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Prepared at the
**DEPARTMENT OF BIOLOGY
UNIVERSITY OF OREGON
EUGENE, OREGON**

D. melanogaster

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EDITOR'S COMMENTS

Once again the pressure of the ever-increasing number of contributions has made it necessary to take measures to keep the size within the limits imposed by the method of reproduction and the binding, and the funds available for this work. This has been done by postponing those stock lists which have not materially changed since they appeared in either DIS-34, or DIS-35.

It is my opinion that the usefulness of DIS might be increased if the scope of the notes were broadened somewhat to include categories which may be of considerable interest to Drosophila workers, but which have no other outlet at the present time. I have therefore included several notes which may be taken as illustrations of the sort of thing I have in mind. The note in this issue on inversions in D. pseudoobscura covers a theoretical point relevant to the entire argument concerning the distribution of inverted sequences among the chromosomes of that species, and others as well, which might be important to anyone planning to do experimental work along this line. A second note treats in detail a calculation made in 1936 by Sturtevant and Beadle which I think should be in the record somewhere. In DIS-34 I have a note which represents a complete and unequivocal retraction of a previous note and in DIS-35, one which extends and explains a regularly published paper of my own. Undoubtedly all Drosophila workers can think of similar uses to which DIS might be put, and their suggestions and contributions are welcome.

Of course this once again raises the thorny issue as to whether DIS is or is not a publication, one which the editor prefers to avoid but, when pressed, must answer in the negative. Many of the early objections to considering this a publication have now been removed: copies are generally available to anyone who wishes them; complete sets are on file in the libraries of a large number of universities, both U. S. and foreign, as well as in the Library of Congress, and in many instances the notes are referred to in publications just as casually as if DIS were a publication itself. On the other hand, there is no strictly organized reviewing system, proofs are not returned to the writer, and notes are not to be quoted without permission of the author. (Continued on page 179)

AMHERST, MASSACHUSETTS: AMHERST COLLEGE

Corrections and additions to list of Stocks in DIS 34:10.

1 Oregon-R: inbreeding, generation 370 on 6117
 8 Samarkand 204-55: from \$ #7, inbreeding, generation 55 on 6117
 33 lost
 51a ras dy
 51b ras² m/y f:=
 55a sn oc/y f:=
 86 change to: y g^{53d} sd/+. =
 86a y ras² f
 117a bv
 138 lost
 142a ci^D/ey^D
 145 change to Multiple Chromosomes--the se is se^{50k}
 145a +/y f:=;bw:e;spa^{pol}
 150a vg;se^{50k} e^{60k}
 162 lost
 164a sn⁴ oc ptg³/Fm1, y^{31d} sc⁸ wa lz^s B & FM1
 164b v m/FM1, y^{31d} sc⁸ wa lz^s B & FM1

BALTIMORE, MARYLAND: THE JOHNS HOPKINS UNIVERSITY

Note: # = to be selected

Wild StocksChromosome 1

b1	Amherst-34	#c1	br we ec rb t ⁴ / Ins (1) sc ⁸ In ⁴⁹ , B lz ^s wa y ^{31d}
b2	Canton-S	#c2	ec ct ⁶ (s) car/ CLB
b3	Crimea	c3	f B
b4	Florida (inbred)	c4	f/ y
b5	Formosa	c4a	g ^{53d}
b6	Kyoto, Japan	c5	lz ^{50.d} / y
b7	Lausanne-S	c6	sc cv dx v f
b8	Oregon-R150 (mass culture from 150th generation of sib. pair matings)	c7	sc t ² v f Tu car/ y f :=
b9	Salta, Argentina	c7a	v1 (suppressable)
b10	Seto, Japan	c8	w
b11	St. Louis-7 (bw)	c8a	wa
b12	Stephensville	c9	w m f (normal)
b13	Swedish-b	c9a	w m f (xxy)
b14	Tuscaloosa, Alabama	c9b	y ² cho ²
b15	Urbana-S	c9c	y ct f
b15a	Varese, Italy	c10	y ct ⁶ ras ² f
b16	Woodbury, New Jersey	c10a	y sn ³ v ^{36f} (v ^{36f} - unsuppressed)
		c10b	y ² su ^{51c15} ras ² v1 f (v ¹ - suppressed)

Duplicationc11 Dp(1) sc^{s1}, y w fInversions

c12 In (1) In⁴⁹, y fan
 c13 In (1) rst³
 c14 Ins (1) sc⁴ Ins sc^{s1}, y
 c16 In (1) sc⁸, B
 c17 Ins (1) sc^{s1} sc⁸, B w^a
 c18 Ins (1) sc^{s1} In⁴⁹, B f v y/ y f :=
 c20 In (1) y^{3P}, B

Lethals

#c21 car 1^(C1 + 1)/ Ins (1) sc^{s1} sc⁸,
 B w^a
 #c22 car 1^(B2 + 6)/ Ins (1) sc^{s1} sc⁸,
 B w^a
 #c23 car 1^(A3 + 3)/ Ins (1) sc^{s1} sc⁸,
 B w^a

Closed X's

c28 Xc1?, y f :=
 c29 Xc2/ y f :=
 c30 Xc2, y v
 c31 In (1) Xc2 wvc/ y w lz^{s9x}
 y w lz^s/ sc⁸ y ♂

Chromosome 2

d1 al b c sp²
 d2 al dp b pr cn c px sp/ Cy sp
 d2a al dp b pr cn c px sp/ Cy pr cn sp
 d2b al dp b pr cn vg c a px bw mr sp/
 S2 Cy lt³ pr⁺ Bl cn² L⁴ sp²
 d3 al Sp b L³⁴/ Cy
 d4 ap^(49j)/ Cy
 d4a b pr cn
 #d4b b Tft vg/ b vg
 d5 b vg
 d6 Bl L/ Cy
 d6a Bl L/ SM⁵, al² Cy lt^v sp²
 d7 bw (ex.49)
 d7a cn Su-Pm
 Cy cn vg Pm
 d7b cn Su-Pm Tac
 Pm (dp b c?)
 d7c c px sp
 d8 dp⁰²
 d9 L²
 d9a l (2) me
 d10 M^{60.29.29.}/ Cy
 d10a mi/ Pm²
 d11 net S ho/ Cy E-S
 d11a pys
 d11b Pfd/ Ins (2L,2R) Cy S²
 d11c px slt sp

d12 rn/ Cy
 d13 S Sp Bl L/ Cy cn² sp
 d14 stw² (ex.51.6.12a.3)/ Cy
 d14a Tac sp/ Cy sp
 d15 Tft/ Cy

Deficiencies

d16 Df (2) bw⁵/ Cy sp
 d17 Df (2) dp^{v51}/ Cy

Inversions

d18 In (2R) bw^A/ Cy
 d19 Ins (2L,2R) Cy bw^{V2}/ al dp b pr
 cn c px sp
 d20 In (2LR) bw^{V29}/ Cy
 d21 In (2LR) bw^{V30k1}/ Cy
 d22 In (2R) bw^{V30k10}/ Cy
 #d23 Ins (2L,2R) Cy bw^{V34}/ b vg
 d24 In (2) b bw^{VDe 1}/ b lt l cn mi sp
 d25 In (2) bw^{VDe 2}/ Rev l
 d26 In (2) bw^{V13}/ Cy

Chromosome 3

e1 bar-3
 e1a e11
 e2 Gl bx^D/ Inv LVM
 e2a Gl Sb/ LVM
 e3 Ly Sb/ Inv LVM
 e3a l (3) tr/ M⁸ Sb
 e4 M(3) y Gl/ Inv LVM
 e5 M(3) y Sb/ Inv LVM
 e6 p ss bx/ T(2;3) Xa
 e6a red
 e7 ru h th st cu sr e^s ca
 e8 Sb bx^D/ T(2;3) Xa
 e9 se
 e10 se e
 e11 stbrk
 e11a st(ex.b8)

Chromosome 4

#f1 Ce/ ci ey^R
 f2 spa
 f3 svⁿ

Multichromosomal

g1 b(Su-er⁺) bw; st er
 g1a Swedish b erupt
 g1b b(Su-er⁺) Pfd bw; st er
 g1c b(Su-er⁺) Tft bw; st er
 g1d b(Su-er⁺) bw; st er bx^D/st er Pr
 g1e cn bw; e
 g1f ct^{45e} v; bw; e; (ey²)⁺
 g2 Cy/ Pm ds^{33k}; H/ Sb-C
 g3 Cy pr cn/ Pm ds^{33k}; H/ Sb-C

g3a Cy/ Pm; st er Su-tu
 g3b Cy sp/ al dp b pr cn c px sp;
 ci ey^R
 #g3c Cy/ tu bw; st su-tu
 g4 dp; e
 g4a net; ru by
 g5 pr cn; by
 g6 pr cn; by; ci ey^R
 g6a SM1, al² Cy sp²/ Pm; Ubx/ Sb
 g7 Su-er tu bw; st er su-tu
 g8 tu-h su^{DIS23}
 g8a tu bw; er⁺ (su-tu)⁺
 g8c tu bw; Sb bx^D/ T(2;3) Xa
 g8d v¹/ y; Su-er bw; st er
 (v¹ - suppressable)
 g8e y sn v^{36f}/ y; Su-er bw; st er
 (v^{36f} - unsuppressed)
 g9 y; bw; e; ci ey^R
 g9a v; bw; e
 g9b y² v f; bw

Aberrations

#g10 v; In(2R) bw^{V2}/ v; +
 g11 Ins (1) scs1 sc8 B wa; T(2;3) Xa

#g11a T(y;2) J/ px bw sp
 g11b T(y;2;3) F; st/ ri p^P
 #g12 T(2;3) bw^{V5} st/ st
 g13 T(2;3) bw^{V5} st/ T(2;3) p^{Gr} st
 g14 T(2;3) bw^{VDe4}/ Cy
 g15 T(2;3) Me/ ru h th st cu sr e^s
 Pr ca
 g16 T(2;3) p^{Gr}/ Cy
 g17 T(2;3) rn/ Cy sp
 g18 T(2;3;4) bw^{V30k18} Ins (2LR)/
 Cy
 #g19 T(2;3) Cy/ pr cn; by
 #g20 T(2;3)G5 Sp (L³⁴)⁺/ pr cn; by
 #g21 T(2;3)G5 Sp (L³⁴) D1 (Pr)⁺/
 pr cn; by
 #g23 T(2;3) Sp; D1 Pr/ pr cn; by
 #g24 T(2;4)/ pr cn; ci ey^R

Tumor Stocks

tu A₂ cito-pl-st
 tu B₃ (Italy)
 tu-55G^{Jacobs} (on 2R)
 g8a see Multichromosomal listing

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Zoology

Stocks listed in DIS 35 with the following numbers are no longer kept in culture:
 11, 13, 36, 37, 41, 44, 46, 49, 50, and 64.

DeKALB, ILLINOIS: NORTHERN ILLINOIS UNIVERSITY
Department of Biological Sciences

In addition to that listed in DIS 34.

Wild Stock

Oregon R.

Chromosome 1

Ins (1) sc^{S1L}, S. sc^{8R}, wa^a B
 Ins (1) sc^{S1L}, S, sc^{8R}, wa^a B/yf:=
 sc cv v f
 sc⁸ Y(y+)/yB & yf:=
 y

Chromosome 2

sf²
 tu 50^j

bw tu
 cn bw
 ar dp b pr px sp

Chromosome 3

tx
 ru h th st p^P cu sr e^s

Multichromosomal

Cy al² sp²/Pm; Ubx 130/ Sb
 Cy/Pm ds 3^{3K}; H/In^{3R} mo sr
 y f:=; bw; e; ci y^R
 bw; st
 y sc ^{SI} In 49 sc⁸; bw; st p^P

LE MARS, IOWA: WESTMAR COLLEGE
Department of Biology

Wild Stocks

a-1 Oregon-R

e-3 se
e-4 stChromosome 1 (X)

b-1 f
b-2 In(1)dl-49, y w
b-3 Ins(1)sc⁸, dl-49, sc⁸ v f/y f:=
b-4 sc ec cv ptg³ v/y v f car:=
b-5 w
b-6 y sc cv v f car/y f:=

Altered Y Chromosomes

c-1 YB^S (BS YL•bb⁺Y^S)/y v ♂ YB^S
(BS YL•bb⁺Y^S)/y f:=
c-2 Ybw⁺ (YL bw⁺•bb⁺Y^S)/y v; bw ♂
y v; bw ♀

Chromosome 2

d-1 b cn c bw
d-2 bw
d-3 bw⁸¹
d-4 bw^{Am}
d-5 bw^{M58}
d-6 bw^{Mi59}
d-7 bw⁷⁵
d-8 bw⁵⁹
d-9 cn su-Pm/SM1, al² Cy sp²
d-10 Df(2) bw⁵ sp²/Xa
d-11 Ins(2L+2R)Cy, bw^{45a} sp²
or^{45a}/Bl
d-12 px
d-13 px bw sp
d-14 sp
d-15 vg
d-16 vg^U/Ins(2L+2R)Roi, bw^{45a} sp²
or^{45a}

Chromosome 3e-1 e
e-2 ry²Multichromosomal

f-1 y:=/Y; bw; In(3LR)Ubx¹³⁰, Ubx¹³⁰
e^S/st (1;2;3)
f-2 y:=/Y; bw⁸¹; In(3LR)Ubx¹³⁰,
Ubx¹³⁰ es/st (1;2;3)
f-3 y:=/Y, bw⁷⁵; In(3LR)Ubx¹³⁰,
Ubx¹³⁰ es/st (1;2;3)
f-4 y; bw; e; ci ey^R (1;2;3;4)
f-5 y:=/Y; In(3LR)Ubx¹³⁰, Ubx¹³⁰ es/
In(3R)Vno, Vno (1;3)
f-6 bw; st (2;3)
f-7 bw⁸¹; st (2;3)
f-8 bw^{Am}; st (2;3)
f-9 bw^{M58}; st (2;3)
f-10 bw^{Mi59}; st (2;3)
f-11 bw⁷⁵; st (2;3)
f-12 bw⁵⁹; st (2;3)
f-14 bw⁵⁹; In(3LR)Ubx¹³⁰, Ubx¹³⁰ e^S/st (2;3)
f-15 In(2L)Cy, Cy px bw sp/b⁵⁵; st
(2;3)
f-16 In(2L)Cy, Cy px bw⁸¹ sp/b⁵⁵; st
(2;3)
f-17 In(2L)Cy, Cy px bw⁷⁵ sp/b⁵⁵; st
(2;3)
f-18 In(2L)Cy, Cy px bw⁵⁹ sp/b⁵⁵; st
(2;3)
f-19 In(2L)Cy, Cy/b⁵⁵; st (2;3)
f-20 Ins(2L+2R)SM1, al² Cy sp²/Bl;
In(3LR)Ubx¹³⁰, Ubx¹³⁰ e^S/
In(3R)Vno, Vno (2;3)
f-21 px bw sp; st (2;3)
f-22 px bw⁸¹ sp; st (2;3)
f-23 px bw⁷⁵ sp; st (2;3)
f-24 px bw⁵⁹ sp; st (2;3)
f-25 px sp; st (2;3)
f-26 px; st (2;3)
f-27 sp; st (2;3)
f-28 vg; e (2;3)

NEW HAVEN, CONNECTICUT: YALE UNIVERSITY
Department of Zoology

Stock list remains essentially as in DIS-34-14 except for the following corrections and additions:

58 y² wa cv sn^{55a} v +f /M-5
60 wa fw^{49c}/M-5
114 sc^{S1} In S wa sc⁸; In SM1, al Cy sp²/
dp h Pm ds^{33k}; C Sb/Ubx¹³⁰ e^S (H-40)

136 Df(1) w²⁵⁸⁻²¹, see No. 153
154 tra/In(3LR) Ubx¹³⁰ (FMA3/wa v)
155 y² wa m f

PASADENA, CALIFORNIA: CALIFORNIA INSTITUTE OF TECHNOLOGY

Note: The following is a list of additions, losses, and corrections to the list of stocks from this laboratory in DIS 34. The convention for listing new stocks can be illustrated by an example; the new stock, w sn³ m, is given the number 143b and should be inserted after 143 in the Pasadena DIS 34 stock list. Some minor typographical errors in the DIS 34 list will be corrected the next time the full stock list is reprinted.

Stock Additions to DIS 34 list:Chromosome 1

6b. amx lz^s v/ y f : =
 31b. ec ct⁶ s car/ FM6, y^{31d}sc⁸dm B
 60b. lz³⁶ / y f : =
 110b. sn³ lz^{y4} v/ y f : =
 135b. v f Bx^{r49k} car/ y f : =
 143b. w sn³ m

Chromosome 2

200b. al S ast ho/ SM1, al²Cy sp²
 200c. alpha-1 (pP)
 319b. lt std/ SM2, al²Cy lt^vsp²
 346b. pd ll
 381b. SD-5/ SM5, al²Cy²lt^v sp²
 381c. SD-72 / SM5, al² Cy lt^v sp²

Multichromosomal Stocks

641b. b (Su-er⁺) bw; st er (2;3)
 643b. cn; ry²
 647b. Su-er tu bw; st er su-tu (2;3)

Attached-X

652b. y pn / FM6, y^{31d} sc⁸ dm B

Closed-Y

659b. Y^c, bw / X⁺; bw (fb "MYR")

Inversions-X

720b. In(1) dl-49, y Su-Hw Hw m²
 g⁴ / y f w : =
 721b. Ins(1) dl-49, BM1, y sc v
 cu-x BM1

Stock Losses:Chromosome 1

20 Bx^{r49k}/ y f : = (replaced by
 135b)
 33 ec dx/ dl-49, y Su-Hw Hw m² g⁴
 (replaced by 32 and 720b)

Chromosome 2

200 al S ast ho/ Cy, En-S
 (replaced by 200b)
 327 M(2)p/ Cy, al² lt³ l⁴ sp²
 394 Sp J/ In(2L) Cy-t, Su-S dp² pr
 (replaced by 395 and 274)

Chromosome 3

550 ry² (replaced by 643b)

Corrections to DIS 34 list:

For:

653 y² su-w^a bb
 718 dl-49, ty-2 bb¹
 749+ Ins(2L+2R)Cy, (2R)bw^{v34}
 (314, 315, etc.)
 749++ Ins(2L)Cy + (2R)NS (333)
 759+ with st l(3)W ca in)

Read:

y² su-w^a w^a bb
 dl-49, ty-1 bb¹
 Ins(2L+2R)Cy, (2R)bw^{v34} (333)
 Ins(2L)Cy + (2R)NS (345)
 with st l(3)W ca (in 578)

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

Note: Only unusual stocks are listed.

<u>Wild-type</u>	8	ci gvl ey ^R sv ⁿ	22	1 (4) 10k/ci ^D pol
	9	ci ⁺ 3	23	1 (4) 14o/ci ^D
1 Salt Lake City	10	ci ⁺ 4	24	1 (4) 25z/ey ^D
2 Solway	11	ci ⁺ 5	25	spa
	12	ci ^D /ey ^D	26	spa ^{pol}
<u>Chromosome 1</u>	13	ci ^D /spaCat		
	14	ey ^D /Scn	<u>Multichromosomal</u>	
3 lix	15	1 (4) PT-1/ey ^D	27	pr; Mal
	16	1 (4) PT-2/ey ^D	28	y; bw; e; ci ey ^R
<u>Chromosome 4</u>	17	1 (4) PT-3/ey ^D		
	18	1 (4) 4d/ci ^D	<u>Deficiencies</u>	
4 ar/ey ^D	19	1 (4) 5e/ci ^D	29	D _f (4) M-4/ey ^D
4 bt	20	1 (4) 6f/ey ^D		
6 bt ^D /ci ^D	21	1 (4) 7g/ey ^D		
7 Ce ² /spaCat				

URBANA, ILLINOIS: UNIVERSITY OF ILLINOIS
Department of Psychology

Behavioral stocks.

1. Positive geotaxis) over 120 generations of selection for performance in mass
2. Negative geotaxis) screening maze
3. Also other stocks used in geotaxis experiments on reversed and relaxed selection as well as the foundation population.

AUSTRALIA

Adelaide, South Australia: University of Adelaide, Department of Genetics

<u>Wild</u>	19.	y/lz ^{57j}	34.	Ly/D ³
	20.	Xc ² /scS ¹	35.	ss
1. Canton S	21.	X.y ^S /y ^{LC}		
<u>Chromosome 1</u>	<u>Chromosome 2</u>		<u>Chromosome 4</u>	
2. B	22.	al	36.	ci ey ^R
3. sd	23.	al dp b pr c px	37.	ey ²
4. car		sp/Cy pr	<u>Multichromosomal</u>	
5. ct v f	24.	cn	38.	bw; st
6. g ²	25.	b j	39.	v; bw
7. Muller-5	26.	bw	40.	y; Cy/Pm, ds ^{33k} ; H/Sb
8. rb cx	27.	dp	41.	y w; dp
9. sc cv v f	28.	fj wt/Xa	42.	e; bw
10. v	29.	ho	43.	e; vg
12. w	30.	vg	44.	e; dp
13. wa ^{55b}	31.	b vg		
14. wsat	<u>Chromosome 3</u>			
15. w m f	32.	ca		
16. y	33.	e ⁴ wo ro		
17. y w spl				
18. y w ^a sc ec				

Hobart, Tasmania: University of Tasmania, Department of ZoologyWild Stocks203 S / Cy L⁴Canton-S

204 fj wt / Xa T 2:3

205 Cy pr / al dp b pr px sp

206 b cn c bw

Several strains from different places
in Tasmania.Chromosome 3Chromosome 1301 Ly / D³302 ru h th st cu sr e^s ca (ru-cu-ca)101 ct v f⁵102 y / lz^{57j}103 Basc^{54j}/od^{54j}104 sc^{S1} In-S B apr sc⁸ (Basc)105 CLB / y² apr ec cv ct v f

106 y / B

107 Xc² / sc^{S1}108 Xc² y v f109 Xc² v f / y

110 y v f / w

111 B

112 yw / y / sc^{8.Y}113 sc^{S1} In-S apr sc⁸

114 y apr

115 w^{61g}116 y apr / sc^{8.YB-S}Chromosome 4

401 ci

402 ey²Multichromosomal

501 bw ; st

502 vg ; se

503 v ; e⁴ ro

504 y ; cn bw

505 y apr ; bw ; st

506 y v / sc^{8.YB-S} ; bw

507 y ; bw ; st

Chromosome 2

201 cn bw

202 Cy L / Pm ds^{33k}Special601 X.Y^S / Y^{Lc}602 X.Y^S / Y^{Lc} ; bw ; stSydney, New South Wales: Sydney University, CSIRO Animal Genetics Laboratory

Three stocks of sc are held which may be of interest -

1. homozygous for sc w , average scutellar bristle number of something over 4.
2. homozygous for sc w^{bl} , has a scutellar bristle number of 2, this number having been rendered rather invariable by selection.
3. $\frac{sc\ w}{+ w^{bl}}$ x sc w not balanced. In this stock the mean scutellar bristle number of + males is about 7 and ++ females about 8 1/2.

These stocks are referred to as follows: sc w High, sc w^{bl} LV , $\frac{sc\ w}{+ w^{bl}}$ High

For more complete list, see DIS 34.

BRAZIL

Pôrto Alegre: Universidade do Rio Grande do Sul, Departamento de Genética, Instituto de Ciencias Naturais

Chromosome I

yellow vermillion

miniature

scute - crossveinless - vermillion - forked

forked

carnation
vermillion
white
honey
blood
eosin
prune
carmine

Chromosome II

clot
purple
cinnabar

vestigial
vestigial - scarlet
Lobe
brown
lightoid - ltd
purpleoid
St. bw (int).

Chromosome III

sepia; ebony; scarlet; pink.
D. paulistorum: yellow S. Pe 12
D. insularis Guadalupe: ebony

CANADAToronto: University of Toronto

The list published in DIS 35 remains the same, with the following additions:

Chromosome I

B
car
g
m g f
w
y w
y v f
y
W^a
y w m
y v f car
Basc

Chromosome II

bw
b vg
c
cp
px
vg

Chromosome III

bar⁻³
ss
es

sr
st
se h

Chromosome IV

ey²

Multichromosomal

w,e
w, e, pol
Cy, e
bw,e
B w^a bw pol
al dp b pr Bl c px sp/SM al₂ Cy sp²

Inversions

In (1)y⁴ y⁴

In II CQ

Cy

Translocations

T (2,3,) Xa/1(3) Xa R
ri p^P/st T (y,2,3)F

Vancouver, British Columbia: The University of British Columbia, Department
of Biology and Botany

Wild Stocks

1 Urbana-S

Chromosome 1

2 B
3 lz/ClB

4 m
5 w

<u>Chromosome 2</u>	<u>Chromosome 3</u>	<u>Attached X's</u>
6 b pr c px sp	11 e	13 <u>y</u> and w
7 bw		
8 dp	<u>Chromosome 4</u>	<u>Multichromosomal</u>
9 L/Cy		
10 vg	12 ey	14 Cy/Pm; Sb/D

COLOMBIABogotá: Universidad de Los Andes, Departamento de GenéticaWild Stocks

- 1) Sao Paulo
- 2) Oregon-K
- 3) Pacific
- 4) Canton-S
- 5) Oregon-R
- 6) Pavia
- 7) Varese
- 8) Anzyo-Aichi
- 9) Canton Special
- 10) Chausuyama-Aichi
- 11) Hachijijima
- 12) Hikosan-Kyushu
- 13) Hiroshima
- 14) Hita-Kyushu
- 15) Omogo-Shikoku
- 16) Oregon
- 17) Shioya-Hokkaido
- 18) Suzuka-Mie
- 19) Takagicho-Tokyo
- 20) Yonekawa-Yamaguchi
- 21) African Strains

Oregon V (177 generations)
 Samarkand (432 generations)

MutantsChromosome 1

Muller 5
 B
 W
 W^a
 v,
 Yellow body

Chromosome 2

Cy L/Pm (Inbred)
 Cy/L²
 Cy Sp/Pm
 Brown eyes
 Dumpy Wing
 b, pr

Chromosome 3

Curled Wing
 Ebony body
 Sepia eyes

Selection Stock

Lobe (Artificial selection of 1 eye)

Inbred Lines

Edinburgh
 Oregon-R (320 generations)
 b pr (77 generations)
 Lobe la. (3 generations)

Multichromosomal

L⁴ Cy Sp/Pm

FINLANDHelsinki: University of Helsinki, Institute of GeneticsWild Stocks

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

- 5 Porvoo
- 6 Swedish-b

Chromosome 1

- 7 B
- 8 bi ct⁶ g²

9 f
 10 fu/CLB
 11 g² ty & y
 12 In(1)dl-49, y faⁿ
 13 In(1)rst³, rst³
 14 In(1)sc⁴, y sc⁴
 15 In(1)w^{m4}
 16 Ins(1)sc^{S1L}, S, sc^{8R}, sc^{S1} w^a B
 (Muller-5)
 17 lz/FM3, y^{31d} sc⁸ dm B 1
 18 ras²
 19 rb ex
 20 s
 21 sc cv v f
 22 sd; (se)
 23 sn³
 24 spl
 25 w
 26 w^e sn/CLB
 27 wch wy
 28 Xc² f B & y
 29 y ac v
 30 y sn³ bb
 31 y v f
 32 z

Chromosome Y

33 f.YS/YL
 34 X.YL/YS (Neuhaus)
 35 In(1)w^{m4} and extra Y
 36 In(1)w^{m4}; rl and extra Y

Chromosome 2

37 al dp b pr c px sp
 38 al² Cy, InL lt³/b pr Bl lt³ cn²
 InCyR L⁴ sp²
 39 Bl L²/Cy
 40 bw
 41 cn² InCyR cg sp²/InsNS px sp
 42 D³/Payne
 43 dp^T ab² pr Bl rn NSR mr/al² Cy
 cn² L⁴ sp²
 44 dp^{tx} Sp cn²/S² Cy cn² (homoz. InCyR)
 45 fj
 46 fj px
 47 In(2L)Cy, al² ast³ b pr (Cy not
 present)
 48 Ns, b mr/Cy
 49 rl
 50 rn/Cy
 51 rn/Cy cn² sp²

52 rn/Cy Bl cn² L⁴ sp²
 53 rn In(2R)M/Cy cn² sp²
 54 stw
 55 vg

Chromosome 3

56 Bd^G/In(3R)C, l(3)a
 57 D³ Sb/InLP Dfd InRP ca
 58 e
 59 e¹¹
 60 Gl Sb/LVM
 61 In(3R)Dl^B, st DlB/In(3R)P^W, st
 l(3)W^{ca}
 62 In(3R)P^{FLA} (homozygous)
 63 Ly Sb/LVM
 64 Me, InL Sb/ru h D InsCXF
 65 R Ly/In(3L)P, gm
 66 se
 67 se app
 68 se rt² th/Me, InL
 69 tra/Me, T23
 70 W Sb/InsCXF

Chromosome 4

71 ci
 72 ci^W
 73 ey
 74 spa
 75 svⁿ

Multichromosomal

76 Cy/Pm; D/Sb
 77 vg; e
 78 w^{m4}; Cy/ap⁴ vg
 79 w^{m4}; Cy/blt

Deficiencies

80 Df(2)MS-4/SML, al² Cy sp²
 81 Df(2)MS-8/SML, al² Cy sp²
 82 Df(2)MS-10/SML, al² Cy sp²
 83 Df(2)rl^{10a} lt cn/Cy
 84 Df(2)rl^{10a} lt cn/Pm ds^{33k}

Translocations

85 T(Y;2)B/b c
 86 T(Y;2)C/cn³
 87 T(2;3)rn/Cy sp
 88 T(2;3)Xa/Sb Ubx

FRANCE

Lyon: Rhône Laboratoire de Zoologie expérimentale, Faculté des Sciences,
16, quai Claude Bernard

Wild Strains

Oregon R.
 Lyon
 Champetières (inbred)
 Algérie

Gif--sur--Yvette (Seine et Oise): Centre National de la Recherche scientifique,
Laboratoire de Génétique formelle

Chromosome 1

1 - B
 2 - car bb
 3 - car od f
 4 - cv
 5 - cv v f
 6 - rb g
 7 - v
 8 - v f
 9 - Base: sc^{S1B} In S wa^{sc8}
 10 - X^{c2t2}; y f : =
 11 - y f
 12 - y f : = ; y Z w^e
 13 - y v m f dl-49
 14 - y w
 15 - y w spl sn³
 16 - y wa^a cv v f
 17 - y wa^a spl rb
 18 - y²su wa^a bb / dl-49 y w lz / Y B S
 19 - ♂ y³YL / y² oc lz Y^S;
 ♀ y³YL / y ac sc pn co rb cm ct⁶
 sn³ oc ras v m g² f car / sc^{S1} B
 In 49 lz⁵ sc⁸
 20 - Y: y bw⁺ / y v bw
 21 - Y^S · X In E N v y · Y^L st⁸ y⁺
 22 - wa^a
 23 - wa^aB / wa^aB⁺ : = ♀ ; ♂ B
 24 - w^e

Chromosome 2

25 - al
 26 - al dp b pr
 27 - al dp b pr c px sp
 28 - Bl L / Cy
 29 - b pr
 30 - bw

31 - cn
 32 - dp
 33 - dp bw
 34 - j³4e3
 35 - L
 36 - Pm / Cy
 37 - Tft / Cy

Chromosome 3

38 - C³G
 39 - D C X F / Dfd
 40 - ri p^p
 41 - st
 42 - th st cp

Chromosome 4

43 - ey²
 44 - ey^{DciD}
 45 - ey^{Rci}

Multichromosomal

46 - C³G ; cn b
 47 - Cy / Pm ; H / Sb
 47 - cv ; e
 49 - cv v f ; e
 50 - sc^{S1B} InS wa^a sc⁸ ; Cy / Pm ; H / Sb
 51 - ♂ sc Y^L / sc w B Y^S } Cy In / S Sp
 ♀ y f : = } ab²1td
 52 - Sifter : S Sp-T (2-3) P-. In S D
 X F / SM1. al²Cy ; D1 H e pⁱ
 53 - T (2-3) E / Cy- R
 54 - tu ; e
 55 - tu ; w
 56 - v f ; e

GERMANY

Berlin-Buch: Deutsche Akademie der Wissenschaften zu Berlin
Institut für Experimentelle Krebsforschung, Genetische
Abteilung, Lindener Weg 70

Wild Stocks

- 1 normal (Berlin wild)
 2 normal (England)

Chromosome 1 (X)

- 3 w
 4 w sn³
 5 wbf
 6 w^a
 7 w^e
 8 w^{co} sn²
 9 wch wy
 10 w^{m4}
 11 gt w^a
 12 y
 13 y³⁰³
 14 y w
 15 y pn
 16 y cv v f
 17 y w bb
 18 y fa wy² g²
 19 f
 20 sc
 21 sc rb cv
 22 sc ec ct
 23 spl
 24 m
 25 B
 26 car bb Y; bb
 27 v
 28 cv
 29 car
 30 faⁿ
 31 ct

32 fu^{ff}/ClB

33 yy/+

34 yy/w^e35 yy/x^c

36 y w f/+

37 y w f/B

38 +/ClB

39 v/ClB

40 y w/ClB

41 w^e bb¹/ClB42 sc ec cv ct⁶ v s² fcar bb¹/ClB43 l⁷/dl-49, y^{Hw} w^{lz}44 sc^{S1} In S w^a sc⁸45 sc^{S1B} In S w^a sc⁸ = M-5

46 sc ec ct v g f

47 sc ec ct v g

Chromosome 2

48 j

49 bw

50 bw^{pp}

51 b cn vg

52 L²/Cy53 S Sp ab² ltd/NS px Sp

54 b pr vg a sp

55 vg

56 bw cn

57 al dp

Chromosome 358 e¹¹

59 st

60 p^p61 Dfd^{r-L}62 ru h st Dfd p^p ss e^s

63 ri

64 ss^a

65 jv se

Chromosome 466 ey²67 ci ey^LMultichromosomal68 Bld w^a/w; Cy69 e¹¹; vg70 w; e¹¹

71 cn; ss

72 v; bw

73 w; ss

74 y^{si} sc⁸ In S y^{3P}; al² Cy
 lt³ sp²/dp b Pm; ru h
 D³ In CXFa Sb In (3R)Tumor Stocks75 sc e¹¹ tu 49h76 tu^gRingchromosome77 sc⁸. Y/y B ♂

y f : ♀

78 X^{C2}, yv ♂

y f : ♀

79 y In 49 v f ♂

y f : ♀

Berlin-Buch: Institut für Medizin und Biologie, Genetische Abteilung
 and

Berlin-Dahlem: Institut für Genetik der Freien Universität Berlin

Note: Correction to the list in DIS-34 (pp. 32-33).

Discarded or lost:

80 Cy al² lt³ L⁴ sp²/+;
 C M¹ Sb C/+

81 fj px sp; p^p/C M¹ Sb C
 93 Df(4)M4/ey^D

Hamburg, Von-Melle-Park 10: Zoologisches Staatsinstitut und Zoologisches MuseumWild Stocks

1 Oregon-S

Chromosome 410 ey²Chromosome 1

2 w

3 y/f

Multichromosomal11 y; ey²12 bw; ey²13 bw;ss;ey²Chromosome 2

4 bw

5 dp b

6 Pm L Cy

Triploid14 y² sc w^a ec/FM4, y^{31d} sc⁸ dmBChromosome 3

7 cu

8 ss

9 Sb/H Payne

Hamburg-Eppendorf: Universitäts-Frauenklinik, Strahlenbiologische AbteilungWild Stocks

1 normal (Berlin wild)

y sc^{S1} In⁴⁹ sc⁸/ bw/ st p^p
B/yChromosome 1 (X)

2 CLB/+

3 sc^{S1} B InS w^a sc⁸4 sc⁸ Y/y f x sc⁸ Y/Xc2 y v

5 w

6 X^c/CLBMultichromosomal

7 cn; ss

Attached-X

8 y

Mariensee: Max-Planck-Institut für Tierzucht und TierernährungWild Stocks

Berlin

Oregon-R

Canton-S

wch
w^{co}sn²
w^{col}
w^h
w^{sat}
w^t
fChromosome 1+3v; ry²Chromosome 1

B

lz^{37h}lz³

v

ma-l

ma-l^{bz}

ma-l/y f:

w

w^aw^{bf}w^{Bwx}Chromosome 2cl
S/Cy, E-S
po²
cn
bwChromosome 1+2

ma-l; cn

Chromosome 3gl
gl³
ru
ro
st
Dfd^r-L
mah
ma
ca
ry¹
ry²
rb
cu, kar

	<u>Chromosome 2+3</u>	<u>Chromosome 4</u>
se		
ri		
ss	cn; ry ²	ey
L/D	(Sb; Ubx ¹³⁰)/Xa	ey ^R
red	(Cy SM1; Ubx ¹³⁰)Xa	

Münster/Westf.: Institut für Humangenetik der Universität Münster

Wild Stocks

a1 + Crimea
a2 + Oregon R

Chromosome 1 (X)

b1 In⁴⁹ sn^{x2} & y f:=
b2 ras⁴ m/Clb
b3 v
b4 w
b5 y² wa sn⁵ B & y
Aa f:=

Combinations of scute or similar
inversions

d1 ("Binsc") sc^{S1} B In⁴⁹ sc⁸ & y f:=
d2 ("new Binscy") y sc^{S1} B In⁴⁹ sc⁸

Altered Y's sometimes with mutants
in X

f1 sc⁸.Y/y^{S1} sc⁸ B f In⁴⁹ v
f2 sc⁸.Y.B^S/y² wⁱ ct⁶ & y f:=

Sterilizer ("sz") Stocks

f3 ("sz +") Y^{Lc}/X.Y^S
f4 ("sz bw") Y^{Lc}/X.Y^S; bw
f5 ("sz c") Y^{Lc}/X.Y^S & y v f.=; c
f6 ("sz e") Y^{Lc}/X.Y^S & y v f.=; e

Chromosome 2

g1 bri
g2 bw (iso 2, 1959)
g3 bw^D

g4 c
g5 c bw
g6 cn (iso 2)
g7 S Sp Bl L^{rm} bw^D/dp^{txI} Cy, InsO
pr cn²
g8 vg (iso 2,3)
g9 vg bw
g10 vg^{-D}/SM5, al² Cy lt^v sp²

Chromosome 3

h1 Df(3) sbd¹⁰⁵/Xa
h2 e¹¹
h3 red (Malpighians)
h4 ri p^P
h5 se
h6 st

Multiple Chromosomes

X,3

j1 wa ♂ & y v f.=♀; tra/D InsCXF
j2 ct-t²; ctⁱp^P ♂ & sc ctⁿ oc ptg
car v ct¹, In In⁴⁹ snx² . =♀
j3 ct-t²; ctⁱ/rip^P ♂ & y f.=; rip^P ♀

2,3

j4 bw; e

X,2,3

j5 y sc^{S1} In⁴⁹ sc⁸; bw; st p^P
(to cross by sc⁸.Y/y B
for losses,
l's & T's)

Tübingen: Max Planck-Institut für Biologie

Wild Stocks

1 Sevelen (Zürich)
2 Berlin (Marburg)
3 Oregon R

Chromosome 1

4 B
5 Df(1)bb In(1)bb⁻, y sl² bb⁻/FM4,
y^{31d} sc⁸ dm B (Extra Y's)

5a Df(1)bb In(1)bb⁻, y sl² bb⁻/ FM4,
y^{31d} sc⁸ dm B */sc⁸.Y
(* FM4-chromosome recessive lethal)

6 Df(1)bb In(1)bb⁻, y sl² bb⁻ sc⁸
bb w^a

7 e
8 In(1)w^{m4}, w^{m4}/ybb⁻
9 sc^{S1} B InS w^a sc⁸ (Muller 5)
10 v/ybb

11 w
12 w/y
13 w^e bb^l/w^e bb^l; yst, bb + w^e bb^l/Y
(extra Y's)

14 w^e bb^lsc⁸ bb w

15 y v f

16 y w bb

17 y² w^a sc ec / y

18 y w (lz)/In(1)X^{C2}, w^{vc} f

19 y bb^l 3a/sc⁸.Y + RM, y w

Multichromosomal Stocks

20 (X,Y,3) sc⁸.Y InEN y; ru h D
InsCxF /ru tra p

Attached-X

* RM, y w (19)

21 sc v f /Y + f.Y^S

22 Y

* Y/w (12)

* Y/y²w^asc ec . (17)

23 y f/Y^S + g² B.Y^L/Y^S

24 y² su-w^a w^a bb/ v f B, X.Y

25 y² su-w^a w^a bb/ sc⁸.Y/X^{C2}, y v

Attached-X.Y

* f.Y^S (21)

* g² B.Y^L ... (23)

* v f B.Y ... (24)

26 X.Y^L (A-2), y w.Y^L/Y^S.Y^S

Closed X

* In(1)X^{C2}, w^{vc} f (18)

* X^{C2}, y v (25)

Triploid

27 y w/Basc

Deficiencies

* Df(1)bb (5, 6)

* Df(Y)bb (8)

* Df(Y)yst (13)

Altered Y's

* ybb (10)

* ybb⁻ (8)

* yst (13)

* sc⁸.Y (19, 25)

* Y^S (23)

* Y^S.Y^S (26)

GREAT BRITAIN

Cambridge: University of Cambridge, Department of Genetics, Milton Road

Note: Only stocks not generally available in this country are listed.

Chromosome 1

1. flp
2. ptg²
3. ras²
4. y.f.car

Chromosome 2

5. al.dp.b.pr.cn.px.sp.
6. CyL⁴/d.b.
7. CyL⁴/Sp.

Chromosome 3

8. by.cu.
9. cu.kar.
10. h.ri.
11. Mé/Sb.
12. th.ri.kar.Sb.
13. th.st.cp.Sb.
14. ve.h.eyg.cp.

