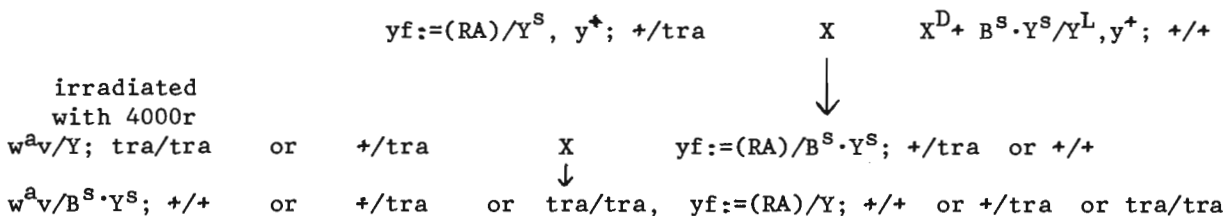
Terzaghi, Eric and E. Novitski.

An attempt to produce fertile "transformed" males.

Because of its obvious technical utility and its intrinsic developmental genetic interest, a number of attempts have been made in the past, by several investigators,

to obtain a fertile "transformed" male (X/X; tra/tra). The authors have performed a large-scale radiation experiment in an attempt to produce the desired "transformed" male.

In a large scale radiation experiment, it is of great benefit to have an experimental set-up in which a large number of individuals may be tested for the desired characteristic with a minimum of time expended. Towards this end, the following mating scheme was designed, in which the progeny of the last cross select themselves in respect to the desired characteristic, fertility.



Among the progeny of the first mating, no fertile males are expected, hence, rigorous and frequent virgin collection is eliminated. Among the progeny of the second mating, no fertile males would normally be expected except in that case when the appropriate dominant mutation had been induced, or the appropriate recessive allele of "transformer" had been induced.

Contingencies which would produce undesired fertile males, such as breakdown of the double X (reversed acrocentric) or non-disjunction of the X and Y in the irradiated male, have been met, respectively, by making certain that both arms of the double X contain lethals and including the Bar Stone fragment in the chromosomal complement of the mates of the irradiated males. Thus, in the latter case, any zygote getting both the X and the Y from the male parent, would get either the Bar Stone fragment or the double X from the mother. The former combination produces a sterile male, and the latter is a super-female.

In order to attain the maximum yield of offspring per female and to conserve labor, a culture technique suggested by Spencer was employed. In this system, 100 to 200 females, plus the appropriate number of males, were placed in a quarter pint milk bottle with the standard cornmeal-molasses medium. They were allowed to remain for two days, and then were transferred to a fresh bottle. Two days later, a small wad of Kleenex (two sheets), saturated with a thick solution of fresh bakers yeast, was placed on the food in the bottom of the bottle. Females were kept and used until they died, only making certain that the initial number of flies per bottle was maintained, by combining bottles when necessary.

To date, approximately 150,000 potential transformed males have been tested, with no case of the desired type of fertility yet appearing. There were, however, occasional cases of spurious fertility due either to contamination or to lack of virginity somewhere in the sequence of matings, where it was essential to have virginity.

Tokunaga, Chiyoko. Notes on the sex chromosome constitution of oogonial cells in gynanders.

During the course of development a female zygote of D. melanogaster heterozygous for a ring X-chromosome may lose the ring chromosome from a cleavage nucleus, thus developing into a gynander. If the differentiation of germ cells should be the result not of their own genotype but of that of the somatic tissues of the gonads it is conceivable that XO oogonia could occur in the ovaries of gynanders.

175 gynanders were obtained among the progeny of a cross between $y\ ac\ sn^3$ females and the ring X-chromosome carrying $X^{c2}\ f\ car$ males. In order to increase the frequency of gynanders among the progeny, $y\ ac\ sn^3$ virgin females were aged for eleven days at 17°C before mating. Of these 175 gynanders, one had underdeveloped gonads, eleven had one testis and one ovary, 43 had a pair of testes, and 120 had a pair of ovaries. The oogonia of 83 gynanders with 2 ovaries each were examined for their X-chromosomal constitution by means of the smear method, after fixation with aceto-lacto-orcein. 33 of these individuals clearly showed two X chromosomes, seven more seemed to have two X chromosomes, and the remaining 43 specimens did not show good mitotic metaphase figures. Oogonial mitotic figures of eight gynanders with one ovary and one testis were also investigated. One showed two X chromosomes clearly, while seven smears were unsuitable for cytological analysis.