INDIA

Hyderabad: Osmania University, Radiation Genetics Project
aided by Department of Atomic Energy
(Government of India)

Wild Stocks

1. Oregon-K
2. Madras

11. b cn
12. cn bw
13. dp bw

Chromosome 3Chromosome 1

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

1. Gl Sb/D
2. st

Chromosome 4Chromosome 2

1. Cy/ Bl L^2
2. dp b cn bw
3. Cy Bl L^2 / LVM
4. dp
5. bw
6. cn
7. b
8. dp b cn
9. dp b cn
10. dp b bw

1. ey

Multichromosomal

1. y sc^{S1} In 49 sc^8 ; dp b cn bw -
 0.1. dp b cn bw X:II
2. y sc^{S1} In 49 sc^8 ; Cy Bl L^2 -
 0.1. Cy Bl L^2 X; II
3. y sc^{S1} In 49 sc^8 ; bw st
 0.1 bw st X:II:III
4. bw st II&III
5. yy bw e ey X:II:III:IV:

ISRAEL

Jerusalem: Hebrew University of Jerusalem

Wild Stocks

- Berlin
 Canton - S
 2 wild strains from various parts of Israel
 4 isogenic strains derived from wild populations in Israel

- lz/C1B
 lz^A & y f:=
 m
 pn²
 rb cx v
 sn
 sc t²v f & y f:= (Bloomington)
 spl cm ct⁶
 v
 v g f
 v ras
 w
 w^a
 w^av
 w^e
 w^m-4000 (Pavia)
 w^m-6000 (Pavia)
 w^m4
 X.Y^L/Y^S (Birmingham)
 X.Y^L/Y^S (Neuhaus)

Chromosome 1

- Basc (Muller -5)
 B
 BB
 B/y
 f
 fB
 f.Y^S/Y^L (Finland)
 g²
 Hw^{49c}/Basc

y^L/f.Y^S & sc v f (Bloomington)
y^{56k}

y ac_{sc} pn sn (Stockholm)
y ct⁰/B^{SY}.sc⁸ & yf:= (Oster)
y t² y f (Bloomington)

y sc^{S1B} In49 sc⁸ & yf:= ("Binscy"-Bloomington) Chromosome 3
y sc^{S1B} In49 ctⁿ⁵ sc⁸ ("Binscty"-Bloomington)

y w
y w sn/y
y w^a v f
y w^a spl & y f:=
y w^a spl m & y f:=
y

Chromosome 2

b cn
b lt bw
b pr vg
Bl L/Cy
bw
bw D (Hinton)

cl
cn bw
Cy/Pm
Cy L/Pm
dp (Texas)
dp^T Sp cn bw sp/S²(ls⁺) Cy, InL cn bw sp
(Bloomington)

dp^T Sp cn In NSR mr/S² ls Cy pr Bl cn²
I⁴ bw sp² (Bloomington)

dp^{tx} Sp cn/S² Cy, InCy L cn

ds^{38k}/Cy L

ds^{52k}/Cy L

fes ms b cn sp/dp^{tx}I Cy, 05 pr cn²
(Bloomington)

fes ms cn sp /net dp^{tx}I Cy b pr Bl lt³
cn² L⁴ sp² (Bloomington)

lt bw

ms cn bw/dp^{tx}I Cy pr Bl lt³ cn² L⁴ sp²
(Bloomington)

net bw mr crs/al² dp^{tx}I Cy Misl pr Bl
lt³ cn² L⁴ sp² (Bloomington)

pr

px

S fes Sp ms ta cn mr crs/al² In, Misl
dp^{tx}I Cy pr Bl cn² L⁴ sp² (Bloomington)

sp J L⁴ Pin/SM1 al² Cy sp² (Purdue)

sp² bs²

spt

stw²

stw³/ T (y:2)

10 second chromosome lethal balanced
over Cy L
melanotic tumor strain (e¹⁴⁴) homo-
zygous for a wild second chromosome

e
e¹¹ (Purdue)
Gl Sb/ Ins ^{L/M}
p
pP
ri pP

ru h th st cu sr e^S ca ("rucuca")

se

se e

sed

ss

st

th st cp

Chromosome 4

ci ey

ey²

Multichromosomal

1;2

Bld w²/ w; Cy

v; bw

y; Cy L/Pm

1;2;3

X.Y InEN In49 y; cn bw; e (no free Y)
(Bloomington)

y In49 v; bw; e (Bloomington)

2;3

bw; st

cn bw; ri e (Bloomington)

Cy L/Pm; H/Sb

Cy/Pm; D/Sb

fes ms cn sp/Cy 0; h ri e^S/Me ri
(Bloomington)

pr; st

2;3;4

bw; e; ci ey^R (Bloomington)

Not located

D - like

ITALYMilano: Universita' di Milano, Istituto di GeneticaWild Stocks

- 1) Canton - S
- 2) Chieti - v
- 3) Crkwenika
- 4) Gaiano
- 5) Jaslo o. c.
- 6) Moltrasio
- 7) Oregon - R
- 8) Pavia
- 9) S. Maria
- 10) Suna
- 11) Urbana
- 12) Valdagno
- 13) Varese

Chromosome 1

- 14) B
- 15) fan
- 16) NB⁴S
- 17) ptg
- 18) sd
- 19) w^a
- 20) w^{bl}
- 21) w^e

Chromosome 2

- 22) a px sp
- 23) ab
- 24) ast⁴ dp cl
- 25) b cn vg
- 26) blt
- 27) blt^S
- 28) bw ba
- 29) c wt px
- 30) cn
- 31) ft
- 32) ll²

33) net

34) so

35) so² b cn36) So^C

37) spt

Chromosome 3

38) cp

39) gl³

40) mwh

41) obt

42) ri-s se ss k e^S ro43) ru b st p^D ss e^S

44) ru

45) ve

Multichromosomal46) px^{43j} oo; ru jv se st caNot localized47) tg (formerly abab⁴⁹)InversionsChromosome 1:

48) CLB/+

49) CLB y/y Hw m² g⁴50) l(1) 7/dl 49 y Hw m² g⁴

51) Muller-5

Chromosome 2:

52) Cy sp/Pm

53) Cy E-S/S

54) Cy pr/db

55) M(2) 1/In(2R) Cy

56) M(2) B/In(2L) t, 1(2) B

57) spd gt-4/Gla

Chromosome 3:

58) H/Sb sr In(3R) Mé

59) ltr/ Sb sr In(3R) Mé

60) R Ly/In(3R) P, gm

Multichromosomal:61) 1.⁴ Cy sp/Pm ; H/Sb
sr In(3R) MéDeficiencies62) Df(2) Px² Df(2) Px,
bw sp/SMI, al² Cy sp²63) Df(2) bw⁵ Df(2) bw⁵
sp²/Xa64) Df(2) Px Df(2) Px/Df
(2)P ; Dp(2;3) P/In
(3R) Mo, sr ; w^eStocks selected for tumor
manifestation

65) tu A1

66) tu B1

67) tu B2

68) tu B3

69) tu B4

70) tu C1

71) tu C2

72) tu C3

73) tu C4

74) tu C5

75) tu D

76) tu Aspra

77) tu mwh

78) tu Oregon

79) tu So^C

80) tu w

81) tu y Hw

Roma: Istituto di Genetica Facalta di Scienze Dell' Universita
Citta UniversitariaWild Stocks

- A 1 Canton - S
- 2 Oregon - R

Normal X Chromosome

- B 1 car bb
- 2 N²⁶⁴⁻¹⁰⁹ / In (1) dl-49, y Hw m²g⁴

3. pn

4 sc cv v f B / y f : =

5 sw

6 w^a7 w^B w x8 w^{bl}9 w^{cf}10 w^{cf} / y f : =11 w^{co}

- 12 w^{col}
 13 w^{cp}
 14 w^e dy / y v f car
 15 w^r
 16 w^{sat}
 17 y ac sc pn / y f : =
 18 y cv v f car
 19 y faⁿ sn³
 20 y sc w^{col} spl f / In (1) rst³,
 rst³ f
 21 y w^a spl rb
 22 y l²⁵⁹/y² su-w^a w^a bb/sc⁸.Y
 23 y²wcf

Chromosome 2

- C 1 b cn^c bw
 2 Bl L²/ SM5, al² Cy lt^v sp²
 3 bw
 4 bw^D
 5 cn bw
 6 Sb J L² Pin / SM5, al² Cy lt^v sp²
 7 Cy Bl L / d l
 8 Cy Bl L / Sp Pin

Chromosome 3

- D 1 ca K - pn
 2 Gl Sb / L V M
 3 H²/ In (3R) Vno, Vno
 4 ru h th st cu sr e^s ca
 5 sc ss K e^s ro
 6 st C 3 G ca/ In (3LR) Ubx¹³⁰,
 Ubx¹³⁰ e^s
 7 st sr e^s ro ca

Multichromosomal

- E 1 bw; st (2;3)
 2 In (1) AM, y²/ FM6, y^{31d} dm B;
 SM1, al² Cy sp²/ Bl; In (3R)
 Vno, Vno / In (3LR) Ubx¹³⁰
 Ubx¹³⁰ e^s (1;2;3).
 3 y; sv^h (1;4)
 4 y; ru h th st pP cu sr e^s (1;3)
 5 y; bw; st (1;2;3)
 6 y; pol (1;4)
 7 yf : = : ci ey^R (1;4)
 8 lys rc ; (2;3)

Triploid

- F 1 y¹²⁵⁹ / FM4, w f/ FM4, w f

Inverted X Chromosomes

- G 1 In (1) dl-49, v^{of} f
 2 In (1) dl-49, w lz
 3 In (1) dl-49, y Hw m²
 4 In (1) dl-49, y Hw m²g⁴

- 5 In (1) sc^{4L}, sc^{8R}, y sc⁴⁺⁸w^a
 m car
 6 In (1) sc⁷ AM
 7 In (1) sc⁸ sc⁸
 8 Ins (1) sc⁸, dl-49, 3C-4EF,
 15DE-20, y^{31d} sc⁸ dm B (FM6)
 9 In (1) sc⁸, dl-49, y^{31d} vof f
 10 In (1) w^{m4}, w^{m4}
 11 In (1) y sc⁴ w^a m car
 12 In (1) 481 (12E-F; 14B),
 y bb¹ 481

Deficiency and Duplications

- H 1 Df (1) N⁸/ dl-49, y Hw m²
 2 Df (1) N⁸/dl-49, y Hw m² g⁴
 3 Df (1) N²⁶⁴⁻³⁹ wch/ FM4, y^{31d}
 sc⁸ dm B
 4 Df (Y) Ybb-

Translocations

- I 1 T (1;4) B^S (16 Al), y² cv v
 B^S car/ y f : =
 2 T (1;4) w^{m5} (3C3), w^{m5}
 3 T (2;3) bw^{V4}; bw^{V4}

Closed - X

- L 1 X^C, y/ y f : =

X Chromosomes with a Y Arm Attached

- M 1 X Y^L (C-2), y cv v f car
 bb-, Y^L
 2 X Y^L (A-3), sc cv v . Y^S
 3 Y^{SX} (FR-1), Y^S y cv v f
 4 Y^S X (P-7), In (1) EN, Y^S y f

Attached - XY

- N 1 X Y^L.Y^S (108-9 Parker), y²
 su-w^a Y^L. Y^S
 2 X Y^S.Y^L (110-8 Parker), y²
 su-w^a w^a Y^S.Y^L y⁺
 3 X Y^S.Y^L (129-16 Parker), y²
 su-w^a w^a Y^S.Y^L y⁺
 4 Y^{SX}. Y^L, Ins (1) EN, dl-49,
 Y^S car f v y .Y^L

Altered Y

- O 1 sc⁸.Y:bw⁺ (Y^L bw⁺.bb⁺Y^Sac⁺y⁺)
 2 y sc⁸. Y (y ac⁺Y^L. bb⁺ Y^S)
 3 Y B^S.y⁺ (B^S Y^L. bb⁺Y^S y⁺)
 4 Y^{Su-Var}
 5 Y: bw⁺ (Y^L bw⁺ . bb⁺Y^S)
 6 Y^C : bw⁺ (MYR)
 7 Y w⁺
 8 y⁺Y w⁺
 9 y⁺Y w⁺B^S

JAPANAnzoy, Aichi: Nagoya University, Department of Animal Breeding, Faculty of AgricultureWild Stocks

1. Anzoy-Aichi
2. Canton Special
3. Chausuyama-Aichi
4. Hachijojima
5. Hikosan-Kyushu
6. Hiroshima
7. Hita-Kyushu
8. Omogo-Shikoku
9. Oregon
10. Suzuka-Mie
11. Takagicho-Tokyo
12. Yonekawa-Yamaguchi

Chromosome 1

13. Bx
14. ec ct⁶ g² bb¹ / C 1 B
15. f
16. m
17. m⁵⁸ⁱ
18. sc^{S1} B InS w^a sc⁸ (Muller 5)
19. sc^{S1} B InS w^a sc⁸ 1(1)59 / y w m f
20. v
21. w
22. y⁵²¹
23. y w m
24. y w m f
25. y w m f / y C 1 B

Chromosome 2

26. b
27. bw

28. bw (from population bell No. 33)
29. cn
30. Cy / bw (M)
31. Cy / bw (T)
32. Cy bw / bw
33. Cy / 1(2)50c
34. Cy bw / 1(2)50c
35. Cy / Pm
36. dp^x
37. dp^v b
38. Pm b
39. Pm / 1(2)50c
40. vg
41. vg^{Nw} Hia / T(2,3) S^M Cy

Chromosome 3

42. cu
43. e
44. Sb

Multichromosomal

45. Cy / 1(2)50c ; Sb
46. Cy / 1(2)50c ; Sb cu / cu
47. Pm / 1(2)50c ; cu
48. v ; bw

Unanalyzed

49. Dichaete like
50. brown like
51. jaunty like

Kyoto: Kyoto University, Faculty of Science, Department of ZoologyWild Stocks

- South Africa (6)
Sweden (1)
Switzerland (1)
U.S.A. (30)

Common Stocks

- Canton-S
Oregon-RS

Chromosome 1From different natural populations

- Formosa (1)
France (5)
Israel (3)
Italy (2)
Japan (29)
Spain (2)

- 1 B
- 2 car
- 3 ec ct⁶g²bb¹/ClB
- 4 f
- 5 Muller-5
- 6 v
- 7 w
- 8 w^a

9 w^e
 10 w m
 11 y
 12 y w m f

Chromosome 2

13 b
 14 b gp
 15 bw
 16 bw (Nanzenji)
 17 ce
 18 cn
 19 cn bw
 20 dp
 21 dp^x
 22 S/Cy E-S
 23 vg
 24 vg^{no}

Chromosome 3

25 ca
 26 Confluent-3
 27 cu
 28 e¹¹
 29 e¹¹se
 30 ro
 31 ru h tu st cu sr es ca
 32 se
 33 se (Nanzenji)
 34 Hn^{r3}
 35 ss^a
 36 st
 37 wo

Chromosome 4

38 svⁿ

Multichromosomal1;2

39 v; dp

1;3

40 w; e¹¹
 41 v; bar-3

2;3

42 bw; e
 43 bw; st
 44 dp; bar-3
 45 vg; Hn^{r3}
 46 vg; bar-3

2;3;4

47 cn; ca; gvl

1;2;3;4

48 y; bw; e; ci ey^R

Lethal Stocks

49 1-(1)us-2
 (ultra sonic induced)
 50 1-(1)us-48
 (ultra sonic induced)

51 Df(2)px²/Cy L⁴sp²

Special Stocks

52 st sr cr ro ca
 53 tu^{36e}
 54 tu bw
 55 tu st

56 Y/Basc/T(1;2), T(2;3) }
 57 Y/wm/T(1;2), T(2;3) } XXY with
 two trans-
 locations

Mitaka, Tokyo: International Christian University, Biology Department

Wild Stocks

Tokyo

Chromosome 1

1 : y, m, ywmf, w, w^e, w^a
 2 : bw, vg
 3 : cu, e

KOREAKwangju: Chonnam National University, Department of BiologyWild Stocks

- 1 Kwangju
- 2 Oregon-R
- 3 Swedish-C

Chromosome 1

- 4 lz^{37h}
- 5 w
- 6 xc² v y:=
- 7 y
- 8 y w f
- 9 YLc/y w Y^S & y v f
- 10 Muller 5

Chromosome 2

- 11 Bl L²/Cy, sp²
- 12 Bl L²/Cy, bw sp²
- 13 cn
- 14 ds S G b pr/Cy, al² lt³ L⁴ sp²
- 15 lt std/SM1, al² Cy sp²

- 16 M(2)S7/SM5, al² Cy lt^v sp²
- 17 Pin Pfd/Cy, sp²
- 18 Pfd/Ins(2L+2R)Cy, S²
- 19 pr
- 20 Sp Bl L/Cy, sp²
- 21 Sp bw^D/SM5, al² Cy lt^v sp²
- 22 vg

Chromosome 3

- 23 p^D bx sr e^s
- 24 ro
- 25 se h
- 26 W Sb/Ins(3LR)Cx

Chromosome 4

- 27 pol

Stock list, other species

- 1 D. virilis
- 2 D. lutea
- 3 D. rufa

Seoul: Chung-Ang University, College of Liberal Arts & Sciences,
Department of BiologyWild Stocks

- 1 Oregon-R
- 2 Urbana-S
- 3 Kongju (3 strains)
- 4 Seoul

Chromosome 1

BB
Bx

w
y

Chromosome 2

b
cn
j
vg

Chromosome 3

e¹¹
ro
ru
se

Inversion

Muller-5

Seoul: Seoul National University, Department of ZoologyWild Stocks

1. Canton-S
2. Kongjoo (Korea)
3. Oregon-R
4. Quilpart (Korea)
5. Seoul (Korea)
6. Swedish-C
7. Urbana-S

Chromosome 1

BB
Bx
spl
t
w
wBx
y
yw

Chromosome 2

bj
BL²/cy
c
cn
j
vg

<u>Chromosome 3</u>	ru ca	<u>Multichromosomal</u>
e se e ¹¹ se ro	<u>Inversion</u> Muller-5	y vg

Seoul: Sung Kyun-Kwan University, Department of BiologyWild Stocks

- 1) Oregon-R
- 2) Seoul (Korea)
- 3) Swedish-C

Chromosome 1

- 4) BB
- 5) w
- 6) y

Chromosome 2

- 7) c
- 8) cn
- 9) j
- 10) pfd/Sm-5

Chromosome 3

- 11) ca
- 12) ro
- 13) ru cu ca
- 14) se h
- 15) ve h th

Chromosome 4

- 16) pol

Inversion

- 17) Muller-5

Seoul: Yonsei University, Department of BiologyWild Stocks

- 1 Canton-S
- 2 Oregon-R --- Isogenic
- 3 Oregon-R-C --- Isogenic
- 4 Oregon-S
- 5 Samarkant (Japan)
Isogenic (300 generations)
- 6 Seoul-1 (Korea) --- Isogenic
- 7 Seoul-2 (Korea) --- Isogenic
- 8 Suwon (Korea)
- 9 Swedish-C
- 10 Yangdong (Korea)

Chromosome 1

- 11 B
- 12 bi ct⁶ q²
- 13 bo
- 14 br
- 15 Bx³
- 16 cm
- 17 ec
- 18 ec dx
- 19 fa
- 20 rg
- 21 sc cv v f

- 22 sc cv v eq
- 23 sn³
- 24 svr
- 25 t
- 26 t² v f
- 27 v
- 28 v car
- 29 w
- 30 w^a
- 31 w^{bf}2
- 32 w^{ch}
- 33 w^{co} sh²
- 34 w^{col}
- 35 w^e bbl/C1B
- 36 y
- 37 y ac v
- 38 y sc mf²
- 39 y² cv v f
- 40 M-5/y sc⁸ y
- 41 M-5/y ac Sn³ cn

Chromosome 2

- 42 a px or
- 43 a px sp
- 44 ab
- 45 al

46 al bc sp²
 47 al b pr an vg a sp²/ In(2LR)Cy,
 L⁴ sp²
 48 al dp b pr blt bw/ SM5, al² Cy
 ltv sp²
 49 al dp b pr c px sp/Cy,pr
 50 al dp b pr Bl c px sp/ SML al²
 Cy sp²
 51 b
 52 b lt wxt bw
 53 b vg
 54 bw
 55 bw ba
 56 Bl/Cy, bw^{45a} sp² or^{45a}
 57 Bl L/SM5, al² Cy ltv sp²
 58 c
 59 c wt px
 60 cl
 61 cn bw
 62 Cy/Pm
 63 ex
 64 ho
 65 L
 66 L⁴
 67 pd
 68 pr
 69 pr cn ox/SM5, al² ltv Cy sp²
 70 rh
 71 so
 72 vg
 73 wt

Chromosome 3

74 aa h
 75 bul
 76 ca
 77 cp in ri p^p
 78 cu
 79 cv-c sbd²
 80 D/Gl
 81 gl
 82 Gl sb/LVM
 83 h
 84 jv
 85 p
 86 ra
 87 ro
 88 ru
 89 ru h st p^p ss e^s
 90 ru h th st cu sr e^s ca
 91 ru^g jv se by
 92 Sb/In(3LR) Ubx¹⁰¹
 93 se
 94 se h

95 ss
 96 st
 97 th
 98 th st cp

Chromosome 4

99 bt
 100 ci
 101 ci gvl bt
 102 ey
 103 ci gvl ey^{Rsvn}
 104 pol
 105 spa

Multichromosomal Stocks

106 br³ dxst;ed Su-dx(1;2)
 107 lz^D/dl-49,m² g⁴; Cy/Pm(1;2)
 108 v; bw(1;2)
 109 w; vg(1;2)
 110 M-5, Cy/Pm Sb/Ubx(1;2;3)
 111 ptg; px pd; su-pd(1;2;3)
 112 bw;st(2;3)
 113 Cy/pm; D/Bb(2;3)
 114 Cy/Pm Sb/Ubx(2;3)
 115 lys rc; ss(2;3)
 116 vg; se(2;3)
 117 se h; ci ey^R(3;4)

Attached-X

118 br ec/y^{3d}
 119 y/g² ty

Deficiencies

120 Df(3)sbd¹⁰⁵/Xa
 121 Df(2)al Cy, En-S

Duplications

122 Dp(2;3)S

Inversions

123 Muller-5
 124 Ins(1)sc^{S1L}, s, se^{8R} wa B.
 125 Vg^{nw} Hia/SM5, al² Cy lt^L sp²
 126 Vg^u/Roi, bw sp or
 127 A/In(3R) hp hp

Translocations

128 T(1;2) Bld/ClB
 129 T(2;3) Xa/Sb bx^D

SOUTH AFRICAJohannesburg: University of the Witwatersrand, Department of ZoologyWild Stocks

1	Bethulie
2	Bloemfontein
3	Canton-S
4	Cape Town
5	Cedara
6	Drakensberg
7	Florida
8	Graaff-Reinet
9	Inhaca Island
10	Johannesburg
11	Kalahari
12	Kariba Dam
13	Limpopo
14	Nelspruit
15	Nyasa Lake
16	Oregon-R
17	Stanford Lake
18	Stellenbosch
19	Tzaneen
20	Umgazi River
21	West Rand
22	Western Province
23	Zoutpansberg

Chromosome 1

24	bi ct ⁶ g ²
25	bo
26	B
27	car
28	car ²
29	cm
30	cm car
31	cm g ³ car
32	ct v
33	ct v dy g
34	ct ⁶
35	cv ct
36	cv sc
37	ec
38	ec ct ⁶ v g ³
39	f
40	f ⁵ m
41	f ⁵ v
42	g
43	g ²
44	g ³
45	m
46	pn ²
47	ras dy
48	ras ²
49	rb
50	rb car

51	rb cm g ³
52	rb cm car
53	rb cx
54	rb g ³
55	rb g ³ car
56	sc
57	sc ec cv ct ⁶ v
58	sc ec cv ct ⁶ v g ² f
59	svr w ^a /FM3 y ³ id ^{sc} 8 dm B 1
60	v
61	v ³⁶ f
62	w
63	w m
64	w m f
65	w ^a
66	w ^a 3
67	w ^a 4
68	w ^a rb
69	w ^{bl}
70	w ^{ch}
71	w ^{co} sn
72	w ^{col}
73	w ^e
74	w ^e 2
75	w ^e 3
76	w ^e car
77	w ^e g ³
78	w ^e rb
79	w ^e rb car
80	w ^{sat}
81	w ^t fw
82	w ^w f ⁵
83	w ^w rb
84	y
85	y g ⁴
86	y m
87	y w
88	y w m
89	y ² su-w ^a w ^a bb
90	y ² w ^a w

Chromosome 2

91	albaspr/Cy L ⁴ sp ²
92	al dp b pr Bl c px
	sp/SM1 al ² Cy sp ²
93	a sp ²
94	b
95	b pr cn
96	b pr cn a
97	bw
98	bw ^{2b}
99	bw ^D
100	c

101	c px
102	cn
103	cn ³⁵ k
104	cn vg
105	cl
106	cl ^{50a}
107	dke c
108	dp
109	lt std/Cy sp ²
110	lt stw ³
111	ltd
112	pd
113	pr
114	pr ^{42d}
115	px
116	sf ²
117	sp
118	Su-H/Cy, pr
119	tk sf ² abb
120	vg
121	vg ^{dn}

Chromosome 3

122	ca
123	cd
124	cu kar
125	D/Gl
126	e
127	e ^s
128	ma fl
129	mah
130	p
131	p ^p cu
132	p ^p cu sr e ^s
133	res
134	ru
135	ry
136	se
137	sr
138	st ^{sp}
139	su ^B -pr/In(3R)C, e;pr
140	th st
141	th st p ^p

Multichromosomal

142	bw; e; ci ey
143	bw; ci ey
144	bw; st
145	Cy/Pm, ds ^{33k} ; H/In(3R)
	Mo; sr
146	g ³ ; bw
147	g ³ ; st
148	g ³ ; st p ^p

149 ras ² ; st	<u>Attached-X</u>	165 In(1)rst ³ , y rst ³
150 rb; bw		car bb
151 rb; ry	160 f B/ suS ² -v-pr v	166 In(1)w ^{m4}
152 rb; se	161 y/+	167 In(1)w ^{m4} ; st
153 rb; st	162 y ² su-w ^a w ^a bb /	168 Ins(1) sc ^{S1} , S, sc ^{S1}
154 car; ry	sc ^{4L} sc ^{8R}	w ^a B sc ⁸
155 car; se		
156 vg; se	<u>Inversions</u>	<u>Translocations</u>
157 w ^e rb; se	163 In(1)A99b	169 T(1;3)0 ⁴ , D/CLB
158 w ^w ; cd	164 In(1)dl-49, y fa ⁿ	170 T(1;4)w ^{m5}
159 y; bw; e; ci ey		

Pretoria: University of Pretoria, Department of Genetics

Wild Stocks

Beckett St., Pretoria
 Durban (contains In(1), In(2L), In(3R))
 Government Ave., Pretoria
 Graaff Reinet
 Inhaca
 King William's Town
 Koedoeskop
 Mark 2, Pretoria
 Mequatling
 Pietersburg 1 (contains In(1), In(3R))
 Pietersburg 2
 Pretoria
 Stellenbosch (contains In(3R))
 Windhoek (contains In(3R) and se)

Chromosome 2

bw
 cn
 cn bw
 vg

Chromosome 3

D/G1
 e
 se

Attached-X

+/y

Chromosome 1

In(1) dl-49, y faⁿ
 v
 w
 w^a
 w^{bl}
 w^e
 w m f

Multichromosomal

bw; st
 Cy/Pm; H/In(3R)
 T(1;3)0⁴; D/CLB
 y bw ci e ey

SWEDEN

Uppsala 7: University of Uppsala, Institute of Genetics

Wild Stocks

1. Algeria
2. Amherst-3
3. Bayfordbury
4. Boa Esperanca, Minas Gerais, Brazil
5. Canton-S
6. Crimea
7. Curitiba
8. Florida
9. Formosa
10. Gruta, Argentina

11. Hikone-R (resistant to BHC, DDT, parathione, nicotine)
12. Karsnäs
13. Kochi-R (resistant to parathione)
14. Oregon-R
15. Salvador, Bahia, Brazil
16. Samarkand
17. San Miguel, Buenos Aires, Argentina
18. Stäket
19. Tunnelgatan
20. Ultuna
21. Örebro

Chromosome 1 (X)

101. B
102. B/y:=
103. BB car; sc⁸ Y/y f:= ; sc⁸ Y
104. Bⁱ
105. ct
106. cv
107. cv sn³
108. ec
109. ec ct v g
110. ec ct v f
111. f
112. f B od car/ y f:=
113. f BB; sc⁸ Y/ y f:= ; sc⁸ Y
114. f Bⁱ Bⁱ/ y f:=
115. f od sy car
116. fu ff/ ClB
117. g (Sweden)
118. g²
119. In (1) w^{m4}
120. lz/ ClB
121. ma-1/ y f:=
122. od car
123. sc z w^{17G2} ec/ y w f:=
124. sc z ec
125. sc² mottled
126. sc^{S1} B InS w^a sc⁸ (Muller-5)
127. sc^{S1} InS w^a sc⁸
128. sn³
129. sp-w
130. sp-w²
131. su-w^a w^a
132. v g
133. w
134. w cv
135. w cv sn³
136. w sn³
137. w^a su-f/ y f:=
138. w^{a4} / y f:=
139. w^{bf} f⁵
140. w^{bf2}
141. w^{bl}
142. w^{Bwx}
143. w^{ch} wy/ y f:=
144. w^{co}
145. w^{co} sn²
146. w^e
147. w^{e2}
148. w^{e2} en-w^e/ y f:=
149. w^h
150. w^h ct
151. wⁱ yb
152. w^{sat}
153. y
154. y ec ct v f
155. y rst³ car
156. y f Eb/ sc^{S1} B InS w^a sc⁸

157. y² sc w^a w^{ch} fa/ y w f:=
158. y² sc wⁱ
159. y² sc wⁱ w^{ch}/ y w f:=
160. y² su-w^a w^{a2} w^{ch} spl/ y f:=
161. y² w^a
162. y² w^a w
163. y² w^a ec
164. y² w^{bf} spl sn³/ y f:=
165. z
166. z w^a/ y w f:=
167. z w^{a3}/ y w f:=
168. z w^{11E4}

Chromosome 2

201. bw
202. bw^D
203. Cy/ Pm
204. Cy/ S
205. fes Alu lt/ al² Cy lt³
206. nw²/ Cy RNS
207. pr
208. S/ NS, px sp
209. vg
210. vg bw

Chromosome 3

301. ca
302. cd
303. D³/ InP
304. D/ Sb
305. e¹¹
306. kar²
307. ri ss
308. ri²
309. ri² ss
310. ro
311. ru h st p^D ss e^s
312. ry
313. ry²
314. ry² cd
315. se
316. ss
317. st
318. st p
319. st ry
320. st ss e¹¹

Chromosome 4

401. ey
402. svⁿ

Multichromosomal

501. w^{ch}; Su-w^{ch}/ Cy (1;2)
502. w^{col}; bw (1;2)

503. w^e ; cr-u/ Cy (1;2)
 504. y^{S1} sc⁸ InS y³P; al² Cy
 lt³ sp²/ dp b Pm¹;
 ru h D³ InCXF ca/ Sb
 In (3R) (1;2;3)
 505. y' w spl; Cy; Ubx¹³⁰/
 Xa (1;2;3)
 506. bw; cd (2;3)
 507. bw; st (2;3)
 508. Cy/ S; D/ InP (2;3)
 509. L sp; th (2;3)
 510. L²+/; sp; th (2;3)
 511. sp; th (2;3)

Deficiencies and Duplications

601. sc z Df(1)w²⁵⁸⁻⁴⁵/ FM4
 602. y² Df(1)w²⁵⁸⁻⁴⁵/ FM4

603. XY', g² B; Y"/ y / Y" (Stern)
 604. Df(1)w²⁵⁸⁻⁴⁵, y w spl dm;
 Dp(1;3)w^{Vco}/ y w f:=
 605. Dp(1)is/ y f:=
 606. Dp(1;2R)w^{+51b7}
 607. Dp(1;4)w^{+51c20}
 608. Dp(1)w^a/ y w f:=
 609. Dp(w^{a4}/ w^a)/ y f:=
 610. Dp(w^{bf}/ w^a) ec/ y f:=
 611. sc Dp(1)z^{59d15}/ y f:=
 612. z Dp(w^{a4}/ w^a)/ y f:=

Translocations

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

NEW MUTANTS

Report of A. Chovnick and A. Schalet

A large number of X-ray induced rosy mutants are under study in this laboratory (see notice DIS 34, page 122). Some of these mutants have been reported in prior notes (Schalet and Chovnick, DIS 34; Chovnick, Schalet and Kernaghan, 1961), and others will be discussed in future reports. We are using the designations ry^4 through ry^{73} for existing mutants. In view of Hubby's report of a new mutant, ry^3 (DIS 35), we have changed the designation of our ry^3 to ry^{3a} .

Report of K. S. Gill

$fs(3)1^{59a}$: female-sterile in the third chromosome 59a Gill, 1959. 3-47⁺. Pub. Gill, 1960, Anat. Rec., 138:351; Gill, 1961, Ph.D. Thesis, Yale Univ. X-ray induced. Females completely sterile; eggs laid die in early cleavage. Males fertile. RK3.

$fs(3)2^{59a}$: female-sterile in the third chromosome 59a Gill, 1959. 3-11⁺. Pub. Gill, 1960, Anat. Rec., 138:351; Gill, 1961, Ph.D. Thesis, Yale Univ. X-ray induced. Females completely sterile; rare breakthroughs may develop into adults. Oogenesis incomplete, usually stopping in early phases of vitellogenesis. 89% of the follicles contain 32 cells instead of the normal number 16. The (32) cells of an incipient cyst are enclosed in two chambers (twin chambers) in 6% of the cases. Males partially sterile. Viability low. RK3.

$fs(3)3^{59a}$: female-sterile in the third chromosome 59a Gill, 1959. 3-25⁺. Pub. Gill, 1960, Anat. Rec., 138:351; Gill, 1961, Ph.D. Thesis, Yale Univ. X-ray induced. Females completely sterile. Oogenesis incomplete: most follicles stop development during yolk deposition (after stage 9). Males fertile. RK3.

$fs(3)4^{59a}$: female-sterile in the third chromosome 59a Gill, 1959. 3-59⁺. Pub. Gill, 1960, Anat. Rec., 138:351; Gill, 1961, Ph.D. Thesis, Yale Univ. X-ray induced. Females completely sterile. Oogenesis incomplete: follicles generally cease development early in vitellogenesis (at or before stage 9). Primary compound chambers in which 2, occasionally 3, incipient cysts are enclosed occur in about 6% of the cases. Males completely sterile. Adult fat body hypertrophied; body size reduced; occasionally metathoracic legs with tibiae more curved than normal and tarsi crooked. Viability low. RK3.

$fs(3)5^{59a}$: female-sterile in the third chromosome 59a Gill, 1959. 3-49⁺. Pub. Gill, 1960, Anat. Rec., 138:351; Gill, 1961, Ph.D. Thesis, Yale Univ. X-ray induced. Females completely sterile. Oogenesis incomplete: ovarioles contain excessive numbers of follicles which usually stop development at or before stage 9. Males fertile. RK3.

Report of E. H. Grell

nw^D : narrow-Dominant. E. H. Grell, 59f. 2-83⁺. From an X-rayed Canton-S male. The wings tend to be longer and more narrow than normal. The phenotype is highly variable and except for slightly squared wing tips sometimes approaches wild type. The viability of heterozygotes ($nw^D/+$) is below normal and homozygotes are entirely inviable; nw^D/nw is also lethal. RK2.

Report of Afton M. Hansen and Eldon J. Gardner

scrp:scarp Hansen and Gardner 1960. 2-74⁺. Pub. Hansen and Gardner, 1961, Genetics 46:869. Appeared in a "wild" Cockapontett stock that had been subjected for several days of high temperature. This stock was apparently homozygous for the scrp gene at the time the phenotype was detected. Expressivity is variable. Usually the ventral one-third of the eye is flattened and differentiated from the dorsal two-thirds by a horizontal depression. Tufts of vibrissae are often present on the anteroventral border of the eye. Occasionally the eyes are reduced. Growths may be present. Either or both eyes are affected. The ommatidia are shorter in the area of depression. The expression completely overlaps the wild-type at 25° C., with an average penetrance of 80 per cent at 30° C. Temperature effective period at 30° C. is from the forty-second to sixty-eighth hour of development, with the entire period at 30° C. necessary for maximum penetrance. Viability good. RK4.

Report of T. Imaizumi

Cf-3 : Confluent-3 60j. 3-66.2. Dominant, homozygous lethal; arose spontaneously as I male in the cross of Oregon-RS x b vg, an allele of Delta, phenotype of heterozygote very likely to that of Delta, good viability; homozygote die at late stages of embryo and partially at early larval stage. Perhaps with deficiency.

Report of P. T. Ives

m^{61e} : miniature^{61e}. Ives, 61e23. Like m. Induced by 1 kr γ radiation in an Oregon-R/rucuca sperm which was deposited on day 4 of an exhaustive mating schedule.

Functional allelism to m established by A. B. Burdick, who is also investigating its pseudoallelic properties.

Report of P. T. Ives

e^{60k} : ebony^{60k}. Ives, 60k25. Like e. Spontaneous in vg;se^{50k}.

Report of Shanta V. Iyenyar

blt:ballet, X-chromosome, not localised; X-ray induced in young male, recessive, wings one third the normal length stretched outwards and slightly upwards, wing tip broadened, venation markedly altered as in fused; viability of the male impaired but not to the same extent as females since only one female homozygous for ballet has been found but died one hour after emergence. Mutation does not seem to be affected by temperature.

nr:narrow wing-rough eyes, X-chromosome not localised, X-ray induced. Wings like tapered on II chromosome, eyes smaller, oval in outline and uniformly more rough. Both sexes have been noticed, recessive, does not seem to be affected by temperature.

Report of E. B. Lewis

Correction to DIS 34:51. The mutant alpha-1 of H. W. Lewis and its description should appear under a separate heading entitled "Report of H. W. Lewis."

Report of Y. Maeda

uex: unexpanded Maeda, Y., 5813. 2-. Spontaneous in local stock (Kobe) of wild. Wings are wrinkled and crumpled as those of newly emerged flies, about 1/2 length of normal size, like pu, often inflated as balloon. Tibia and tarsi in 3rd legs are irregularly shortened and gnarled, so the mode of creeping is abnormal. Posterior scutellars bent toward median. The data from the preliminary linkage analysis by crossing with cn or bw indicated that the approximated position of 2-55⁺ for uex (+ 724, cn 27, uex 14 and cn uex 663 from cn uex/+ + x cn uex, and + 535, bw 367, uex 233 and uex bw 258 from uex bw/+ + x uex bw). Male viability is somewhat low. RK2.

Report of E. Ortiz

w^{57b}: white^{57b} Ortiz, 57b. From Canton-S wild type male, treated with ethyl-urethane, as 8 males. Behaves as an allele of our stock of w. Eye colour snow white. Good viability and fertility. RK1.

w^{58a}: white^{58a} Ortiz, 58a. Spontaneous as 14 males in our laboratory stock of vg. Behaves as an allele of our stock of w. Eye colour snow white. Good viability and fertility. Kept in stock as w^{58a}; vg. RK1.

lz^{61g}: lozenge^{61g} Ortiz, 61g. From Oregon-R wild type male, X-ray induced, as 22 males. Eye oval, smaller than wild type, all facets run together into smooth surface. Eye colour dark brown, with darker rim. Homozygous female highly infertile. RK2.

Report of Verena Rohr

r^S: rudimentary Swiss Hadorn, 59d. Spontaneous in cross +/+ x ma-1/. Wings obliquely truncated, often arclike and blistered with medial and lateral marginal bristles sparse and ruffled. Often more than one bristle on one socket. L₄ and L₅ shortened. Males fertile, females sterile. Wing size much more reduced in homozygous females than in hemizygous males. Expressivity similar to r^{39k}, but with great variability. r^S/ male offspring from different crosses much influenced by genotypic milieu. Compounds r^S/r^{39k}, r^S/R⁹ semilethal with abnormous marginal wing bristles. Compound r^S/r¹² subvital, some individuals with normal lateral marginal wing bristles. RK₂.

LINKAGE DATA

Report of M. M. Green: Gene sequence at the X chromosome tip in Drosophila melanogaster.

From a cross of ♀♀ y^2 sc w^a ec/y^2 $su-w^a$ w^{a4} X ♂♂ a single y^{+sc+w^a} ♂ was found. Subsequent crosses showed that the ♂ was y^2 sc w^a and carried a short duplication of the X tip inserted in chromosome 3. This duplication in all probability arose as a spontaneous X-3 translocation in a parental + ♂ of which only the X part translocated to chromosome 3 was found. In addition to y^2 and sc , the duplication covers $su-w^a$ but not dor , thereby establishing the probable order as y^2 , sc , $su-w^a$, dor . Inadvertently this duplication was lost.

Report of Afton M. Hansen and Eldon J. Gardner

Scarp-eyed (scrp) flies were mated with curly, plum, stubble, dichæte flies. All individuals in the F_1 generation had wild-type eyes. Flies from this mating with curly wings and stubble bristles were then back-crossed with scrp flies and their progeny allowed to develop at 30° C. The progeny with scarp eyes had either stubble bristles or wild-type bristles and wild-type wings. Scarp eyes did not occur in combination with curly wings. Thus, scrp was located in the second linkage group.

Scarp-eyed flies were mated with aristaless, dumpy, black, purple, curved, plexus, speck flies and the wild-type F_1 females were back-crossed with al dp b pr c px sp males. Flies of each of the 12 possible single crossover gametes were collected and mated with scrp males or females. Their progeny developed at 30° C. When scarp-eyed flies appeared in the progeny, the parent was scored as a carrier of scrp. A summary of data from two experiments is included in the following table:

Genotype tested	Number of successful matings	Number that were carriers of <u>scrp</u>
<u>+ dp b pr c px sp</u> al dp b pr c px sp	8	0
<u>al ++ ++ + +</u> al dp b pr c px sp	17	17
<u>+ + b pr c px sp</u> al dp b pr c px sp	12	0
<u>al dp + + + +</u> al dp b pr c px sp	16	15 ^a
<u>+ + + pr c px sp</u> al dp b pr c px sp	9	0
<u>al dp b ++ + +</u> al dp b pr c px sp	13	13
<u>+ + + + c px sp</u> al dp b pr c px sp	53	2

(Hansen and Gardner, table--continued)

Genotype tested	Number of successful matings	Number that were carriers of <u>scrp</u>
<u>al dp b pr + + +</u> al dp b pr c px sp	23	22
<u>+ + + + + px sp</u> al dp b pr c px sp	19	16 ^b
<u>al dp b pr c + +</u> al dp b pr c px sp	30	0
<u>+ + + + + sp</u> al dp b pr c px sp	13	11 ^c
<u>al dp b pr c px +</u> al dp b pr c px sp	6	0
<u>al dp b pr c px sp</u> al dp b pr c px sp	6	0
<u>+ + + + + + +</u> al dp b pr c px sp	13	12 ^d

- a only 5 offspring
- b only 8, 5, and 3 offspring
- c only 2 and 19 offspring
- d only 14 offspring

These data showed that scrp was located near c, but to the left. Three of 76 crossovers between pr and c (21 map units) were also crossovers between c and scrp. Crosses with lobe-recessive showed that scrp is not an allele of L^r. Scrp is located at approximately 74 units from the left end of the second chromosome.

STOCK LISTS

AMHERST, MASSACHUSETTS: AMHERST COLLEGE

Correction to list in DIS 34:

D. simulans: Amherst, Mass. 1961

BALTIMORE, MARYLAND: THE JOHNS HOPKINS UNIVERSITY

a1	<u>D. funebris</u>	a6	<u>D. simulans</u> , Lima, Peru, a ⁵
a2	<u>D. hydii</u>	a7	<u>D. simulans</u> , Lima, Peru, a ¹⁰
a3	<u>D. simulans</u>	a8	<u>D. simulans</u> , New Orleans
a4	<u>D. simulans</u> , La-3	a9	<u>D. simulans</u> , South Africa
a5	<u>D. simulans</u> , La-4	a10	<u>D. virilis</u>

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Zoology

D. virilis

Stocks listed in DIS 34 with the following numbers are no longer kept in culture: 4, 37, and 39.

DeKALB, ILLINOIS: NORTHERN ILLINOIS UNIVERSITY
Department of Biological Sciences

Stock Lists, other species: Essentially as in DIS 34, but have added:

D. tripunctata - several local samples
D. immigrans - several local samples

LINCOLN, NEBRASKA: THE UNIVERSITY OF NEBRASKA
Zoology Department

D. affinis: Alabama, Georgia, Florida, Iowa, Kentucky, Louisiana, Minnesota, Mississippi, Missouri, Nebraska, New York, North Carolina, South Carolina, Tennessee, and Texas. All wild-type strains except for several with CO₂ sensitivity (virus-caused).

D. algonquin: Massachusetts and Minnesota.

D. athabasca: Massachusetts, Maine, Alaska, Minnesota.

D. narragansett: New York.

LOS ANGELES, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Botany

D. pseudoobscura

Lethal strains: A number of lethal strains of various gene arrangements of the third chromosome from Southern California and Guatemala, which are currently being tested. Lethal strains are currently being established from wild samples from Bogota, Colombia.

Wild strains (homozygous and isogenic for Chromosome 3 inversions):

Standard (1) San Jacinto Mountains
 Arrowhead (1) San Jacinto Mountains
 Arrowhead (1) Texas
 Chiricahua (2) San Jacinto Mountains
 Tree Line (1) San Jacinto Mountains
 Tree Line (4) Guatemala
 Pikes Peak (2) San Jacinto Mountains
 Pikes Peak (1) Texas
 Santa Cruz (2) San Jacinto Mountains
 Santa Cruz (1) Guatemala
 Oaxaca (2) Guatemala
 Cuernavaca (3) Guatemala
 Vandeventer (2) San Jacinto Mountains
 Thomas Mountain (1) San Jacinto Mountains
 Pinyon (1) San Jacinto Mountains (carries lethal factor, maintained heterozygously balanced with marked Standard chromosome)

Chromosome 1

Cnv (Convergent veins)
 Pt w^e
 Pt mg²
 y sn v co sh
 sd (scalloped)
 upd
 er (erect bristles)

or pr spr (Standard)
 or pr spr (Arrowhead)
 or px (Arrowhead)
 or px pr (Arrowhead)
 or ru (Tree Line)
 or pr vg (Arrowhead)
 or Bl L pr cv (AR) / lethal (CU)
 or Bl L Sc pr cv (ST) / lethal (CU)
 or Bl px^D (AR) / or L (SC)
 or Bl Sc ru pr cv (ST) / or L (SC)
 or L Sc pr (ST) / lethal (CU)
 or Bl px^D (AR) / lethal (CU)
 or Bl L Sc (VA) / lethal (CU)

Chromosome 2

gl²
 pcv¹
 ubx
 ga
 upt gl
 upt bx Ba gl (In) 1 / 1
 upt bx Ba gl (In) 1 / Dl ubx gl² bv
 pcv¹ ubx cd gl² bv

Chromosome 4

inc j hk
 tg^c

MultichromosomalChromosome 3

or (Standard)
 or (Santa Cruz)
 pr (Standard)
 pr (Arrowhead)
 or pr (Standard)
 or pr (Santa Cruz)
 or pr cv (Standard)

gl: or pr (ST)
 gl²: or (SC)

Other species:

D. hydei San Jacinto Mountains
D. tolteca Guatemala
D. biopaca Guatemala

NEW HAVEN, CONNECTICUT: YALE UNIVERSITY
Department of Zoology

D. americana americana: Independence, Ohio; Western
D. americana texana: Florida
D. ananassae: Cristobal
D. bifasciata: Pavia, normal and sex-ratio

D. busckii: Abingdon, Pa.
D. equinoxialis: Puerto Rico, normal and sex-ratio
D. flavomontana: Yampa River, Colo.
D. funebris: Rexburg, Idaho; Stockholm, Sweden; Upperville, Va.; Yucatan; white
D. gibberosa: South Mexico
D. hydei: New Haven, Conn.; Zurich, Switzerland
D. laticola: Fairbanks, Minn.
D. littoralis: Switzerland
D. melanica
D. montana: Cottonwood Canyon, Utah; LU
D. nebulosa: Haiti, normal and sex-ratio
D. nigromelanica
D. novamexicana
D. paramelanica
D. paulistorum: Belem; Bucamaranga; Cantareiras; Lancetilla; Trinidad
D. pseudoobscura: Pinon Standard
D. repleta: Philadelphia, Pa.
D. simulans
D. virilis: Japan
D. willistoni: Barbados-3; Belem; Recife-3; Recife-6; Recife Pop. 168; ebony; pink; white eye; sex-ratio
Zaprionis vittiger: South Africa

PHILADELPHIA, PENNSYLVANIA: WOMAN'S MEDICAL COLLEGE

D. robusta

A. Homozygous stocks (in addition to those listed in DIS-34):

<u>Stock designation</u>	<u>Chromosome and Arrangement*</u>			<u>Origin of wild strain</u>
	<u>X</u>	<u>2</u>	<u>3</u>	
IPK 22A7	11	SS	SS	Pokagon State Park, Steuben County, Indiana
IPK 61C5	11	SS	SS	"
IPK 108B2	S1	3S	S1	"
IPK 108B6E	S1	3S	SS	"
IPK 117B7D	11	3S	S1	"
IPK 122G1	S1	3S	SS	"
IPK 122K3C	12	3S	SS	"
IPK 209D2D	S1	SS	SS	"
IPK 227F	11	S1	SS	"
OBH 158A1	12	SS	SS	Broadview Heights, Cuyahoga County, Ohio
OBH 171B1D	11	SS	SS	"
OBH 178C,D	1S	SS	SS	"
OW 75C2,5	SS	SS	SS	Wooster, Wayne County, Ohio
OW 92B2A	12	SS	SS	"
OW 101E5	1S	SS	SS	"

*Following notation in Carson, H. L., 1953, Genetics 38:168.

B. Wild strains (not inbred): Ten or more each from Alabama, Indiana, Ohio, Mississippi, and South Carolina.

RALEIGH, NORTH CAROLINA: NORTH CAROLINA STATE COLLEGE
Department of Genetics

D. arizonensis

4 strains from Tucson, Arizona:

50 isofemale strains collected October and November, 1961:

Tucson, Arizona (2)
Catalina Mountains (8,000 ft.), Arizona (1)
Patagonia, Arizona (6)
Magdalena, Sonora, Mexico (29)
Cornelio, Sonora, Mexico (1)
Hermosillo, Sonora, Mexico (10)
Desembogue, Sonora, Mexico (1)

Mutant strains:

White eye - chromosome 1
Lobed eye - chromosome 3

D. psuedoobscura

isofemale strains:

Bryce, Utah (8)
Ferron, Utah (6)
Gunnison, Colo. (6)
Lemon Cave (9)
Mather, Calif. (8)
Mono, Calif. (7)
(74 inbred lines from the above strains)
(multiple-isofemale cage populations from each locality above)

New isofemale strains:

Patagonia, Arizona (1) 1961.
San Felipe, B. Calif., Mexico (1) 1961
Chiriaco Summit, California (2) 1961

Other species

D. aldrichi - Hermosillo, Sonora, Mexico (1961)
D. americana
D. hamatofila
D. hydei
D. longicornis
D. mojavensis - Chocolate Mts., California
D. mulleri - Austin, Texas
D. mulleri - (G-207 - 4th chromosome inversion)
D. persimilis
D. putrida - Raleigh, N. C.
D. ritae
D. simulans
D. texana
D. virilis (3 strains)

AUSTRALIA

Sydney, New South Wales: The University of Sydney

Addition to list in DIS 34:62:

"D. husckii": Sydney; Melbourne

BRAZIL

Pôrto Alegre: Universidade do Rio Grande do Sul, Instituto de Ciencias Naturais

D. willistoni

Wild strains from: Praia do Leste (Paraná), Ilha das Cobras (Paraná), Col. São Pedro (Rio Grande do Sul), Itapeva (Rio Grande do Sul), Manaus (Amazonas), Tubarão (Sta. Catarina), Maranguapé.

Chromosome I

w^ey sn In ru (analyzer stock)

yellow

w,

w^h

sepia

w^elz

Chromosome II

abb bw (analyzer stock)

SHkabbbw (In)/lethal (analyzer stock)

abbpw bw

Em abbbw/abbbw

20 wing eye and other mutants from natural populations unirradiated or irradiated with Co60.

Chromosome III

pink (analyzer stock)

Δ pink (In)/lethal (analyzer stock)

ebony

ebony mixed with other mutant

30 other of the same origin as those listed for Chromosome II.

Several "semilethals" and "normal" viable strains, homozygous for the second and third chromosomes.

Other Species

D. ananassae - Cassarongongo (Bahia), Tabatinga (Amazonas), Sacavem, Boa Viagem (Maranhão), Belém (Pará).

D. bandeirantorum - Itatiaia (Rio).

D. bocainensis - Praia do Leste, Ilha das Cobras (Paraná), Pitanga (Bahia), Eldorado (R.G.Sul), Jacú (Sta. Catarina), Belém (Pará).

D. capricorni - Itatiaia (Rio), Eldorado (R.G.Sul), Ilha das Cobras (Paraná).

D. cardini - Costa Rica H 15-1, Panamá H 7918, Belém (Pará), Eldorado (R.G.Sul), Pedras (Bahia).

- D. equinoxialis - Belém, Içana, Tefé (Amazonas).
D. fumipennis - Pedras (Bahia), Paranaí (Paraná).
D. gaúcha - Cordoba (Argentina) (from prof. Brncic).
D. immigrans - Cassarongongo (Bahia).
D. insularis - Islands of St. Kitts and Guadabepe (from prof. Dobzhansky).
D. kikkaway - Eldorado (R.G.Sul).
D. nebulosa - Lima, Tingo Maria (Perú), Natal (R.G.Norte), São Luiz (Maranhão),
 Angra dos Reis (Rio), Eldorado (R.G.Sul), Guarapará (Espírito Santo),
 Pitanga, Pedras (Bahia).
D. neocardini - Angra dos Reis (Rio).
D. neoleptica - Itatiaia (Rio).
D. neomorpha - Trinidad.
D. paulistorum - Colônia S. Pedro (R.G.Sul), Ilha das Cobras, Praia do Leste (Paraná),
 Manaus (Amazonas), Florianopolis (Sta. Catarina), Belém (Pará),
 Sapé (Minas), Coroico (Bolivia), Tarapoto, Tingo Maria (Perú),
 San Salvador (El Salvador), Bucaramanga (Colombia), Trinidad
 (from prof. Dobzhansky).
D. pallidinennis - Eldorado (R.G.S.).
D. pararepleta - Eldorado (R.G.Sul), Ilha das Cobras, Paranaí (Paraná), Jacú
 (Sta. Catarina), Pedras (Bahia).
D. pavani - Vallenar Vina del Mar (Chile).
D. polymorpha - Eldorado, Itapeva (R.G.Sul), Ilha das Cobras (Paraná), Jacú
 (Sta. Catarina), Pedras (Bahia), Boa Viagem (Maranhão).
D. prosaltans - Eldorado (R.G.Sul), Pitanga (Bahia), Praia do Leste (Paraná),
 Guarapará (Espírito Santo), Jacú (Sta. Catarina), Boa Viagem
 (Maranhão), Belém (Pará).
D. saltans - Itatiaia (Rio), Pitanga (Bahia).
D. simulans - Eldorado (R.G.Sul), Angra dos Reis (Rio), Pedras (Bahia), Tabatinga
 (Amazonas), Boa Viagem (Maranhão), Porto Platon, Serra do Navio
 (Território do Amapá).
D. sturtevant - Ilha das Cobras, Praia do Leste, Paranaí (Paraná), Pedras (Bahia),
 Itatiaia (Rio), Belém (Paraná), Boa Viagem, Sacavem (Maranhão).
D. tropicalis - Palma, Maranguapé (Brazil), Trinidad 330.
D. tropicalis cubana - Townsend.

COLOMBIA

Bogotá: Universidad de Los Andes

South American Species:

- D. equinoxialis: Tefe (State of Amazonas)
D. prosaltans: Cantareira (State of S. Paulo); Sangre Grand (Trinidad)
D. sellata: Cuba; Huickehugan (Mexico)
D. simulans: Aspra and Pavia (Italy). Stocks selected for tumor manifestation:
 tu A; tu B1; tu B3; tu C; tu Aspra
D. sturtevant: Puerto Rico; Cantareira (State of S. Paulo)

FRANCE

Lyon (Rhône): Laboratoire de Zoologie expérimentale,
Faculté des Sciences, 16, quai Claude Bernard

Other species: D. funebris
D. buskii

GERMANY

Berlin-Buch: Deutsche Akademie der Wissenschaften zu Berlin
Institut für Experimentelle Krebsforschung,
Genetische Abteilung, Lindenberger Weg 70

Other Species:

80	<u>D. funebris</u>	84	<u>D. funebris</u> wy : III
81	<u>D. funebris</u> ev : 1. Chromosom	85	<u>D. simulans</u> : v
82	<u>D. funebris</u> st : autosomal	86	<u>D. virilis</u> : wild
83	<u>D. funebris</u> ci : I	87	<u>D. busckii</u>

Berlin-Buch: Institut für Medizin und Biologie, Genetische Abteilung
 and

Berlin-Dahlem: Institut für Genetik der Freien Universität Berlin

Note: Additions and corrections to the list in DIS 34 (p. 64)

Lost: 102 D. repleta: wild

Additions: D. funebris

Chromosome 1

Chromosome 3

105 ci

106 wy

Tübingen: Max Planck-Institut für Biologie

D. busckii
D. hydei
D. miranda
D. persimilis
D. pseudoobscura
D. simulans
D. virilis

ISRAEL

Jerusalem: Hebrew University of Jerusalem

D. immigrans Brisbane
D. immigrans Israel
D. simulans
D. subobscura Küsnacht
D. subobscura Eilon (Israel)

ITALY

Milano: Universita' di Milano, Istituto di Genetica

Drosophila simulans

Stocks selected for tumor manifestation:

Wild type from Pavia
 Wild type from Aspra

tu A
 tu B1

tu B3
 tu C
 tu Aspra

JAPAN

Tokyo: Tokyo Metropolitan University
Department of Biology

D. ananassaeWild stocks

- | | |
|---|-----------------------------|
| 1. Texas | 21. 2L-A; 3L-A ^M |
| 2. TL ₁ | 22. 3L-A ^{C104} |
| 3. TL ₃ | 23. 2L-B; 3L-A ^H |
| 4. TL ₄ | |
| 5. TL ₃₋₄ | <u>Mutants</u> |
| 6. TL ₃₋₁₁ | 25. st f |
| 7. Barro Collorado, Panama 69
(Low elevation) | 26. ru |
| 8. Barro Collorado, Panama 74
(Low elevation) | 27. pxd |
| 9. Turrialba, Costa Rica 101
(High elevation) | 28. st f ru ² |
| 10. Turrialba, Costa Rica 104
(High elevation) | 29. st f se |
| 11. Turrialba, Costa Rica 125
(High elevation) | 30. y f |
| 13. Christobal, Panama | 31. b |
| 15. Baton Rouge, Louisiana | 32. Bn-R |
| 16. Hawaii-H | 33. bw-R |
| 17. Calcutta, India | 34. fu |
| 18. 2L-A ^H | 35. j |
| 19. 2L-B ^H | 36. px |
| 20. 2L-A; 3L-A ^H | 37. S |
| | 38. se |
| | 39. sn |
| | 40. wy |

D. bifasciataWildAutosomal

- | | |
|-------------------------------|----------------|
| 1. Akkeshi (3 strains) | ag |
| 2. Asakawa (1) | ar |
| 3. Gotokuji (1) | ar ic |
| 4. Hakkoda (3) | ar ob |
| 5. Kisokomagatake (1) | ar ro |
| 6. Kitazawatoge (1) | arp |
| 7. Kumotoriyama (3) | bn |
| 8. Meakan (1) | ca ro |
| 9. Nishitappu (2) | cn |
| 10. Ohkurayama (1) | ic |
| 11. Pavia, Italy (1) | ic cn |
| 12. Pfynwald, Switzerland (1) | M ob |
| 13. Shibunoyu (1) | ob |
| 14. Taisetsuzan (2) | orr |
| 15. Tanigawadake (1) | ps yh |
| 16. Tsukubasan (1) | vi |
| | yh (5 strains) |

MutantCytoplasmicSex-linked

a y
f
y

Sex-ratio, Italy
 Sex-ratio, Japan (6 strains)

Other Species

<u>D. americana</u>	Wild	1 strain
<u>D. ambigua</u>	Wild	1 strain
<u>D. auraria</u>	Wild	6 strains (Type A)
<u>D. busckii</u>	Wild	3 strains
<u>D. chinoi</u>	Wild	4 strains
<u>D. funebris</u>	Wild	2 strains
<u>D. hydei</u>	Wild	4 strains
<u>D. immigrans</u>	Wild	5 strains
<u>D. kikkawai</u>	Wild	5 strains
<u>D. lutea</u>	Wild	17 strains
<u>D. miranda</u>	Wild	1 strain
<u>D. novamexicana</u>	Wild	1 strain
<u>D. obscura</u>	Wild	1 strain
<u>D. pseudoobscura</u>	Wild	5 strains
<u>D. pseudoobscura</u>	Mutant	4 strains
<u>D. pulchrella</u>	Wild	1 strain
<u>D. simulans</u>	Wild	1 strain
<u>D. suzukii</u>	Wild	4 strains
<u>D. takahashii</u>	Wild	37 strains
<u>D. tristis</u>	Wild	1 strain
<u>D. virilis</u>	Wild	6 strains
<u>D. virilis</u>	Mutant	5 strains

NETHERLANDS

Groningen: State University of Groningen, Genetical Institute, Haren (Gr)

Stock List - Species:

<u>D. pseudoobscura</u>	various strains homozygous for AR. CH. ST.
<u>D. immigrans</u>	(wild 1959)
<u>D. mercatorum</u>	(wild 1961)
<u>D. repleta</u>	
<u>D. hydei</u>	(wild 1961)
<u>D. busckii</u>	(wild 1961)

SOUTH AFRICA

Johannesburg: University of the Witwatersrand, Department of Zoology

D. persimilis

Porcupine Flat

D. pseudoobscura

Several strains of Standard, Arrowhead, Chiricahua, Treeline, Pikes Peak, and Santa Cruz

gl
or
se
tb b v se pp
v

D. simulans

Drakensberg
Free State
Inhaca Island
Johannesburg
Kalahari
Limpopo

Mkuzi Game Reserve
Nyasa Lake
Stellenbosch
Umgazi River
West Rand
Zoutspansberg

Other species

D. busckii: Inhaca Island
D. funebris: Witwatersrand, Natal
D. hydei: Inhaca Island, Natal
D. nebulosa: Brazil
D. séguyi: Limpopo River
D. willistoni: Brazil
se

D. yakuba: Northern Transvaal,
Inhaca Island
Zaprionus ghesquierei
Z. tuberculatus: various strains
Z. vittiger: various strains

SPAINBarcelona: Universidad de Barcelona, Centro de Genética Animal y Humana

D. ambigua. Several Spanish stocks.
D. busckii. Barcelona.
D. buzzatii. Armentera (Spain).
D. funebris. Several Spanish stocks.
D. hydei. Barcelona.
D. immigrans. Barcelona.
D. mercatorum mercatorum. Barcelona.

D. mercatorum pararepleta. Jijuca (Brazil).
D. phalerata. Several Spanish stocks.
D. repleta. Barcelona.
D. simulans. Barcelona.
D. subobscura. Several Spanish stocks;
mutant stocks.
D. testacea. Barcelona.
Parascaptomyza disticha. Barcelona.

Madrid: Centro de Investigaciones Biologicas, Laboratorio de Genética

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

SWEDENUppsala 7: University of Uppsala, Institute of Genetics

Drosophila littoralis
Drosophila hydei
Drosophila funebris
Drosophila subobscura

RALEIGH, NORTH CAROLINA: NORTH CAROLINA STATE COLLEGE, DEPARTMENT OF GENETICSD. arizonensis

l: lobed eye. Coulson, K59. Third chromosome recessive, spontaneous in A7.7a stock. Eye reduced in size, with notch in anterior edge. Eye reduction variable from slight notch to nearly eyeless. Eye color slightly duller and darker than wild type.

Alderson, T., and M. Pelecanos.

The mutagenic activity of ethylating agents by the larval feeding method in the presence and in the absence of ribonucleic acid.

It has been shown (Alderson, 1960a) that formaldehyde exhibits no mutagenic activity towards Drosophila melanogaster, by the larval feeding method, unless ribonucleic acid is present in the treatment medium, whereas the presence or absence

of ribonucleic acid has no influence on the mutagenic activity of urethane. A 6-amino group alkylation of adenylic acid by formaldehyde in the treatment medium has been shown (Alderson, 1961) to be the responsible reaction for the mediation of the mutagenic activity of formaldehyde in Drosophila: adenylic acid may be present as any of its free mononucleotides or bound in the ribonucleic acid polynucleotide, but the mutagenic activity of formaldehyde is completely dependent on the presence of adenylic acid within the treatment medium.

The responsible mutagenic alkylation of adenylic acid by formaldehyde obviously does not occur to a sufficient extent in vivo for the reaction product to significantly increase the mutation rate. Yet, even under physiological conditions, alkylation would be expected to take place. One reason for lack of effect may be that the extent of the reaction required is not attained under physiological conditions; for example, in the case of formaldehyde, dimerisation by methylene bridging of adenylic acid is postulated as the effective mutagenic alkylation (Alderson, 1960b). In the case of other mutagenic alkylating agents, the alkylation is obviously attained in vivo. Szybalski (1961) has shown that the mutagenic activity of triethylene melamine in bacteria is probably mediated by in vivo alkylation of precursor thymidylic acid since the in vitro reaction product of triethylene melamine and thymidylic acid is found to be mutagenic.

In vitro alkylation of purine and pyrimidine bases is thus implicated as an important mechanism in chemically-induced mutagenesis, especially in view of the mutagenic activity of several N-methyl xanthines in bacteria (Novick and Szilard, 1952), and that of caffeine (1,3,7-trimethyl xanthine) in Drosophila (Andrews, 1959).

Since mutagenicity by alkylating agents may be mediated by both in vivo and in vitro reactions, an attempt to separate these reactions by culturing Drosophila larvae in media containing or lacking purine and pyrimidine components was carried out. Further, the recent chemical characterisation and isolation of the products of alkylation of the nucleic acids and their constituents nucleotides (Lawley, 1960) might reveal the essential in vitro alkylations concerned in mutagenesis.

Two ethylating agents, diethyl sulphate and ethyl methanesulphonate, were chosen for a preliminary study. Both ethylating agents were tested for mutagenic activity in the presence (0.4%) and in the absence of ribonucleic acid using a chemically defined and aseptic treatment medium (see Alderson, 1960a, for the composition). Diethyl sulphate (B.D.H. 99-101 per cent) and ethyl methanesulphonate (supplied as a 0.5 M saturated solution by the Chester Beatty Research Institute) were added when the temperature of the treatment medium was 60° C. In most experiments Oregon-K eggs were sterilised using Sang's (1956) method and transferred as newly hatched larvae to the treatment medium for their entire larval life; in one experiment the larvae were treated for the first 48 hours of larval life and then transferred onto ordinary laboratory food to complete their development. Hatching males were individually examined for sex-linked recessive lethal mutations by the Muller-5 technique; two broods were used, each with two females for three days.

Neither ethylating agent showed a significant difference in its mutagenic activity whether ribonucleic acid was present or absent from the treatment medium. (Table 1.)

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Table 1.

Medium	Mutagen	Concentration	Length of treatment of larvae	Larval development time in days	Per cent survivors	First Broods				Second Broods			
						Total	Lethals	%	X ²	Total	Lethals	%	X ²
No RNA	Diethyl sulphate	$3.2 \times 10^{-4}M$	0-48 hrs.	9-9.5	50.25	337	2	0.6		325	5	1.54	
With RNA				8-8.5	81.0	364	1	0.28	0.4	310	3	0.96	0.37
No RNA	Diethyl sulphate	$2.0 \times 10^{-4}M$	Entire larval life	11-12	54.8	432	8	1.85		438	6	1.4	
With RNA				9-9.5	57.5	434	15	3.45	2.16	332	10	3.0	2.5
No RNA	Diethyl sulphate	$2.6 \times 10^{-4}M$	Entire larval life	15-16	36.0	378	8	2.10		266	3	1.13	
With RNA				11	73.0	406	18	4.40	1.7	202	6	3.0	2.0
No RNA	Ethyl methane-sulphonate	$5.0 \times 10^{-6}M$	Entire larval life	14-16	48.5	1020	62	6.10		1416	88	6.20	
With RNA				10	61.25	1118	56	5.00	1.7	1380	74	5.36	0.93

Band, H. T. Comparison of viabilities and variances for homozygous chromosomes and heterozygous combinations using different matings in the test cross generation.

To investigate the genetic load in many species of *Drosophila*, it is possible to study the wild chromosomes in homozygous condition and in random combinations. Derivation of the chromosomes often presents the investigator with a choice, however.

Matings in the test cross generation may be made such that two genotypes are produced in the resulting progeny of four genotypes. The latter method, introduced by Wallace (1956), produces a "control" genotype in every culture--a type free of the wild chromosome(s) being studied. Thus, chromosomes and combinations can be scored by the usual % wild type viability measurement, by ratio of wild type to "control"; or by other ratio measurements which can be devised.

In the latter method, however, Spassky et al (1960) found the ratio of wild type to control quite variable so used % wild type viability as the measurement of chromosome performance. Other investigators, Dobzhansky, Krimbas, and Krimbas (1960) and Greenberg and Crow (1960) have used other ratios obtainable among the four genotypes, though the former also used the ratio of wild type to control.

Counting four genotypes takes longer than counting two per culture, and the variability accompanying the +/-control ratio limiting the usefulness of this measure. At the suggestion of Dr. P. T. Ives, chromosomes from the August 1960 collection from the S. Amherst *D. melanogaster* population were used to compare the two types of test crosses.

Since only 52 of the 274 wild males received proved fertile, data on the frequency of drastic (1e + sle) chromosomes would be of questionable value, so is omitted. Derivations followed standard procedure. For the test crosses two types of matings were made for each chromosome line: Cy x Cy and Cy x Bl, with one culture being scored for each type of mating. Analysis was confined to second chromosomes only.

Of the nondrastic chromosomes recovered (% +/- greater than 17% by Cy x Cy matings; % +/- greater than 12 1/2% by Cy x Bl matings), 13 chromosomes were chosen and 7 combinations devised to compare: (1) genetic, environmental, and sampling variances of viabilities obtained with the two methods; (2) the accuracy of viability measurement using three replicate cultures versus that obtained by counting only one culture.

Matings were as before: Cy x Cy and Cy x Bl with 3♀ and 3-4♂ as parents in each line or combination. Parents were transferred every 24 hours for three days to give three replications. Counts were made three times during the emergence period.

One chromosome, 2912, was discovered later to be semilethal by Cy x Cy tests, though of low quasinormal viability by Cy x Bl tests. Another chromosome, 2935, was sterile in Cy x Cy repeat tests. Hence, computations of average viability and variances for homozygous chromosomes have been made in two ways for the Cy x Bl tests: (a) including all thirteen chromosomes; (b) excluding 2912 and 2935.

The formulae given by Dobzhansky and Spassky (1953) and Wallace and Madden (1953) have been used to partition total variance (T) into its environmental (E), genetic (G), and sampling (S) components. Comparison of variances for the two methods are shown in Table 1. Total number of flies counted in each type of test cross is also included.

Table 1

	mean viability	T	E	S	G	no. counted
Heterozygotes						
Cy x Bl	25.9%	13.25	33.02	5.49	-25.26	2860
Cy x Cy	34.4%	23.87	32.90	8.77	-17.79	2036
Homozygotes						
(a) Cy x Bl	22.2%	40.53	12.99	4.75	22.79	5054
(b) Cy x Bl	22.6%	31.49	8.24	4.76	18.49	4354
Cy x Cy	30.1%	51.79	25.20	9.47	17.12	3358

In both heterozygotes and homozygotes total and sampling variances are lower using the Cy x Bl method. Environmental variances in heterozygotes are the same for either method, but lower in homozygotes with the Cy x Bl type of mating. In heterozygotes, genetic variances are likewise lower using these matings. In homozygotes genetic variances are approximately the same for either method.

For both methods heterozygotes show highest viability and least genetic variance--the typical picture when heterozygotes and homozygotes are compared. However, for this particular set of chromosomes, larger environmental variances are found among heterozygotes. This is sometimes observed in the data of others.

Data on the means of homozygous chromosomes tested are shown in Table 2. Data on 2912 are included in the table. Mean viability of 2935 was 24.1%, original viability measurement 25.0% in Cy x Bl tests. Correlations between average viability from the three replicates per chromosome line (\bar{x}_3) and viability previously computed from scoring the original test cross culture (x_0) are similar for both methods, 84.9% for Cy x Bl matings and 82.5% for Cy x Cy matings.

Table 2

Line:	2905	2908	2912	2913	2915	2916	2920	2927	2928	2929	2936	2943
Cy x Bl												
\bar{x}_3	26.2	22.1	15.0	24.2	26.1	18.1	21.0	20.9	21.1	17.5	26.6	24.8
x_0	29.5	24.7	15.4	28.0	26.2	15.2	20.1	25.2	24.3	13.5	26.4	21.0
Cy x Cy												
\bar{x}_3	32.5	29.6	10.0	32.3	29.3	29.0	34.2	33.8	29.2	28.1	19.3	33.3
x_0	28.8	29.1	9.1	29.8	28.1	20.7	31.0	27.3	25.9	22.9	18.3	24.7

Hence, for scoring viability for each chromosome derived homozygously from a natural population, counting only one culture appears satisfactory as an index of performance and enables the rapid computation of the frequency of drastic (le + sle) in the population. Either method gives comparable results.

However, this tells nothing about performance in heterozygous condition, nor enables comparisons between heterozygotes and homozygotes. For this, the Cy x Bl method, with replicated observations, is to be preferred, primarily because variances of viability measurements are generally less than, and certainly no more than equal to, those obtained with Cy x Cy scoring. In addition, a variety of ratio comparisons can be made. So the Cy x Bl cross for D. melanogaster, or the method by which four genotypes are scored among the progeny, is the more versatile. It is this method which has been employed in subsequent analyses of chromosome samples from this population.

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Band, H. T. Preliminary evidence that variation in temperature affects viability of heterozygous wild type flies.

The correlations between drastic (le + sle) frequency and temperature variables of range and mean detected by Band and Ives (1961) suggest a dynamic relationship between environment and genetic structure of the S. Amherst

D. melanogaster population. To determine if environment could influence viability of different kinds of heterozygotes, a small preliminary experiment was conducted using second chromosomes derived from the August 1960 collection. Crosses between chromosome lines were of two types: Cy/i x Cy/j and Cy/i x Bl/j such that the wild type (i/j) progeny were known to be genotypically drastic/drastric (d/d), drastic/nondrastric (d/nd) or nondrastric/nondrastric (nd/nd). Five crosses were constructed to yield d/d

progeny, 12 to give d/nd progeny and 6 to give nd/nd progeny. The same crosses were tested by both methods, and 2 Cy ♀♀ and 2-3 Cy or Bl ♂♂ used for each cross. Parents were transferred every 24 hours for 4 days to give 4 replicate cultures. Oviposition was at room temperature. Replicates B and D were kept at 25° C.; A and C were transferred between 17° C. and 25° C. constant temperature incubators. The F17/25 environment corresponds to a narrow range environment according to range data given in Band and Ives (1961). Transfers were made to alternate temperatures every 24 hours. Progeny were counted 3 times during the emergence period. Wild type viabilities for the 3 kinds of heterozygotes are shown in Table 1. A = average viability computed from Cy x Cy matings; B = average viability computed from Cy x Bl matings; C = the ratio of wild types produced to $1/2 (Cy + Bl) + 1$. The ratio is based on the total number of flies within observed genotypes in each of the 3 heterozygous combinations.

Table 1

Viability of the wild type progeny in the two environments						
	C25			F17/25		
	A(%)	B(%)	C	A(%)	B(%)	C
d/d	30.6	20.5	0.82	33.0	26.1	1.06
d/nd	35.4	24.0	0.90	33.3	29.1	1.15
nd/nd	33.8	26.1	0.98	32.3	25.9	0.91

Drastic/drastic (d/d) heterozygotes have lowest viability at C25 but improve in the narrow range F17/25 environment. In this latter environment, however, nd/nd wild types have lowest viability. Otherwise, the two methods show slightly different results. By Cy x Cy crosses, d/nd heterozygotes appear decidedly superior at C25, but at F17/25 little difference is noted between different heterozygotes, though nd/nd is lowest in viability. By Cy x Bl methods, the different genotypes react differently in each environment; viability changes determined by % wild type viability are substantiated by ratio results.

Due to the small number of chromosomes involved and the small number of progeny realized, chi-square comparisons have been made using total number of flies and total number of wild types obtained in each method for the 3 kinds of heterozygotes. Even so, only 16,831 flies have been counted in the entire experiment.

Between environments, three comparisons are of interest: change in viability of d/d heterozygotes, of d/nd heterozygotes, and of nd/nd heterozygotes. For Cy x Bl crosses, d/d viability has improved significantly in the F17/25 environment from the level shown at C25, likewise d/nd. For both $P < .005$. No difference is detected between nd/nd viabilities at the two temperatures. For Cy x Cy matings, only the viability of d/d shows significant changes between the two environments, with $.025 < P < .05$.

Within environments both methods indicate that the different heterozygotes are significantly different in viability at C25; $P < .005$ for each method. At F17/25 only the results from the Cy x Bl crosses indicate significant differences between viabilities of the different heterozygotes. Again $P < .005$.

The outcome of this small experiment thus indicates: (1) that environment can influence heterozygous viabilities and so may affect the genetic structure of the population through selection at the heterozygote level; (2) heterozygotes carrying drastic chromosomes (either d/d or d/nd) can be favored in narrow range environments; (3) again indicates the Cy x Bl method to be more sensitive to genotypic differences than the Cy x Cy method.

In contrast to the viable mutant, dumpy, which is genotypically designated as dp^{ov1} , or ov^1 , the double mutant $dp^{o2} dp^{v2}$ or o^2v^2 (which was prepared from the heterozygote, $ed dp^{v2} / dp^{o2} cl$) manifests a more extreme wing and thorax effect. Furthermore, the mutant ov^1 is viable with all lethal members of the dumpy series, but the double mutant o^2v^2 is inviable with some of the lethal alleles. For this reason a study of the dumpy lethals was made with o^2v^2 at 22° C. and at 28° C. The results in Table 1 strongly suggest that these lethals can be classified in three ways. In one group the lethal acts as a dominant in the compound with o^2v^2 at both temperatures. The absence of a change in activity with temperature for the lethals in this group is characteristic of that class of mutants designated as amorphs (lacking any detectable activity), in Muller's classification of gene action (Muller, 1932, Proc. 6th Int. Congr. Genet. 1:213-255). The alleles in the first group include olv^w , olv^{54e} , lv^1 , and lv^{x3} . The second group of alleles shows a greater manifestation of lethality at 28° C. than at 22° C. These might be considered hypomorphic, with a partial normal activity of the lethal allele restored at lower temperature, just as is the case for the (o) and (v) effects. These mutants include l^m and ol^m . A third group of mutants is more difficult to classify. For example, olv^{bm} shows little difference in temperature response with a suggestion of a reversal of sensitivity. The mutant ol^s , in contrast to ol^m , is much more viable with o^2v^2 at 28° C.

When the lethals are tested in compound with the allele dp^{cm2} or cm^2 the response is quite unexpected -- cm^2 unquestionably reverses the manifestation of the lethal at these temperatures. This is not the case, however, for the (o) and (v) effects manifested by cm^2 in its viable compounds, which show an exaggerated mutant expression at higher temperatures.

The developmental basis for this variation in expression of the lethal effect is not apparent from these data. However, the o^2v^2 and cm^2 alleles provide a useful classifying technique for phenotypically similar mutants and for determining the quantitative degree of expression of the lethal effect in the dumpy series.

Table 1. Lethal expression with temperature in the dumpy allelic system

P ₁ Cross	28° C.			22° C.		
	non-Cy	Cy	% viable	non-Cy	Cy	% viable
$\frac{olv^1}{Cy} \times o^2v^2$	0	351	0	0	70	0
$\frac{olv^w}{Cy} \times o^2v^2$	0	202	0	0	254	0
$\frac{olv^{54e}}{Cy} \times o^2v^2$	0	249	0	0	478	0
$\frac{lv^1}{Cy} \times o^2v^2$	0	518	0	0	268	0
$\frac{lv^{x3}}{Cy} \times o^2v^2$	0	24	0	0	29	0
$\frac{l^m}{Cy} \times o^2v^2$	2	872	0.31	4	152	2.56
$\frac{ol^m}{Cy} \times o^2v^2$	1	259	0.38	26	99	20.8
$\frac{ol^s}{Cy} \times o^2v^2$	13	73	15.1	43	100	30.7
$\frac{olv^{bm}}{Cy} \times o^2v^2$	3	236	1.25	1	584	0.17
$\frac{olv^w}{Cy} \times cm^2$	87	304	22.2	5	478	1.03

(Carlson and Falk, Table 1--continued)

P ₁ Cross	28° C.			22° C.		
	non-Cy	Cy	% viable	non-Cy	Cy	% viable
$\frac{olv^{54e}}{Cy} \times cm^2$	27	211	11.3	13	594	2.14
$\frac{olv^{bm}}{Cy} \times cm^2$	76	409	15.7	2	518	0.39
$\frac{olv^1}{Cy} \times cm^2$	44	426	13.3	4	714	0.56

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Castiglioni, M. C., and Raimondi G. Rezzonico.
Cultivation of *Drosophila* cells in synthetic medium.

the lymph gland, both in larval stage. For studying the ganglia the following wild stocks have been chosen: Barese, S. Maria and Aspra 52. For the lymph gland, stocks S. Maria, yw and Chieti, where the structure of the gland is very similar (lobes of the first pair of loose structure but fairly well developed).

Cultures were set in a drop of medium put on the coverslip, which is kept underneath, while the slide is above. The culture medium for Insects according to Kuroda and Tamura (1956) has been used.

The ganglia have been kept in culture for 28 days; the lymph gland for 21 days. In both cases, the culture medium has been replaced every 7th day. Each time both the organ and the cells released from it have been pipetted and transferred in a drop of fresh medium. Cells adhering to the coverslip have been washed, stained with May Grünwald-Giemsa, classified and counted.

Using this method it is possible to detect stable differences between the stocks, as far as the behaviour of the organ and of the cells is concerned. Since the culture conditions are strictly controlled, cultivation in vitro permits to recognize entirely new physiological properties at cellular level, which are apparently genetically controlled.

Chen, P. S., and C. Bachmann-Dien.
Studies on the transamination reactions in the larval fat body of *D. melanogaster*.

In our previous studies on the biochemical effects of lethal factors in *D. melanogaster* various abnormal patterns of free amino acids in

the lethal homozygotes were reported. Since the amino acid pattern is characteristic for each mutant, it seems that the effect is locus-specific (for a general review, see Hadorn 1955). In order to obtain more insight into the intermediary metabolism of amino acids in *Drosophila* and as a basis for comparison between lethal and normal individuals, studies on the transaminase activities in the wild type have been carried out in our laboratory. Fat bodies from ten +/- larvae aged about 96 hours (at 25° C.) were dissected out in ice-cold insect Ringer's solution and homogenized. The homogenate was incubated in a sodium-potassium-phosphate buffer solution (0.067M; pH 7.56) at 38° C. with one keto acid (α -ketoglutaric acid, oxalacetic acid, pyruvic acid or glyoxylic acid) as the amino acceptor and one amino acid (aspartic acid, glutamic acid, α -alanine, leucine, threonine, glycine, valine or arginine) as the amino donor. In addition, pyridoxal phosphate was added as

	Days after irradiation											
	1	2	3	4	5	6	7	8	9	10	11	12
Adults 12 h. control	% 0'8	1'7	0'4	0'9	0'6	0'4	0'6	0'6	0'6	0'7	0'6	1'4
Adults 6 d.	% 14'4	11'8	14'2	12'7	26'2	33'0	46'6	52'0	62'6	32'0	18'5	10'7
Adults 12 h.	% 6'7	6'7	7'0	11'0	29'8	38'0	39'8	44'0	7'5	1'3	1'1	0'9
Pupae 48 h. (500 r.)	%		10'8	16'5	24'6	24'0	22'6	32'6	5'1	1'7	1'3	
Prepupae 2 h.	%				40'5	55'5	58'5	50'0	8'1	1'5	1'1	
Larvae 72 h.	%					42'2	46'0	33'6	2'0	1'2	2'4	
Larvae 48 h.	%						24'0	2'4	1'4	1'2	0'9	
Larvae 24 h.	%							1'7	1'1	1'4	1'2	

From these data it appears quite likely that meiosis (beginning with pupation) has been sampled between the fourth and fifth days after irradiation. From the displacement of the peak value from day 7 to day 9 a. i. it is concluded that it may correspond to the most damaged cytes located at the end of the sampled cytes. It is also apparent that the value corresponding to the 8th day after irradiation includes a higher proportion of cytes as the number of germ cells increases during development. The subsequent drop in mutation rate coincides with the appearance of gonial mitosis (24 h. old larvae) and germ cell mortality. There is an apparent delay of the speed of the spermatogenesis probably due to the increase of the number of germ cells during development.

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Gardner, E. J., and A. M. Hansen.
Further studies on the transfer of the tumorous head maternal effect in D. melanogaster by injection.

Gardner, Turner and Berseth (Genetics 45:905-913, 1960) injected extract prepared from tumorous head females into the thorax of Lausanne females and mated them with tu-h males. Eighteen

per cent of the progeny expressed the tumorous head trait as compared to less than four per cent for the controls from uninjected females. These results were interpreted to indicate that the maternal effect had been transferred by the injection of tu-h female extract.