Thus no mitotic figures were found which showed oogonial cells of XO constitution among the ovaries studied.

Wolff, M. and A. Coughlin. Tests for meiotic drive in interspecific hybrids.

The crosses recorded here were designed to detect possible cases of abnormal chromosome segregation in interspecific hybrids of D. pseudoobscura and D. persimilis.

The rationale behind this test is to check on the possibility that during the divergence of these two species, there occurred in one or the other of the two a case of meiotic drive which became fixed and now would be undetectable by ordinary tests.

GENERAL PROCEDURE: Successive backcrosses with progeny counts each generation were carried out more or less extensively for a large number of cross types. For each type of cross, tests for possible abnormal chromosome segregation in the female and male were carried out in separate lines of backcrosses. For convenience, the following terminology was used: female Backcross -- female progeny with stock male
male Backcross -- male progeny with stock female

Mass matings were used in the original hybrid crosses. Pair matings were set up for each successive female Backcross. From the progeny of each generation of female Backcrosses, mass male Backcrosses were set up until successful crosses were obtained. Then a separate line of pair male Backcrosses were set up.

Where recessive markers were followed, + progeny were backcrossed to the original stock carrying the mutant. Where dominant markers were utilized, progeny showing the character were mated to the original wild type stock.

Although crossing-over was partially suppressed by normal or, for chromosome IV, special stock inversions, unequal recovery in the progeny of females might indicate unequal recovery of only the chromosomal region adjacent to the loci involved.

CHROMOSOME II:

Original Matings:

Iv. gl^2/gl^2 D. pseudoobscura females x wild type #12 D. persimilis males.

Iw. gl^2/gl^2 D. pseudoobscura females x wild type #21 D. persimilis males.

Iy: up Ba $gl/up+gl$ D. pseud. females x wild type #21 D. persimilis males.

(Dominant marker followed)

Results:

Iv. 1st female Backcross -- Equal recovery $-\frac{gl^2}{gl^2+}$
283/281

Iw. Equal recovery. Combined data through 3rd female Backcross $-\frac{gl^2}{gl^2+}$
456/463

Iy. From 18 successful 1st female Backcross matings, 612 Ba and 924 Ba⁺ progeny were obtained. However, practically all of the excess Ba⁺ progeny came from only ten matings. Unfortunately, the female Backcross matings from these ten were singularly unsuccessful; the 2nd female Backcross yield were small and no successful 3rd female Backcrosses were obtained.

Matings from the remaining eight 1st female Backcross matings were more successful. These and subsequent backcrosses gave equal recovery in both sexes. Combined data for the 2nd through 6th female Backcrosses:

Ba		Ba ⁺	
female	male	female	male
1160	829	1225	931

CHROMOSOME III:

Original Matings:

If. or (ST) D. pseudoobscura females x wild type #12 D. persimilis males

Ih. or (ST) D. pseudoobscura females x wild type #15 D. persimilis males

Ij. or (ST) D. pseudoobscura females x wild type #21 D. persimilis males

Results:

1st female Backcross: In all three there appeared to be an excess of or⁺ over or progeny:

	or	or ⁺
If	1181	1453
Ih	508	636
Ij	463	686

Subsequent Backcrosses:

	or	or ⁺
If. Equal recovery - 2nd B.C.	714	799
3rd B.C.	2374	2413
Ih. Equal recovery 2nd Backcross -	167	164

No further matings.

Ij. While results of the 2nd female Backcross indicated possible preferential recovery of or⁺, subsequent backcross generations gave approximately equal recovery:

	or	or ⁺
2nd B.C.	419	543
3rd B.C.	618	691
4th B.C.	705	769

Backcrosses

If and Ij. Numerous successful male Backcrosses were obtained from 1st female Backcross progeny. In these and subsequent male Backcross generations there was approximately equal recovery of or and or⁺.