When this line of investigation was resumed several months later it was not possible to transfer the maternal effect by injections of tu-h female extract, tu-h female hemolymph, or tu-h oöplasm into Samarkand and Canton wild-type females. Experiments involving the original Lausanne stock were then repeated. Tumorous head flies appeared among the progeny of all the control crosses as well as among those of injected females. The Lausanne stock in our laboratory now carries the tu-1 gene or an allele of tu-1 that has presumably entered through mutation or contamination. A Lausanne S stock obtained from the California Institute of Technology was also found to carry tu-1 or an allele of tu-1, but the tu-1 gene was not present in a Lausanne S stock obtained from Johns Hopkins University. More than two thousand progeny were produced from 16 females of this stock that had been injected with tu-h female extract and mated with tu-h males. Less than one per cent of these expressed the tu-h phenotype. This result was comparable with that of the controls.

Another series of experiments was designed in which the extract was prepared in a more concentrated form and maintained in an ice bath during preparation and injection. Injections were made into either the thorax or abdomen of Samarkand and Cockaponsett females. Progeny from the injected flies did not express the tumorous head phenotype in greater proportions (less than one per cent) than the controls. The transfer of the maternal effect was not accomplished under the conditions of this experiment.

Glass, H. Bentley. The mutagenic effect of a 5-r dose of x-rays.

The experiment in progress, reported in DIS-33 and DIS-34, has been completed with scoring of the mutations from 50

exactly balanced and coded control and irradiated series of tests. The total number of individuals scored is 1,360,948. As previously reported, each parent is exposed to 5r (dose rate about 40r/min), irradiated males are crossed with irradiated females, and dominant Minutes are scored in the F₁. In 32 of the 50 replications of the experiment, the Minute mutations in the treated series have exceeded the number in the control series. The cumulative results are as follows:

Minutes in unirradiated control series	334/684,160	0.049%
Minutes in irradiated series	383/676,788	0.057%

The difference between the irradiated and control series is thus 0.008% in contrast to the prediction of 0.005% based on extrapolation downwards from dosages of 1000r and 2000r. A simple X^2 test yields a value, for one degree of freedom, of 3.92: $P = .047$ (when cultures with clusters of more than 3 Minutes are excluded from the data, inasmuch as clusters cannot be produced by the irradiation of mature spermatozoa and oöcytes). When cultures with more than 2 Minutes are excluded, as an alternative statistical correction, the difference is statistically strengthened. $X^2 = 4.60$: $P = .032$

The viability and/or fecundity of the irradiated series is significantly lowered, by 7,540 flies, or 1.1%.

Glassman, E. Some observations on the prune-killer gene.

In DIS 33:136 there was reported a method for selecting back mutations at the prune locus using the following cross:

$$pn \cdot = X \text{ lJ1/sc}^8 \cdot Y; K\text{-pn}$$

The following observations of interest have been made.

1. The main progeny group following irradiation are the half-translocations, which suggests that this system might be used to test the effects of various agents on this group of chromosome aberrations.

2. A second, smaller group, consists of males derived by non-dysjunction in their fathers, which suggests that this system might have applications here, too.

3. It is found that the $pn \cdot = /sc^8 \cdot Y; K\text{-pn}/+$ female progeny die as pupae. This is unexpected since males which are $pn; K\text{-pn}/+$ die in early second instar. This points to an interesting sex difference in the expression of these genes.

Glassman, E., J. D. Karam,
E. C. Keller, Jr., and J. McLean.
Gene dosage relations at the
 $ma\text{-}1$ and ry loci.

Assays of xanthine dehydrogenase in flies heterozygous for ry (ie ry^+/ry^1 and ry^+/ry^2) show that these heterozygotes have about 40-70% of the activity of normal. Thus, the ry mutants appear to

be similar to other genes in which the heterozygote has lower enzyme activity than the homozygous wild-type.

On the other hand, $ma\text{-}1^+/ma\text{-}1$ heterozygotes show an activity of xanthine dehydrogenase which is greater than or equal to the wild-type. It is felt that the increase of activity in heterozygotes is associated with autosomal heterozygosis since males derived from $\pm \times ma\text{-}1$ also show greater activity than \pm . The reason for this increase is not understood but it might be due to an increase in body mass or to an increase in vigor associated with heterozygosis.

That $ma\text{-}1^+/ma\text{-}1$ flies are probably equivalent to $ma\text{-}1^+/ma\text{-}1^+$ is substantiated by the insertion of $ma\text{-}1^+$ -bearing chromosomal fragments derived either from the $T(1;4)B^S$ translocation or the duplication $dp(1;3)B^S3$ into various diploid combinations of $ma\text{-}1^+$ and $ma\text{-}1$. The results demonstrate essentially no difference between the following genotypes:

$ma\text{-}1^+/ma\text{-}1/ma\text{-}1$
 $ma\text{-}1^+/ma\text{-}1^+$
 $ma\text{-}1^+/ma\text{-}1^+/ma\text{-}1^+$

In other words, one dose of $ma\text{-}1^+$ produces an amount of xanthine dehydrogenase activity which is equal to three doses of $ma\text{-}1^+$.

If the analysis of Jacob and Monod on the lac region of E. coli is applicable, then one might conclude from the above that ry is a structural gene for xanthine dehydrogenase and $ma\text{-}1$ is a regulator gene. The regulation is expressed

not through a repressor substance, but through an internal inducer which is not the substrate. However, the fact that the *ma-1* locus does have mutants which form a CRM and which show complementation, both of which are attributes of a structural gene, is not consistent with this hypothesis. Another possibility is that the *ma-1* locus regulates the activation of the already formed enzyme molecule. Many models based on this assumption can be suggested, but none of them are subjectable to experimental analysis at the present time.

Goldberg, A.¹, A. Schalet, and A. Chovnick.
On the lethality of double mutants of *Hn^{r-3}* and various *ry* mutant alleles.

Taira has reported that the double mutant chromosome, *Hn^{r-3} ry* behaves as a recessive lethal (DIS-34). If the synthetic lethality of *Hn^{r-3}* and

ry applied as well to combinations of *Hn^{r-3}* and other rosy alleles, then the lethal effect might be used as the basis for a highly efficient system designed to select for pseudoallelic recombinants at the rosy locus. Moreover, selective systems could be developed for the study of reverse mutation of *Hn^{r-3}* and rosy alleles, and for sex-linked suppressors and dominant suppressors of both *Hn^{r-3}* and *ry*. Consequently, we synthesized five chromosomes bearing *Hn^{r-3}* and each of five different rosy alleles (*ry¹*, *ry²*, *ry⁴*, *ry⁶*, and *ry⁹*) in order to check for lethal effects of the mutant combinations (the three chromosomes with *ry⁴*, *ry⁶*, or *ry⁹* also carried *cu kar*). Since both *Hn^{r-3}* and *ry* affect pterine metabolism, the chemotypes of all genotypic combinations were examined by direct chromatography of heads and abdomens.

The mating scheme, run in parallel for all rosy mutants, was designed to provide an unambiguous answer to the question of the lethality of *Hn^{r-3} ry* double mutants. Heterozygous females were produced, *Hn^{r-3} ry⁺sr/+ ry +*, and back-crossed to *Hn^{r-3} ry⁺sr/Hn^{r-3} ry⁺sr* males. Single male offspring of the phenotype henna, non-stripe (crossovers between *Hn^{r-3}* and *sr*) were crossed to *ry² 126 Sb Ubx/In(3)DcxF* females to distinguish between the crossover classes *Hn - ry*, and *ry - sr*. Males of the former class would be *Hn^{r-3} ry sr⁺/Hn^{r-3} ry⁺ sr* and would be distinguished from the latter class by producing rosy offspring. In those vials which did produce rosy offspring, there was no significant deviation from the expected frequency of rosy offspring. From vials which did produce rosy offspring, *ry Sb Ubx* males (*Hn^{r-3} ry/ry² 126 Sb Ubx*) were crossed to *M34 Dfd ry¹/In(3)DcxF* females, yielding *Hn^{r-3} ry/In(3)DcxF* males and virgin females, which were mated. In the final cross, the expected ratio of *Hn^{r-3} ry/In(3)DcxF* to *Hn^{r-3} ry/Hn^{r-3} ry* progeny is 2:1. The following table shows ratios obtained for all combinations of *ry* mutants grown at 26° C.

	<i>Hn^{r-3} ry¹</i>	<i>Hn^{r-3} ry²</i>	<i>Hn^{r-3} ry⁴</i>	<i>Hn^{r-3} ry⁶</i>	<i>Hn^{r-3} ry⁹</i>
<i>Hn^{r-3} ry¹</i>	229/88				
<i>Hn^{r-3} ry²</i>	357/156	439/152			
<i>Hn^{r-3} ry⁴</i>	442/191	368/160	409/130		
<i>Hn^{r-3} ry⁶</i>	357/190	501/206	319/105	364/0	
<i>Hn^{r-3} ry⁹</i>	449/194	395/193	344/140	334/94	379/105

From the data presented, it can be seen that the double mutant *Hn^{r-3} ry⁶/Hn^{r-3} ry⁶* behaves as a lethal, but all other combinations are viable.

Of some interest are observations made on fluorescent pterines found in the various genotypes. For all rosy alleles tested, the double heterozygotes, $Hn^{r-3} +/+ ry$, appear phenotypically wild type, but exhibit a considerable increase over Oregon-R controls in content of sepiapterine, and the spot containing biopterin and 2-amino-4-hydroxypteridine. Examination of all viable double mutant homozygotes and all combinations of rosy mutant heterozygotes (homozygous for Hn^{r-3}) revealed that they contained large amounts of sepiapterine, a considerable increase in the spot containing both biopterin and 2-amino-4-hydroxypteridine, and were lacking isoxanthopterin.

¹Mr. Alfred Goldberg is an undergraduate student at Harvard College, Cambridge, Massachusetts. His participation in this study was made possible by The National Science Foundation Undergraduate Research Participation Program conducted at The Biological Laboratory, Cold Spring Harbor, during the summer of 1961.

Goldschmidt, Elisabeth. The effect of silver nitrate on a melanotic tumor stock.

Rapaport (1939) first produced a phenocopy of the mutant yellow by adding soluble silver salts to the food of developing larvae. Yaffe (1956) ascribed this phenocopy

to a blocking of tyrosinase activity by silver ions. He demonstrated the effect in vitro in a mixture of prepupal hemolymph with tyrosine solution. The blackening of this mixture is inhibited by the addition of silver nitrate.

In view of this specific influence of silver on melanin formation it appeared paradoxical that small amounts of silver nitrate were also reported to produce melanic inclusions in *Drosophila* (Sand & McDonald, 1954).

In order to test the effect of silver on the dark pigment of the 'pseudotumors' of *Drosophila melanogaster*, a stock with high tumor penetrance was grown on standard corn molasses agar medium containing silver nitrate. The emerging adults were scored for normal or 'yellow' body pigmentation and for presence or absence of tumors. Flies scored as non-tumorous on first inspection were cleared in 10% potash in order to reveal any tumors that had escaped attention. The results are summarized in the following table:

Medium	No. bottles	Total flies	Non-melanotic				Melanotic				Total melanotic %	With pale integument %	With pigmented integument %
			Males		Females		Males		Females				
			No.	%	No.	%	No.	%	No.	%			
Regular (Control)	6	3753	325	8.7	245	6.5	1439	38.3	1744	46.5	84.8	0	100.0
+ 0.0125% AgNO ₃	5	1307	2	0.2	5	0.4	655	50.1	645	49.3	99.5	99.0	1.0 ⁺
+ 0.0250% AgNO ₃	6	860	2	0.2	4	0.5	446	52.0	409	47.6	99.3	99.0	0.1 ⁺⁺

+ 5 males, 8 females all melanotic

++ 1 male, melanotic

It is seen that silver nitrate concentrations which produce 100 per cent phenocopies give also rise to a drastic reduction in the number of emerging flies. Among the survivors the tumor incidence reaches almost 100%.

It cannot be decided whether the salt kills off most non-tumorous individuals along with a good many tumorous ones or whether it kills at random and raises tumor incidence among the survivors. The increase in tumor penetrance may well be due to an oxidizing effect of the silver ion becoming reduced to metallic silver. In the 'suppressor-erupt' stock, at all events, tumor incidence is much enhanced by various oxidizing agents (Plaine, 1955).

Especial interest attaches to the prevailing phenotype in the silver nitrate cultures. This is the fly exhibiting the pale integument, while harboring one or several deeply pigmented tumors.

This result may indicate that an enzyme which is essential for cuticular melanogenesis is not involved in the pigmentation of the cell aggregates forming the tumor sheath (Rizki & Rizki, 1959). Such an assumption gains support from the feeding experiments of Plaine and Glass (1955), who found tyrosine, the classical substrate of melanogenesis, to have little effect on tumor penetrance, while tryptophane feeding greatly enhanced the incidence of the pigmented aggregates.

Alternatively, the silver ions may fail to reach the aggregates at the critical stage of their blackening. The present experiment does not exclude this possibility. It should also be noted that the mouth hooks of larvae in silver nitrate cultures appear as dark as those of untreated individuals.

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Gottschewski, G. H. M., and W. Querner.
Spreading of injected fluorochromes in
explanted cephalic complexes of different
larval stages.

Earlier investigations demonstrated the influence of the brain on the differentiation of the eye-imaginal-disc during development. This influence could be nervous or humoral. In order to show the

eventual spreading of substances from the brain to the eye disc, we injected smallest amounts ($0,5 - 5 \times 10^{-10}$ ml) of fluorochrome-solutions in different parts of explanted cephalic complexes, taken from late second to late third larval stages. With a new arrangement of the Zeiss-photomicroscope for mixing normal light and UV-light in any proportion we could observe both, the structures of the tissue by phase contrast and the spreading of the fluorescent substances within the tissue. In a first series we injected in the late 3. larval stage either of 4 spots: posterior part of the hemisphere, anterior part, which becomes the medulla, eye-imaginal-disc, and antennal-disc. After injection in the posterior hemisphere, the substances spread in this part only, nothing reaches the anterior part of the neighboring hemisphere or the supraoesophagale ganglion, in spite of the connection between all parts. Likewise, the fluorochromes injected in the anterior part of the hemisphere do not spread into the posterior part, indicating a barrier inside the hemisphere, not allowing substances to pass. However, the fluorochromes pass from the anterior part of the hemisphere to the eye disc and from there to the antennal disc rather quickly. Vice versa, substances injected in

the eye-disc or in the antennal disc only spread in these two organ-Anlagen (then still connected). However, they do not pass in the brain or in the second eye-antennal disc of the explant. These results demonstrate, that the transport of the injected fluorochromes between brain and eye-disc is only allowed in one way (brain towards eye), the other one (eye towards brain) is blocked. In a second series we injected in earlier stages, where the frontal sac is in full action, its ends slide as a mucous layer on each hemisphere and the connection between brain and eye-disc by the nerve cord is not yet strong. Because of the difficulty to carry the injection-needle to the small eye-disc without stripping off the fluorochrome in the mucous frontal sac we only injected in the hemisphere. The same barrier in the hemisphere as in the late 3. stage was found. The substances do not pass from posterior to anterior and vice versa. And as in the first series the fluorochromes are spreading from the anterior brain part to the eye disc, and from there to the antennal disc. In contrast to the first series they spread from there to the second eye-antennal-Anlage, again ending nevertheless at the second hemisphere; thus demonstrating the same barrier from eye to brain as in the late 3. larval stage.* We assume, that other substances may pass from the medullar part of the brain to the eye as well as the fluorochromes, and that, consequently, there is a way to influence the differentiation of the eye by substances from the brain.

*Fixation of the explants by Formol or Alkohol after injection does not change the spreading effect of the fluorochromes, both barriers are visible before and after fixation.

Greenberg, Rayla. Two new cases of SD found in nature.

Natural populations of Drosophila melanogaster from two new localities have now been shown to exhibit segregation distortion.

Among 503 males sent from Berea, Kentucky, by Dr. Frank Seto, 24, or a frequency of .048, carry the SD gene; in a group from DeKalb, Illinois, collected by Dr. Jack Bennett, 1 among 184 tested males, or .005, show distortion. The finding of such a low frequency in the latter locale indicates that other populations previously scored as negative (e.g. by Mange, 1961) may, on more extensive investigation, be found to contain SD. Indeed, the frequencies given here are minimal since a few additional SD's have appeared after successive backcrossing of the wild flies to a laboratory stock, suggesting that suppressors present in the males collected may now have been eliminated.

The Berea SD's examined conform in the following properties to the original SD discovered in Madison, Wisconsin (see Sandler, Hiraizumi and Sandler, 1959), and to the Baja California SD found by Mange (1961).

- 1) males heterozygous for a putative SD chromosome and a cn bw chromosome show mean segregation ratios ("k" values) of .99 in favor of the SD-bearing chromosome.
- 2) heterozygous females produce mean k values of .52.
- 3) one or more inversions are present in the right arm of the SD chromosome, as shown by suppression of crossingover between cn and bw.
- 4) SD action is inhibited when heterozygous with the Cy inversion.
- 5) a heterozygote between a Berea SD and a Madison SD exhibits no distortion.

These further findings of meiotic drive indicate its widespread occurrence and possible ubiquity in natural populations of *Drosophila*. Additional studies are being done, with particular interest in the question of sensitivity of wild non-SD chromosomes to the action of SD.

Grell, R. F., and E. H. Grell.
A correction to the cytology of
the rearrangement associated
with Glazed.

Bridges and Li (Morgan, Bridges and
Schultz, 1936), Carnegie Year Book 35:293,
and also quoted in Bridges and Brehme,
1944) describe the rearrangement asso-
ciated with Glazed as a single pericentric

inversion of the second chromosome with breakpoints at 27E and 51D. This inversion
should permit crossing over between the tip of 2L and the breakpoint at 27E. Recent
crossover tests failed to detect any recombination in this region among 12,727 flies.
Salivary gland chromosome analysis reveals two additional breaks in the Glazed
chromosome at 22D and 33F and which appear identical with those of In(2L)Cy.
Furthermore, the heterozygote, Gl^a/In(2L)Cy shows a single pericentric inversion
difference between the two chromosomes. It seems probable that the Glazed inversion
was originally induced in an In(2L)Cy chromosome. The correct arrangement of the
Glazed chromosome appears to be: 2L tip to 22D/33F to 27E/51D through the centromere
to 33F/22D to 27E/51D to 2R tip. Ins(2LR)Gl^a is a good balancer for all of 2L and
the proximal half of 2R.

Hadorn, E., and I. Faulhaber.
Range of variability in cell
number of larval salivaries.

Earlier studies of our laboratory showed
that some larval and pupal lethals affect
the cell number of the salivary glands.

This finding points to an early action
of the mutant genotype during embryogenesis. Several genotypes have now been inves-
tigated by counting the nuclei in Gomori stained whole mounts of salivaries. Thereby
a rather high variability was found which seems to depend on a polygenic basis. The
following table shows a few examples from cultures kept at 25° C. on standard food
and under equal population density conditions.

Genotype	Range of variability in single lobes	Mean for both lobes per individual	n
Wild stock Sevelen	107 - 144	256.7 ± 2.87	15
lg1/Cy	90 - 153	247.3 ± 2.07	67
lg1/lgl not outcrossed	90 - 148	237.5 ± 2.26	73
lg1/lgl after outcrossing	97 - 160	257.3 ± 2.45	61
l 73 a larval lethal	109 - 174	293.0 ± 2.57	73

There is a distinct correlation between the cell number of the two lobes within an
individual. The cell number increases when the time of embryonic development is
prolonged by keeping the freshly laid eggs at 18° C. instead of 25° C. Thus the
high numbers found in some lethals (at 25° C.) might result from the fact that these
genotypes develop more slowly than normals.

Hanks, G. D. Selection for reduced recovery of yellow, white attached X females in D. melanogaster.

An unusually low rate of recovery of females occurred when pair matings of yw attached X females to Canton S⁺ males were made. One mating in particular gave 74% males and only 26% females. Cultures

with the lowest recovery of females were selected when possible for the mating of the progeny with the following summary of results:

Cross	No. of Cultures	Males	<u>yw</u> Females	% ♂	% male range of individual cultures
1. <u>yw</u> ♀ X Canton S ⁺ ♂	16	1357	1132	55.3	49.3-74.0
2. <u>yw</u> F ₁ ♀ X F ₁ + ♂	6	481	325	59.6	41.1-75.4
3. <u>yw</u> F ₂ ♀ X F ₂ + ♂	7	453	139	76.5	60.4-96.9
4. <u>yw</u> F ₃ ♀ X car, ru ♂	5	392	66	85.6	74.2-95.8
Controls:					
		Males	Females	%	
1. al, ru ♀ X F ₂ + ♂		1060	1040	50.5	
2. bw, st ♀ X F ₂ + ♂		1248	1269	49.6	

It is not known whether the low rate of recovery of females can be accounted for by their mortality, but apparently the effect is inherited in such a manner that only the female parent produces the effect. Thus the attached X itself or something which interacts with it is suggested to be the cause of the low recovery rate of females. Unfortunately the stock was lost when the furnace overheated at the University of Utah.

Hansen, A. M., and E. J. Gardner.
New eye phenotype in D. melanogaster
expressed only at high temperature.

A new recessive mutant in D. melanogaster has been found in a wild Cockapontsett stock. It was given the descriptive name scarp and the gene was symbolized scrp (current DIS).

Linkage studies placed the scrp locus at 74+ in the second chromosome. Scarp overlaps wild-type completely at 25° C., but the penetrance is approximately 80 per cent at 30° C. A temperature effective period has been established that extends from the forty-second hour to the sixty-eighth hour after fertilization when development begins at 30° C. The full 26 hours at 30° C. are necessary for maximum penetrance.

A number of "wild" laboratory stocks and one sample from a natural population were tested for the presence of scrp. Scarp was present in three of the laboratory stocks, but two of them were derived from the same stock and had been separated for about three years.

Examinations of histological sections of scarp eyes showed that certain ommatidia are shorter than others. The scrp gene presumably controls this condition. There is a considerable time lapse between the temperature effective period and the time at which the ommatidia elongate. Further investigation is necessary to determine the intermediate steps.

The effects of high temperature upon the frequency of expression of welt, lobe, and lobe-recessive have been examined. A temperature of thirty degrees centigrade significantly increased the frequency of expression in the F₁ heterozygotes from crosses of lobe or lobe-recessive with wild-type. Homozygous welt was found to be lethal at 30° C.

Heed, W., J. Russell, and D. Harrington.
Diversity and density of *Drosophila* in
the immediate vicinity of Tucson with
special reference to *D. pseudoobscura*.

The following list of species has been
accumulated in 41 irregular collecting
trips during a 3 1/2 year period (1958-61)
within a 20 mile radius of Tucson, Arizona.

The two main habitats collected (chiefly
by lard cans containing old bananas) are the pine and fir in the Santa Catalina
Mountains (6-9000') and the cactus and riparian in the desert (2-5000').

The three most abundant species (above 1000 individuals) in the total
collection of 20,766 individuals and 32 species are *pseudoobscura* (50%), *hamatofila*
(22%) and *simulans* (6%). The six next most common species (400 to 700 individuals)
are each 2 or 3% of the total. They are *melanogaster*, *pseudoobscura*-like, *victoria*,
longicornis, *rubrifrons* and *hydei*. The seven next most common species (100 to 300
individuals) are each .5 to 1% of the total. They are *nigrospiracula*, *macrospina*,
carbonaria, *macroptera*, *nigrospiracula*-like, *innubila* and *azteca*. The remaining
16 species (one half of the total) are less than 70 individuals each. They are
tenebrosa (65), *californica* (56), *Leucophenga varia* (35), *grisea* (28), *arizonensis*
(15), *Clastopteromyia inversa* (10), *Gitona bivisualis* (8), *Chymomyza* sp. (6),
busckii (5), *Scaptomyza graminum* (5), *melanopalpa* (3), *montana* (2), *bifurca* (2),
Gitona americana (2), *nigrohydei* (1), and *Leucophenga pulcherrima* (1).

Of the 32 species collected, 1/3 of them (11) are restricted to the mountains,
1/3 of them (10) are restricted to the desert, and 1/3 of them (11) are found in both
habitats. A total of 22 species were collected in the mountains and 19 in the desert.
D. pseudoobscura represents 71% of the 13,038 individuals collected in the mountains.
It represents 14% of the 7,728 individuals collected in the desert and is the third
ranking species there. *D. hamatofila* makes up 12% of the mountain fauna and ranks
second there and 38% of the desert fauna where it is the most abundant species.
D. simulans is second in abundance in the desert (14%) but ranks 13th in the mountains
(0.3%).

The main difference in species abundance at the two elevations is that the
desert contains a higher frequency of common species (11 species are each 2% or more
of the number of individuals in the desert). In the mountains only 4 species reach
a frequency of at least 2%. The differential is due to the swamping effect of
pseudoobscura in the mountains. Does *pseudoobscura* really affect the frequency -
distributions of other species' abundances in the mountains as compared to the desert?
Reference to the table shows a surprising similarity in the two faunas in the ranking
of species if *pseudoobscura* is deleted from the mountains. There are now one
abundant species (42%), two common species (12 to 17%) and the remainder fall off
gradually from about 6%.

Species Ranked According to Density

MOUNTAIN (without <i>pseudoobscura</i>)		DESERT	
N = 3,735 (23 collections in four different months)		N = 7,728 (18 collections in seven different months)	
1. <i>hamatofila</i>	42.3%	37.6%	<i>hamatofila</i>
2. <i>pseudoobscura</i> -like	17.2	14.2	<i>simulans</i>
3. <i>rubrifrons</i>	12.4	14.1	<i>pseudoobscura</i>
4. <i>longicornis</i>	5.5	7.3	<i>victoria</i>
5. <i>hydei</i>	4.9	7.1	<i>melanogaster</i>
6. <i>macroptera</i>	4.8	4.4	<i>longicornis</i>
7. <i>melanogaster</i>	3.4	3.9	<i>nigrospiracula</i>
8. <i>innubila</i>	2.7	3.4	<i>macrospina</i>
9. <i>tenebrosa</i>	1.7	3.1	<i>hydei</i>
10. <i>californica</i>	1.5	2.5	<i>carbonaria</i>
11. <i>azteca</i>	1.4	1.9	<i>nigrospiracula</i> -like
12. <i>simulans</i>	1.0	0.5	<i>azteca</i>
13. <i>grisea</i>	0.7	0.5	<i>Leucophenga varia</i>
0.1% or less, 8 species		0.1% or less, 6 species	

The data indicate that the large pseudoobscura populations (larvae and/or adults) in the mountains possibly control over-all abundance within the other species, at least at the traps, but by the criterion of the desert fauna, they have little effect on the number of species or on their relative abundance. It appears that the only interaction here is that of random crowding at the site of collection.

The two new species, one similar to pseudoobscura, and the other, a morphological and ecological sibling of nigrospiracula, will soon be described.

Hess, Oswald. Scute⁸ as Y suppressed lethal factor.

In the course of cytological investigations of spermatogenesis in X/O ♂♂ of D. melanogaster (Meyer, Hess and Beermann, 1961) we found that sc⁸/O ♂♂ are lethal. Four different X chromosomes carrying the sc⁸ mutation were tested, namely Muller-5 (scS1B InS w^asc⁸), FM 4 (y³ld sc⁸dm B), sc⁸bb w^a, and sc⁸bb w. These males were crossed with y²su-w^aw^a bb/O ♀♀. From these crosses in F₁ XX/Y daughters and sc⁸/O sons are expected in the ratio 1:1. The actual ratio found, however, from three crosses apiece, is shown in the following table:

Paternal X	XX/Y	X/O	Sex ratio	
			♀♀	♂♂
Wild Berlin (control)	1015	1278	100	126
M-5	1005	111	100	11
FM 4	1070	71	100	6,6
sc ⁸ bb w ^a	1074	2	100	0,19
sc ⁸ bb w	1028	24	100	2,3

The penetrance of the lethal effect of sc⁸ in X/O ♂♂ is below 100%. It varies between 89 and 98% in our experiments. We have not yet established whether the observed percentage of sc⁸/O break throughs (Durchbrenner) are characteristic for the type of sc⁸ chromosome used. Since dead pupae have not been found in the crosses the critical period of the lethal effect must be earlier in development.

The lethality of sc⁸ is partially suppressed by fractional Y chromosomes, as is shown in the following table for Y^S:

Paternal X	XX/Y	X/Y ^S	Sex ratio	
			♀♀	♂♂
Wild Berlin (control)	975	1044	100	107
M-5	1316	943	100	72
FM 4	1136	661	100	58
sc ⁸ bb w ^a	1241	989	100	79
sc ⁸ bb w	1087	791	100	73

Similar ratios were found with sc⁸/Y^S.Y^S and sc⁸/ybb- ♂♂.

The most likely hypothesis from these results is that sc⁸ is a lethal variegated position effect. Lindsley et al. have found that after X-raying of D. melanogaster ♂♂ with 3-4 kr 20-25% of the resulting lethals are suppressed by the Y chromosome and therefore overlooked in the usual tests. Another argument for this hypothesis is, that the sc⁸ mutation meets the basic requirement for a V-type position effect,

namely it has in the X chromosome an inversion with one break in the euchromatin and another in the heterochromatin. We did not find any significant difference between cultures maintained at 22, 25, and 28° C., but we do not consider this to be a strong argument against the hypothesis. As many investigators have found, the amount of heterochromatin, especially in the Y chromosome, is a much stronger modifying factor than temperature differences.

Hildreth, P. Influence of different Y chromosomes on secondary nondisjunction in *D. melanogaster*.

Females heterozygous for a wild type X chromosome from a Samarkand stock and an X chromosome of the composition $y^2sc^{S1}B\ In49\ v\ w^a\ sc^8$ were tested for

the frequency of X-chromosomal nondisjunction and segregation of the X's when Y chromosomes of different types were present in the females. The Y chromosomes used were (1) a normal unmarked Y, (2) $sc^8.Y$, (3) $sc^8.Y.B^S$ and (4) y^+BY (a chromosome which arose in one of our experiments and has not yet been analyzed). Since this was only a preliminary test no attempt was made to isogenize the stocks. Larger-scale experiments are planned in which these and other Y's will be used and the genetic background will be strictly controlled.

Individual female inversion heterozygotes, each bearing one of these Y chromosomes, were mated with males carrying a normal Y chromosome and having the X chromosome marked with y and w. The corrected nondisjunction rate was highest, 66.1%, in females carrying the normal Y chromosome. The frequencies decreased to 59.3% in the presence of y^+BY , to 54.9% in the presence of $sc^8.Y$, and to 49.8% in the presence of $sc^8.Y.B^S$.

Table I indicates the percentage of recovery of the chromosomes singly and in combinations. Because of the markers used it was not always possible to ascertain whether or not the Y chromosome from the female was present. This was true entirely for the unmarked Y and partially for the $sc^8.Y$ chromosomes.

Table I

Percentage recovery of Y and X chromosomes

P female	No. of offspring	Nondisjunction		Single X recovered		Total recovery			
		Corrected total	Recovered		++++	$y^2B\ v\ w^a$	% of X's		
			XX	Y			Y	++++	$y^2B\ v\ w^a$
$y^2B\ v\ w^a$ ++++ Y	709	66.1	20.6	28.8	with Y - without Y -	- - -	-	50.6	49.4
$y^2B\ v\ w^a$ ++++ y^+BY	966	59.3	19.2	23.1	with Y 15.8 without Y 16.4	7.9 - 17.6	46.8	53.6	46.4
$y^2B\ v\ w^a$ ++++ $sc^8.Y$	930	54.9	16.6	21.3	with Y - without Y -	9.7 - 20.8	-	50.7	49.3
$y^2B\ v\ w^a$ ++++ $sc^8.Y.B^S$	642	49.8	12.5	20.7	with Y 18.1 without Y 17.1	11.4 - 20.2	50.2	52.0	48.0

In each instance the $y^{2B} v w^a$ chromosome is recovered nearly as frequently as the wild type chromosome, indicating in these cases that the viability effects of the two chromosomes are similar. The recovery of the Y chromosome from the female approached or surpassed 50% in those cases in which the presence of the Y could be ascertained, indicating relatively good viability of individuals carrying the Y chromosome.

When apparent nondisjunction of the X's took place the Y chromosome was always recovered in higher frequency than the two X's, as had been observed by Sturtevant and Beadle (1936) in their tests of several different inversion heterozygotes for secondary nondisjunction. The inversions used by them were not as complex as the one used here, however. The more complex inversion would lessen the chance for pairing between the X's and therefore decrease the opportunity for crossing over. This in turn would mean a low frequency of anaphase bridges to account for death of eggs containing these, and therefore should not contribute greatly to the frequency of patroclinous males in our experiment. A test of inversion heterozygotes without Y chromosomes failed to produce patroclinous males in higher frequency than matroclinous females.

Another interesting aspect is the frequency with which the X chromosomes are recovered singly with and without the Y chromosome. According to random expectation when one X chromosome is retained in the egg nucleus, then 50% of the time it should be the inverted X and 50% of the time it should be the wild type X. When the inverted X is retained, then 50% of the time the Y chromosome should be retained with it. The same is true for the wild type X, thus the four classes should occur with equal frequency. As is seen in Table I, the frequencies of wild type X chromosomes with and without the Y are nearly equal but there is great inequality in the frequencies of $y^{2B} v w^a$ chromosomes recovered with the Y and without it. Table II gives the expected and the observed ratios for the nondisjunctional and other classes.

Table II

P female	Y	: XX	+ + + + : + + + + / Y : $y^{2B} v w^a$: $y^{2B} v w^a$ / Y			
Expected	1	1	1	1	1	1
Observed						
$y^{2B} v w^a$ + + + + Y	1.39	1.00	-	-	-	-
$y^{2B} v w^a$ + + + + $y^{+}BY$	1.21	1.00	2.09	2.01	2.31	1.00
$y^{2B} v w^a$ + + + + $sc^8.Y$	1.28	1.00	-	-	2.14	1.00
$y^{2B} v w^a$ + + + + $sc^8.Y.BS$	1.66	1.00	1.50	1.58	1.78	1.00

It appears that the rate of secondary nondisjunction is influenced by the Y chromosome and that the normal Y is associated with the highest degree of nondisjunction, while the $sc^8.Y.BS$ is associated with the least degree. The wild type X chromosome is recovered as frequently with the Y as without it, but the inverted

X is recovered approximately twice as often without the Y as it is with the Y. From the total recovery of the Y and each of the two X chromosomes it seems unlikely that viability differences could account for this latter effect. It is possible that some mechanism causes the Y to be lost frequently from its association with the $y^2B v w^a$ chromosome but not from its association with the wild type X chromosome.

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

Hiraizumi, Y. Low viability induction by the segregation distorter (SD) locus; preliminary note.

In a heterozygous SD male, SD causes a breakage in its partner chromosome, perhaps at SD^+ . This broken chromosome is eliminated in some stage before fertilization, thus more than 50% (usually 95% or more) SD-bearing

chromosomes are transmitted to the next generation. Here a question arises whether the SD^+ -bearing chromosomes found in the F_1 -generation are 1) those which were not affected at all by SD action or 2) those which recovered from the break. If 2) is the case, then we may expect some changes, perhaps viability reduction, in the SD^+ -bearing chromosomes from heterozygous SD males. Accordingly, $SD/cn\ bw$ (and $SD^+/cn\ bw$ as a control) males were crossed to $cn\ bw/In(2L)\ Cy\ cn\ bw$ females. In each set of experiment the $cn\ bw$ chromosomes in the heterozygous SD and SD^+ males in P-generation were derived from a single, lethal free chromosome and the remaining genetic background had been uniformized before the present experiments. From the F_1 of these matings $cn\ bw/In(2L)\ Cy\ cn\ bw$ males were chosen to cross individually to $cn\ bw/In(2LR)\ Cy$ females, and the $F_2\ cn\ bw/In(2LR)\ Cy$ sibs from each F_1 mating were mated to test the homozygote viabilities of $cn\ bw$ chromosomes in comparison with their Cy heterozygotes. For the significance test the observed percentage of $cn\ bw$ homozygotes ($= r$) in the F_3 in each culture vial was transformed according to the relation $r = \sin^2 R$. The results are summarized in the table. Figure in parenthesis is the percentage of $cn\ bw$ homozygotes corresponding to each \bar{R} ($=$ average of R) value. The lethal-bearing $cn\ bw$ chromosomes (indicated as +1 lethal etc.) were excluded from computing \bar{R} . Each experimental set was made at a different time, but in each set the $cn\ bw$ chromosomes from the $SD/cn\ bw$ males showed, on the average, reduced viabilities ($p < 0.01$). It is interesting to note that original SD ($= SD-72$ and $SD-5$; strong SD) lines caused more viability reduction than recombinant SD (weak SD) lines, although the difference was not statistically significant. The detailed mechanism for this is not yet fully understood, but a small deletion accompanied by the breakage-reunion event could be responsible.

Table

Exp. set No.	\bar{R}					
	Original SD	No. of cultures	Recombinant SD	No. of cultures	SD^+	No. of cultures
1	31.16 (26.8)	8 (+1 lethal)	31.49 (27.3)	20 (+1 lethal)	34.34 (31.8)	18
2	31.69 (27.6)	12	32.50 (28.9)	38	33.52 (30.5)	37
3	31.80 (27.8)	16	32.42 (28.8)	52 (+1 lethal)	33.38 (30.3)	31
Total	31.62 \pm 0.34 (27.5)		32.30 \pm 0.28 (28.6)		33.63 \pm 0.27 (30.7)	

Hoenigsberg, H. F., Y. Garcia Cortés, and D. Ortiz Rubio. The male and the female choice in studies of sexual preference in D. melanogaster mutants.

During recent studies of sexual behavior in Drosophila melanogaster mutants the authors found D. melanogaster Cy/BL to establish very definite preferences both in various elements of courtship which prompt response, and in copulations. The male choice method consisted in placing the male to choose between two females; one of his own type and the other a wild D. melanogaster female. Such preferences were also present in D. melanogaster Cy Pm but to a lesser extent, and not for all elements of courtship. Nevertheless, the phenomenon in Cy Pm resulted in courtship discrimination with subsequent nonrandomness in mating but extended to those elements of courtship which most elicited the lowering of the female threshold barrier. Moreover, the authors completed the studies by making the female choice as well. The results, which will be published elsewhere, show female choice preferences like those already apparent by the male choice method. In other cases we found discrimination in the other direction indicating a lesser importance of the female behavior as the deciding condition in sexual preferences.

Hoenigsberg, H. F., Y. Garcia Cortés, and D. Ortiz Rubio. The degree of sexual preference in D. melanogaster Cy BL and the fitness associated with it.

The authors, interested in the evolutionary consequences of the mating preferences of D. melanogaster mutants, are studying in various experiments, still in course, the adaptive "peak" resulting from each case of nonrandomness in each separate mutant.

Hoenigsberg, H. F., and N. J. Díaz. Differential induction of phenodeviants by heat treatment in D. melanogaster mutants.

Several mutants of D. melanogaster were treated in their egg stage with temperatures which ranged from 15° C. - 32° C. every day for 4 generation. At the end of the 4th generation various phenodeviants of various sorts began to appear. A very high frequency in the lobe mutant was found. This indicated to us that at least a fundamental pathway in ontogenesis had been altered by the procedure. Furthermore, comparisons of this effect in the lobe mutant of D. melanogaster with other mutants of the same species indicated that there is a fundamental "potential" difference in the ontogenetic pathway, or its "canalization," of the lobe mutant and the others here tested. The extent of the phenodeviations from the "normal" pattern was different in different organs affected by the fluctuating temperatures. The complete experiments will be published elsewhere next year.

Hollander, W. F. Two mosaics.

(1) From our stock of y w/Basc triploid females and Basc males, a well-formed male was obtained having the left half of the body yellow, left eye white, right eye bar apricot (= Basc). This male lived a week, but failed to produce any progeny. The simplest explanation seems to be simultaneous fertilization, by normal Y-bearing sperms, of a binucleate egg with one diploid nucleus and one haploid nucleus (suggestion of Peter E. Thompson). The resulting intersex side might have been shifted toward normal maleness by coexistence with the male side, but not enough to permit fertility.

(2) In our attached-X stock #82 a male was obtained with the left eye reddish-colored, similar to "coral," the right eye white (typical color of males of stock). Mated to 5 virgin sibs he produced 15 sons with the new red eye color, and 100 sons white-eyed. Next he was mated to attached-X white-eyed females, and produced 52 sons, all white-eyed and yellow-bodied. It appears likely that this mosaic was somatically mutant from white to coral (?), including also a small portion of the germ cells. The new eye color is being maintained.

Hollander, W. F., and Michael F. Festing.
Equational exceptions from roughex males.

Matings of roughex males (rux^{60d} - see
DIS 34:50) with attached-X females have
produced 17 homozygous roughex daughters

in 9447 progeny examined. Any associated sex-linked markers also became homozygous.
Secondary non-disjunction from these females has been below expectation; further
tests are in progress.

Hunter, Alice S. Abnormal sex ratio
in wild Drosophila pseudoobscura.

During recent months, collections of
Drosophila have been made in various
natural localities in the vicinity of

Bogotá, Colombia. In one of these collecting sites, a relatively high percentage
of Drosophila pseudoobscura has been found in the collections made over a period
of four months. The site, which we call "Pine Woods," is located at an altitude
of 2,700 meters, with an average temperature of 15° C. Since Bogotá is located at
a latitude of 4° North there is little or no variation in temperature throughout
the year. There are, however, two rainy seasons, one March-April and the other
October-November.

An inherited "sex ratio" condition is well known for D. pseudoobscura, but
since this results in the production of all female offspring it could not be related
to the high percentage of males collected in the Pine Woods. These collections are
made by sweeping over a bait which consists of fruit skins and wastes of a wide
variety of local fruits such as banana, pineapple, papaya, guava, mora, oranges
and curuba. The data follow:

Month	Total collections	D. pseudoobscura		Chi Square
		females	males	
July	13	38	173	87
August	24	93	237	64
September	44	724	1,000	44
October	37	282	605	118
Total	118	1,137	2,015	122

It is obvious that the deviation from the expected 1:1 ratio is large each
month and also for the total. It seems to us that such an abnormal ratio in a total
of over 3,000 flies is worthy of investigation. Therefore both field and laboratory
studies have been started in order to ascertain the basis of this abnormal sex ratio.

Collections are being made at another site roughly 400 meters from the Pine
Woods. These collections are being made from traps which contain pure banana bait.
Although there is a slight preponderance of males in these collections, the sex
ratio is much closer to 1:1. This suggests that a comparison should be made of the
different methods of collection, different types of bait and different ecological
conditions of the collecting sites.

As a start in the laboratory investigations, isolated wild females collected
in the Pine Woods were grown at outdoor temperature under optimal food conditions.
Counts of the F₁ from 54 different females showed that 47 produced a normal 1:1 ratio,
while only 7 females produced offspring which varied significantly from the expected
1:1 ratio of males to females. Of these, 4 showed a higher percentage of females,
and 3 a higher percentage of males. This suggests that environmental rather than
hereditary factors are involved in the abnormal sex ratio found in the wild popula-
tions of D. pseudoobscura.

Imaizumi, T. On a strain of XXY of D. melanogaster with two translocations.

From genetical and cytological analyses, it becomes clear that females of the strain derived from a male of wild Miyazu strain irradiated by X-ray (previously reported)

have two translocations in addition to the chromosomal constitution of XXY: one is a half-translocation between X and II, the other a mutual translocation between the rest of II and III. In the half-translocation, the distal 1/3 of the left arm of II including cn is locating at the end of X; the rest of II is broken at the middle of the left arm and translocated with III. Thus, some genes on the left arm of the original II are linking with X, and some with III. The strain can be preserved by following crosses; Y/Basc/T(1;2) x Y/Basc or Y/w m/T(1;2) x Y/w m (the translocation between II and III is always contained in those females). In various crosses of this strain, the total mortality reaches 82-89%. The details will be reported in the "Cytologia."

Iyengar, Shanta V. A male Drosophila mosaic for the Y^{bw+} chromosome.

In an experiment where regular F₁ males (expected type) carried vermilion on the X-chromosome and were homozygous for brown eyes, it was found that among several

white eyed exceptional males whose phenotype is due to the loss of the bw⁺ gene or the entire Y carrying it, one was fertile. On being mated to virgins from a stock homozygous for brown all his male offspring have a phenotype (bw⁺) which proves that they have the bw⁺ gene (by covering bw/bw) as well as in their being fertile showing they have the Y chromosome. It is apparent that the loss of the Y chromosome occurred from the primordial tissue forming the eyes but did not occur in his germ tissue on either side.

Kaneko, A., T. Shima, and E. Momma. Drosophila species in Utoro and Habomai, eastern Hokkaido.

Collections were made with the use of traps for three days in the middle of August, 1961. A total of 831 specimens belonging to 24 species was obtained. In Utoro, a

northern side of the Shiretoko Peninsula, the collection showed that dominant species were represented by D. lacertosa, D. auraria, D. nigromaculata and D. okadai. In Habomai, lying south to Utoro at a distance of 100 km, D. nigromaculata was the only dominant species showing the frequency of 66% (Table 1). The difference in distribution between the two localities is mostly attributed to the flora in their habitats.

Table 1

Species	No. of flies		
	Utoro	Habomai	Total
<u>D. nigromaculata</u>	93	194	287
<u>D. lacertosa</u>	157	7	164
<u>D. auraria</u>	132	15	147
<u>D. okadai</u>	89	0	89
<u>D. testacea</u>	24	0	24
<u>D. histrioides</u>	1	19	20
<u>D. suzukii</u>	3	10	13
<u>D. coracina</u>	12	0	12
<u>D. brachynephros</u>	2	9	11
<u>D. ezoana</u>	5	3	8
<u>D. funebris</u>	2	0	2
<u>D. moriwakii</u>	1	0	1
<u>D. nipponica</u>	1	0	1
<u>D. tenuicauda</u>	1	0	1
<u>D. trivittata</u>	1	0	1

(Kaneke, Shima, and Momma, Table 1--continued)

Species	No. of flies		
	Utoro	Habomai	Total
<i>D. sexvittata</i>	1	0	1
<i>D. sp.</i> (fenestrarum group)	1	0	1
<i>D. spp.</i> (two different species)	8	0	8
<i>Scaptomyza graminum</i>	0	3	3
<i>S. polygonia</i>	1	0	1
<i>Parascaptomyza disticha</i>	0	29	29
<i>Amiota variegata</i>	1	0	1
Total	537	294	831

Kang, Yung Sun, and Lee, Hei Yung.On *Hirtodrosophila macromaculata*
sp. nov. from South Korea, with
7 text-figures.

External feature:

Male and Female; Body: Dark brown,
about 2.5 - 3 mm long, with remarkable
black stripes on mesonotum.

Antenna brownish yellow, 3rd joint broad and large, with greyish long hairs. Arista has about 8 branches including a small fork, one being below it. Ocellar triangle and occiput darker. Carina narrow and flat. Perioral black and one prominent bristle. Cheek dark yellow, about $1/8$ as broad as the greatest diameter of the eye. Orb_2 about $1/5 orb_1$. Palpus dark brown, with only one long apical bristle.

Head: Eyes dark red, with short piles.

Mesonotum (Fig. E): yellowish brown, with 4 black longitudinal stripes, inner pair interrupted at posterior and outer pair interrupted at anterior. Broaden black spot below intrascutal suture. Scutellum black, with posterior portion brownish yellow. Thoracic pleura largely dark brown spots. Humerals two. Acrostical hairs 8 rows. Cross distance of dc. about third the length distance. Anterior scut. slightly divergent. Sterno-index about 0.4.

Legs: Brownish yellow, ultimate femora and tarsal joints dark brown.

Preapicals prominent on hind tibia. Apicals on middle. Wings fuscous.

Wings (Fig. A): Three rather large and distinct spots in the wing. One spot distributes around the anterior cross vein starting from the proximal end of the wing, another one in the central part of the wing extending from radius $2\frac{1}{3}$ to media $3\frac{1}{4}$ and surrounding the posterior cross vein, and the last one extends from the distal end up to nearly the middle part of the wing, covering the distal parts of marginal cell and submarginal cell, with a small puncture

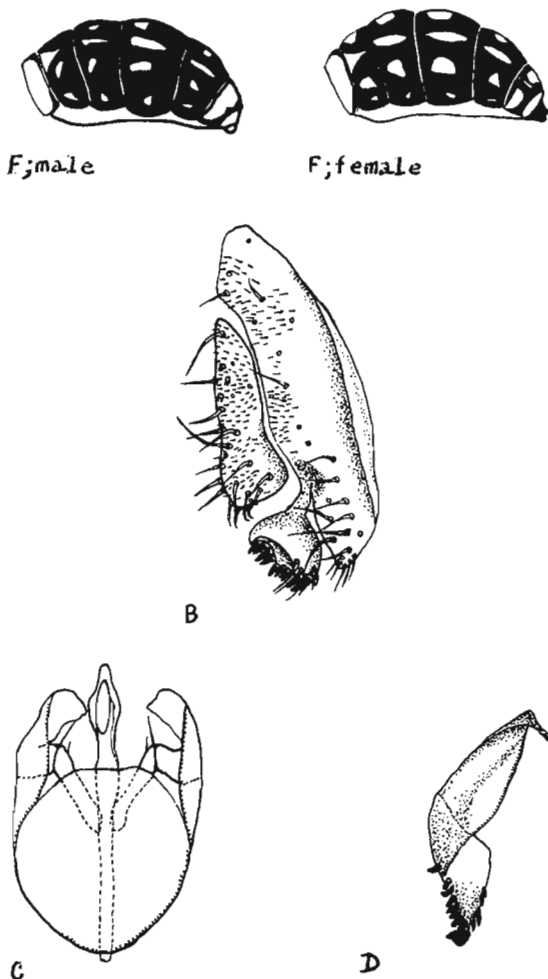
in the region of marginal cell. The distal end of the last spot draws a curve forming a concave connecting media 1 and radius $4\frac{1}{5}$ and the central end forms also a concave. C-index about 1.3; 4V-index about 1.4; 4C-index about 1.4; 5X-index about 1.0; C1-bristles 2; C3-bristles on basal $7/10$. Halter white.



E



A



Abdominal tergites (Fig. F): Brownish yellow, with black patches. 1 tergite brownish yellow, black at lateral corners; 2T brownish yellow, with broad caudal black band, which is deeply incised at middle and with 4 brownish yellow spots at lateral sides; 3-5T with 4 brownish yellow spots on each lateral segment and one rod shaped spot on each middle side. 6-7T brownish yellow.

Periphallic organ (Fig. B): Genital arch yellow, elongate, somewhat triangulate at lower tip, and with about 25 strong setae, upper portion densely hairy. Clasper yellow, broad, narrowing basally, and with about 10 teeth in a row, and about 6 secondary teeth arranged in 2 rows. Anal plate yellow, narrow and broad below, separated from genital arch, and with about 26 stout hairs including numerous short hairs.

Phallic organs (Fig. C): Aedeagus elongate, apically swollen in lateral view, flattened and elliptical. Anterior paramere brownish yellow, elongated, fused to novasternum. Novasternum brownish yellow, and nearly quadrangle, and with a spine on the inner edge. Posterior paramere seems to be absent. Ventral fragma brown, semielliptic, and rounded at tip.

Egg-guides (Fig. D): Lobe yellow, tapering at tip, and with about 14 marginal brown teeth. Basal isthmus brownish yellow, thick and short.

Holotype: Male, Kwang nung, Kyngki province, South Korea, 1 Male 1, June, 1961.

Allotype: Female, collected together with holotype.

Paratype: Kwang nung, Male 16 and female 12.

Distribution: South Korea.

Collecting method: Net sweeping on the decayed trees.

Kaplan, William D., V. E. Tinderholt, and D. H. Gugler. The number of sperm present in the reproductive tracts of Drosophila melanogaster females.

Fuelgen-positive sperm heads is much less than the impression given by a fully-packed seminal receptacle or the paired spermathecae in which the sperm, with their extremely long tails, are contained. A count of sperm heads in these structures gave a maximum of about 650 in eight females examined. A mass of sperm cells is also present in the vagina and, two hours after a single copulation, this mass contained about 300 sperm cells. The total in this one female was, however, 750. We are now studying the way in which this vaginal sperm mass is utilized to replenish sperm in the seminal receptacle and the spermathecae.

In studying radioautographs of the female reproductive tracts for the presence or absence of labelled sperm, it was noted that the number of sperm present was not so great as was expected on the basis of earlier published reports. The number of

Kikkawa, H. Strain differences in proteolytic enzyme activities in D. melanogaster.

and strains. Strain differences seem to be due to qualities and quantities of the enzymes. Of interest is that a strong inhibitor of trypsin is contained in the body fluid.

Proteolytic enzymes of *Drosophila* are mainly involved in digestive glands. Their activities are controlled by various factors such as sexes, developmental stages

King, R. C. Vitellogenesis in *Drosophila*.

spheres between 1 and 3 microns in diameter which belong to two classes. The alpha yolk sphere contains proteins (extractable from sections by pepsin, trypsin, or papain) and acidic lipids (which appear to be relatively unsaturated). The second type of yolk, comprising the beta spheres, is devoid of protein and contains (1) periodic acid-Schiff-positive polyglucosans which are extracted from sections by alpha amylase, (2) alcian blue-positive, acidic polysaccharides, and (3) lipids (which are relatively saturated). Under the electron microscope the dense alpha spheres are seen to be covered by a double walled envelope; whereas the pale beta spheres appear to be devoid of an enclosing membrane. Beta yolk spheres often coalesce with one another.

Cytochemical studies have shown that the mature ovarian oöcytes of *Drosophila melanogaster* and *D. willistoni* contain large yolk

The oöplasm contains myriads of mitochondria and lipid droplets which are just above the limit of resolution of the light microscope. In electron micrographs the mitochondria are seen to be ellipsoidal, and many are embedded in the cortex of the beta spheres. The lipoidal bodies have a stellate appearance. Stacks of annulate lamellae of the sort illustrated in Growth 22:323, Fig. 26, and isolated filaments of endoplasmic reticulum occur commonly also. The background oöplasm contains proteins, polysaccharides, lipids, glycoproteins, lipoproteins, and ribonucleoproteins. The so-called periplasm of the egg represents a region filled with layers of membranes arising from convolutions of the plasma membrane.

Cytological observations indicate that during vitellogenesis a stream of cytoplasmic material flows in a posterior direction through the cells of the nurse chamber and finally into the oöcyte through pores which connect all these daughter cells. These pores are so large that particles the size of mitochondria and lipoidal droplets easily pass through them. Alpha yolk spheres are first seen in the oöcyte during stage 8. During maturation of these spheres the lipids they contain appear to increase in concentration and/or to become less soluble in organic solvents (perhaps through formation of lipoprotein complexes). Immature alpha spheres contain a considerable amount of polysaccharide which is later lost. The alpha spheres are believed to arise by the growth while in the oöplasm of membrane enclosed droplets a few tenths of a micron in diameter. These precursor particles may arise in turn from tiny blebs which are pinched off the envelopes of the nurse cell nuclei and are subsequently carried into the oöcyte.

The beta spheres arise during stage 13 (some 4 hours after the alpha spheres first make an appearance) from smaller particles of similar morphology. It appears that as the beta sphere grows the carbohydrates it contains become more resistant to extraction, since in osmium-fixed material they are lost from the small particles but retained in the larger masses. However, formalin-containing fixatives retain the polysaccharides of the precursor particles. The source of these beta sphere precursors is currently under study.

Koch, R., and H. Burla. Dispersal rates in *Drosophila subobscura* and *Drosophila obscura* in relation to factors of environment, sex and age.

According to the method of Sakai et al. (Evolution 12, 1958, pp. 93-101), the two species, *Drosophila subobscura* and *D. obscura*, have been compared in reference to their dispersal capacities.

At 25° C. the dispersal rates are higher for *D. obscura* than at 18°, whereas it is the inverse for *D. subobscura* at the two temperatures. The maximum dispersal

rates for D. obscura have been shown to be at lower relative humidities. Presumably higher humidities are more optimal and the activity is reduced in the range of such preferred humidities. Furthermore, the case is the reverse for D. subobscura, their activity being increased at higher humidities. For both species the activity is greater on fresh food than it is on old food and is greater when the flies are starved than when well fed. Younger adults are more active than the older, as are the males when compared with the females. For both species the activity has been shown to be reduced in low air-pressure.

In general, D. subobscura reaches higher dispersal rates than D. obscura, while D. obscura is more sensitive to all factors except air-pressure. Consistently, D. obscura has been shown to be the less resistant species in reference to unfavorable conditions.

Generally the dispersal activity was increased by conditions of environment which were considered not to be optimal for the respective species.

Koref-Santibañez, Susi.

A comparative study of courtship behavior in some species of the mesophragmatica group of *Drosophila*.

Courtship behavior has been analyzed in the following five species of the mesophragmatica group: D. viracochi from Machu-Picchu (Peru); D. mesophragmatica, from Machu-Picchu (Peru); D. gasici, from Arica (Chile);

D. pavani, from Bellavista (Chile) and D. gaucha from Rio Grande do Sul (Brazil). The general courtship pattern in all five species is very similar, and follows the ritual described by Spieth (1952) for other species of the subgenus *Drosophila*. Nevertheless, there are differences which allow the individualization of each species. Thus, D. viracochi males have a slower wing vibration movement; D. gasici males circle and touch antennae of females much more profusely than do those of other species. D. mesophragmatica males and females display very little activity and all their movements are slow. Another characteristic for each species is the duration of copulations, which are significantly different in all.

When, by means of the "male choice method," individual males are allowed to discriminate between a female of their same species, and a female of any of the other four, the following facts are observed:

a) The general activity of both males and females increases significantly over that observed when only individuals of the same species are together.

b) All males court their own females for a longer period than they do foreign females.

c) Only some courtship elements are used significantly more towards their own females, while others do not seem to be discriminative. Thus, D. gasici males discriminate against D. gaucha, D. viracochi, and D. mesophragmatica females, using all courtship elements more towards D. gasici females; D. pavani males discriminate when confronted with D. viracochi and D. gasici; D. gaucha males discriminate against D. viracochi females while D. viracochi and D. mesophragmatica males use all elements equally towards their own or towards foreign females.

d) As regards mating, the males of almost all the species copulate only with the sister females. Only D. pavani and D. gaucha mate almost indiscriminately with one another.

The comparative analysis of the courtship behavior of the different species included in the mesophragmatica group agrees with the phylogenetic relationships previously determined by the morphologic and cytogenetic studies (Brncic and Koref-Santibañez, 1958, and Brncic, 1959). It may be postulated, that as regards divergence of courtship behavior, D. gasici is the most distant, as both males and females discriminate highly, and the ritual itself is the most diversified. D. pavani and D. gaucha, which are the most closely related (they are sibling species), discriminate very slightly against each other, but markedly against the other species. The apparent lack of preference shown by D. viracochi and D. mesophragmatica males may be due to their general low activity, or to the fact that in them, the females are the more discriminate.

The higher activity of males and females of each of the five species when confronted with individuals of any of the other may be tentatively interpreted as following: the males may receive repulsory stimuli from the foreign females which may increase their excitation and obliges them to a greater activity, raising also the stimuli threshold of the females, thus conditioning longer courtship time and greater utilization of all the elements which make up the courtship ritual.

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Lefevre, G., Jr., and Ulla-Britt Jonsson.
Sperm relationships in twice-mated
D. melanogaster females.

D. melanogaster females that mate with two different males usually produce offspring by both males. However, as various investigators have noted, a

considerable degree of individual variability is evident in the results. As a consequence, a number of differing conclusions have been offered in regard to questions of sperm displacement, sperm mixing, and the sequence of sperm utilization. Reinvestigating this problem, we have identified an important source of such variability, and we are now able to define some consistent features in the activities that follow double matings.

Individual virgin males were encouraged to mate in succession with several 2 to 3-day-old virgin females. Males will often proceed energetically with their task, fertilizing as many as 5 successive females in a period of 3 to 4 hours. Then, each female of the sequence (which we designate #1, #2, #3, etc.) was presented with a second, genetically different, virgin male. If a remating did not ensue during an observation period of about 4 hours, the female was left with the second male until the next day.

As a rule, #5 females, as well as the majority of #4 females, mated for the second time almost as readily as they did originally as virgins. The exceptions, particularly with #4 females, in which remating did not take place usually occurred when the first male had spent an unduly long time carrying out the sequence of matings. The #3 females, in contrast, remated much less readily than did #4 and #5 females; nonetheless, some were receptive to a second male soon after the completion of the first mating, others within 24 hours. The #1 and #2 females, however, rarely remated the same day, but a reasonable number of rematings occurred within 24 hours.

After an observed remating, or after having been with a second male for 24 hours, each female was removed to a new vial and then subcultured, generally at 2-day intervals, for 10 or 12 days. The resulting offspring were counted and classified as to paternity, and the hatches were compared with those obtained from females that had mated only once. Two striking conclusions arise from the comparisons: (1) once-mated females produce just as many offspring as do twice-mated females, and (2) after a remating, the number of progeny sired by the first male is always less than he would have produced had the female not remated with a second male.

Additional females were dissected at intervals after mating, ranging from a few minutes to a few days. Soon after mating, the ventral receptacle and the two spermathecae are completely filled with sperm in #1 and #2 females, most often noticeably less full in #3 females (with considerable variation), more sparsely filled in #4 females (indeed, frequently, they were empty), and almost always empty in #5 females (occasionally, a very few sperm were observed). Further, the number of sperm transferred by a male in his first or second mating, perhaps three or four thousand, far exceeds the capacity of the female sperm storage organs. Long after they are completely full, great quantities of motile sperm can be seen in the uterus, oviduct, and even in the ovary. Rough estimates indicate that only between 10 and 20% of the sperm deposited in a #1 female are actually stored, but perhaps as many as half of the stored sperm are subsequently used in fertilization. In #3 females, on the other hand, relatively fewer excess sperm are present; yet, occasionally their storage organs are about as full as in #1 and #2 females. In #4 females, virtually all of the smaller number of sperm transferred are stored, and in #5

females the few sperm that are sometimes transferred find their way into the receptacle and spermathecae within a matter of minutes.

Dissections of males following the various matings showed that the decline in the number of sperm transferred with each successive mating was not so much a matter of exhausting the seminal vesicles of sperm, but rather was correlated with the condition of the accessory glands. Even after five matings, the vesicles have many motile sperm left, but the accessory glands are completely collapsed and devoid of secretion. When the sequence of matings proceeds more slowly than usual, as it sometimes does, or when the male is deliberately rested between matings, then even in a fifth mating an appreciable number of sperm may be transferred.

Stained whole mounts of dissected female sperm storage organs always showed sperm distributed throughout the receptacle. The sperm heads do not congregate at the distal blind end, but are dispersed more-or-less randomly throughout the length of the receptacle, and in fact may be identified "inbound" and "outbound" at all levels. Even during the period shortly after mating when sperm are being stored and many are still in the uterus, outbound sperm heads can be detected in the basal end of the receptacle near the entrance. Thus, a circulation of sperm appears to exist in the receptacle, so that even as some sperm are coming in, others are going out. Clearly, after sperm enter the receptacle, they do not simply proceed along as far as they can go and remain there until all the space in the receptacle is occupied; rather, throughout the period when sperm are in the uterus, we believe that sperm are continually entering the receptacle, reversing direction at any level, and even returning to the uterus, perhaps later to re-enter the receptacle, perhaps not. Eventually, the excess sperm thrash off in unrewarding directions (up the oviduct or out the vagina), leaving only the better oriented ones finally accommodated within the storage organs. It is abundantly evident, in any event, that the female sperm storage organs are normally filled to capacity by a single copulation.

Upon remating, the sperm circulation (which appears to continue between matings in the same manner) results in the emergence from the receptacle of previously stored sperm where, in the uterus, they are diluted by the vastly greater quantity of sperm newly deposited by the second male. The likelihood of re-entrance of the first sperm is thereby greatly reduced. Finally, when the excess sperm have vanished and all sperm possible have been stored, sperm from the first male still present in the receptacle are quite diminished in number.

Remating surely does not result in the storage of a double quantity of sperm, nor do the two kinds of sperm form any sort of layers in the receptacle; but rather remating results in the displacement of a greater or lesser proportion of the sperm originally present. It is not apparent from dissections that a similar activity occurs in the spermathecae, but judging from the offspring produced by remated females, which sometimes contain very few sired by the first male, we are inclined to suspect that to some degree it does.

Lefevre, G., Jr., and Ulla-Britt Jonsson.
The effect of cold shock on *D. melanogaster* sperm.

In 1949, Novitski and Rush (Biol. Bull. 97:150-157) reported that fertilized *D. melanogaster* females can be deseminated by an exposure to

sub-zero temperatures. An effective treatment was -10°C . for 10 minutes. At the same time, they stated that males subjected to the same treatment were not affected in regard to their subsequent fertility, at least.

It seemed paradoxical that mature sperm stored in the female should be killed by cold treatment, while similar sperm stored in the male should be immune. Reinvestigating the effect on males, we have found that exposure to -10°C . for 10 minutes does in fact inactivate all of the fully mature, motile sperm stored in the seminal vesicles of the male, exactly paralleling the effect on mature, motile sperm stored in the ventral receptacle and spermathecae of females. For a period of 24 hours or so after treatment, no motile sperm can be detected in the male reproductive organs, nor are any sperm transferred to the female during

copulation. The male regains fertility, however, as the apparently more resistant, less mature, immotile sperm in the testis mature and enter the seminal vesicle, becoming available then for insemination. If the males are first irradiated with 4000r, then exposed to the cold treatment, a virtually complete sterility occurs from which there is little or no recovery.

Dissections indicate that the treated male is unable to expel from the vesicles the mass of dead sperm produced by the cold shock. This results in a greater or lesser obstruction to the passage of newly maturing sperm, and occasionally motile sperm can be seen in the testis itself, having been unable to descend into the vesicle. Thus, none of the treated males regain a normal degree of fertility, and in particular are unable to inseminate more than one or two females within a 24-hour period. There is no loss of sex drive, however, and treated males will copulate repeatedly without transfer of sperm. In fact, unless such males are removed from the presence of females and rested between matings, it is very rare to find a given male capable of successfully inseminating more than one female. It would appear that the dead sperm masses in the seminal vesicles so impede the passage of motile sperm into the ejaculatory duct that an appreciable period of time is required to build up the volume of sperm required for a normal insemination.

Cold-treated females, on the other hand, are able to expel the dead sperm from their sperm storage organs, so that following a reinsemination, there is no significant effect of subsequent fertility.

Lewis, E. B. Salivary gland chromosome analysis of segregation distorter lines.

Analysis of segregation distorter chromosomes SD5 and SD72 (of Hiraizumi, Crow and Sandler) indicates that each carries an inversion in the distal part of the

right arm of the second chromosome apparently identical with In(2R)NS. The SD5 chromosome carries an additional inversion in 2R having one breakage point somewhere in the region extending from 45C to 45F and another in region 49A. The SD72 chromosome lacks the latter inversion but has, in addition to In(2R)NS, a small pericentric inversion with one breakage point in the euchromatic region of sections 39 or 40 of 2L and the other breakage point close to or within euchromatic section 42A of 2R.

Lovellette, E., and F. Ratty. Comparisons of inbred and random bred larval survival to 1200r.

The following experiments were conducted to determine whether first instar larvae of various genotypes express a differential survival to acute X-irradiation. This

work compares the survival of four lines including a random bred (line 1), a hybrid formed from random females and inbred males (line 2), a hybrid formed from inbred females and random males (line 3), and an inbred (line 4).

Five hundred first instar larvae were placed on a one inch plaque of standard *Drosophila* medium, and then exposed to 1200r (220 kv, 20 ma) or used as controls. Survival is herein defined as the proportion of larvae developing into the adult stage. The results presented in the following table are based upon the average number of larvae which hatched from an original plaque of 500.

Line	Total sample	CONTROL (avg./500) Total			Total Sample	IRRADIATED (avg./500) Total		
1	7,000	214	211	425	11,000	159	195	354
2	7,000	205	217	422	11,500	107	125	232
3	7,000	220	206	426	9,500	72	120	192
4	7,000	214	219	433	14,500	77	113	190

line 1(random x), line 2(random x inbred),
line 3(inbred x random), line 4(inbred x).

These results indicate that the average total survival among the four controls is quite uniform. In the irradiated samples survival is highest in line 1, while the groups derived from the inbred strain do not survive as well.

Possible interpretations of these observations might be:

(1) Differences in the rates of development which would result in lines derived from the inbred group being in a different stage of development at the time of irradiation and thereby having a lower survival. Critically timed studies on first instar larvae of ages 2, 4, and 6 hours indicate this effect is probably not relevant.

(2) If survival is dependent upon a maternal effect the survival of line 1 would be expected to approximate that of line 2, since they both have random bred mothers, while that of line 3 should approximate that of line 4, both having had inbred mothers. This relationship is indicated for the combined survival of lines 3 and 4; however, the survival of line 1 is significantly higher than line 2 which does not support a strict maternal effect hypothesis. The survival of irradiated females from line 2 does not differ significantly from those of lines 3 and 4 which also would not be indicative of a maternal effect.

(3) Possibly survival is related to the genotype in a particular line. Differential survival of lines 2 and 3 seems to indicate that the males of line 3 do not survive as well as those of line 2. These males differ only with respect to their X chromosome. This suggests that lower survival is partially a function of the X chromosome from the inbred line--possibly related to the fixation of deleterious recessives therein. However, this explanation would not account for the high survival of line 4 females in relation to those of lines 2 and 3.

(4) In addition, survival is probably also related to epistatic effects between the autosomes and the X chromosome of the inbred line. When these chromosomes are homozygous they tend to increase survival, as indicated by the comparative values of lines 3 and 4.

Löönd-Luchsinger, S.

The riboflavin content in Malpighian tubules of D. hydei.

Chromatographic and fluorometric methods were used in determining the riboflavin quantities in the Malpighian tubules of larvae, pupae and imagoes. Two maxima were

found, one at pupation time and the other at the time of eclosion. A sex difference becomes apparent only in imagoes, where females contain about twice the quantity of riboflavin as males. Adding riboflavin to the standard food results in a strong increase of the substance in Malpighian tubules of larvae and pupae. On the other hand, the feeding of riboflavin to imagoes leads to almost no increase of this substance in their Malpighian tubules.

Malogolowkin, Ch. A new sibling species of the D. willistoni group.

A new species of the subgenus Sophophora, morphologically very similar to D. willistoni and D. paulistorum has been found in the

states of Guanabara, Rio de Janeiro, Bahia, Salvador and in Pernambuco, Recife. This species crosses to D. willistoni and to strains of D. paulistorum from the Andean-South-Brazilian group of species. This species is being studied at the Department of Zoology of Columbia University and a formal description, together with genetic and cytological data, will be published elsewhere.

(This investigation is being supported by a fellowship from the Pan American Union.)

Malogolowkin, Ch. A new transitional race in Drosophila paulistorum.

The species Drosophila paulistorum is known to be a complex of six races or incipient species sharing varying degrees of reproductive isolation. Five of the races

produce completely sterile F1 hybrids, if they can be crossed at all. The sixth race, termed Transitional by Dobzhansky and Spassky, produces fertile hybrids with

at least some strains of the other races. Now, the Amazonian race, which lives in the northern part of South America, from Belem to Panama, showed very strong reproductive isolation from the Andean-South-Brazilian race, which occurs from Colombia and Peru, to southern Brazil. Now collections made by myself in Central and Northeastern Brazil have disclosed the existence of a new Transitional race, which crosses and yields fertile hybrids of both sexes with the Amazonian as well as with the South-Brazilian strains. Strains of the new Transitional race have been isolated from populations of Ceara (Maranguape), and Bahia (Salvador), and may occur in other regions as well.

(This work has been assisted by grants from the Conselho Nacional de Pesquisas of Brazil and from the Rockefeller Foundation.)

Mettler, Lawrence E. Fertility relationships of recombination-hybrid males from the cross of D. mojavensis and D. arizonensis.

Baker (1957) demonstrated that hybrid males from the cross arizonensis-mojavensis were sterile and that the reciprocal mating (mojavensis-arizonensis) produced partially sterile males. Population studies have

indicated that introgressive (recombination) hybrid males may be partially fertile when the initial cross is arizonensis-mojavensis. The present investigation is to determine if the sterility is due to a simple X-Y chromosome unbalance, or if autosomal recombination and/or the cytoplasm influences fertility. The acquisition of a spontaneous white-eyed (X chromosome) mutant, which is apparently closely linked to the region in the X chromosome which differs in the two species by a paracentric inversion, makes such a study possible.

Cross A was arizonensis (white eye)-mojavensis and cross B was the reciprocal mojavensis-arizonensis (white eye). The F₁ females from these two crosses were backcrossed to mojavensis males and to arizonensis (white eye) males. The 4 backcrosses produced 8 classes of male progeny in respect to the cytoplasm and the X and Y chromosomes:

	Y	X	cyto.	per cent fertile
Cross A backcrossed to <u>arizonensis</u>	ariz	ariz	ariz	4.0
Cross B backcrossed to <u>arizonensis</u>	ariz	ariz	moja	4.0
Cross A backcrossed to <u>arizonensis</u>	ariz	moja	ariz	4.0
Cross B backcrossed to <u>arizonensis</u>	ariz	moja	moja	6.0
Cross A backcrossed to <u>mojavensis</u>	moja	ariz	ariz	24.0
Cross B backcrossed to <u>mojavensis</u>	moja	ariz	moja	20.0
Cross A backcrossed to <u>mojavensis</u>	moja	moja	ariz	42.0
Cross B backcrossed to <u>mojavensis</u>	moja	moja	moja	77.0

Fertility tests were made by placing 100 males of each class individually with 3 virgin mojavensis females. The number (per cent) of those cultures producing offspring are listed above.

Those males carrying mojavensis Y chromosomes and X chromosomes are more fertile. The cytoplasm appears to have some effect especially when the X and Y chromosomes are both mojavensis. The fact that the combination moja-ariz-ariz (which is similar to the F₁ sterile males from the cross arizonensis-mojavensis) shows 24 per cent fertility indicates an influence of autosomal recombination. Crossing over cannot be ruled out. The arizonensis arrangement is a simple inversion and it is being identified by a mutant locus. Certain backcross progeny with white eyes may actually carry the mojavensis X chromosome arrangement. If this is true the problem becomes more interesting. It would mean that the sterility loci are closely related to those loci differing by an inversion in the two species. Experiments are now in progress to verify these results and to ascertain the amount of crossing over.

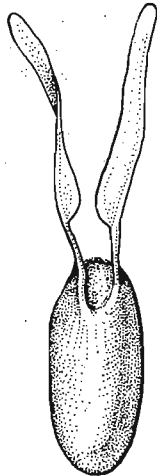
Mettler, Lawrence E. Locating mutants by crossing species with chromosome differences.

crossed to D. mojavensis (which differs from arizonensis by paracentric inversions in two of the four major autosomes). The F_1 of this cross was wild type. The F_1 hybrid females were backcrossed to homozygous mutant arizonensis males. Half of the progeny of this cross showed the mutant phenotype. Those flies showing the mutant phenotype were intercrossed and the salivary chromosomes of 20 of the progeny larvae were examined to determine which chromosome (2nd, 3rd or neither) was always homozygous for the arizonensis arrangement. All third chromosomes were such. Thus, the mutant was found to reside in this chromosome.

An autosomal recessive mutant was recently found in D. arizonensis. In order to ascertain the autosome in which the mutant resides, the homozygous mutant strain was

Mettler, Lawrence E.
Drosophila pachea.

description of Drosophila pachea Patterson & Wheeler 1942. The species was placed in the hydei subgroup of the repleta group. These flies did not breed in the



D. pachea

A single record of 2 males and 2 females collected about 3 miles north of Hermosillo, Sonora, Mexico (August, 1941) led to the laboratory. Recently (October, 1961) 4 males were collected near Hermosillo and 1 female near San Felipe, B. C., Mexico. These were classified as pachea by Dr. W. B. Heed. The female produced a few eggs on banana media but they did not hatch. The egg is rather small (1.76 mm. including filaments) with 2 relatively large, flat blade-like filaments which are as long as the body of the egg. Since all described eggs of the repleta group are characterized by 4 thread-like filaments, and since pachea lacks the general feature of mesonotum spotting, it is suggested that this species might belong in the subgenus Sophophora instead of Drosophila. Further collections and possible culturing will be attempted to help ascertain its true position.

Milkman, R. D. cve phenodeviants in the progenies of wild inseminated females.

collected at Syracuse. Five hundred flies in the F_2 of each line were examined for posterior crossvein defects. The distribution of frequencies of such defects implies a polygenic basis for this deviant phenotype. The number of crossveinless flies of a total of 500 examined are presented, ranked as follows: 30, 17, 13, 12, 10, 6, 4, 3, 3. In addition, 5 strains contained 2 cve flies, 7 contained 1, and 8 contained none. These results are consistent with previous findings. Attempts to select true-breeding cve strains from the top 5 strains are in progress.

The analysis of a polygenic cve strain selected from the progeny of a wild inseminated female in the Ann Arbor experiment shows that all autosomes are involved. The second chromosome is more important, its homozygous state being a necessary and sufficient condition for the appearance of crossvein defects. Two genes between

An experiment similar to a previous one in Ann Arbor (Science 131:225-226) has been conducted on the progenies of 29 wild inseminated Drosophila melanogaster females

the Star locus (1.3) and the Sternopleural locus (22.0) seem to be involved. The X chromosome, on the other hand, favors the production of normal crossveins more than the X chromosome in Oregon R. In contrast to a cve strain previously analyzed, not all the alleles have increased expression at 18°, although some of them do.

Milkman, R. D. Protection against phenocopying by pre-treatment at high temperatures.

Resistance, both to the production of crossvein defects and to death at 40.5° C., has been described for pre-treatment in the 31°-38° range. Although temperatures

throughout this range are comparably effective in conferring resistance to death at the higher temperature, a high temperature coefficient has been found for conferring resistance to transfiguration. The Q_1 seems to be around 1.3 based on comparison of the effectiveness of treatment at various durations at 38.5°, 36.5°, 34.5°, and 32.0°. It is necessary to return pupae to room temperature for a short time, such as 2 minutes, in order for the protection to take effect. Pre-treatments lasting as little as 10 seconds at 36.5° have a measurable effect. Short pre-treatments at 40.5° also have a protective effect in terms of subsequent treatments at the same temperature, if the interval is of the order of 2 hours. Longer pre-treatments at 40.5° do not protect in the same way. It is possible to think of several states of a protein, whose interconversion is akin to denaturation, as the basis for these phenomena, and experiments are in progress to test this hypothesis further.

Momma, E., A. Kaneko, and T. Shima. Rate of emergence of pupae irradiated at various stages in D. virilis.

The sensitivity for X-rays to spermatogenesis was analysed preliminarily. A total of 663 pupae was irradiated with 2000r in various developing stages, and

the rate of their emergence was examined. The results are shown in Table 1. All the pupae irradiated within 20 hours after pupation died before the emergence. With the pupae irradiated 20 hours or more after pupation, emerged flies tended to increase gradually in number. Normal ratio of emergence was observed in pupae irradiated 40 hours or more after pupation. A few decrease of the ratio was observed in pupae irradiated 58 and 122 hours after pupation.

Table 1

Age of pupae at irradiation after pupation (hours)	No. of irradiated pupae	No. of emerged flies			Rate of emergence (%)
		Females	Males	Total	
2- 15	127	0	0	0	0
16- 25	77	0	1	1	1.3
26- 35	41	12	6	18	43.9
36- 45	29	18	9	27	93.3
46- 55	30	14	15	29	96.8
56- 65	89	32	35	67	75.3
66- 75	26	12	10	22	84.7
76- 85	60	31	29	60	100
86- 95	11	4	6	10	90.8
96-105	72	25	36	61	84.8
106-115	8	4	4	8	100
116-125	4	3	0	3	75.0
126-135	37	13	19	32	86.6
136-145	3	2	1	3	100
146-155	44	31	6	37	84.2
156-160	5	2	2	4	80.0
Total	663	203	179	382	57.7
Control	558	243	253	496	88.9

Moree, Ray. Relative fecundity involving the *e* locus in *D. melanogaster*.

Male and female carriers of $+/+$, $+/e^{11}$ and e^{11}/e^{11} were crossed in all possible combinations, each combination being made in a separate culture bottle. The 9 combi-

nations were distributed over 160 bottles in a $(1:2:1)^2$ ratio. To minimize larval inviability each culture contained but 5 parental flies of each sex. The results in terms of total number of progeny per combination were as shown in Table 1. Dividing twice the number of homozygotes by the number of heterozygotes gave the relative fecundity coefficients shown in Table 2. Corrections for a small amount

Table 1

$\delta\delta$ \ ♀♀	$+/+$	$+/e^{11}$	e^{11}/e^{11}	Total
$+/+$	8646	18991	7504	35141
$+/e^{11}$	19795	43419	15386	78600
e^{11}/e^{11}	9011	21066	7332	37409
Total	37452	83476	30222	151150

Table 2

	$+/+$	$+/e^{11}$	e^{11}/e^{11}
♀ parents	0.897	1	0.724
♂ parents	0.894	1	0.952
All parents	0.896	1	0.835

of larval inviability changed the fecundity coefficients only very slightly. As a matter of interest the frequencies of the progeny genotypes $+/+$, $+/e^{11}$ and e^{11}/e^{11} were, in that order, 0.25, 0.52 and 0.23. The requisite statistical tests (made on the numbers and on the logarithms of numbers when that was necessary) support the obvious conclusion that the heterozygotes are heterotic and that the relative fecundities for the genotype sequence $+/e^{11} > +/+ > e^{11}/e^{11}$. This sequence is similar to that for relative viabilities (Moree and King, Genetics, in press) and helps to explain the long known fact that the *e* alleles are maintained at low frequency for long periods in cage populations.

Moriwaki, D., et al. A shift of sex-ratio in the progeny from irradiated males in *Drosophila melanogaster*.

It has been reported that effect of irradiation on the mutation rates changes depending on the different stages of germ cells. Paying attention to this point, we investigated the shift of the sex-ratio in the progeny of

males irradiated at different stages of the male germ cells in *D. melanogaster*. Using the isogenic Oregon-R wild strain, male flies of 4 $\frac{1}{2}$ hour-old were irradiated with 1000r, 2000r and 3000r and crossed with the same wild type females of about 3 day-old. The crossings were made immediately after irradiation, and every other day thereafter with new virgin females, continuing up to 16 days after irradiation to obtain 8 different classes. Every class was divided into four groups, one control and three treated; thus 32 groups in total were made for 8 classes. In the next generation, the number of males and females were noted and the sex-ratio in each group was calculated. The counted flies amount to about 700,000, and the sex-ratio in each group changes dependent on the dose as well as the germ cell stage. The sex-ratio shifts to the lowest level in the group where progeny come from males of 6 - 8 days after irradiation (class IV). In table, regression coefficients are given, showing relationship between sex-ratio and dose with respect to every class. It can be said that in each class sex-ratio depresses in proportion to dose, and the coefficient is - 0.0237 per 1000r in the average.

Regression coefficients (b)

Class	b/1000r	Class	b/1000r
I	- 0.00597	V	- 0.02506
II	- 0.01473	VI	- 0.02702
III	- 0.03495	VII	- 0.01969
IV	- 0.06013	VIII	+ 0.00277
Average		- 0.02371	

Moriwaki, D., and H. Ikeda.
Disturbance of "sex-ratio"
condition by X-ray irradiation.

In several species of *Drosophila*, "sex-ratio"
condition has been analysed in various ways.

"Sex-ratio" flies of *D. bifasciata* in
Japan have been kept more than about sixty
generations in our laboratory, producing almost only female progeny. The note
reported here concerns whether X-ray irradiation can disturb "sex-ratio" condition
in *D. bifasciata* or not. Normal males irradiated by X-rays (2000r, 4000r, 6000r)
in each generation were mated with "sex-ratio" females successively for several
generations. Their progenies were tested in each generation, but no male offspring
could be detected. On the other hand, "sex-ratio" females which had been exposed
to X-rays (2000r, 3000r) were mated with normal untreated males over a series of
generations. Although no male appeared in the F_1 generation, in the F_2 , for the
first time, twelve per cent of the treated females raised progenies comprising some
degree of males. Using the sister flies of the exceptional males without treatment,
sex ratio in the next generation was examined, where some were realized as quasi-normal
ratio and others behaved in the manner typical to the original "sex-ratio" strain.
The result indicates that X-ray irradiation can inactivate the "sex-ratio" factors
in egg cytoplasm originated from "sex-ratio" females. Further it is almost similar
to results obtained in the previous investigations on temperature cure of the
"sex-ratio" condition in *D. bifasciata* (Magni, 1953; Moriwaki and Kitagawa, 1957).

Mukai, T., and S. Chigusa.
Radiation-induced mutation rates
of polygenes controlling the
number of sternopleural bristles
in *D. melanogaster*.

The males of an isogenic line (Burdick's
W160) and an inbred Oregon R (Hiraizumi's
M-Oregon) were acutely irradiated with
X-rays and γ -rays at 500r. Immediately
after irradiation, the irradiated males
were mated to the females of the same

lines. The numbers of sternopleural bristles in females and males which hatched
on or before the 13th day after the mating were scored. Therefore, the heterozygous
effects of radiation-induced mutations were tested. The experiments are still in
progress, but the results at hand are reported here.

The data of females only were analyzed by using a technique in which the
means, variances, and the third moments about the means of the distribution patterns
of sternopleural bristle numbers are used. The summarized results are presented in
the table.