CHROMOSOME IV:

Original Matings:

Im. in hk j Cy (inv. IV)/1 D. pseudoobscura females x wild type #15 D. pers. males

Io. in hk j Cy (inv. IV)/1 D. pseudoobscura females x wild type #21 D. pers. males

Results:

	Cy	Cy ⁺
Backcrosses: Im. Equal recovery 1st female Backcross:	228	245
Io. Equal recovery through 5th Backcross.		
Combined data:	3214	3316

male Backcrosses: Several successful backcrosses were obtained from the 3rd female Backcross progeny of Io. These and the 2nd male Backcrosses gave approximately equal numbers of Cy and Cy⁺ progeny. Combined data:

Cy	Cy ⁺
419	364

X CHROMOSOME - RIGHT ARM:

Original Mating:

Wild type AH D. pseudoobscura males x se D. persimilis females

Result: 1st female Backcross - only one successful mating.

se	se ⁺
61	59

No successful 2nd female Backcross matings.

X CHROMOSOME - LEFT ARM:

Original Matings:

IIf. y sn v co sh D. pseud. females x wild type #12 D. persimilis males

IIE. y xn v co sh D. pseud. females x wild type #21 D. persimilis males

It. Pt y sn v mbl D. pseud. females x wild type #21 D. persimilis males

(F₁ females mated to y sn v co sh D. pseudoobscura males)

Is. Pt y sn v mbl D. pseud. females x wild type #19 D. persimilis males

Dominant marker followed. (Pt males almost always y sn v)

Results:

II f. 1st female Backcross only: Apparent excess of y^+ over y progeny: $\frac{y}{209} \mid \frac{y^+}{324}$
 Also an excess of sh^+ over sh progeny: $\frac{sh}{199} \mid \frac{sh^+}{344}$

III e. 1st Backcross only - Excess of y^+ over y progeny among both females and males.

y^+		y	
females	males	females	males
300	216	91	124
516		215	

Also a somewhat higher recovery of sh^+ over sh among the progeny.

sh^+		sh	
females	males	females	males
250	161	141	179
411		320	

II t. Excess of $+$ over y xn v progeny. $\frac{y \text{ sn } v}{331} \mid \frac{+}{707}$

II s. 1st female Backcross - Equal recovery: $\frac{Pt}{307} \mid \frac{Pt^+}{282}$

Subsequent female Backcrosses: Equal recovery. Combined data 3rd through 6th female Backcrosses:

Pt	Pt ⁺
2059	2261

SEX RATION IN THE 3rd CHROMOSOME CROSSES:

(1) If. or (ST) D. pseud. females x wild type #12 D. persimilis males

Unfortunately no sex counts were made until the 3rd female Backcross.

3rd female Backcross:

Twenty-four matings were set up from nine 2nd female backcross matings. Overall, there was an approximately 2:1 ratio of females to males (3274 females to 1513 males) and an equal number of or^+ and or males (763 or^+ to 750 or males). Matings from two of the nine 2nd female Backcross matings seemed to show segregation for (a) high female excess and (b) only slight female excess. In the other lines, all matings gave off-ratios of approximately 2 females to 1 male or higher.

Subsequent Backcrosses:

Whenever only a slight excess of females appeared, all subsequent backcrosses descending from that mating gave similar sex ratios. When the ratio of females to males was high, the next backcross gave apparent segregation of approximately even and high female to male ratios. In some instances, or females from matings giving a high excess of females were mated to stock or D. pseudoobscura males. These also showed an apparent segregation for approximately even and high female to male ratios.

(2) Ij. or (ST) D. pseudoobscura females x wild type #21 D. persimilis males

This series consistently showed a relatively slight excess of females. The 3rd female Backcross gave 775 females to 699 males and counts from subsequent backcross generations were similar.

(3) Ih. Or (ST) D. pseudoobscura females x wild type #15 D. persimilis males

Data from only two 2nd backcross matings:

	females	males	males	
			or	or ⁺
Mating I	94	55	29	26
Mating II	147	35	16	19

Conclusion: There appears to be no evidence for any instance of meiotic drive incorporated into either of the two species.

Würgler, Friedrich E. Modification of x-ray induced embryonic mortality by different anoxia conditions before and during irradiation of uncleaved D. melanogaster eggs.

Continuing our work on the oxygen effect in Drosophila zygotes (see Ulrich & Würgler, DIS-33) a more detailed analysis of the influence of environmental gas conditions before and during irradiation has been made. In earlier experiments we have exposed eggs (which were 10 - 20 minutes old) to an air or a nitrogen current 1 minute before and during the 3 minutes lasting irradiation. In the experiments reported here the influence of prolonged pretreatment and change of gas atmosphere during irradiation has been tested; the age of eggs at the beginning of irradiation (3 minutes; 1000r; 50 kV; 10 mA) was again 10 - 20 minutes. A wild stock (Berlin wild) was used.

a) nitrogen treatment without irradiation

Unirradiated controls in air showed an embryonic mortality of 6.4%. 1 - 7 minutes nitrogen treatment increased the mortality linear with the duration to 8.7%. Beyond 8 minutes mortality increased more steeply. After 20 minutes in nitrogen 55.7% of the embryos died. Straight lines calculated by linear regression for these data were used to correct the following results (two different lines being calculated from (1) the interval 1 - 7 minutes and (2) the interval 8 - 20 minutes. In irradiation experiments anoxia was never extended over more than 8 minutes.

b) irradiation with nitrogen pretreatment

With 1 minute pretreatment and irradiation in nitrogen we found an embryonic mortality of 54.9% (n = 739). Longer (up to 5 minutes) and shorter (1/2 minute) pretreatment did not significantly change the results (summarized pretreatment experiments: 55.1%). The same was true even if there was no pretreatment at all: 54.7% mortality (n = 2488).

If the irradiation in air was preceded by exposure to nitrogen for 5 minutes, the mortality (84.1%; n = 1418) was not different from that without pretreatment (83.6%; n = 2426).

c) change of gas environment during irradiation

The rapid exchange of gas between egg and environment demonstrated under b) allowed for a change of the gas conditions within the egg during the 3 minutes lasting irradiation. In a first series X-rays were applied without an interval 1 minute (either the first, second, or third) in nitrogen and 2 minutes in air; in a second series 2 minutes in nitrogen and 1 minute in air. As compared to 3 minutes irradiation in air practically the same decrease of embryonic mortality was found in the 3 experiments of series 1. The same was true in series 2, where the decrease was more pronounced. Therefore the results of each series are summarized. Thus an embryonic mortality of 77.2% (1 minute N₂) and 69.2% (2 minutes N₂) was found. This agrees well with the expectation of 77.2% and 68.0%. This expectation is derived from the hypothesis of independent realization of radiation-damage caused under aerobic and anaerobic conditions. The calculation of the exact data were based on the findings that dose-action curves in air (ULRICH 1960) and nitrogen (WÜRGLE 1960) can be approximated by curves of the form $y = 1 - e^{-kd}$ (y = mortality; D = dose).

d) conclusions

- 1.) The replacement of gas inside D.m. zygotes by N₂ is achieved within a few seconds.
- 2.) A nitrogen pretreatment up to 5 minutes has no influence on the embryonic mortality induced by irradiation of 10-20 minutes D.m. eggs.
- 3.) If during part of the irradiation time the air atmosphere was replaced by nitrogen, the embryonic mortality decreased proportionately to the length of the N₂-fraction. This agrees with the assumption of independent realization of the radiation-damage caused under aerobic and anaerobic conditions.

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Zimmering, S. and H. J. Muller.
Studies on the action of the dominant female-lethal Fl and of a seemingly less extreme allele, Fl^s.

Tests were made to determine whether the female lethal Fl (Muller and Zimmering, 1960, Genetics 45: 1001-1002) still acts as a complete lethal when present in pseudo-males having two Fl-containing

X-chromosomes and two third chromosomes containing tra ("transformer of sex," Sturtevant, 1945, Genetics 30: 297). It was found that the sex transformation failed to save the lives of these flies. Similarly, in flies heterozygous for Fl, the viability of pseudo-males (XX, but homozygous for tra) was as much reduced by the dominant action of Fl as was the viability of their non-transformed sisters that had the same X-chromosome composition but were heterozygous for tra.