Treatment	Isogenic W160		Inbred Oregon R
	X-rays	γ -rays	γ -rays
No. of genomes tested	1088(975)	1653(1392)	2290(2415)
No. of mutations per individual	0.284	0.112	0.091
*Variance increase rate	$4.87 \times 10^{-4}/r$	$1.98 \times 10^{-4}/r$	$2.24 \times 10^{-4}/r$
**Mutation rate	$1.14 \times 10^{-6}/\text{locus}/r$	$0.41 \times 10^{-6}/\text{locus}/r$	$0.36 \times 10^{-6}/\text{locus}/r$

* heterozygote basis

** assuming 500 loci

() no. of tested genomes in the control

To our surprise, the relative biological effectiveness of X-rays to γ -rays turned out to be about three. The detailed studies of this problem are now under way. The polygenic mutation rate is higher than that of major genes as previously described by Burdick and Mukai (1958), and the variance increase rate is also higher than the estimates obtained previously by several investigators. This is supposed to have been caused by the different response of the males and females to the irradiation, i.e., the variance of females was increased by irradiation while that of males was not increased but decreased. In spite of this phenomenon, they used pooled data of the males and females.

Mukherjee, A. S., and R. C. Strohman.

A preliminary study on the chromatographic behavior of the heterozygous and homozygous conditions of a mutant and that of wild type *Drosophila melanogaster*.

In an attempt to make a comparative study of the chromatographic pattern of mutants in heterozygous and homozygous conditions we selected vestigial due to the following reason.

In the description of vestigial (vg) wing mutation it has been reported that this mutant is temperature sensitive.¹ The wings are completely vestigial at room temperature or below, but at 30° C. and above the wings tend to be stretched to take a normal phenotype. This is an example of the fact that the phenotypic expression of the gene might be governed by the metabolic rate. With this in mind and encouraged by the work of Buzzati-Traverso² we attempted, first, to distinguish between the flies homozygous for vestigial and those heterozygous for the same gene, and between these and normal flies (OregonR+), with respect to their amino acid constituents and then tried to correlate the differences with the assumed phenotypic change caused by the temperature.

The temperatures selected for this experiment were as follows: 18° C., 22 ± 1° C. (as room temperature), 30° C. and 33° C. Unfortunately, the flies rearing at 33° C. and above either became sterile or did not give rise to any living adult. However, they were reared at 33° C. to be used for chromatographic purpose. As regards the methods for resolution of the difference between the various types of flies, we adopted the ascending chromatographic technics, as given by Buzzati-Traverso, with the following modifications. We used Whatman filter paper No. 1 and No. 42. Six decapitated flies, at a time, were washed in 95% ethyl alcohol, boiled in distilled water for a minute or two, and then squashed at a spot on the paper. Males and females were separately squashed and recorded. The chromatogram was developed in a mixture of n-propanol and 1%-ammonia in 2:1 ratio for a period of 18 to 24 hours. The R_f values are being presented in the table and discussed later. The flies obtained from the vestigial stock were, however, not tested for their isogenicity; but, rather, they were collected every time from the F₂ with wild type inbred in the laboratory for a long period of time. This leaves an open question of other genes affecting the chromatographic pattern. However, an over-all picture can be obtained considering the relative qualitative and quantitative differences.

Results and conclusion:

The homozygous vestigial males and females differ from both heterozygous and normal flies. The difference is mainly based on the number and color of the spots and on their R_f values. The spots were of two types. In all the cases both fluorescent and ultra-violet-absorption spots were obtained. These results have been summarized in the table. There is a great difference in the R_f values of the fluorescent spots of males and females, within a given genotype.

Quantitative aspect:

There is a reduction in the R_f values for fluorescent spots obtained in the cases of flies raised at 33° C. as compared to those raised at room temperature, except for +/+ male and vg/+ female, which show an increase. The significance of the increase in +/+ male is, however, very poor. Distinction is possible between +/+, vg/vg and vg/+ from their R_f values both at room temperature and at 33° C. It is interesting to note that vg/vg and +/+ can be more easily distinguished from

those heterozygous for *vg*, than between each other. There is a considerable change for both UV-absorption as well as fluorescence from room temperature to 33° C.

Qualitative aspect:

There is a great difference in the kinds of fluorescent spots in the different genotypic conditions of the flies used. These are presented in the table. One point needs to be mentioned here, that while at room temperature the qualitative differences are very pronounced, at 33° C. these differences seem to be minimized. Another experiment, done with *vg*^{No} (vestigial-notched) mutant shows similar qualitative differences between the male and the female and also between this and other genotypic conditions (see the table).

Our results agree, in general, with those of Buzzati-Traverso. In our case we can further distinguish, by UV-absorption spots, the qualitative and quantitative nature of patterns in the different genotypes of the flies. The fluorescent spots might be either due to tyrosine or tryptophan or both. Both of these show fluorescence in conditions similar to those of the experiment.

In our experiment we could not get the variable phenotypic expression of the mutant *vestigial* in different temperatures (stretching of wings at high temperature). However, these preliminary results indicate that the study on the relationship between the temperature sensitivity of the gene and the chromatographic analysis of its products would probably be a very interesting topic for careful research.

¹Bridges, C. B., and Brehme, K., 1944, The mutants of *Drosophila melanogaster*. Carnegie Inst. of Publ.

²Buzzati-Traverso, A. A., 1953, Proc. N. A. S. (Wash.), 39.

Table: Summary of the results of the chromatographic behavior of vestigial and wild type flies of *Drosophila melanogaster* in different genotypic and sexual conditions. F = fluorescent spots, UVA = ultraviolet-absorption spots, Rm° C. = room temperature, ++ = color (bluish or yellowish in the bright spots) and separate, -+ = no color but separate spots, -- = no color, no separate spots but fused and continuous, = not done, * = in separate expt.

Conditions	R _f values at Rm° C. for		R _f values at 33° C. for		Number of spots			
	F	UVA	F	UVA	F at		UVA at	
					Rm° C.	33° C.	Rm° C.	33° C.
MALE								
+/+(Oregon R+)	0.447	0.259	0.47	0.16	3(-+) (2 fused)	4	3	2
vg/vg	0.427	0.269	4(++)	3
vg/+	0.5	0.3	0.456	0.253	2(-+)	3,5*	3	3,2*
vg ^{No} /vg ^{No}	0.447	0.154	5(++)	1
FEMALE								
+/+(Oregon R+)	0.638	0.212	0.33	0.291	3(-+)	2	3	3
vg/vg	0.627	0.221	0.356	0.277	1(--)	2	2 (fused)	3
vg/+	0.241	0.308	0.493	0.372	1(--)	3	3	3
vg ^{No} /vg ^{No}	0.52	0.163	2(-+)	2

Munz, P. Xanthindehydrogenase activity in D. melanogaster (Oregon-R).

The enzyme activity in homogenates from different stages and sexes was determined by measuring the amount of newly-formed isoxanthopterin after 90 minutes incubation with 2-amino-4-hydroxypteridine as substrate. This reaction product was found in the following amounts (relative units of fluorescence): Pupae with faintly yellowish eyes ♀ 11; ♂ = 12. Imagos immediately after eclosion ♀ = 13; ♂ = 8. Imagoes three days old ♀ = 49; ♂ = 28. These figures are relative to fresh weight. It is remarkable that females exhibit a higher enzyme activity than males though males contain much more isoxanthopterin than female imagos.

Narain, P. Effect of age of female on the rate of egg production in D. melanogaster.

To estimate the period during which a female should be tested for her egg production level, about 70 females of a strain of D. melanogaster derived from Nai Basti (India) were studied for their daily egg production for life-time (about 40-50 days) starting from the first day of egg laying. The flies were raised on a standard medium evolved by Burdick (1954) which was also used for testing the females' level of egg production. The eggs were collected on food (coloured green) placed on card-board chips treated with paraffin. A little live yeast was put on the food-chip which was inserted in test tubes of size 6" x 0.7" where a female and her partner were kept. The food-chips were changed daily (usually after 24 hours) and the number of eggs laid therein were counted on a stereoscopic binocular microscope with 10 x magnification. The flies were kept at 25° C. ± 1° C.

It was observed that the daily egg production showed rapid increase in the first few days reaching the peak on the 4th day of egg laying. Thereafter the daily egg production gradually decreased till 40th day of egg laying. The declining pattern of egg production after the attainment of peak was found to follow the following exponential:

$$Y = 66.56 e^{-0.0269 t}$$

where Y represents the number of eggs corresponding to a particular day (t) of egg laying. It is apparent from the equation that after attaining the peak, the daily egg production fell, on an average, at the rate of 2.7% per day.

The results of comparing the mean egg production over life-time (average 52.5 eggs/day) with mean egg production taken over various 3- and 10-day periods for a set of 37 females are shown in the table below:

Comparison of means of egg production over life-time and different periods

Days of egg laying	Average egg production per day	Rank correlation coefficient between the two means
2nd to 4th	68.1	0.54**
3rd to 5th	74.0	0.67**
4th to 6th	71.5	0.69**
5th to 7th	70.0	0.75**
6th to 8th	66.0	0.66**
7th to 9th	64.3	0.76**
8th to 10th	63.6	0.72**
1st to 10th	64.0	0.79**

**Significant at 1% level

Each of the rank correlation coefficient was found to be significantly different from zero. When tested for homogeneity, the various rank correlation coefficients were found to differ insignificantly among themselves. An estimate of the weighted values of rank correlation coefficients based on Fisher's z-transformation was found to be 0.71.

Thus, these findings revealed that the age of females has a significant bearing on their rate of egg production. There is a peak (4th day of egg laying) in their life-time when the females lay eggs with maximum rate. It is also evident that to take advantage of the *Drosophila* flies in breeding at short intervals of 8-10 days and the special feature of 3-day egg laying periods, the females should be tested for egg production when they are 6 to 8 days of age which is equivalent to 4th to 6th day of egg laying. Females of this age group will also be having sufficient vigour for producing their progenies. These findings varied but little with those reported by Gowen and Johnson (1946) and adopted by Bell *et al.* (1955), Rasmuson (1956), and Robertson (1957) in regard to the best period for testing females for their egg production levels.

Similar results have recently been obtained by Prabhu and Bhat (unpublished 1961) in *D. ananassae* also.

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Narain, P., C. Joshi, and S. S. Prabhu.
Response to selection for fecundity in
D. melanogaster.

In a quantitative character like fecundity, which is closely connected with fitness and which is less heritable, heritability being estimated at 5 to 15% by Bell *et al.*

(1955) and at 18% by Robertson (1957), the response to a selection pressure is expected little and still less if the selection is applied to a laboratory population of *Drosophila* flies which has been under mass culture for 20 to 25 generations. The selection would also cease to be effective after few generations, the number of generations depending upon the level of heritable variation present in the initial population to which the selection is applied. In such a case to maintain the degree of heterozygosity and hence to keep the selection effective, a slight modification in the 'mass' selection method may be helpful. This was tried in the present investigation.

Selection was practised in two laboratory populations, initially derived from different localities in India, viz., Nai Basti and Matunga. Five cultures were set up each generation and selection was made in either directions with total of 100 females each generation, 4 females being selected out of 20 females in each culture. A control was also run simultaneously. While making pair matings from each culture, the mates of the females were taken in a random fashion from the other remaining cultures instead of the same. Further, after selection, the eggs

laid by the selected females were mixed and distributed at random in almost equal numbers in the cultures set up for the next generation. This method ensured minimum chances of inbreeding in the population, though inbreeding, as such, cannot be completely dispensed with in a closed population. The females were tested for their egg production level on 4th to 6th days of egg laying and the method of testing and other details were as reported by Narain (unpublished, 1961).

The results of selection experiments conducted for 10 and 7 generations respectively in Nai Basti and Matunga stocks of D. melanogaster expressed in terms of average egg production per day per female in each generation are shown in the table below. The level of average egg production in the initial foundation stocks of Nai Basti and Matunga were 85.3 ± 1.50 and 37.5 ± 1.36 eggs per day respectively. The standard errors are based on 'within' culture variations.

Table 1
Average fecundity (No. of eggs/day) with
standard error in the selection
experiments

Generation No.	Nai Basti			Matunga		
	High	Control	Low	High	Control	Low
1	71.8 ± 1.35	64.9 ± 2.06	61.1 ± 1.62	54.1 ± 2.59	52.7 ± 1.98	46.6 ± 2.22
2	82.7 ± 1.50	77.4 ± 1.69	70.1 ± 2.07	53.2 ± 1.24	52.1 ± 1.69	44.5 ± 1.96
3	76.1 ± 0.88	69.6 ± 1.42	65.9 ± 1.04	73.3 ± 2.01	67.2 ± 2.16	65.3 ± 2.02
4	67.6 ± 0.80	65.9 ± 1.71	63.3 ± 1.12	57.2 ± 1.98	55.8 ± 1.48	45.8 ± 2.27
5	88.9 ± 1.22	80.5 ± 2.12	80.3 ± 1.32	72.9 ± 2.10	69.5 ± 2.55	50.8 ± 3.40
6	79.3 ± 0.94	70.9 ± 2.61	69.6 ± 1.29	66.2 ± 1.90	57.2 ± 2.60	56.2 ± 1.82
7	67.2 ± 0.97	65.6 ± 0.96	58.5 ± 1.26	50.6 ± 1.63	50.4 ± 1.46	47.2 ± 2.46
8	40.3 ± 0.68	34.9 ± 0.72	30.4 ± 0.67	-	-	-
9	46.9 ± 1.13	38.1 ± 1.58	36.4 ± 1.19	-	-	-
10	52.6 ± 1.40	51.6 ± 1.43	44.1 ± 1.32	-	-	-

The data in Table 1 indicate that there are wide fluctuations from generation to generation even in the control series where no selection was practised. The difference in "high" and "low" lines in Nai Basti stock, is quite high in second generation being 12.6 ± 2.55 whereas it falls down to quite a low figure of 4.3 ± 1.38 in fourth generation. Thereafter it fluctuates and does not go below 8.5 ± 1.92 of 10th generation. In Matunga stock, the situation is different, the maximum divergence being 22.1 ± 3.99 in the 5th generation whereas minimum being 3.4 ± 2.95 in the 7th generation. To get an idea of how the response to selection behaved as a fraction of selective force applied (which was measured by selection differential), the ratio of divergence (between high and low) with the cumulative selection differential which is frequently referred to as realized heritability (Falconer, 1960) was worked out after each round of selection. The results after each round of selection are shown in Table 2.

Table 2

Realized heritability for egg production
after each round of selection

Round of selection	Nai Basti			Matunga		
	Cumulative selection differential	Divergence	Realized h^2	Cumulative S. D.	Divergence	Realized h^2
One	36.7	10.7	0.29	27.6	7.5	0.27
Two	56.5	12.6	0.22	44.6	8.7	0.19
Three	89.9	10.2	0.11	66.5	8.0	0.12
Four	110.2	4.3	.04	90.5	11.4	0.12
Five	129.4	8.6	.07	116.6	22.1	0.19
Six	157.3	9.7	.06	146.6	10.0	0.07
Seven	180.8	8.7	.05	172.2	3.4	0.02
Eight	202.2	9.9	.05	--	--	-
Nine	216.5	10.5	.05	-	-	-
Ten	238.3	8.5	.04	-	-	-

It is apparent from the above results that the response to selection was substantial in the initial few generations as judged by the values of realized heritability. After four rounds of selection in Nai Basti stock and after six rounds of selection in Matunga the response ceased to be of appreciable magnitude.

These results indicate that in a character like fecundity which is largely determined by environment, the effect of the modified 'mass' selection is to deviate the 'high' and 'low' lines initially to a significant extent and then preserve the heterozygosity at a level characteristic of the character, i.e., exhibiting only 5 to 7% of variation as genetic. Such a situation is likely to arise in characters largely determined by non-additive actions of genes, viz., dominance and epistasis.

- References: Bell, A. E., Moore, C. H., and Warren, D. C. (1955). The evaluation of new methods for the improvement of quantitative characters. Cold Spr. Harb. Symp. Quant. Biol. 20:197-212.
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Narise, T. Genetic studies
on migrating activity in
D. melanogaster.

A number of inbred strains which differed from each other with regard to random- and mass-migrating activities were used for the present genetic study (for details

of material and methods refer to DIS 30, p. 149, 32, p. 153, and 34, p. 94). The findings obtained in the present experiment are: 1) Selection for high migrating activity was quite effective and the selected lines were found to be quite active in either mass-migration or random-migration. 2) Both the migrating activities were genetic characters and showed a dominance effect in some cases but not others. 3) By the method of substitution of II and III chromosomes, both migrating activities were found to be highly controlled by genes included in these chromosomes.

Nash, D. Selection for changes in the manifestation of the Hairless mutant.

macrochaetae has been studied. Selection has altered the susceptibility to loss of all macrochaetae except the median orbitals. Directional selection using four sub-culture, rotationally mated, populations has increased the mean numbers of bristles lost from four to ten, and reduced it to two. Breeding from the flies possessing sockets at all sites at which bristles are lost, and selecting for increased loss amongst this class of flies, has resulted in a 95% level of socket presence (base stock 65%) and a mean loss per fly of 7.5 bristles; the loss is concentrated at the eight sites where sockets were usually or commonly present in the base population.

Selection for the presence of one geographical group of bristles, the anterior and posterior orbitals, simultaneously with selection for absence of a second group, the anterior and posterior verticals, and for the converse, has met with limited success. It is considered that the limitation is a function of the developmental system; the verticals are developmentally associated and hence can react similarly to selection, but the posterior orbital is more closely associated with the verticals than with the anterior orbital.

The series of associations deduced from reactions to selection coincides with the series of bristles placed in order of percentage socket presence at sites where bristles are missing. Since there is no simple spacial relationship between the associated bristles in either of these series, and since the difference between presence and absence of sockets is probably a function of the time during bristle development at which the mutant is effective, it is suggested that there exists a developmentally significant temporal inter-relationship between bristles.

The effect of selection upon the
manifestation of Hairless

Stock	Base stock		Increased loss		Decreased loss		Orbital loss Vertical presence		Vertical loss Orbital presence		High socket presence	
Bristle	(gen 0)		(gen 19)		(gen 19)		(gen 19)		(gen 19)		(gen 19)	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
ant. vertical	25	98	83	100	1	100	14	100	72	100	95	100
post. vertical	21	98	84	99	3	100	28	85	48	98	70	100
post. orbital	39	88	97	49	4	92	52	64	40	97	86	100
post-vertical	99	61	100	35	91	96	100	27	100	54	100	89
ant.orbital	14	3	92	5	2	0	80	1	16	12	19	85
ocellar	9	1	31	0	3	0	37	0	20	0	6	0
(med. orbital)	.1	0	.3	100	0	-	0	-	1	50	1	33

Column (a) shows the percentage loss of bristles at a particular site; (b) shows the percentage of the sites at which the bristle vestiges are present.

Each percentage based upon data collected from 80 flies of each sex; i.e., 320 bristles. For a description of the selection criteria, see text.

Nash, Donald J. Fertility studies involving miniature-dominant.

The definitive description of miniature-dominant (m^D) by Slatis (Genetics, 1954) revealed that viability was about 20% to 50% in hemizygotes and 5% in homozygotes and that there was very low fertility in homozygous females. The present note includes results of further investigations involving this mutant. The foundation stocks for these experiments were developed from stock No. 65 from Pasadena (DIS-34). The stock is maintained by breeding m^D/FM^3 females to m^D males. In matings of m^D/m^D by m^D flies from the stock cultures approximately 35% of single-pair matings were fertile. Mass selection from the fertile matings has yielded stocks in which the per cent fertile matings is between 60% and 70%. It is possible to get direct comparisons of the relative viabilities of the m^D homozygotes compared to the hemizygotes as these are the only two classes of progeny segregating in these matings. A total of 3456 progeny were counted, comprising 817 females and 2639 males indicating m^D/m^D females are only 31% as viable as m^D males.

A preliminary study indicates that fertility is not impaired in $m^D/+$ females.

Nöthiger, R. Sepiapteridine and riboflavine in *Drosophila*.

The occurrence of riboflavine and sepiapteridine in the heads, testis and Malpighian tubules of *Drosophila melanogaster* (wild type "Sevelen" and homozygous sepiia-mutant) has been qualitatively investigated. By purifying the extracts with several solvents in column-chromatograms small quantities of practically pure riboflavine and sepiapteridine were obtained. The results are given in the table in a semiquantitative form.

genotype	organs	riboflavine	sepiapteridine
wild type	heads	+	++
	testis	+	++
	Malpigh.	++++	+
sepiia-mutant	heads	-	+++++
	testis	-	++
	Malpig.	++++	+

Sepiapteridine was found in all of the tested organs of the two genotypes, while riboflavine was not detectable in the heads and testis of the sepiia-mutant. Therefore it appears that the sepiia-locus not only affects the pteridines but also the flavines (although probably only quantitatively), since riboflavine was easily found in the heads and the testis of the wild type.

In both genotypes the Malpighian tubules contain much riboflavine. The UV-spectrum of this substance was found to be identical with that of synthetic riboflavine.

Novitski, E. A comment on the accumulation of inversions in natural populations.

Terzaghi and Knapp (Evolution, 1960) have shown that if *D. pseudoobscura* is heterozygous for inversions in only one pair of chromosomes, there is only a very small amount of zygote mortality but that if there are heterozygous inversions in two or more independent chromosomes, mortality is high, running up to fifteen per cent. This is similar to the result reported earlier for *D. melanogaster* (Cooper, Krivshenko and Zimmering) but is of particular interest for this species because it provides a simple explanation for the accumulation of inversions in the third

chromosome only of pseudoobscura: if a certain chromosome, as the third, by chance becomes variable in sequence, there will be no great selective disadvantage (indeed, it may provide an advantage through heterosis), but if subsequently any other chromosome (as the second) suffered an inversion, the new sequence would immediately be at an extreme disadvantage because of the great zygote mortality elicited when this new sequence found itself in a heterozygote in combination with an inversion heterozygote in the other chromosome and, similar to the argument applicable to the gradual decrease of the least frequent allele when a heterozygote has reduced adaptive value, inversions in any chromosomes other than the one with the fortuitous headstart will be eliminated.

While this is an attractive scheme to account for the situation in D. pseudoobscura, and a few other species in which variability is almost completely limited to one chromosome, it does not appear to hold for other species (D. robusta, for instance) where inversions appear to be distributed generally throughout the complement. It is then a natural, and important, question as to whether this difference has its basis in a different behavior of the chromosomes in the two types, or whether other (possibly non-genetic) factors (such as an initial geographic isolation of inversions in non-homologous chromosomes) played some role.

It is possible of course to answer this question for each species by a direct experimental attack; however, the writer would like to make a simple observation that suggests strongly that the chromosomes in all reasonably well studied species of *Drosophila* will behave the same and that the answer to this question must rest in the second alternative.

The zygote mortality described above is undoubtedly caused by non-homologous pairing, followed by irregular disjunction of non-homologous chromosomes. As has been emphasized recently by Sandler and Novitski (Genetics, 1956), such pairing manifests itself quite obviously in 3N meioses, where the frequency of balanced gametes is considerably lower than one would expect if the distribution of the single member of each trivalent towards a given pole were simply $(1/2)^N$ as elementary texts tell us it should be. If the mortality is high in a species like *melanogaster* with three major autosomes, then it should be very much greater (certainly more than doubled) for a species with four major chromosomes because of the greater possibilities for non-homologous pairing in the first place, and the reduced fraction of balanced gametes among all possible combinations in the second. It is now clear why the writer was unsuccessful in producing a permanent 3N stock of D. pseudoobscura (with four major chromosomes) although the initial 3N female did produce one 3N offspring (DIS 20). Similarly, it undoubtedly is not fortuitous that 3N lines of simulans can be maintained without difficulty, but that a 3N line in D. virilis is unknown despite the great amount of work done on this form, especially in Japan, by the most acute observers. The one apparent exception to this trend (to my knowledge) is the case of polyploidy reported by Stalker (Genetics, 1942), but this strengthens rather than weakens the argument, since it occurred in americana which has two pairs of its elements joined, thereby shifting the form from the five chromosome group to the three chromosome group like melanogaster.

The conclusion, then, is that the absence of fertile triploid stocks in species other than those with three major autosomes suggests that in most if not all species of *Drosophila* we would expect to find pairing potentialities conducive to the accumulation of inversions in some one chromosome (as in the third of pseudoobscura) at the expense of the others.

Novitski, E. A note on Sturtevant and Beadle's 1936 inversion paper.

For years I have puzzled over the explanation given on pp. 584-586, Genetics, 1936, concerning the behavior of tandem metacentric chromosomes. Questions directed

to the two authors have been of no help, because of the long time that has elapsed since that paper was written, and the loss of the notes on which that section was based. Others also have been mystified by their calculations, so I should like to report partial success, at least, in unravelling their puzzle.

The question is: how is the figure of 90.8%, given as the best estimate of the frequency of single crossing over in their tandem metacentric, arrived at? We know now, of course, that their experiment was confounded by non-random disjunction, which was unrecognized at the time. For this reason, the number of recovered crossovers was excessive, in fact, was greater than the number of non-crossovers and by their method of calculation should have amounted to 114% single exchanges (or four times the number of recovered rings, 313, divided by the number of patroclinous males, 1098). A second method of computation for the singles is to take the excess of males over females, 445, multiplied by four because each lost egg comes presumably from that one fourth of the exchanges producing dicentrics, the product then to be divided by twice the number of patroclinous males, 1098. This gives a value of 81.06%. (At this juncture an arithmetical slip seems to have been made, because to get the right answer we must use the value of 81.6%.)

Now we are confronted with the nasty problem of having two distinctly different estimates from the same data, 114% and 81.6%. This is resolved by, first, reducing the 114% to 100% since the first figure is clearly impossible, and then averaging 81.6% and 100% to get 90.8%!

It might also be mentioned that Table 15 on which the logic of the arithmetic is based is in error by having several figures reversed (undoubtedly a typesetter's error) and that for a better approach to this problem of estimating exchange in tandem metacentrics, one should refer to the author's paper on non-random disjunction (Genetics, 1951).

Ogaki, M. Inheritance of heat tolerance in D. melanogaster.

Two strains of Hikone-H and Mino-H have been reared at higher temperature than 30° C. through more than three years.

These stocks are able to breed at 31° C. successively, but heat susceptible stocks, for instance B;e11, se ss and others, reproduce no fly at the same temperature in the next generation. Genetical analysis of heat tolerance indicated that this character was polygenic, but the major gene was recognized as dominant, and located around the spindle fiber attachment on the third chromosome. It is notable that this locus is almost the same place as that of the nicotine sulfate resistant gene in D. melanogaster. It is also suggested that the selection pressure to nicotine sulfate increases the heat resistability without contact to heat. This seems to imply that the main gene of heat tolerance is very closely related to the nicotine resistant gene. Otherwise it is assumed that the heat tolerance might be a kind of vigor tolerance manifested by the same gene as that of the nicotine resistance.

Ogita, Z. Genetic control of ali-esterase activity in D. melanogaster.

Methylbutyrate was scarcely hydrolyzed by homogenates of two mutant strains (bw;st ss and bw;st;svⁿ), while homogenates of wild type strains have high

methylbutyrate-splitting capacity. From the results of genetical analyses, it became clear that the low ali-esterase activity was controlled by a recessive factor on the 3rd chromosome. Although it is still premature to conclude that the low activity is controlled by only one gene, it may be called ali for convenience sake. It is very interesting that the activity of the hybrid (ali/ali⁺) obtained from the crosses of low activity flies (ali/ali) and high activity flies (ali⁺/ali⁺), reveals an intermediate level of the parent strains.

The cholinesterase activity, however, showed no difference among these strains. In fact, the distribution of ali-esterase in parts of the body was distinctly different from that of cholinesterase. The results suggest that these esterase activities are controlled by different genes.

Okada, T. "Speed index" shown by the apodemes of drosophilid flies.

There is known to occur a phenomenon among systematic groups of Drosophilidae that the length of the apodeme of aedeagus shows

gradual decrease in accordance with the gradual development of the aedeagus itself. In other words, the phallosomal index (ratio in length of the aedeagus and its apodeme) tends to increase in the more advanced forms. A similar pattern of decrease is seen in the ratio in length of the basal plate and the stalk of the ejaculatory apodeme. The increase of these ratios should be of high biological significance, since it turns out to bring the more speedy contraction of muscles attached to the apodemes by means of shortening distance of muscular action. Therefore, this phenomenon can be interpreted by that of "speed index" proposed by Lull for the ratio in length of the metacarsus III and the femur, which becomes higher in the more speedy mammals as shown by N. American fossil horses.

Okada, T. "Compensatory adaptation" of the ejaculatory apodeme of drosophilid flies.

Although the general process of differentiation of the ejaculatory apodeme of the drosophilid flies seems to be the gradual shortening of the stalk in accordance with

the development of the plate, as expressible by the phenomenon of "speed index" (see above), in some forms, e.g., the members of the subgenera *Sophophora* and *Lordiphosa*, the stalk remains elongate and the effectiveness of muscle contraction seems to be compensatorily attained by the centripetal shifting of the junction of stalk on the plate. This kind of structural differentiation concerning a certain functional adaptation may be called "compensatory adaptation."

Oksala, T. A. The effect of autosomal inversion heterozygosity on crossing-over frequency in the X chromosome of *D. melanogaster*.

It is well known that inversion heterozygosity produces an increase in crossing-over frequencies in nonhomologous chromosomes. This increase has been found to be most striking around the centromere and at the

tips of the chromosome arms, being less pronounced in the central regions of the arms. When the X chromosome has been the affected chromosome and the relative increases in crossing-over frequencies have been determined in different regions along its whole length an essentially U-shaped curve has been obtained (e.g. Schultz and Redfield, Cold Spring Harbor Symp. Quant. Biol. 16, p. 184, fig. 5, 1951).

However, in papers dealing with this phenomenon the long central part of the X has not been closely marked. Therefore, the present author has carried out a more detailed analysis concerning the region from crossveinless to forked. This region was divided into five subregions (see below) and each of them was tested separately in four parallel experiments: the standard autosomal homozygote as a control, the In(2L+2R)Cy heterozygote, the In(3L+3R)P heterozygote, and the combination of these two autosomal inversion heterozygotes. The following relative increases (in per cent) from the control were found:

Region	Curly	Payne	Curly; Payne
cv - sn	18.3	19.5	56.6
sn - lz	3.1	4.8	33.5
lz - m	37.7	44.7	87.1
m - g	9.8	12.1	55.2
g - f	14.8	37.4	50.4

The curve computed from these data together with the data for the ends published by Schultz and Redfield is not U- but rather W-shaped with a fairly conspicuous peak in the very middle of the one armed chromosome (more or less around vermilion). This result is very much what one should expect on the basis of the tetrad analysis carried out by Redfield (Genetics 42, p. 723, Table 4, 1957). This analysis showed that in the presence of autosomal inversions the singles in the X tend to be replaced by triples (and to smaller extent by quadruples). When there are three crossovers in the same tetrad it is but natural that they are, due to interference, situated as far from each other as possible at even intervals, i.e. at both ends and in the middle. This state of affairs is reflected in the three-peaked curve obtained.

Further interpretation of the above findings is possible on the basis of the hypothesis put forward by the present author in an earlier connection (Oksala, Cold Spring Harbor Symp. Quant. Biol. 23, pp. 197-210, 1958).

Oshima, C. The persistence of deleterious genes in natural populations of D. melanogaster.

The second chromosomes were isolated from several Japanese wild populations by using the method of completely marked inversion.

The relative frequencies of chromosomes carrying lethal, semi-lethal, subvital and normal genes were estimated. The results obtained in 1959 had been reported in DIS 34 (p. 99). A similar sampling of second chromosomes from the same populations was carried out also in 1960 and the different classes of deleterious chromosomes were similar in relative frequencies. There was apparently no fluctuation between samples collected in 1959 and 1960. The lethal chromosomes have been maintained in the Cy balanced system in the successive generations. Diallel crosses were performed with all lethal strains to determine the allelic rate within and between populations. After maintaining the lethal chromosomes during the year 1959, they were subjected to cross-testing with new lethal chromosomes isolated in 1960 from the same populations. The allelic rates underwent scarcely any change during one year, but they seemed to have increased slightly in 1960. Most interesting was that the two lethals isolated in 1959 were found again in 1960 (allelic rate: 0.87 per cent). This finding shows that the two lethals have been maintained in the same population at least for a year.

Oshima, C. Dieldrin resistance in D. pseudoobscura.

About ten strains of each five kind of chromosomes for homozygous ST, AR, CH, TL and PP, which had been established by

Prof. Th. Dobzhansky from flies collected in California in 1959, were transferred to us in 1960. Several strains having the same chromosomal arrangement were mated, and female and male flies in the offspring were tested separately with test papers, containing 0.8, 0.4, 0.2, 0.1 and 0.05 per cent Dieldrin. Ten flies were exposed to test paper for one hour in a small vial and then transferred into another vial containing wet filter paper. After 24 hours, the number of dead flies was scored. Such a test was repeated ten times. On the other hand, two different chromosomal strains were crossed and the hybrid offspring were tested by the same method described above. The mortalities obtained in the experiments were transformed into arc-sine units and analyzed statistically. From the results of analysis of variance, the difference between chromosomal strains was highly significant and the order in resistance was observed as follows: TL > PP > CH > ST > AR. Flies having the relatively rare chromosomes such as TL and PP in California would be more resistant than flies having common chromosomes. The significant differences between resistances of both sexes and mortalities in doses were observed expectedly, but the difference between resistances of monochromosomal strain and hybrid strain could not be detected significantly.

Parsons, P. A. A biochemical polymorphism in Drosophila melanogaster.

Ebony (e^e) flies have less tyrosinase than wild type flies. Flies can be rapidly tested for tyrosinase content by growing larvae on the tyrosinase inhibitor

phenyl-thio-carbamide (P. T. C.), and ascertaining the lethal concentration of P. T. C. Recently, in an Oregon-R stock of flies an allele at the ebony locus, reacting to P. T. C. in a similar way to e^e flies, has been found. This "ebony" allele is wild type in body colour. The Oregon-R stock is polymorphic for this allele with a gene frequency of 13%. Flies collected in the wild in September, 1961, at Eugene, Oregon, have also turned out to be polymorphic. Hence the allele in the Oregon stock probably came from the wild population from which the stock was derived. Other polymorphic stocks found so far are Oregon-K, Kaduna (from Africa) and Bikini with gene frequencies of 45%, 40%, and 67% respectively, while a Canton-S stock from the California Institute of Technology is 100% "ebony." Flies collected in the wild near Cambridge are also polymorphic. Hence this polymorphism is probably very widespread in the wild. It probably explains, in part, the variability of tyrosinase estimations found in different stocks.

The selective basis of the polymorphism is unknown, but two observations deserve mention, namely (1) the gene frequencies in the wild Cambridge flies has not varied much during the summer of 1961 and (2) there is a degree of male sterility of the "ebony" homozygotes in the Cambridge flies. It is, however, remarkable that the "ebony" allele has probably persisted in the Oregon Laboratory population for at least 35 years, since the Oregon-R stock was collected at Roseburg, Oregon, in 1925 or before by D. E. Lancefield (Bridges & Brehme, 1944).

Pelecanos, M. Induced oögonial lethals in Drosophila.

A simple method for the induction of high frequencies of oögonial sex-linked recessive lethal mutations by larval feeding with

diethylsulphate is reported.

Tests for the mutagenicity of chemicals are usually carried out on males. By the larval feeding method, only one chemical (chloroethyl methanesulphonate) has so far been shown to have a mutagenic effect on females (Auerbach and Sonbati, 1957).

Larvae were cultured on an aseptic medium with the following composition:

Glucose	10%
D. C. L. Yeast	10%
Agar	3%

The medium was autoclaved and 0.4 per cent propionic acid was added at 65° C., and diethylsulphate at 60° C. The medium was dispensed as 25 ml portions into sterile bottles. Oregon-K eggs were sterilised using Sang's (1956) method, and one hundred newly hatched larvae were transferred to each culture. Hatching males and females were collected as virgins and tested for sex-linked recessive lethal mutations by the Muller-5 mating method. Four three-day broods were studied; each male was mated with two females and vice versa. Table 1 shows the results obtained over four broods for treated males and females. A high rate of sex-linked recessive lethals was maintained for both sexes over the four broods tested.

Results so far available do not allow comparisons between the effects of larval treatment in males and females, since both spermatogonia and primary spermatocytes are present during the male larval period, whereas only oögonia occur in the female larvae. However, the significant heterogeneity X^2 in females suggests that there might be stages of different sensitivity among the larval oögonia.

- References: 1) Auerbach, C., and E. M. Sonbati. DIS 32, p. 109 (1957).
2) Sang, J. H. (1956). J. Exp. Biol. 33, 45.

Table 1.

Concentration of diethylsulphate 0.5% (Molarity 3.2×10^{-4}). Treatment throughout the larval life. Temp. 25° C.

Time of Development				9 days		
♂♂ Treated				♀♀ Treated		
% Survival				50.50		
Broods	No. chrom.	No. lethal	% lethal	No. chrom.	No. lethal	% lethal
1st	672	83	12.35	856	135	15.77
2nd	626	63	10.06	864	136	15.81
3rd	514	43	8.36	514	99	19.26
4th	422	52	12.32	672	143	21.28
Total	2234	241	10.79	2906	513	17.65

Heterogeneity X^2 3 D.f.

for ♂♂ = 6.01 (0.20 > P > 0.10)

for ♀♀ X^2 = 11.91 (0.001 > P > 0.01)

Phillips, B., and E. A. Carlson.

Time of action of the lethal effect in the dumpy series.

Various alleles of the dumpy series manifest a lethality as homozygotes or as compounds derived from interallelic crosses. Several such lethal-bearing alleles were mated to

Ore R wild type flies and their heterozygous F_1 progeny were used for examination of the time of action of the lethal effect. In most of these tests, the progeny were mated in vials and transferred to petri dishes containing a sugar and bactoagar medium treated with streptomycin. This system permitted a relatively germ free development on a transparent food medium.

In Table 1 the flies were examined for their egg hatchability after fertilization. These results suggest that the homozygous alleles ol^S , lv^1 , and olv^1 all die in the embryonic stage (before the egg collapses with the emergence of a first instar larva). The mutant l^m , as a homozygote, survives this stage and dies at a later stage (the third larval instar). In heteroallelic crosses, a partial complementation for the lethality exists for most of the crosses. Thus l^m/ol^S , ol^S/olv^1 , and l^m/olv^1 show little mortality in the embryo. In lv^1/ol^S , and l^m/lv^1 there is less lethality in the embryo than for homozygous lv^1 . Only lv^1/olv^1 maintains a high mortality in the embryonic stage.

In Table 2, the mortality is shown to exist prior to the pupal stage, based on the survivors from pupae to adults in these crosses. This is important because both the oblique wing (o) and the thoracic vortex (v) effects of the dumpy series are manifested at the pupal stage at slightly different times (see Carlson and Falk, this issue). In Table 3, the lethality not manifested at the embryonic stage is shown to exist primarily during the third instar larval stage.

The mechanism for the lethality may differ, not only in those instances where their time of action differs, but in those cases where the same apparent stage is affected. Thus uncollapsed eggs of ol^S/ol^S and olv^1/olv^1 genotypes are yellow, but eggs of lv^1/lv^1 genotype are white. The color of uncollapsed l^m/l^m embryos (which account for only a small portion of the total lethality of this compound) is also white. No attempt was made in this study to examine first and

second instar larval mortality, but they must be slight, judging by the results of Tables 1 and 3. Because of the small numbers used in each test, no attempt was made to subtract the control values from the experimental cultures. This provides a maximal lethality which is, of course, higher than the corrected values which should have the frequency of the control lethality subtracted.

Table 1

Maximum lethal frequency of dumpy alleles during embryonic stage

Cross	Total eggs laid	Uncollapsed eggs	Maximal % lethality
$l^m/+ \times l^m/+$	274	9	3.3
$ol^s/+ \times ol^s/+$	133	31	23.3
$lv^1/+ \times lv^1/+$	130	28	21.5
$olv^1/+ \times olv^1/+$	177	42	23.7
$lv^1/+ \times ol^s/+$	132	8	6.1
$l^m/+ \times lv^1/+$	127	13	10.2
$lv^1/+ \times olv^1/+$	149	23	15.4
$l^m/+ \times ol^s/+$	124	2	1.6
$ol^s/+ \times olv^1/+$	126	3	2.4
$l^m/+ \times olv^1/+$	169	9	5.3

Table 2

Absence of lethality during pupation for dumpy alleles

Cross	Eggs laid	Pupae	Adults
$l^m/+ \times olv^1/+$	183	124	122
$ol^s/+ \times ol^s/+$	109	68	66
$l^m/+ \times l^m/+$	227	122	119
$olv^1/+ \times olv^1/+$	192	137	120
$lv^1/+ \times lv^1/+$	104	73	70
$+/+ \times +/+$	64	50	50
$lv^1/+ \times ol^s/+$	187	131	129
$l^m/+ \times lv^1/+$	145	97	93
$ol^s/+ \times l^m/+$	99	59	59
$olv^1/+ \times lv^1/+$	129	99	93
$ol^s/+ \times olv^1/+$	100	58	56

Table 3
Maximal frequencies of lethality at different developmental stages for dumpy alleles

Cross	Eggs laid	Lethality stage A	Third instar larvae	Lethality stage B	Pupae	Lethality stage C	Adults	Maximal per cent lethality
+ / + x + / +	55	9.1	50	3.64	48	0	48	12.7
olv ¹ / + x olv ¹ / +	92	25.0	69	16.4	54	5.4	49	46.8
l ^m / + x l ^m / +	105	4.8	100	23.8	75	0	75	28.6
l ^m / + x olv ¹ / +	122	4.1	117	38.7	82	0	82	32.8
ol ^s / + x olv ¹ / +	185	10.8	165	31.4	107	0.5	106	42.6

Stage A = lethals occurring from embryo to second instar larvae

Stage B = lethals from third instar larvae to beginning of pupation

Stage C = lethals during pupation

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Pipkin, S. B. Spontaneous mosaics in the progeny of triploid females.

Several spontaneously occurring rare mosaic types have been observed in the progeny of triploid females in the course of experi-

mental work. These verify the previously known facts of sex determination and fail to give indication of hormonal effects of tissues of diverse origins upon one another except in the last case. Certain mosaics demonstrate the simultaneous loss of X, II, and III. Descriptions of the mosaics follow: (1) Triploid-diploid female. This individual, coming from a cross of ru, T(3,4) 30/ca, T(3,4) 28 males with y^2 ; ru ca triploids, had a gray body and displayed the mutants ru and ca on both sides. Both eyes possessed the large eye facets typical of 3A tissue. The left wing showed the coarse wing texture characteristic of 3A tissue and was slightly longer than the right wing which exhibited the fine texture of diploid wings. All primary and secondary sexual characters were female. It is presumed that the genetic composition of the left side was $+y^2/y^2$; II/II/II; ru, 30L+ca, 28R/ru ca/ru ca (hyper-triploid for the short section between T(3,4) 30 and T(3,4) 28.) The right side was 2X2A, having lost one y^2 X chromosome, one II, and either an intact III or both the 30L and the 28R fragments of III.