Tests of the genetic factors determining the dominant female-lethal effect of Fl in crosses of our y v stock ("b120") have shown that all the major chromosomes (X, II, and III) of this stock play an important and synergistic role in producing the effect. In daughters of females heterozygous for some or all of these three chromosomes little or no dominant lethal effect was produced except when all three of the chromosomes were present together in the given mothers. The effect was a maternal

one, tending to kill the heterozygous Fl (but not the non-Fl) daughters of all classes, provided that the mothers contained these intensifiers in all three chromosomes, in at least single dose. The intensifiers themselves were partially dominant, in that mothers homozygous for them gave a higher lethality of daughters heterozygous for Fl than did mothers heterozygous for them. When virgin females carrying the intensifiers are kept at a comparatively high temperature (35°C) for 36 or more hours prior to egg laying, the mortality of their heterozygous Fl daughters, derived from eggs laid at 25°C within the next three days, relative to that of their brothers, is considerably reduced (in the cases studied, from about 96% to 80%). Little reduction of mortality is produced when the exposure to warmth is allowed to last only 24 hours. Other cases of genes that have a maternal effect in killing daughters but not sons have been reported by Redfield (1924, 1926), Gowen and Nelson (1942), Gowen (1949), and Bell (1954), but in these cases there was no finding of a primary female-lethal gene, corresponding to Fl, that had to be present in the female that was herself subject to the lethal action.

In the experiment on pseudo-males the stock that had been used to provide the tra gene (our stock "j22") had had females with attached X's and males whose single X-chromosome contained w^a . The crosses of this stock unexpectedly showed that the w^a -containing chromosome also carried an allele of Fl. We are, for reasons to be given below, denoting this as Fl^s (a symbol superseding our earlier, unpublished designation, Fl^2). It was found that compound females, one of whose X-chromosomes carried Fl and the other Fl^s , invariably died. However, when crosses were made of Fl^s males to stocks y v ("b120") and w ("b69"), which on crossing to Fl males had given a high mortality of daughters, i.e., a high dominance of Fl, no such lethality occurred among these daughters. That is, Fl^s , unlike Fl, failed to act as a (partially) dominant lethal. That this difference was not sufficiently explained by autosomal modifiers was proved by experiments in which the autosomes were appropriately substituted by the aid of chromosomes having inversions and markers. Similarly, parts of the X far from Fl were ruled out. It was further found that Fl^s , unlike Fl, is not, when in its original setting, lethal even in the female homozygous for it, despite its lethality when "in compound" with Fl. Fl^s does sometimes act as a lethal to females homozygous for it, however, when taken out by crossing over from its original genetic setting, but the number and loci of the modifying genes here involved have not been worked out.

In the crosses that gave rise to homozygous Fl^s females it was found that these females are invariably sterile (hence the superscript s). Their abdomens remain unenlarged, like those of homozygous fcs females, while they appear normal in other outward respects. Like the lethality of Fl, the sterility of Fl^s is to a certain extent and under some conditions dominant, inasmuch as heterozygous Fl^s females are found in some crosses to have a high frequency of sterility. Such sterility has not thus far been observed among heterozygous Fl females. Further studies are however needed to determine definitely whether there are differences in the action of Fl and Fl^s when they are in exactly the same genetic setting.

Whereas Fl arose within Inversion-49, Fl^s is in an X-chromosome of normal structure. It must therefore have arisen as a result of a spontaneous mutation independent of that which produced Fl. Both these genes have been found by linkage tests to be slightly to the left of oc. Fl^s has been located more exactly as lying between cm and ct, nearer to cm, inasmuch as only 2 out of 10 tested crossovers between cm and ct^6 proved to have been between the loci of cm and Fl^2 , while the rest were between the loci of Fl^2 and ct, thus placing Fl^s at approximately 19.1 in the X-chromosome map.

Thus far, tests for female lethality have been made in our laboratory, by Robert Baum and by Marcia Henning, of a considerable number of our stocks in which the X-chromosome of the male had been kept confined to males by having them always crossed to females with attached X's. Thus far, no cases of female lethality have been found other than those in which the original Fl allele had been present as a result of the common origin of the Fl-containing region of the given X-chromosome with that of the X-chromosome of the stock in which Fl had first been discovered. These results suggest that mutations of the type in question are comparatively rare.

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Browning, Luolin S. and Edgar Altenburg.
Weighing of dehydrated *Drosophila* as
a counting method.

The counting of many thousands of individual flies is one of the major technical problems involved in experiments in which there is a low incidence of the phenomenon

being studied -- for example, visible mutation rates at specific loci and pseudo-allelic crossingover. A method which eliminates this task without the use of special equipment and furnishes counts reliable within a standard error of about 3% has been developed.

Flies are grown by the "vat method" (D.I.S. #33, p. 177), from 8,000 to 20,000 offspring being obtained from several transfers of the same parents (usually 200 to 400 parents). Since the flies that hatch first in a vat may be twice as large as those hatching later, due largely to better cultural conditions, it was found necessary to establish a rigid routine for emptying, examining, and handling the flies. Three examinations of the etherized offspring are made per vat -- on the 14th, 17th and 20th days -- after which the vat is discarded. After each examination, the offspring are immediately killed with ether, placed in an open milk bottle in an incubator at 55°C and kept for at least 24 hours. All the flies from a given vat are eventually added to the bottle, and after dehydrating, the bottle is transferred to a dehumidifier until a convenient time for weighing on an analytical balance. Flies kept for as long as three months in the dehumidifier showed only a slight change in weight. It was found that various mutant strains in laboratory use had different weights and sex ratios, and although this variation was not great, it was calculated by actual counts for different experiments. The table below shows the results of such a sampling, together with the weights after dehydration of each sample of counted flies coming from a single vat. (The female parent flies in this experiment carried w^a in one X chromosome and w^{Bwx} in the other, and were also heterozygous for y and spl , as well as for the Cy and Ubx inversions in the second and third pair of autosomes; the male parents were Basc males containing $sc w^a B$.)

Females			Males			Females and Males		
No.	Weight (Grams)	No. per Gram	No.	Weight (Grams)	No. per Gram	No.	Weight (Grams)	No. per Gram
4,637	1.2920	3,560	3,903	0.8543	4,580	8,540	2.1463	3,980
2,455	0.7533	3,260	2,184	0.4851	4,500	4,639	1.2384	3,750
1,973	0.8127	2,436	1,563	0.4139	3,780	3,536	1.2266	2,880
-	-	-	-	-	-	2,797	1.0342	2,700
-	-	-	-	-	-	2,551	0.7686	3,320
-	-	-	-	-	-	2,241	0.7296	3,080
2,072	0.6431	3,220	1,724	0.3969	4,320	3,796	1.0400	3,650
-	-	-	-	-	-	2,832	0.7465	3,790
2,406	0.7313	3,290	2,091	0.4749	4,420	4,497	1.2062	3,730
-	-	-	-	-	-	5,375	1.6378	3,280
-	-	-	-	-	-	4,141	1.2296	3,375
-	-	-	-	-	-	5,657	1.6760	3,380
13,543	4.2324	3,200	11,465	2.6251	4,370	50,602	14.6798	3,450
								± 113(S.E.)

There is a range in yields among the twelve sample vats of from 2,200 to 8,500 flies, with a range in flies per gram of from 2,700 to 3,980. The average number of flies per gram for all the vats is $3,450 \pm 113$, or $3,450 \pm 3.3\%$. Thus in 67% of all experiments involving 50,000 or more flies, the total number of flies (obtained by multiplying the weight of dehydrated flies by 3,450) would be expected to be in error by no more than 3.3%, and by no more than 6.6% in 95% of such experiments.

It is sometimes desirable to scan rapidly a large number of flies for rare mutants in one sex but without separating them as to sex. The table shows that the number of females represented by one gram of dehydrated males and females can be reliably estimated. For example, the sex ratio based upon a count of about 25,000 flies (columns one and four in the table) was 0.54 females: 0.46 males, 13,543 females being included in a total weight of 7.3508 grams of both males and females. This indicates that one gram of both males and females contained 1,845 females, a figure very similar to that of 1,863 females obtained when the average number of males

and females contained in one gram of both males and females (3,450) based upon a count of over 50,000 flies is multiplied by the sex ratio factor of 0.54.

Mickey, G.H. Nigrosine as an aid for staining brain and salivary gland chromosomes.

We have found that the addition of nigrosine (alcohol soluble -- National Aniline) to aceto-orcein stain enhances the staining of chromosomes remarkably. Larval

ganglion chromosomes are stained intensely enough to be examined and photographed without the use of phase contrast microscopy. The procedures are as follows:

1) Aceto-orcein stain is prepared by dissolving one gram of orcein dye (Allied Chemical) in 100 ml of 45% acetic acid in an Erlenmeyer flask, with reflux condenser attached, and heating for one hour without boiling. The solution is cooled, filtered, and stored in refrigerator.