(2) Triploid-diploid female. This individual arose from a cross of ca, T(3,4) 85C/ru, T(3,4) 89E males with y^2 ; ru ca triploids. The mosaic possessed gray body color throughout, and both eyes were red (not claret) and not-roughoid, but they exhibited diploid facet size. Both wings, on the other hand, showed coarse 3A texture. All primary and secondary sexual characters were female. The fly presumably started out with $+y^2/y^2$; II/II/II; 85CL + 89ER/ru ca/ru ca (a hyper-triploid for the short section between T(3,4) 85C and T(3,4) 89E). Subsequent loss of one y^2 X, one II, and one intact ru ca III chromosome produced the diploid eyes.

(3) Triploid-intersex. This individual was found in the homozygous y^2 ; ru ca triploid stock and was therefore homozygous for these three mutants. Both eyes were the same size and shape and appeared like those of a triploid female. The right wing was distinctly shorter than the left but both wings showed coarse 3A texture. The right foreleg bore a sex comb of 8 prongs, the usual size of intersex sex combs in this stock. No sex comb was on the left foreleg. The abdomen was typically female, not bent or misshapen, and genitalia were female. Presumably the anterior portion of the right or intersex side arose from tissue which had lost 1X from the 3X3A intersex complement.

(4) Triploid-diploid female-intersex triple mosaic. This individual occurred in the progeny of T(3,4) 28/ ru ca males with y^2 ; ru ca triploid females. It is thought to be the result both of double fertilization and chromosome loss. The left wing was a mere stub. The head, forelegs, left half the thorax, left half plus the right distal half the abdomen were gray (not yellow²). The left eye was triploid (3A) in facet size and showed the mutants ru and ca. The top one fifth of the right eye was likewise ru ca and triploid in facet size. The lower four fifths of the right eye was not-ru, red (not-ca) and diploid in facet size. The right half the thorax, right anterior half the abdomen were yellow. The right wing was clearly of 3A texture. All primary and secondary sexual characters were female. Presumably the left gray, ru ca triploid part of the body arose from a union of sperm bearing a gray X and intact ru ca chromosome III and one chromosome II with a diploid egg possessing y^2/y^2 ; II,II; ru ca/ ru ca. The yellow tissue including the 3A right wing, right half of thorax, and right anterior half of abdomen was apparently intersex, arising from the same zygote as the gray half except for loss of the gray X chromosome. The red, not-ru (not-roughoid) part of the right eye must have arisen by fertilization of a haploid second oöcyte (one y^2 X/one II/one intact ru ca III) by a sperm carrying a gray X, one II/ and the T(3,4) 28 chromosome fragments bearing the normal alleles of ru and ca. An alternate explanation of the non-roughoid, red (not-ca) lower 4/5 of the right eye is that this tissue is haploid, derived from the development of above sperm alone.

(5) Intersex-hypointersex mosaic. This individual arose from a cross of ru, T(3,4) 12/ca, T(3,4) H3 males and y^2 ; ru ca triploids. It was typically intersexual throughout. The eyes showed 3A texture on both sides, but the left eye was

not-roughoid, claret; the right eye, roughoid and claret. The mosaicism of the eyes was unmistakable since the mutant *ru* narrows the eye and disturbs facets in the intersex eye considerably more than in a diploid *ru* eye. Body color was y^2 throughout. Wings were slightly outstretched and had coarse 3A texture. Genitalia were of the fragmentary male type; anal plates, of the female type. Sex combs of 8 and 9 prongs were present on the right and left forelegs respectively. The fly is presumed to be the result of union of a sperm bearing a Y chromosome/one II/ and the ca, T(3,4) H3 chromosome III with a diploid egg containing y^2/y^2 ; II/II; and *ru* ca III/*ru* ca III followed by the loss of the H3L fragment in the tissue giving rise at least to the right eye. This mosaic is interesting because the H3L fragment represents half of chromosome III, and a hypointersex lacking such a long fragment does not ordinarily live. Even if the right eye were derived from a different syngamy; i.e., from the union of sperm bearing a Y chromosome; one II; and *ru*, 12L + H3R with a diploid second oöcyte carrying y^2/y^2 ; II/II; *ru* ca/*ru* ca, then hypointersex tissue would result. This hypointersex tissue would lack in triplicate one dose of the section of the III chromosome between T(3,4) 12 and T(3,4) H3 and would not be expected to survive except as mosaic tissue. In this mosaic alone may we observe evidence of a "hormone effect" since the right eye hypointersex tissue survived in the mosaic fly.

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Pozzi, L. V., S. Giavelli, G. P. Sironi, and E. Gallucci. Frequency of recessive sex-linked lethals in *D. melanogaster* spermatogenesis, in O_2 , N_2 and air, with 600 r and 1200 r.

spermatids. Gas treatment is given before, during and after irradiation. N_2 treatment removes the sensitivity peak, that is different developing stages do not show significantly different mutation frequencies. Maximum O_2 effect is found in meiotic stages. The frequency pattern of the two doses in regard to gas atmosphere is quite constant.

Using a mating system of one irradiated male to three Muller 5 females, with renewed matings every 24 hours, a sensitivity spectrum of different sperm cells stages is scored, which, in air, shows a peak corresponding to

Reitan, P. J., and M. E. Annan. The effects of dehydration on the frequency of irradiation induced embryonic abnormalities in *Drosophila*.

eight days following treatment and increased the number of cross-over like exchanges in the X chromosome and gross chromosomal rearrangements. It was suggested that the increases noted were probably due to induced lethal mutations. The work reported here has demonstrated an increase in the number of induced embryonic abnormalities in the eggs of females that had been desiccated prior to irradiation. Abnormalities noted include only those in which a substantial number of cells had been produced and in which the developmental sequence was interrupted prior to sixteen hours of development.

It has been demonstrated by Herskowitz that the dehydration of *Drosophila* females prior to their exposure to irradiation increased the frequency of mortality of eggs laid during the first

Virgin *Drosophila melanogaster* females were desiccated, desiccated and irradiated or irradiated only. Desiccation was carried out by exposure to 25% humidity for six hours. Irradiation consisted of exposure to 3,000 r or 4,000 r from a cobalt-60 source at 40 r per minute. Eggs were collected at eight hour intervals during the first five days following treatment. They were divided so that some were used for embryological examination and the remainder for the determination of hatchability. Eggs for embryological examination were allowed to develop for 16 hours, then fixed in Carnoy's and stained with iron hematoxylin.

The table shows the results from three experimental groups. Hatchability data was highly variable in all treatment groups; however, the number of gross developmental abnormalities noted among the eggs which had clearly undergone development was consistently greater in the embryos from desiccated and irradiated flies. Desiccation alone did not have any effect on the embryonic development. The data support the hypothesis that dehydration increases the number of irradiation induced lethal mutations.

Experimental Group		No. of embryos	No. unfertilized eggs	No. grossly abnormal	% of developing embryos showing gross abnormalities
1	ax	45	8	6	16.2
3,000 r	bx	82	27	3	5.4
2	ax	197	74	22	17.8
4,000 r	bx	263	100	14	8.5
3	ax	525	124	37	9.2
4,000 r	bx	338	68	22	8.1
Combined	ax	767	206	65	11.6
	bx	683	195	39	7.9

ax - denotes desiccated and irradiated

bx - denotes irradiated only

(This work was supported by grant A-2162 from the National Institute of Health.)

Roberts, Paul. Autonomy of a claret nondisjunctional ovarian transplant in D. melanogaster.

Ovaries from third instar larvae of the genotype y/y; can^d/can^d were transplanted into third instar Oregon-R female larvae.

Each survivor bearing an ovarian implant was mated, after eclosion, to a y B male. All but one of the females so mated produced no yellow or exceptional offspring due to failure of the transplanted ovary to become connected to an oviduct. However, from a female giving both y and y⁺ progeny of both sexes, all of the 59 y⁺ progeny were regular. Of the 20 y progeny obtained, three were exceptional males and one was a gynandromorph. The percentage of exceptional progeny (20%) from a homozygous can^d ovary developing in a wild type female is not significantly different from the percentage of exceptions obtained in this laboratory from mating homozygous can^d females.

Ronen, Amiram. Induced somatic recombination in the third chromosome of Drosophila melanogaster.

An attempt was made to study spontaneous and induced somatic recombination in the third chromosome of Drosophila melanogaster in flies of various genotypes.

All individuals investigated were heterozygous for the same marker gene, Sb, carried on a structurally normal chromosome. Their X- and second chromosomes were either homozygous for the standard arrangement or heterozygous for various inversions. Third instar larvae of each genotype were given an X-ray dose of 1170 r (190 r per minute) at 80-90 hours after hatching and were then allowed to pupate. Individuals of each genotype were kept

as unirradiated controls. The adult flies were searched for normal bristles, assumed to be due to somatic crossing-over between the locus Sb and the centromere.

The bristles were scored according to a schedule fixed in advance. This schedule included 40 specified bristles on the head and thorax in the first series of the experiment, while 34 bristles of each individual were examined in the second experimental series (some of the bristles, numbers 8, 13 and 14 in Table 1, having been found to give only a small frequency of normals).

The frequency of normal bristles on the head and thorax was as high as 0.08 per irradiated fly among 4970 flies, but not a single normal bristle was found in 1590 unirradiated controls. The frequency of flies exhibiting normal bristles did not differ significantly between the various genotypes (the over-all χ^2 test did not indicate any significant deviation from homogeneity), but different bristles showed different frequencies of normals (Table 1). It should be remembered that most of the bristles examined in these experiments (except for the humerals) are derived from the dorsal meso-thoracic imaginal disc.

The interpretation of the results on the basis of induced somatic crossing-over is complicated by several factors. The most important among these is the complete and unexpected absence of spontaneous recombination in the unirradiated controls.

However, it should be stressed that the frequency of normal bristles observed in the irradiated individuals appears too high to be accounted for by induced somatic back mutations. Even on the assumption that normal bristles may be due to induced mutations to suppressor genes of Sb at up to 10 different loci, in addition to back mutation at the Sb locus itself, the average mutation rate per r per locus would still have to be as high as 1.4×10^{-7} in order to account for the observed effect.

Table 1
The frequency of normal bristles among various bristles
of the thorax and head, after irradiation

No.	Bristles	No. tested in each fly	Experiment I		Experiment II	
			No. Normal	Mean	No. Normal	Mean
1-4	ocellars, orbitals, verticals, post verticals	16	56	3.5	44	2.75
5	ant. dorsocentrals	2	12	6.0	8	4.0
6	post. dorsocentrals	2	15	7.5	12	6.0
7	ant. postalars	2	19	9.5	9	4.5
8	post. postalars	2	7	3.5	-	-
9	ant. scutellars	2	18	9.0	5	2.5
10	post. scutellars	2	10	5.0	12	6.0
11	supralars	2) 33	2.75) 23	5.75
12	ant. notopleurals	2				
13	post. notopleurals	2				
14	prescutellars	2				
15	humerals	4)		12	3.0
Total		40	170		125	

Schepers, A. M. An interaction in Pteridine metabolism between garnet and brown genes in D. melanogaster.

of wild type, a garnet allele resembling g^2 , a brown allele, and the double mutant $g; bw$. Both mutants were found to differ from wild type only quantitatively in respect to their pteridine patterns. In particular, 2-amino-4-hydroxypteridine is present in both of them although in lower concentration than in wild type. The double mutant, on the other hand, contains no detectable quantities of this substance. The activity of xanthine dehydrogenase which has been implicated in formation of pteridines, was determined following the method described by Mitchell, and measured fluorometrically. Enzyme activity in the double mutant was found to be present though reduced with respect to wild type.

Two-dimensional paper chromatograms using various solvents were made of extracts from 25 heads. All flies used were three days old. A fluoroscopic comparison was made

Schulten, G. M. A case of aberrant sex-ratio in D. melanogaster.

the ratio shifts towards normal, with a $1\text{♀} : 1.5\text{♂}$ ratio at 17°C .

When the males of this stock were crossed to unrelated stocks, the aberrant sex-ratio did not reappear in F_1 or subsequent generations. Also, crosses of "sex-ratio" females to unrelated males gave normal sex-ratios, save in the cross to $Cy\text{-Oster}/Pm; Ubx^{130}/Sb$ males in which case there was a deficiency in females of the genotype $Cy\text{-Oster}/Kr$ resulting in a ratio of $1\text{♀} : 2.5\text{♂}$.

Substitution, in the "sex-ratio" stock, of a Pm -chromosome for the Cy -chromosome, resulted in a ratio of about $1\text{♀} : 1.5\text{♂}$. After substitution of a Pm -chromosome for the Kr -chromosome, a ratio of about $1\text{♀} : 3\text{♂}$ was obtained. The sex-ratio became normal in all cases where the X -chromosomes of the "sex-ratio" stock had been replaced by X -chromosomes from other sources.

It is tentatively assumed that this temperature-sensitive deviation from a normal sex-ratio is caused by anomalous behaviour of a mutant X -chromosome depending on the presence of the Cy and Kr second chromosomes.

A Kr/Cy stock which had been selected for a high penetrance of Kr gave in contrast to the original stock a sex-ratio of about $1\text{♀} : 4\text{♂}$ at 25°C . At lower temperatures

Schwinck, I. Drosoppterin formation and semi-lethality of the mutant rosy in temperature experiments.

The pleiotropic pattern of the mutant rosy (ry) includes the following characters: (1) reduced amount of drosoppterins (red eye pigments), (2) nonautonomous formation of drosoppterins as demonstrated in transplanta-

tion experiments, increased drosoppterin formation at low breeding temperature, (4) semi-lethality in the late pupal stage and during emergence of the fly, (5) aberrant morphology and function of the Malpighian tubes, (6) no xanthine dehydrogenase activity and no accumulation of isoxanthropterin and uric acid. In course of the study of cause and relation of these characters and their dependence on temperature, first the critical time for the manifestation of drosoppterin quantity was determined. For the strain $v; ry^2$ this was found to be the very late pupal stage, as revealed by changing the breeding temperature from 18°C to 26°C , and vice versa, at the following stages: (a) 1.larval stage, (b) early 3.larval stage, (c) prepupa, (d) pupa 36 hours after pupation, (e) pupa with beginning red pigment formation, (f) imago 0-2 hours after emergence. It seems rather interesting that the critical time determining the drosoppterin quantity is the period when actually the drosoppterin pigments are deposited in the eyes. -- Furthermore, the temperature effect on the drosoppterin quantity and on the semi-lethality of the late pupae and emerging flies was studied in various rosy alleles and compounds, as well as in isogenic $cn; ry^2$ strains. With decreasing temperature the drosoppterin formation increases parallel to increasing viability of pupae and young flies. For the radiation induced rosy alleles ry^4 , ry^6 , ry^8 and ry^9 (Chovnick, A., A. Schalet, and R. P. Kernaghan, Rec.

Genetics Soc. America 30, p. 68, and Genetics 46, p. 858, 1961) the temperature effect on both, the drosoprotein formation and the semi-lethality, is similar to ry^1 and ry^2 ; the death rate in the late pupal stage being about 40-60% for a breeding temperature of 26° C. as compared with below 5% for ry^+ strains. However, for the compounds of these rosy alleles the semi-lethality at 26° C. decreases in certain crosses below 5%, although the eye color is typical for 26° rosy-breed and the aberrant morphological structure of the Malpighian tubes could not be distinguished from those of rosy strains with low emergence rate. A rather strong influence of the genetic background on the temperature dependence of the drosoprotein quantity and the pupal semi-lethality was found in experiments with various isogenic cn ; ry^2 strains. Crossing flies from certain isogenic strains with flies from the original ry^2 stock resulted in an improved viability at 26° C., the pupal death rate being about 10% in the F_1 as compared with about 50% in the parental stocks raised in the same incubator.

(Supported by a grant from the USPHS RG-7464.)

Seki, T. Absence of beta-alanine in hydrolyzate of the pupal sheaths of ebony mutant of D. virilis.

After washing in water and drying in the air, 50 mg of pupal sheaths were homogenized with 80% ethanol and filtered and washed with 80% ethanol, followed by 99%

ethanol. The residue was hydrolyzed in 6 N HCl at 110° for 24 hours in a sealed tube.

The fluid was evaporated rapidly by using rotary evaporator and dissolved in 2 ml of deionized water. One ml of the solution was added on a column of Amberlite CG-120 (H form, 0.8 X 140 cm) and eluted with 1.2 N HCl. The effluent was collected in fractions of 2.9 ml. Each fraction was neutralized with sodium hydroxide solution and assayed according to the method of Yemm and Shen.

No beta-alanine was detected in the hydrolyzate of pupal sheaths of ebony mutant strain, in contrast with that of wild strain, in which a considerable amount of beta-alanine was present. Similar results were obtained with black pupa mutant strain of Musca domestica and with sooty mutant of Bombyx mori.

Sherwood, Eva R. All-male offspring from heatshocked cultures.

Accidental exposure of a few hours' duration to heatshock of 31° C. of four bottles with crosses of $y^2 sc w^a ec$, $sc^8.Y \text{ ♀♀}$ x $ywB \text{ ♂♂}$

yielded only male progeny (a total of approximately 250-300) of ywB , $sc^8.Y$ constitution, except for 2 females of the expected genotype. At 25° C. approximately equal numbers of male and females were produced. Repeated single pair crosses resulted in the same sex ratio effect, when the shock was given on the 4th, 5th or 7th day after the start of the cultures. Those shocked before the third day after mating had no offspring.

Heatshock to crosses involving different attached and unattached X chromosomes, as well as the $sc^8.Y$ carried in male or female showed that the particular attached XXs of $y^2 sc w^a ec$ constitution were responsible for the lack of female progeny in the next generation.

Shima, T., A. Kaneko, and E. Momma. Hatchability of eggs during varying lapses from the time of mating in D. virilis.

Preliminary examinations were made in order to analyse the sensitivity for X-rays to spermatogenesis. Fifteen young virgin females were mated singly in vials each with a single young male for three days,

and then the females were kept without males. Eggs were laid and their hatchabilities were observed every day during 15 days after the separation from males. As shown in Table 1, the largest number of eggs (29.5 per single female) were observed on the 5th day of single culture, the smallest one (1.4) being on the 8th day. Hatchability

of the eggs laid on every day was about 90 per cent within the first ten days. From the 11th day on, however, it showed a remarkable decrease (6%). No egg hatched out from those laid after the 12th day. Dissected seminal receptacles showed a rapid decrease of sperm after the 11th day.

Table 1

Hatchability of eggs, based on 15 females

Days after mating	No. of eggs laid	No. of hatched eggs	Hatchability (%)
1	86	81	94
2	120	107	89
3	93	86	93
4	90	86	96
5	443	434	98
6	167	158	95
7	357	346	97
8	20	15	75
9	50	42	84
10	266	244	92
11	235	14	6
12	93	0	0
13	28	0	0
14	173	0	0
15	51	0	0
Total	2272	1613	71

Snyder, L. A. The effect on TEM-induced mutations and translocations of storing treated spermatozoa in the female.

with no sensible change in frequencies of sex-linked recessive lethals or chromosome losses. The post-copulatory vaginal douche treatments of females admit to uncertainty in interpretation since the chemicals used are highly penetrating and would be expected to reach the ovaries of the females in which treatment was carried out. Similar experiments were repeated, using inter-abdominal injection of day-old males, and the results were in agreement with those reported earlier. Using 2×10^{-4} triethylene melamine in 0.7% saline resulted in a 4-fold increase in translocations after 3 days storage in the female of treated spermatozoa, with no increase in sex-linked recessive lethals or chromosome losses.

Studies by Schalet (1955: Genetics 40, 594), and Herskowitz (1956: Genetics 41, 605) on the effects of storing nitrogen mustard- or triethylene melamine-treated spermatozoa in females, revealed sharp increases in translocation frequencies,

Sondhi, K. C. Selection for an invisible pattern of macrochaetes in *Drosophila*.

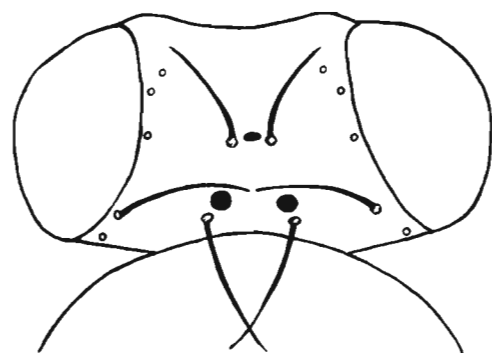
ocelli on top of the head in *Drosophila subobscura*. Earlier experiments had shown that if selection for higher numbers of structures were practised on a population homozygous for the mutant, it was possible to obtain a population in which a high

The present experiments describe the effect of selection on the expression of the ocelli-less mutant, a sex-linked recessive, variable for the number of bristles and

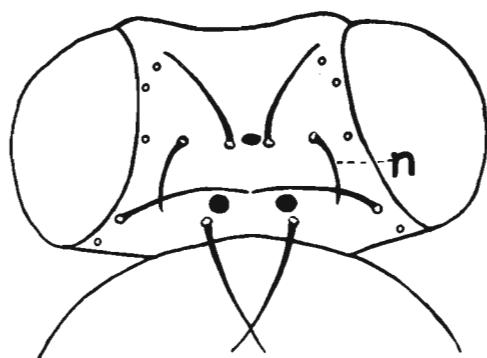
proportion of flies had a wild-type phenotype. There was found to be a partial barrier at the wild-type phenotype, preventing progress beyond the "score" of six bristles and three ocelli (Fig. 1 A). The frequency of repeated bristles

(two or more bristles lying close together at the site normally occupied by one) was found to be much greater than in the foundation population. In a few flies more than three ocelli were seen. In the present experiments an attempt was made to observe the effect of continued selection, in the hope that an increase in the frequency of genes for higher score might bring forth the expression of new structures.

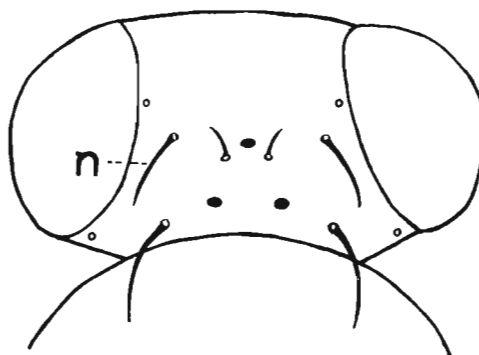
The results of the present experiment show the appearance of a novel pair of bristles. These bristles (Fig. 1 B) always appear at a specific site and have a definite orientation. The "neomorphs" are normally absent in *Drosophilidae* but they are present (Fig. 1 C) in a family closely related to it, the *Aulacigasteridae*.



A



B



C

Figure 1

The pattern of the macrochaetes and ocelli in A, wild-type *Drosophila*; B, selected line; C, *Aulacigaster leucopeza*; n, neomorph; o, the positions of macrochaetes; o, ocelli.

The appearance of these structures as a consequence of continued selection on the mutant population, after the wild-type phenotype had been reached, suggests that a particular frequency of genes for higher score is required for their expression. This is also suggested by the higher frequency of neomorphs in females, which have a higher mean number of bristles and ocelli than males.

A hypothesis is postulated, similar to that suggested by Stern (1954, Proc. 9th Intern. Congr. Genet., 6:355-369), to explain the origin of neomorphs in terms of an unvarying "prepattern" which determines the positions of these structures and

a common "precursor" of bristles and ocelli which must be present in the required amount if structures are to be formed. It is suggested that if the amount of the precursor is increased to a certain threshold, a new bristle is formed in response to the peak of the prepatter which is present in the wild-type flies, but to which wild-type cells are not competent to respond.

Sperlich, D. Hybrids between D. melanogaster and D. simulans in nature.

the males of the two species can be easily detected and separated, the females are practically identical in appearance. In order to determine the frequency of females in nature, cultures from individual females, mated already in nature, were reared. Out of a total of 141 cultures the examination of the male offspring showed that 81 (57.5%) of the females belonged to D. melanogaster and 53 (37.5%) to D. simulans. The remaining 7 cultures (5%) gave only sterile female offspring of a typical appearance, most probably resulting from the cross D. melanogaster ♀ x D. simulans ♂ in nature. This frequency of cross-matings is surprisingly high and cannot be explained just as a chance happening. It may be noted that crosses D. melanogaster ♀ x D. simulans ♂ with Lipari-strains are almost always successful also in the laboratory. It seems that the sexual isolation between these two species at the island is not so strong as on other places, perhaps on a genetical basis. But we could not establish the existence of the reciprocal matings in nature.

In *Drosophila* samples caught in May/June at the island of Lipari (South Italy) the most frequent species observed were D. melanogaster and D. simulans. Whereas

Stern, Curt, and Eva R. Sherwood. Can primordial germcells of the genotype $\bar{X}XY$ produce functional sperm?

somes from $\bar{X}Y^S Y$ males (Neuhaus, 1937, Genetics) suggests that spermatogonia with two X chromosomes in males of non-tra genotype can develop normally. An attempt was made to discover gonosomic mosaics in which the soma is normally XY male and part or all of the testes contain primordial germcells with two X chromosomes. Would they form functional sperm? Frost (1961, DIS 35:81) has shown that somatic double nucleus mosaics occur among the offspring of 3N females with a low frequency. Therefore, sons of 3N females $y^2 sc w^a ec$, FM4 mated to FM4, $sc^8.Y$ were progeny tested. These sons were somatically FM4, $sc^8.Y$. Would an occasional individual contain functional germcells derived from an attached-X female pronucleus fertilized by a $sc^8.Y$ sperm? If so, $y^2 sc w^a ec$ daughters should be observed among the offspring of the FM4, $sc^8.Y$ males mated to attached-X females of the yf genotype. None of 3,744 FM4 males, mated in groups of 6, produced $y^2 sc w^a ec$ daughters. In a similar experiment, none of 4,740 ywB sons from the cross yf x ywB when mated to $y^2 sc w^a ec$, $sc^8.Y$ females produced any yf, $sc^8.Y$ daughters. Thus, either none of the total of nearly 8,500 males were gonosomic $\bar{X}XY$ -XY mosaics, or no functional sperm is formed by cells derived from $\bar{X}XY$ primordial germcells. Only experiments on a much larger scale than those reported here can furnish a decision.

Spermatogonia with two X chromosomes in transformed phenotypic tra male individuals are unable to develop into functional sperm (Seidel, 1960, Naturw.) but the recovery of clusters of sperm with attached X chromo-

Stevenson, Richard. Altitudinal distribution of inversion heterozygotes in D. robusta.

preparations were made, and, at the time, the samples were considered too small to be statistically significant. The data, however, may be of some interest to other workers.

During the summers of 1950, 1951, 1952, and 1954, collections of D. robusta were made from several sites on Unaka Mountain in northeastern Tennessee. Salivary gland

The accompanying table shows the frequencies of inversion heterozygotes at different elevations. These data are in sharp contrast to those of Stalker and Carson (1948), who collected in the Smoky Mountains.

It is suggested that the Unaka collections were, in the main, from marginal populations (in the sense of Carson, 1955). During the 1920's a fire destroyed much of the forest land, and most of the collecting sites were in or very near areas that had not yet restocked. Selection pressure at these sites was evidently quite severe. The collecting sites have been described elsewhere (Stevenson, 1952).

Large collections could not be made because of the inaccessibility of some of the stations, and, later, several of the stations were disturbed by logging operations.

- References: Carson, H. L. (1955) The genetic characteristics of marginal populations of *Drosophila*. Cold Spr. Harb. Symp. Quant. Biol. 20:276-287.
 Stalker, H. D., and Carson, H. L. (1948) An altitudinal transect of *Drosophila robusta* Sturtevant. *Evol.* 2:295-305.
 Stevenson, R. (1952) Altitudinal distribution of species of the genus *Drosophila* (Diptera) on Unaka Mountain, Tennessee-North Carolina. *J. Tenn. Acad. Sci.* 27:97-103.

Table to show the frequencies of inversions in *D. robusta* at different altitudes on Unaka Mountain, Tenn.

Altitude	n	XL	XL-1	XL-2	XL-3	XR	XR-1	XR-2	
2200 ft.	36	.92	.052		.026	.89	.056	.056	
2500	48	1.00				.96	.042		
3000	44	.98	.021			1.00			
3500	9	1.00				1.00			
3760	2	1.00				1.00			
4080	63	.92	.091			1.00			
4200	24	.83	.007			1.00			
4400	26	1.00				.81	.038	.154	
4800	10	1.00				1.00			
		2L	2L-1	2L-2	2L-3	2R	2R-1	3R	3R-1
2200	36	.81	.083	.083	.028	.97	.028	.86	.139
2500	48	.79	.145	.016	.049	.98	.016	.90	.105
3000	44	.95	.023		.023	.91	.091	.98	.023
3500	9	.67			.333	1.00		.89	.112
3760	2	1.00				1.00		1.00	
4080	63	1.00				1.00		1.00	
4200	24	.92	.042	.042		1.00		.80	.200
4400	26	.88			.116	1.00		.73	.270
4800	10	1.00				1.00		.80	.200

Stone, L. E. Structure and variation of the salivary gland chromosomes in *Drosophila affinis*.

Standard salivary gland chromosome maps have been prepared for *D. affinis* using a homozygous strain from central Nebraska. The chromosomes of this species

have seven large euchromatic arms and a dot-like element. These represent a V-shaped X-chromosome (LX and SX) and four pairs of autosomes, which includes a J (3L and 3S), a near-V (2L and 2S), a rod (4) and a dot-like element (5).

Chromosomal variation is being investigated by mating males from various laboratory stocks to females carrying the Standard sequence. A number of slides have been prepared with lactic-acetic orcein, but the best success has been obtained with a modification of Cohen's Sudan Black B reagent (Cohen, 1949, Stain Technol., 24:177-184). Preliminary data on chromosomal variation has been gathered from a number of strains representing 35 localities in 18 states (Table 1).

Table 1

State	Number of Strains	Sequences besides Standard*
Alabama	3	SX/SX-1
Florida	4	LX/LX-1; 4/4-3
Georgia	2	SX/SX-1
Iowa	2	4/4-2
Kentucky	1	?*
Louisiana	10	LX/LX-1; SX/SX-1
Massachusetts	1	?*
Minnesota	3	LX/LX-1; SX/SX-1
Mississippi	2	LX/LX-1; 4/4-2
Missouri	2	LX/LX-1; SX/SX-1
Nebraska	53	LX/LX-1; LX/LX-2; LX/LX-4; 4/4-2
New York	1	SX/SX-1
North Carolina	1	LX/LX-3; 4/4-2
Ohio	1	4/4-2
Oklahoma	1	LX/LX-1; SX/SX-1
South Carolina	2	LX/LX-1; SX/SX-1
Tennessee	2	?*
Texas	2	LX/LX-1

*In addition to the non-Standard sequences listed in Table 1 a complex configuration has been found in the distal part of chromosome four. It has been found in at least one strain from each of the states listed in the table. Since the pairing in this configuration is so variable, it has not yet been determined how many different inversions may be involved or what differences there may be between strains.

Excluding the dot-like element there are four rather long arms that have been found to contain only the Standard sequence in all of the strains tested to date.

Strangio, V. A. Recessive lethals, sex chromosome loss, and nondisjunction followed simultaneously.

sex-chromosome loss and nondisjunction of X and Y chromosomes. This has been achieved by the use of a modified Muller-5 (Basc) stock in which the Bar marker had reverted to wild type. The daily brood technique involved the mating of fresh virgin females of this stock to irradiated males (800r) carrying a normal X and the doubly-marked $y^+ Y B^S$. In the F₁, sex chromosome loss and marker deletion from the Y were scored as exceptional non-Bar males, primary nondisjunction of X and Y as Bar females. Impregnated F₁ females were placed individually in minimilks to provide an F₂ from which the sex-linked lethal frequency was obtained. If the first

The incidence of sex-linked recessive lethals induced at various stages in the spermatogenesis of D. melanogaster has been investigated simultaneously with the induction of

appearance of induced nondisjunction is accepted as a valid reflection of the irradiation of early meiotic stages, then the condensed results presented in the following table indicate two radio-sensitive peaks during spermatogenesis, one in spermatids (5th day) for recessive lethals; the other in spermatocytes (7th day) for breakage-loss aberrations. Detailed discussion will be published elsewhere.

Aberration	Daily Brood				
	4	5	6	7	8
% lethals	4.67	5.43	3.61	2.92	3.30
% non Bar ♂♂	0.23	0.23	0.84	1.70	1.47
% Bar ♀♀	0.00	0.00	0.00	0.43	0.17

Strangio, V. A. Pseudo-allelism at spineless-aristapedia locus.

The investigation of possible pseudo-allelism at the spineless-aristapedia locus is in progress by means of a crossover selector

technique. The work is at present being hampered by the low fertility of the selector cross males.

Takaya, H., and S. Kaji. On the inheritance of the erosion eye in D. melanogaster.

Previously the authors (1959, 1960) reported that in several strains of D. melanogaster spontaneously there occurs definite deficiency of the compound eye. The occurrence is quite

seldom on standard medium, but may be considerably increased on experimental media containing soybean powder or monosodium glutamate. Further it has been shown that these deficient eyes, once appeared, are inherited if inbred by means of sib-mating, while crossing of them by the wild type generally fails to be reproduced. Especially it was striking that the eye-deficiency is inherited not only by the same degree but also by higher ones. Therefore, if inbreedings were properly continued, gradual diminution and, in extreme cases, complete disappearance of the compound eye eventually results. On this account, these are designated as 'erosion eye.' Since these facts suggest that the erosion eye is a hereditary character which represents peculiar mode of inheritance, further evidences were collected in the following experiments.

In an attempt to prepare a pure strain of fly which produces no erosion progeny, a selection experiment was carried out with wild type flies of Oregon. In the pair matings carried out randomly with flies of this stock, frequency of the erosion eye was about 0.3%. Later it was recognized that among flies of this stock there are ones the compound eyes of which represent slight irregularities in outline. Mating of these was liable to produce erosion offspring more often. In the present selection, therefore, special care was taken not to choose these aberrant flies. Even in this way erosion offsprings were found to be produced in the first 3 generations of selection. But in later generations erosion flies were ceased to be produced, and in none of the offsprings successively examined during 50 generations the eye deficiency was met with (Table 1). However, when offspring flies produced in every generation of this selection were reared on the medium containing 10% of soybean powder, occurrence of the erosion progeny was not only increased in rate but also continued in still later generations. In this medium the rates obtained were 2 or 3 times higher than those in the standard medium, and the occurrence was continued till the F₆ generation. Among the offsprings produced in later generations than this, there was none which presented erosion eyes. Therefore, it may be safe to assume that the offspring flies produced in relatively later generations of the selection can be regarded as a pure strain which are free from the erosion-eye producing factors.

In a second experiment crossing was tested between erosion flies and wild types. The erosion flies were chosen from the strains kept in our laboratory and the grade of erosion was very low, the facets of the compound eye counting about 600. The wild types were of 2 sources: one from unselected strain and the other from offsprings of the F₅₀ generation of the pure strain. When the crossing was done by using wild types of the unselected strain, erosion offsprings were produced, being found in 4 out of 13 matings examined. Rates of the occurrence considerably varied in individual matings, but the rate averaged throughout the cases was 0.44%. The same crossing tested on the soybean medium produced erosion progeny far more frequently than on the standard one. The average rate amounted 1.42% (Table 2A). When wild type flies of the pure strain were used in the crossing, all the offspring flies produced failed to represent the eye deficiency being normal so far as they were reared on the standard medium. But the same crossing tested on the soybean medium brought about erosion flies invariably in all the matings examined. Rates of the occurrence were nearly equal in individual cases, and, moreover, they were very high, the average being 2.29% (Table 2B).

From the results above mentioned it can be surmised that spontaneous occurrence of the erosion eye is due to certain hereditary factors which are widely distributed among population of Oregon stock. By means of the inbreeding properly carried out for several successive generations, these factors can be swept off so that the flies are quite unable to produce erosion progeny. These flies again acquire the ability to produce erosion offsprings if they were crossed with the flies possessed with the factors. Further it was striking that the phenotypic representation of the erosion eye is shown to be influenced by the medium on which flies were reared.

Table 1

Frequencies of erosion flies produced in selection experiment carried out by means of pair-matings of wild type flies

Generation	Number of total flies	Number of erosion flies	Per cent of erosion flies
F ₁	634 (508)	2 (11)	0.29 (2.17)
F ₂	410 (573)	0 (7)	- (1.22)
F ₃	402 (425)	1 (2)	0.25 (0.47)
F ₄	480 (492)	0 (3)	- (0.61)
F ₅	573 (260)	0 (1)	- (0.40)
F ₆	649 (487)	0 (1)	- (0.25)
F ₇	214 (522)	0 (0)	- (-)
F ₈	526 (613)	0 (0)	- (-)
F ₉	139 (439)	0 (0)	- (-)
F ₁₀	225 (341)	0 (0)	- (-)
F ₅₀	607 (735)	0 (0)	- (-)

Numerals without parentheses represent results on standard medium and those within parentheses results on soybean medium.

Table 2
Results of crossing erosion flies by wild types
of different strains

Standard medium			Soybean medium (10%)		
Total flies	Erosion flies	%	Total flies	Erosion flies	%
A. Wild type flies of unselected strain					
1597	7	0.44	1412	20	1.42
B. Wild type flies of selected strain					
1474	0	-	1221	28	2.29

Thompson, Peter E. The basis for high "nondisjunction" from maroon-like females.

Females homozygous for the mutant maroon-like (ma-1) produce unusually high frequencies of apparent primary nondisjunction. The exceptional offspring which result are

predominantly male; the frequency of exceptional females is not appreciably greater than the normal rate (see Spieler, Rec. Gen. Soc. Am., 1961). In this study crosses of y^2 ma-1/ y^2 ma-1; T(3;4)89E/+, y^2 ma-1/ y^2 ; T(3;4)89E/+, and y^2 ma-1/ y^2 ma-1 females to w^a males were made to test whether the X's tend to interact with unpaired autosomal elements when the translocation is present. The disruption of homologies by heterozygous T(3;4)89E results in appreciable frequencies of haplo-4 and triplo-4 offspring. If failure of pairing is the basis of nondisjunction in ma-1 lines, the availability of nonhomologous elements for pairing should enhance the effect (Grell and Grell, 1960). The progeny of the above crosses were:

Series A: y^2 ma-1 / y^2 ma-1; T(3;4)89E/+ \times $w^a\sigma\sigma$

+ ♀♀	y^2 ma-1 $\sigma\sigma$	y^2 ma-1 ♀♀	$w^a\sigma\sigma$
diplo-4 haplo-4	diplo-4 haplo-4	diplo-4 haplo-4	diplo-4 haplo-4
2058 91	1691 62	2 0	24 0
excep. ♀♀ = .09%	excep. $\sigma\sigma$ = 1.35%	haplo-IV = 3.9%	

Series B: y^2 ma-1/ y^2 ; T(3;4)89E/+ \times $w^a\sigma\sigma$

+ ♀♀	$y^2\sigma\sigma$	$y^2\text{♀♀}$	$w^a\sigma\sigma$
diplo-4 haplo-4	diplo-4 haplo-4	diplo-4 haplo-4	diplo-4 haplo-4
2208 95	1966 79	5 5	13 1
excep. ♀♀ = .43%	excep. $\sigma\sigma$ = .68%	haplo-IV = 4.1%	

Series C. y^2 ma-1/ y^2 ma-1 ♀♀ \times $w^a\sigma\sigma$

+ ♀♀	y^2 ma-1 $\sigma\sigma$	y^2 ma-1 ♀♀	$w^a\sigma\sigma$
diplo-4 haplo-4	diplo-4 haplo-4	diplo-4 haplo-4	diplo-4 haplo-4
2501 0	2082 0	3 0	31 0
excep. ♀♀ = .12%	excep. $\sigma\sigma$ = 1.4%	haplo-IV = 0.0%	

The presence of homozygous *ma-1* and the translocation in Series A did not result in any appreciable increase in X-chromosome exceptions over crosses lacking the translocation (Series C), or in chromosome-4 nondisjunction over crosses where *ma-1* was not homozygous (Series B). Furthermore, no coincidence of X and 4 nondisjunction was observed in Series A; this absence of coincidence even falls below an expectation based on the presence of the translocation alone (see Series B).

It appears that the basis for the occurrence of exceptional types from maroon-like females is not a looseness or failure of pairing. The great predominance of exceptional males over exceptional females suggests chromosome loss or elimination as the underlying mechanism.

Tsukamoto, M. Comparative studies on the oxidation of DDT in *D. melanogaster*.

Relationship between the chemical structure of DDT-like compounds and their oxidative metabolism was investigated. It has been confirmed that DDT and some alkane-type

analogues of DDT, which have a replaceable hydrogen atom in the alkyl moiety between two *p*-chlorophenyl groups, have been metabolized to their corresponding alcohols. On the other hand, *para*-substituted analogues of DDT were rapidly metabolized but the corresponding alcoholic metabolites could not be detected among the recovered fractions.

Toyofuku, Y. Non-random association of inversions in *D. nigromaculata*.

In DIS 34, the author reported that in *D. nigromaculata* in natural population there occur twenty-two different kinds

of chromosomal aberrations, all being represented by heterozygous inversions. Cytological analyses of these chromosomal aberrations have been carried out in more detail. This species showed a variety of inversions in each arm of two or more chromosomes. Frequency distributions of inversions in each chromosome were as follows: C-chromosome showed 34.65% in frequency, D-chromosome ranked next, showing 25.74%, A-chromosome occurred at 24.76% of a total inversion, B-chromosome showed 8.91%, and the X-chromosome was found at the lowest frequency of 5.94%. The results are referred to in table. It is interesting to note that in C-chromosome, for example, the inversion d includes sections 65C - 67B often appeared in combination with the inversion b and inversion e involves sections 67C - 72A, containing new inversion c. This suggests that the distribution of inversions is not random.