2) Aceto-nigrosine stain is prepared by heating 100 ml of 50% acetic acid to boiling point, adding 4 gms of alcohol soluble nigrosine and stirring constantly. It should be boiled for 3 - 5 minutes, or until it acquires a highly viscous consistency. When cooled, it should be filtered, using a water pump filter. Store in closed glass vessel in the dark at room temperature.

3) Aceto-orcein-nigrosine stain for salivary slides is made by mixing 1 ml of aceto-nigrosine and 9 ml of aceto-orcein stain. This must be filtered before each use!

4) Aceto-orcein-nigrosine stain for brain slides is made by mixing 4 ml of aceto-nigrosine and 6 ml of aceto-orcein stain. Must be filtered before each use!

The chief drawback of the stain is the tendency for the nigrosine to precipitate, which necessitates frequent filtering. A temporary squash preparation sealed with cover glass wax will improve with aging a few hours to several days, but should be made permanent if it is to be kept for a longer time. We use the dry ice technique for removing the cover glass and mount in euparal. The stain is permanent and does not fade.

The stain produces superb salivary gland slides, the fine bands on the chromosomes showing very distinctly.

Moyer, S. E., R. E. Comstock and L. H. Baker. Efficient procedures for culturing *Drosophila* in disposable paper containers.

Arnold (Amer. Biology Teacher 19:248-251 and DIS-32 p. 166) described a method for utilizing plastic-lined paper containers for culturing *Drosophila melanogaster*.

We have modified this basic plan to enable greater ease and speed in routine handling of large numbers of containers.

Covers for the containers are now available with a half moon clear plastic window. Ventilation holes, if needed, can be quickly punched with a "pin cushion" made from a rubber laboratory stopper and well spaced common pins.

The medium is anchored in the bottom of the container by a 4" x 4" non-sterilized 8 ply surgical gauze sponge stapled to the sides. This prevents the medium from falling on the flies while the container is inverted during anesthesia.

Anesthesia is accomplished by applying several drops of ether on a small cotton plug stapled into a 1/4 inch hole punched in the side of the container. Since this plug may be an attraction as a site for pupation, it may be desirable to locate it near the top of the container. Further diversion of the larvae from the plug may be achieved by limiting the amount of medium to slightly more than needed to cover the gauze on the bottom. In this way, larvae are attracted to the remaining gauze above the level of the medium. However, ether does not kill pupae occasionally lodged in plugs and possible physiological effects seem negligible.

Although anesthesia is slow with this system, waiting can be eliminated by initiating anesthetization well in advance of anticipated use of a particular culture. There is no danger of over-etherization to flies remaining in containers for an extended period of time prior to examination.

The chief advantage of using these paper containers is in the time and money saved. The cost of the unit and the time required for assembling it by the above procedures is considerably less than the time and cost of labor for washing and sterilizing bottles. Also, each of these containers supports a larger population for a longer period of time than does a half pint bottle. Finally, the containers can be stacked on top of each other for maximum utilization of storage space for *Drosophila* cultures.

von Borstel, R. C. and Margaret M. Fine. A medium suitable for hatchability and eclosion tests.

Since charcoal agar is a nuisance to mix and since it results in reduced viability of *Drosophila* larvae, another agent was sought that would give satisfactory contrast

to the *Drosophila* culture medium for hatchability testing.

The obvious choice was fruit juice and it was found that frozen grape juice concentrate works well. The flies cling to the medium with a tenacity that decries the change of name from ampelophila. Egg production and survival are high, and eclosion tests as well as hatchability tests are possible from the same vial.

The food formula used at Oak Ridge corresponds closely to that used at the California Institute of Technology (Lewis, DIS-34, 117, 1960). To prepare the hatchability medium, one 12-ounce can of frozen grape juice is stirred into every two liters of the *Drosophila* food and the mixture is then poured into vials. This food is just firm enough for egg hatchability testing, and it may be desirable to add more agar if some other food formula is used.

We presume that other fruit juice concentrates could be used as well, and appropriately dark wines could possibly be used, but local regulations are such that experimentation along these lines at Oak Ridge has been severely restricted.

PERSONAL AND LABORATORY NEWS

James Divelbiss is joining the staff of the Biology Department, Westmar College, Le Mars, Iowa in September 1961, and plans to establish a *Drosophila* research laboratory. Since library facilities in the area of genetics are limited he would appreciate receiving any available reprints, new or old.

Edward C. Keller, Jr., has moved from The Pennsylvania State University to The University of North Carolina at Chapel Hill in The School of Medicine, Department of Biochemistry and Nutrition.

Maxi E. Krawinkel, curator of stocks and head technician of the Purdue University *Drosophila* laboratory for the past five years, was killed in an automobile accident while on vacation in Michigan on January 22, 1961. She has been buried near her home in Bern, Switzerland. A memorial book collection bearing her name has been established in the Biology Library, Purdue University.

MATERIALS REQUESTED OR AVAILABLE

The inbred temperature lines of *D. melanogaster* derived from a wild population near State College, Pennsylvania, that are described in the stock list of University Park, Pennsylvania (DIS-34) will be maintained until further notice is given in D.I.S.

James Divelbiss is currently engaged in a pseudoallelic investigation of the brown locus. He would appreciate receiving stocks of any brown alleles except the following which he already has: bw^1 , bw^{81} , bw^{75} , bw^{59} , bw^{M58} , bw^{Mi59} and bw^{Am} . For his address see entry in Personal and Laboratory News.

ANNOUNCEMENTS

Drosophila melanogaster Stock Centers

The National Science Foundation is now supporting two stock centers for the maintenance of strains of *Drosophila melanogaster*. One is located at the California Institute of Technology, Pasadena, California and is under the direction of Professor E. B. Lewis while the other one is located in the Division of Chemotherapy of The Institute for Cancer Research, Philadelphia 11, Pennsylvania and is under the direction of Dr. I. I. Oster. The nucleus of both centers will consist of duplicates of the 800 basic stocks hitherto only maintained in Pasadena. These centers will serve

as a source of supply for virtually any research needs which might arise regardless of field of genetic interest, 2) provide insurance against loss of all or part of their respective collections by some unforeseen catastrophe, 3) allow the centers to make replacements of each other's stocks in the event any have broken down and 4) provide the "marker" and tester stocks and other combinations of mutations useful in research and teaching.

In addition to the 800 basic stocks, the Center in Philadelphia will maintain approximately 1600 other strains. These will include 600 stocks representing the major portion of the strains currently maintained by Professor H. J. Muller at Indiana University, 200 strains which had been maintained by Dr. J. Schultz in the Department of Genetics of The Institute for Cancer Research, and 800 strains consisting of other useful mutations, combinations of them, and multiple alleles of loci of unusual interest obtained from other laboratories or synthesized by the Center.

We would like to suggest that other workers should contribute useful stocks for inclusion in the collections. The main requisites for acceptance of such strains are that they be held in a combination not requiring selection, they represent new loci, alleles of unusual interest, improved balancers, etc., and they be free of mites. Stocks of mutants which overlap wild-type or of biochemical mutants which have no morphological effect should contain an Rk1 marker mutant to serve as a check on the possibility of contamination. In order to avoid the inclusion of too many stocks of questionable usefulness in the permanent collection, consultations with the Subcommittee on Drosophila Stocks will be held from time to time concerning the advisability of adding any of these newly contributed stocks or of eliminating old ones. Those who wish to contribute stocks should send a complete description of the stock to the Center ahead of time preferably on 3 x 5 cards. This will facilitate evaluation of the stock and provide the basic information needed for the stock records.

When a stock has been improved (for example, by introducing a more efficient balancer, or a more useful combination of mutants) the old stock in general will be discarded and replaced by the new one. However, no mutant type, balancer, nor any chromosomal rearrangement will be deliberately discontinued without notice being given in the Drosophila Information Service at least one year in advance.

Requests for stocks for research purposes will be filled as promptly as possible. As heretofore, there will be no charge for this service. It is requested that the receiver return the empty plastic vials in the original mailing carton.

Suggestions for improving the stockkeeping service are welcomed and can be addressed to either Center or to the Subcommittee on Drosophila Stocks.