Chromosome	Inversion type	Part of inversion	Inversion observed	Frequencies (%)	
				1	2
X	a	3B - 9A	1	17	5.94
	b	5A - 9C	4	67	
	c	5A - 9C, 12A - 15B	1	17	
A	a	26A - 33A	5	20	24.76
	b	26A - 36A	1	4	
	c	29A - 32B	10	40	
	d	32B - 34A	1	4	
	e	28B - 30A, 32B - 37C	4	16	
	f	32B - 34A, 35A - 39A	2	8	
	g	(23D - 27A, 28B - 30A, 32B - 37C	2	8	
B	a	42A - 51D	3	33	8.91
	b	49A - 53A	2	22	
	c	52A - 55B	1	11	
	d	48C - 50D, 52A - 55B	3	33	

(Toyofuku, table--continued)

Chromosome	Inversion type	Part of inversion	Inversion observed	Frequencies (%)	
				1	2
C	a	62B - 64A	6	17	34.65
	b	72B - 77A	3	9	
	c	77A - 79D	7	20	
	d	65C - 67B, 72B - 77A	13	37	
	e	67C - 72A, 77A - 79D	6	17	
D	a	88A - 93B	16	62	25.74
	b	97A - 98B	9	35	
	c	90B - 94B, 96A - 97B	1	4	

Valencia, Ruby M., and J. I. Valencia.
Evidence for a non-chromosomal origin
of dominant lethals.

union of pronuclei) turned out to be very close to those obtained by Parker (1959, The University of Texas Publication, No. 5914:113) in stage 14 oöcytes. Lindsley (personal communication) observed that since adding the male genome has little effect, the majority of dominant lethals induced in these stages apparently are not of chromosomal origin. We considered that this idea was worth a careful test and therefore set up a series of irradiations to test dominant lethal frequency in stage 14 oöcytes, using a dose of 500r and exactly the same stocks and crossing schemes that we were using for the fertilized eggs. We also tested mature sperm (ejaculated within 8-10 hours after treatment), using these same stocks and schemes, and have accumulated some data from treated embryos (35-45 minutes after fertilization). The data are as follows:

The frequencies which we obtained for dominant lethals induced by X-rays in recently fertilized eggs (5-15 minutes after fertilization, supposedly before

	Stage 14 oöcytes		Fertilized eggs		Early embryos		Mature sperm	
	No. eggs laid	% dom. lethals*	No. eggs laid	% dom. lethals*	No. eggs laid	% dom. lethals*	No. eggs laid	% dom. lethals*
Treated	6864	67.5	6972	64.7	2128	69.8	6475	18.2
Control	6190	7.8	1519	8.0	1519	8.0	5101	8.4
Corrected		59.7		56.7		61.8		9.8

*i.e. % unhatched eggs. Corrected value represents death in embryonic stage.

The values we have obtained for stage 14 oöcytes are in quite good agreement with the values obtained by Parker (above reference) and the frequency obtained for recently fertilized eggs agrees well with the results of Ulrich (1960, Revue Suisse de Zoologie, 67:287). It seems clear that oöcytes, with one nucleus, recently fertilized eggs, with two nuclei, and embryos, with several nuclei, all have about

the same mortality (around 60%) after 500r. The mortality induced by irradiating sperm is not detectably added to the mortality induced by irradiating oocytes to give a higher mortality after irradiating fertilized eggs. The conclusion that dominant lethals in oocytes and eggs are mostly due to non-chromosomal damage would appear to be in disagreement with the results of Ulrich (DIS 28) who found a drastic difference in LD50 after irradiating the anterior half (containing the nuclei) and the posterior half of the egg, and concluded that the lethality was almost entirely due to nuclear damage. An explanation which would fit both sets of results is that the damage is largely due to injury to some cytoplasmic constituent or constituents, but that these substances are concentrated in the anterior region of the egg during the perfertilization period.

Valencia, Ruby M. Sex ratio after irradiating fertilized eggs.

Fertilized eggs were irradiated with 500r of X-rays (150 kV, 10 mA, 1 mm. Al filter) within 15 minutes after fertilization,

presumably well before union of the pronuclei, for the purpose of observing dominant and recessive lethal damage. All adults hatching from the irradiated eggs were counted in order to calculate postembryonic mortality. We took advantage of this situation and counted the various classes of progeny separately, in order to determine whether or not there was an effect on the sex ratio. The crosses from which the eggs were derived were $y\ sc^{S1}\ B\ In49\ v/y\ oc\ ptg$ females mated with $y\ oc\ ptg$ males or with $y\ sc^{S1}\ B\ In49\ v$ males. All males hatching from these crosses obviously carry an X chromosome irradiated in the maternal nucleus and a Y chromosome irradiated in the paternal nucleus. The females would carry one X irradiated in the maternal nucleus and one X irradiated in the paternal nucleus. Recessive lethals induced in the maternal X would be expected to result in a lowering of the frequency of males hatching. The results were as follows:

	Type male used	Females			Males			
		$y\ B$	$y\ oc\ ptg$	$y\ B\ v$	Total	$y\ oc\ ptg$	$y\ B\ v$	Total
Irradiated	$y\ oc\ ptg$	872	822	---	1694	803	768	1571
	$y\ sc^{S1}\ B\ In49\ v$	1567	---	1200	2767	1464	1231	2695
	Total	2439	822	1200	4461	2267	1999	4266
Control	$y\ oc\ ptg$	1254	1120	---	2374	1144	1112	2256
	$y\ sc^{S1}\ B\ In49\ v$	1178	---	859	2037	1158	1008	2166
	Total	2432	1120	859	4411	2302	2120	4422

A recessive lethal test carried out with the $y\ B$ females hatching from the irradiated eggs showed that 4.6% of them carried a lethal in the X chromosome which was irradiated in the maternal nucleus. If we assume that an equal number of X chromosomes entered into male zygotes as entered into female zygotes, and that these X chromosomes carried an equal number of recessive lethals, we would expect 4.6% of them to have been eliminated. The number of males expected to have hatched would be 4256, very close to the actual number counted (4266). The observed number represents a reduction of 4.4% from the supposed original number of male zygotes (4461). The controls show no reduction of males, but rather a slight advantage over the females.

It is possible, however, that this apparently good agreement with expectation is spurious. It can be seen that the three classes of females and two classes of males have very different viabilities. It would be preferable to have results similar to these for irradiated isogenic wild type eggs.

Wedvik, Hans. The effect of low temperature on fertility of Drosophila melanogaster males.

The experiment is divided into four groups, each containing at least 60 males. The males were taken from a wild type Canton-S stock, which were stored without females

until they were three days old. The males were then given a temperature treatment for half an hour. Immediately after the treatment, each male was mated for a period of 24 hours to five virgin three day old females hybrid for the gene markers, *cn bw; e*".

At the end of the mating period, the females were transferred to black food for egg laying. The males were given a new set of five virgin hybrid females for another 24 hour mating period. This continued over a period of 14 days. In the control group, Group I, the males were stored at a temperature of 22° C. In the experimental groups, Groups II, III and IV, the males were treated for half an hour at a temperature of respectively 7° C., 4° C. and 0° C.

The data given in the table picture the frequency of fertile males on the different days after treatment. As it can be seen from the table, there is no apparent difference between the males treated at 22° C. and 7° C. However, for the two other groups of males there is a marked reduction in the frequency of fertile males starting already on the second day after treatment. Thus, the data indicate that temperatures below 7° C. induce male sterility.

Percentage fertile males

Groups	Temp.	1	2	3	4	5	6	7	8	9	10	11	12	13	14 days
I	22° C.	100	100	100	98	98	96	93	86	86	71	64	53	46	40
II	7° C.	100	100	100	95	95	95	78	70	70	56	56	56	50	45
III	4° C.	100	73	51	39	31	26	19	19	12	10	5	4	1	1
IV	0° C.	100	50	23	12	7	5	5	2	2	1	1	1	0	1

Zürcher, C. Balanced polymorphism and heterosis in crosses of wild type and the mutant *e^{ug}* of D. melanogaster.

Mass-breeding populations of the constitution *e^{ug}/ +* were established at 20°, 25° and 28° C. and the frequencies of the *e^{ug}* allele were determined at intervals of 2 - 4 weeks. The experiments at 25° and 28° C.

were run for 8 months, and those at 20° C. for 4 months. After the first 2 months a constant value of 35 - 40% was obtained for the frequency of the mutant allele. The temperature seems to have no influence on the process of selection under the conditions used in the laboratory. In separate breeding experiments using *e^{ug}/e^{ug}*, *e^{ug}/ +* and *+/+*, it was established that the heterozygotes show a heterotic effect in reference to longevity and resistance to desiccation. Further analyses of viability factors are in progress.

Bender, H. A. Dioxane dehydration of *Drosophila* tissue.

very successful in reducing shrinkage, and in paraffin embedded material, of decreasing splitting during sectioning. Dioxane is compatible with most fixing agents (including osmic fixation), the majority of stains and the Feulgen technique. Tissues should be fixed and washed as usual, placed in dioxane (with two changes) for at least six hours (usually overnight) and subsequently handled as if in absolute alcohol. (Details of the dioxane method may be found in a critique by Mossman, Stain Technol. 12, 4, October, 1937.)

The substitution of dioxane, $O(CH_2CH_2)_2O$, for ethanol in the dehydration of the delicate tissues of *Drosophila* has proved

David, J. A new medium for rearing *Drosophila* in axenic conditions.

characters. Different micro-organisms may develop on this medium and, if so, are always the source of important and uncontrolled variations. In contrast, an aseptic, chemically defined medium is very difficult to use when large number of flies are to be studied. Therefore, another medium, more convenient for quantitative research, has been worked out. This new medium is easy to prepare because not chemically defined and easy to use because aseptic conditions are assured in a simple manner by an antiseptic. The composition is the following:

The live yeast medium used by many authors in *Drosophila* studies is often unsuitable for the exact determination of quantitative

agar	15 g
dried brewer's yeast	96 g (dry weight)
corn meal	96 g (dry weight)
antiseptic (nipagine or tegosept in alcoholic solution)	6 g
water:	to a total weight of 1200 g

Agar is dissolved in boiling water. The other ingredients are then added, and the medium is autoclaved for 15 minutes at $115^{\circ}C$. When autoclaved, the medium is supplemented with water up to a total weight of 1200 g, then mixed and poured in culture tubes.

With this diet, larval development and adult fecundity are as good as those observed with the live yeast medium, and accidental variability is much lower. The new medium has been used for seven years in our laboratory and has proved very satisfactory for genetic and physiological studies in *Drosophila*. More detailed information is available in the following publications:

David, J. (1959) Etude quantitative du développement de la *Drosophile* élevée en milieu axénique. Bull. Biol. Fr. Belg. 93:472-505.

David, J. (1961) Influence de l'état physiologique des parents sur les caractères des descendants. Etude chez *Drosophila melanogaster* Meig. Ann. de Génétique (in press).

Hildreth, P. E., and Cole Brunt. Method for collecting large numbers of fertilized *D. melanogaster* eggs in meiotic stages.

The method to be described permits the transfer of females from one egg collection dish to another with no loss of females and with a minimum of agitation to the females, thus disturbing the egg-laying pattern very

little. Although we have used the method for collecting large numbers of eggs it would also be satisfactory for collections from single females.

Two days before the eggs are needed, virgin females and males are collected and stored separately with approximately 40 flies in each 1/2-pint culture bottle which contains yeasted standard cornmeal-agar-molasses medium. Also on this day, 600 cc of the same type of culture medium should be mixed with 10 g of live Baker's yeast and allowed to ferment for two days. In the morning, two days after having been collected, males from each bottle are shaken (without etherization) into the bottles with the females, giving about 40 pairs per bottle. After about three hours the flies are shaken, without etherization, into tubes used as egg-laying chambers (one bottle of flies per tube) and the tubes are placed immediately on blotting paper for collection of eggs.

The tubes are clear plastic, about 45 mm long and 22 mm in diameter. One end of the tube is covered with a single layer of dacron gauze (through which the females oviposit) and the other end is plugged with cotton after the flies have been shaken in. The cotton should be pushed down to within about 1/2 cm from the dacron gauze to keep the flies near the food. Dacron gauze is used because the fibers do not absorb moisture and do not shrink or expand with moisture changes. The gauze may be held on the tube with rubber bands or the gauze may be glued to or embedded in the plastic with the proper solvent. We have found the latter method to be most satisfactory.

The collection dish consists of a Petri dish lid or base which has been filled with the previously mentioned fermented culture medium. On the surface of this food is placed a Kimwipe or Kleenex-type tissue on which the blotting paper will be placed. Dark green blotting paper cut into rectangles about three inches by four inches is found to be satisfactory for the egg collection as the eggs are readily visible against the dark background. The blotting paper is first soaked in a vinegar solution (nine parts water to one part commercial white vinegar) before it is placed on the collection dish. Both the fermented food and the vinegar solution are necessary to stimulate rapid egg laying. At the end of the egg-laying interval the tubes may be gently lifted, the blotting paper with the eggs removed, a new piece of blotting paper placed on the fermented food, and the tubes placed on the fresh paper for another collection. The eggs may be treated while on the paper, or removed easily with a needle or small brush, or the paper and eggs may be inserted into a bottle containing culture medium and permitted to develop.

During collecting intervals of five minutes we have occasionally been able to collect over 200 eggs (using nine tubes), and average about 100 eggs. In our experiments we normally make 30 to 40 such collections in an afternoon. In a sample of 190 eggs collected in this manner and then prepared with Feulgen's stain it was observed that slightly more than 75% of the eggs were in meiotic stages. Fixation in some cases did not occur until about 20 minutes after the eggs were laid, so the percentage of eggs in meiotic stages at the end of the five-minute collection period would be higher than is indicated. If small quantities of very young eggs are desired it would be best to use fewer flies in each tube and then to select those tubes in which the eggs are being laid rapidly.

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

Kirschbaum, W. F., and Ruby M. Valencia.
Modified egg-collecting technique.

We are using a modification of the egg-collecting technique published by Ulrich (DIS 27). Since for our purpose

we need not manipulate the eggs, we considered it worth while to avoid any manipulation, thus saving time and perhaps avoiding possible damage to the eggs. When we wish to collect eggs from the flies in the bell jar apparatus, we substitute for the food plate a plate containing a block of wood (to fill the space usually occupied by medium) on which is placed a round piece of thin white blotting paper (diameter 5.6 cm.), wetted in vinegar and spread with a thin film of yeast suspended in vinegar. The paper is previously scored with pencil in rectangles (7.5 x 4.5 mm.) calculated to fit the field of the dissecting microscope. After placing the paper

on the wood block, it is scraped sharply between the pencil lines, in the longitudinal direction of the rectangles. The flies like the rough surface to lay their eggs and lay most of them there, thus facilitating the counting later. The papers are simply removed from the plates, irradiated and placed in regular bottles containing medium. The larvae hatching from the eggs very soon crawl down into the medium, since they lack sufficient food on the paper. The papers are removed from the bottles at 48 hours and the eggs counted. (If time does not permit counting at once, the papers may be conveniently stored in petri plates in the refrigerator for several days.) For counting, the papers are moistened from the lower surface, placed on a clear glass plate and examined with a stereo-microscope, using transmitted light and a magnification of 25X. Hatched and non-hatched eggs are easily distinguished.

Levitan, M. Long-distance
Drosophila collecting.

In collecting particular species of Drosophila at some distance from home territory, it is imperative to obtain the largest possible

sample in the shortest possible time. The problem is particularly acute when seeking a species such as D. robusta which is rarely the majority among the flies coming to banana traps. Over the past several years I have found that the following technique modifications usually increase the efficiency and ease of long-distance collections:

Bait: About 5-7 days before they are to be used, very rotten bananas, the kind grocery-men want to throw away, are mashed, the cut-up peels added, and placed in a bucket which has ice-pick size holes punched in the bottom. At a woods the first bucket is hung alone from a branch and the contents allowed to ferment from (or absorb) wild yeasts and bacteria, water of fermentation dripping out. When traveling this bucket is set into another one so that the fermentation water leaks into the one with good bottom. D. robusta and certain other species appear to be selectively attracted to bait that is fermented in this way and is not too wet; even quite dry bait will attract them better than most other species.

Traps: Since D. robusta is most efficiently collected from traps that are left in the woods several days, I use quart Mason jars tied to the trees with wire. Stove-pipe (for example Sears Roebuck catalog #9H9903, 10 cents for a 50 ft.), galvanized, or similar cheap pliable wire is good for this purpose. I attach the wire permanently, tightening it around the neck of the jar with pliers. Rain is kept out by making the wire long enough so that it can be knotted several times a few inches beyond the jar and then passed through the mid-point of a paper plate, the plate resting on the knotted portion. Plasticized paper plates are excellent for this, especially the ones that present a blue or brown surface to the open mouth of the jar. For transport the paper plate can be pulled off, the wire wrapped around the jar, and the jars with the bait in them covered by regular jar caps. Thus they are ready to hang quickly in the next locality.

Mahowald, A. P. Fixation problems
for electron microscopy of
Drosophila embryos.

An electron microscopic study of early embryogenesis in Drosophila is nearly completed and it seems appropriate at this time to pass on information on the fixation

techniques used. The vitelline membrane is impermeable to the usual fixatives used. After trying various chemical and enzymatic methods for attacking the membrane, it was found that very brief treatment with ether (1-2 seconds) or slightly longer treatments with other organic solvents with lower water affinities, e.g. toluene or isopentane, rendered the membrane readily permeable to either OsO_4 or KMnO_4 . The ether-extracted compound is probably a wax since this substance is frequently the water-proofing substance in insects. In electron micrographs a layer about 500A in width was found between the vitelline membrane and the chorion; it is probably this layer that was removed by the solvents. Examination with the electron microscope

showed that embryos treated with ether for as little as one second were seriously injured. Consequently further efforts along this line were stopped and the usual micro-puncture with a fine tungsten needle was used.

Concerning the puncture, a compromise must be achieved between one large enough to allow the fixative to penetrate sufficiently fast and one so large that distortion of the embryo results. This latter difficulty is especially common at the early stages studied, i.e. the blastoderm formation and pre-blastoderm stages. In our studies the chorion was routinely removed with NaOCl so that the specific stage desired could be picked out. The removal of the chorion has the added advantage that the puncture can be made more delicately. However, the chorion is no hindrance to the penetration of the fixative, so this is not a necessary step. As soon as the initial fixation has occurred around the puncture wound, the hole should be enlarged. Experience has shown that both OsO_4 and KMnO_4 render the vitelline membrane brittle; consequently care must be taken that excessive pressure is not used which would result in splitting of the membrane. This fact, however, becomes useful for the next procedure. After most of the embryo has become colored with the fixative, it is helpful if the vitelline membrane is dissected off (this is not possible after permanganate fixation for reasons not known). This last difficult step is not necessary for good fixation if the initial penetration of the fixative was rapid enough, but it has other advantages. Bahr et al. (Exp. Cell Research 12:342-355, 1957) have shown that tissues undergo a 15-20% expansion during short fixations and that during subsequent dehydration with alcohol there is an equivalent shrinkage. Because of the vitelline membrane, however, the expansion in the fixative does not occur. Consequently, after the contraction in the alcoholic series, the embryo is 20% smaller than originally. This shrinkage results in increased cytoplasmic density, thus necessitating very thin sections in order to discern the fine structure. If the vitelline membrane is removed within the first 30 minutes of fixation, an expansion of about 15% still occurs. As a result the fine structure is more easily observed and it can be more readily compared to other tissues.

A second advantage is that infiltration with plastics is more uniform. If the vitelline membrane is not removed, the embryo should be cut in two in 95% alcohol in order to facilitate infiltration. This is imperative with the epoxy resins and is necessary for consistent results with methacrylate.

Because of the dangerous fumes of OsO_4 it must be noted that good ventilation is required during these procedures. During the actual operations, especially if groups of embryos are being worked on, a small fan has been successfully used to prevent the accumulation of fumes at the dissecting scope.

No other modifications of standard preparatory procedures were found necessary.

Mittler, S., and J. Bennett.

A simple food medium that requires no live yeast with the minimum of variables.

to add or maintain live yeast whose growth forms a moist sticky layer over the surface of the food which can trap flies. The highly variable molasses and corn meal of the "standard media" has been eliminated. The formation of harmful excess carbon dioxide by the live yeast and movement of media by the gas has been eliminated.

The medium to be described had been developed by Dr. J. Crow at the University of Wisconsin, and has been used for several years in our laboratories with much success. There is no need

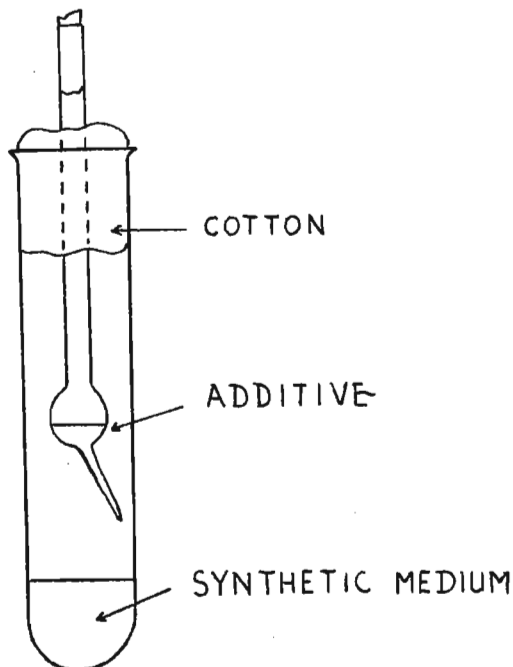
Food Formula

<u>Ingredients</u>	<u>Amounts</u>
Water	1000 ml
Agar	19 gm
Sucrose	54 gm
Dried yeast	32 gm
Propionic acid	5 ml

The agar, sucrose, and dried yeast are added to the warm water and mixed while heated until thoroughly dispersed. The mixture is boiled for 5 to 15 minutes to kill all the yeast (it may be necessary to add 200 ml of additional water at the start to allow for the amount boiled away). The propionic acid is added after the mixture has cooled to 60° C. We use Schlitz Brewing Co.'s "non-debittered dried brewers yeast," obtainable in 100 lb. bags. The large quantity assures long term control of quality of this ingredient.

Sang, James H. A simple method of adding solutions to axenic cultures.

phila larvae at particular ages is simply solved by the device illustrated. Bulb tubes are made by first drawing out cleaned tubing and then blowing a bulb so that



The problem of adding measured amounts of nutrients (or of mutagens or other substances) to germ-free cultures of *Drosophila* larvae at particular ages is simply solved by the device illustrated. Bulb tubes are made by first drawing out cleaned tubing and then blowing a bulb so that a sufficient length of the drawn tube remains to permit its easy fracture by gentle pressure against the side of the culture tube. The bulb is filled by graduated syringe, and stoppered with cotton-wool. Prepared cultures can then be autoclaved and handled in the normal way (Sang, 1956, *J. Exper. Biol.*, 33:45). Alternatively sterile solutions may be introduced into the bulb after autoclaving when this would damage the required additive. The nutrient is added to the medium at the desired time, by breaking the drawn tube against the side of the culture.

TEACHING NOTE

Moree, Ray. Simple demonstration of modified ratios using b and e.

of b and e. That the F_1 is wild type is surprising to many. The F_2 is classified by most students into wild type and "dark," in 9:7 ratio if the sample size is large enough. But some students detect what they consider as different degrees of darkness, so the possibilities of getting a 9:3:4 or a 9:6:1 ratio are pointed out: it can also be indicated that if a simple chemical test were available it might even be possible to recognize a 9:3:3:1 ratio. That the ratio of wild to dark may be about 1:1 in F_2 progeny and 1:3 in testcross progeny from the cross $F_1 \times b/b; e/e$, is usually somewhat surprising, too. The results emphasize the way in which inferences as to interaction, epistasis, etc., depend upon the possibilities of discriminating among the individual progeny of a cross.

Laboratory experiments relating to modified ratios and genic interaction can be made both simple and surprising by using stocks

J. A. Beardmore has moved from the Genetics Department, University of Sheffield, to the Genetical Institute, State University of Groningen, Netherlands.

Professor Hampton L. Carson has recently returned to Washington University, St. Louis, after a period of nine months in Australia as a Senior Fulbright Research Scholar. Most of this time was spent in the Zoology Department at the University of Melbourne; from here, Professor Carson visited other parts of Australia, also New Guinea.

James Divelbiss has moved from the Department of Zoology at the University of Iowa to the Department of Biology, Westmar College, in Le Mars, Iowa, where he is an assistant professor.

Marvin Druger, formerly of the Genetics Group of Columbia University, New York, is visiting the Animal Genetics Laboratory, Sydney University, for one year from mid-August, 1961. Dr. Druger holds a post-doctoral fellowship from the National Institutes of Health and is working on problems of canalization.

Dr. A. C. Fabergé of the Department of Zoology, University of Texas, is visiting the Genetics Laboratory, Biology Department, University of Oregon, until September, 1962.

Lawrence D. Friedman, formerly at the Department of Medical Genetics at the University of Wisconsin, is now Assistant Professor, Department of Biology, Hiram College, Hiram, Ohio.

George D. Hanks has joined the staff of the Department of Genetics, University of Utah, Salt Lake City, Utah.

Jerry Hirsch has been appointed Associate Professor of Psychology in the Department of Psychology, University of Illinois, Urbana, where he will supervise a Ph.D. program in, teach courses in, and direct research on behavior genetics (the major emphasis in the laboratory remains on *Drosophila*). Reprints of, or references to, studies of heredity and behavior (in all species) will be greatly appreciated.

Benjamin Hochman wishes to express his appreciation to those who supplied him with stocks and trapped flies following the loss of his experimental lines and stocks in October, 1961.

M. E. Jacobs is now located at the Biology Department of Eastern Mennonite College in Harrisonburg, Virginia. He was formerly of Bethany College in Bethany, West Virginia.

Edward C. Keller, Jr., has recently moved to the Genetics Laboratory in the Department of Biochemistry and Nutrition, the University of North Carolina, Chapel Hill, North Carolina.

Professor R. C. Lewontin of the Biology Department, Rochester University, New York, is visiting the Animal Genetics Laboratory, Sydney University, for one year from mid-June, 1961. Professor Lewontin holds a Fulbright Scholarship and is working on problems of canalization and population genetics.

Benedetto Nicoletti is organizing a *Drosophila* Laboratory in the new Genetics Department, University of Rome, Rome, Italy. He shall be very grateful to the friends who can send him their old reprints or put his name in their mailing list.

T. M. Rizki, formerly in the Biology Department at Reed College, has joined the Department of Zoology at the University of Michigan as Associate Professor.

B. Sakaguchi has moved on the end of August from Dr. Donald F. Poulson's laboratory at Yale University in New Haven to the National Institute of Genetics, Misima, Japan. He is continuing his work on maternal inheritance of "sex-ratio" condition in *Drosophila*.

L. Sandler will, in June of 1962, move from the Genetics Department, University of Wisconsin, Madison, Wisconsin, to the Genetics Department, University of Washington, Seattle, Washington.

K. C. Sondhi, formerly at the Department of Zoology, University College, London, has been appointed Geneticist at the New England Institute for Medical Research, Ridgefield, Connecticut.

Öistein Strömnaes is on leave of absence from the Institute of Genetics, University of Oslo. He is staying as a research associate at the Department of Botany, University of Chicago, through 1962 until May, 1963.

Victor E. Tinderholt. The Department of Genetics, City of Hope Medical Center, and the Department of Zoology, U. C. L. A., report the sad news of the death of Victor E. Tinderholt. His great courage and ability to enjoy the world about him in the face of grave illness will be long remembered by those who knew this intelligent, sensitive person.

Yasuko Toyofuku (Mrs. Tonomura) was appointed a research member of the National Institute of Genetics at Misima on March 15, 1961.

Heinrich Ursprung has been appointed to the Faculty of the Department of Biology, The Johns Hopkins University, Baltimore 18, Maryland, as an Assistant Professor, effective July 1, 1962.

Dr. Marvin Wasserman, from the University of Texas, returns to the United States in 1962. Dr. Wasserman has spent two years in Melbourne as a member of the teaching staff. During this time he has pursued his studies on the replata group of the genus *Drosophila*, visiting many regions in Australia and New Guinea.

Yukio Yamada, National Institute of Genetics, Misima, Japan, has joined the Population Genetics Institute, Purdue University, as a visiting research professor. He is especially interested in genotype by environment studies with *Drosophila* and *Tribolium*.

MATERIALS REQUESTED OR AVAILABLE

J. A. Beardmore (Genetical Institute, Haren (gr), Netherlands) would like to hear from anyone having stocks of any species of *Drosophila* showing a morphological polymorphism or knowledge of the occurrence of such polymorphism in natural populations.

J. L. Blount (Department of Biology, Mt. Union College, Alliance, Ohio) would be grateful for wild-type strains of *D. melanogaster* whose adult longevity is known or suspected to be of either unusually short or long duration.

B. Burnet and J. H. Sang are studying the factors which alter penetrance and expressivity of *eyeless*. They would be grateful for a stock of *ey*^{D39k} which they have been unable to trace, or for any information about this stock which was last reported on by Hinton, 1942, Amer. Nat., 76:219-23.

F. Mainx (Institut f. Allgemeine Biologie, University of Vienna, Wien IX. Schwarzschanerstr. 17) would appreciate obtaining strains of Megaselia scalaris (=Aphiochaeta xanthina) from different places as well as strains of other species of Phoridae easily bred in the laboratory.

George A. Marzluf would appreciate receiving any stocks containing suppressors of vermilion, purple, and black. His address is: Department of Biology, The Johns Hopkins University, Baltimore, Maryland.

Max Planck-Institut für Biologie, Abteilung Beermann (Tübingen, Germany, Spemannstr. 34) would be grateful to obtain: 1) D. nigrohydei; 2) any mutation of D. hydei; 3) any Drosophila species that can be crossed with D. hydei giving either fertile or sterile hybrids.

R. D. Milkman (Department of Zoology, Syracuse University). If anyone finds it desirable to assign a small selection problem to a student, I should like very much to have any true-breeding polygenic crossveinless strains that may be obtained. It should be possible to obtain such a strain by selection of the progeny of even a few wild flies. This has proven easy in the past.

Dr. Yasuhiro Miyoshi would like to have wild strains from various localities in the United States for studies on tolerance of certain salt concentrations. His address is: Department of Zoology, Faculty of Science, Kyoto University, Kyoto, Japan.

QUOTABILITY OF NOTES

- | | |
|---------------------------------------|--|
| Abrahamson, S. 29:101; 34:70; 34:48. | Lüers, H. 8:86; 13:72; 23:92; 24:86; |
| Angus, D. 35:71. | 26:108; 28:131; 30:132; 33:145; |
| Arnold, L. 32:166. | 34:91. |
| | Lüers, Th. 28:131; 30:132; 30:133. |
| Barish, N. 28:103. | |
| Baumiller, R. 32:113; 33:122. | Mather, Wharton B. 27:101; 33:147. |
| Becker, H. J. 30:101; 30:102; 33:82. | Mead, C. G. 35:89. |
| Belitz, H. J. 28:108; 30:104; 34:72. | Moree, R. 20:66; 20:88; 20:93; 21:69; |
| Bochnig, V. 26:91; 28:108. | 21:87; 21:91; 29:142. |
| Brosseau, George. 29:106a; 29:106b, | |
| 30:106; 30:160; 32:115; | Oksala, T. A. 31:147; 31:149. |
| 32:116; 33:122; 33:123; | |
| 35:73. | Röhrborn, G. 30:148; 33:156. |
| Divelbiss, J. E. 33:128; 35:77, 78. | Rosin, S. 23:97; 25:75; 25:136. |
| Doane, W. W. 32:121; 34:49 | |
| (cf. Doane 35:45b), 35:45a; | Sandler, L. 26:119; 27:111; 28:153; |
| 35:78. | 29:162a; 29:162b; 30:150; |
| | 31:158; 34:103; 35:93. |
| Fox, A. S. 21:85; 21:86; 22:53; | Sandler, I. 30:151; 32:154. |
| 29:116; 35:81. | Seto, F. 31:160; 31:161; 32:157; 33:159; |
| Frost, J. N. 35:81a; 35:81b. | 34:106; 35:94. |
| Fuscaldo, K. E. 35:84. | Stevenson, R. 33:182. |
| | Strangio, V. A. 30:152; 31:163; 34:107; |
| Hannah, A., and C. Stern. 26:104. | 35:96. |
| Hannah, A., and Ø. Stromnaes. 29:121. | Stromnaes, Ø., and A. Hannah. 29:179. |
| Harrison, B. J. 17:60; 28:122a; | |
| 28:122b; 28:123. | Telfer, J. D. 28:161. |
| | Volkart, H. D. 33:100. |
| Jacobs, M. E. 29:126; 31:124; | |
| 32:130a; 32:130b; 32:130c; | Ursprung, H. 33:174; 34:110. |
| 33:140; 35:89. | |

The Genetics Training Committee of the University of North Carolina wishes to announce the availability of the pre- and postdoctoral traineeships for the study of *Drosophila* or Medical Genetics. Persons interested should write to Professor John Graham in care of that institution in Chapel Hill, North Carolina.

King, R. C. A suggestion with respect to translations.

Might it not be useful to have a yearly listing of English translations of foreign language works dealing with *Drosophila* and to have duplicate translations collected in one laboratory (Herskowitz's at St. Louis University, for example), where they could be made available to everyone? Such a system might save a great deal of duplicated effort. Each translation should be OKed by the original author before its release.

Novitski, E., and R. Dorsey. A generalized maximum likelihood program for the IBM 1620.

We are now programming the IBM 1620 to handle maximum likelihood problems of the sort that might concern geneticists. The procedure followed will be the method of least squares as described by Rao, making it possible to solve relatively complex formulations by iteration. Taking the necessary partial derivatives, and forming the matrices, transpose and inverse, will be done internally by the program. Provision will be made to detect insoluble or ambiguous formulations. It would be helpful to us if anyone with a bona fide likelihood problem at the present time would let us know its nature so that we might check our concept of what such a program should be like against the demands of actual cases.

Sokoloff, A. Transfer of *Tribolium* stocks.

The stocks of *Tribolium castaneum*, *Tribolium confusum* and *Latheticus oryzae* have been transferred from the Biological Laboratory, Cold Spring Harbor, to the Department of Genetics, University of California, Berkeley. Several wild type strains for *T. castaneum* and *T. confusum* and one for *L. oryzae* are being maintained. In addition a large number of stocks with sex-linked and/or autosomal markers is available, particularly for *T. castaneum*. Supplies of some of the mutant and wild type stocks are available to those who intend to use them in their genetics courses.

Supported by a National Science Foundation Grant, a conference on Behavior Genetics was held at the Center for Advanced Study of the Behavioral Sciences in Stanford, California, from August 14 through September 3, 1961. The organizing committee consisted of Jerry Hirsch (Psychology, University of Illinois), chairman; Gerald E. McClearn (Psychology, University of California), Benson Ginsburg (Biology, University of Chicago), Howard Hunt (Psychology, University of Chicago).

The other members of the conference were Gordon Allen (Genetics, National Institute of Health), Peter L. Broadhurst (Psychiatry, University of London, England), Jan. H. Bruell (Psychology, Western Reserve University), Ernst W. Caspari (Biology, University of Rochester), Eckhard Hess (Psychology, University of Chicago), John A. King (Zoology, Michigan State University), Daniel S. Lehrman (Animal Behavior, Rutgers University), Gardner Lindzey (Psychology, University of Minnesota), Aubrey Manning (Zoology, University of Edinburgh, Scotland), Robert C. Roberts (Animal Genetics, University of Edinburgh, Scotland), and W. Robert Thompson (Psychology, Wesleyan University).

Guests at some sessions included Sherwood Washburn (Anthropology, University of California), James McGaugh (Psychology, San Jose State College), Mark Rosenzweig (Psychology, University of California), J. Anthony Deutsch (Psychiatry and Psychology, Stanford University), Kenneth Calby (Psychiatry, Center for Advanced Study of the Behavioral Sciences), Leon Otis (Stanford Research Institute), John Clausen (Sociology, University of California), William Meredith (Psychology, University of California), Frank A. Beach (Psychology, University of California), Francis Palmer (Social Science Research Council), and Loise Erlenmeyer-Kimling (Medical Genetics, Psychiatric Institute, Columbia University).

In 1962 a second and final meeting of the conference will be held to complete a volume on Behavior Genetics. It will consist of chapters that were stimulated by last summer's discussions.

At the September, 1961, meeting of the Social Science Research Council, a committee for genetics and social behavior was established. The members of this committee are: Gardner Lindzey (Psychology, University of Minnesota), chairman; Ernst Caspari (Biology, University of Rochester), Theodosius Dobzhansky (Zoology, Columbia University), David Hamburg (Psychiatry, Stanford University), Jerry Hirsch (Psychology, University of Illinois), Gerald McClearn (Psychology, University of California), James Spuhler (Anthropology, University of Michigan).

The expressed purpose and functions of the new committee are "to facilitate and expedite research in Behavior Genetics in whatever manner seems appropriate with particular reference to the application of new knowledge and advanced methods and techniques to the study of human behavior."

DIRECTORY

Geographical

(Alphabetically arranged according to country, city, laboratory.)

ARGENTINA

Buenos Aires

Comision Nacional de Energia Atomica, Claustro de Investigaciones Cientificas,
Laboratorio de Genetica

de Fincati, Wanda Pirovano, Ing. Agr. Research Assistant.
Kirschbaum, Werner F. Research Assistant.
Leon, Williams N. Technical Assistant.
de Marinic, Susana Ercolini. Research Assistant.
Munoz, Enzo Ruben. Research Assistant.
Paz, Bonifacia del Carmen. Curator of Stocks.
Valencia, Ruby Marie. Ph.D. Chief of Laboratory. Radiation genetics.

Buenos Aires

Universidad de Buenos Aires, Facultad de Ciencias Exactas y Naturales

Cacheiro, Néstor. Research Assistant.
Diez, Julio. Research Assistant.
Kaufman, Beatriz. Stocks Curator.
Mazar-Barnett, Beatriz. Student Investigator.
Valencia, Juan I. Professor. Head of Department of Biology.

AUSTRALIAAdelaide, South AustraliaUniversity of Adelaide, Department of Genetics

Mayo, M. Jean. Ph.D. Lecturer.

Hayman, D. L. Ph.D. Lecturer.

BrisbaneThe University of Queensland, Department of Zoology

Mather, Wharton B. Ph.D. Senior Lecturer. Population Genetics, Cytogenetics, Chromosomal Polymorphism.

Angus, D. B.Sc. (Hons.) Graduate Student. Population genetics.

Harlock, Rosalie. B.Sc. Graduate Student. Population genetics.

Spurway, Rosalyn. Research Assistant.

Hobart, TasmaniaThe University of Tasmania, Department of Zoology

Brink, N. G. Research student. Mutation.

Campbell, R. (Mrs.) Research assistant.

Clark, A. M. Professor. Radiation and chemical mutagenesis.

Clark, E. G. (Mrs.) Research assistant.

Knott, B. Technical assistant.

Melbourne, VictoriaThe University of Melbourne, Department of Zoology

Gunson, Mary M. M.Sc. Lecturer. Salivaries.

Strangio, V. A. M.Sc. Senior Demonstrator. Mutation.

Thomson, J. A. M.Sc. Lecturer. Population genetics.

Wasserman, M. Ph.D. Senior Lecturer. Cytology; evolution.

Sydney, New South WalesSydney University, CSIRO Animal Genetics Division, Animal Genetics Laboratory,
Department of Zoology

Rendel, J. M. B.Sc., Ph.D. Chief of Division. Population genetics; selection.

Sheldon, B. L. B.Sc. Agr., Ph.D. Research Officer. Selection; induction of mutations.

Finlay, D. B.Sc. Agr. Experimental Officer.

Sydney, New South WalesUniversity of Sydney, Department of Animal Husbandry

Barker, J. S. F. Ph.D. Senior Lecturer. Population genetics.

Bull, Shane. B.Sc. Agr. Research Assistant. Population genetics.

AUSTRIAVienna IX (Wien IX)Institut f. Allgemeine Biologie, Schwarzspanierstrasse 17

Karlik, Anni. Ph.D. *Melanogaster*: population genetics.

Kunze-Mühl, Elfriede. Ph.D. *Subobscura*: cytogenetics.

Löffler, Erika. Ph.D. *Puliciphora*: genetics.

Mainx, Felix. M.D., Ph.D. Professor. Head of department.

Ruderer, Elfriede. Ph.D. *Megaselia*: genetics.

Ruttner, Friedrich. M.D., Ph.D. Genetics of the honey bee.

Sperlich, Diether. Ph.D. *Subobscura*: population genetics.

Springer, Robert. Ph.D. *Megaselia*: genetics.

BELGIUMLouvainThe University, Agricultural Institute, Laboratory of General Genetics

See DIS 34:127.

BRAZILPôrto AlegreUniversidade do Rio Grande do Sul, Departamento de Genética, Instituto de Ciências Naturais, Av. Paulo Gama.

Antochevizky, Neuza. Technician. Genetic analysis of irradiated populations.
Cordeiro, A. R. Ph.D. Professor. Head of Department. Population's genic and chromosomal polymorphism. Chromatography and races. Natural irradiated populations.
Cordeiro, E. R. Technician.
Ditadi, T. F. (Miss) Technician. Genetic analysis of irradiated populations.
Lewgoy, F. Chem. Eng. Research Assistant. Spectrophotometry chromatography of pteridines.
Ludwig, Maria. Technician. Stockkeeper.
Ludwig, Nilda Conceição. Technician.
Leões, Ana Q. Bc.Sc.Lic. Fellow of Brazil C. of Res. (CNP_q) Poliploidy in vegetal. Alophia (Iridaceae): chromosomal polymorphism.
Maciel, Clara Maria P. Bc.Sc.Lic. Research Assistant Fellow of Brazil C. of Res. (CNP_q) Genetics of recessive sterility. Radiation genetics of natural populations.
Marques, E. K. Bc.Sc.Lic. Research Assistant, Instructor Assistant. Fellow of National Commission of Nuclear Energy (C. N. E. N.). Radiation genetics of natural populations. Competition between irradiated species.
Morales, Nena B. Technician. Cytogenetics of *D. willistoni* and *D. paulistorum*.
Mundt, Carmen S. Bc.Sc.Lic. Fellow of Research. Human genetics. Haptoglobins.
Napp, Marly. Bc.Sc.Lic. Research Assistant. Radiation genetics of natural populations. Polymorphism and radiation.
Ramila, D. Technician. Foodmaker.
Reguly, Maria Luiza. Bc.Sc.Lic. Fellow of Brazil C. of Res. (CNP_q) Radiation genetics of natural populations.
Roiseberg, I. Bc.Sc.Lic. Fellow of Brazil C. of Res. (CNP_q) Human genetics. Hemophilia.
Salzano, F. M. Ph.D. Assistant Professor. Head of the Human Genetics Division of the Department. Human blood groups. Indian population genetics.
Santos, Alda T. D. Administrative Assistant.
Silva, Luiz C. Technician Electronics.
Silva, Tereza M. Technician. Stockkeeper.
Simões, G. V. Technician. Field worker. Human genetics.
Thedy, O. Technician.
Tondo, C. V. E.E. Bc.Sc. Head of Biophysical Division of the Department. Electrophoresis; chromatography in *Drosophila* mutants and human blood groups; development of new techniques.
Trogildo, D. N. Technician. Stockkeeper.
Winge, Helga. Bc.Sc. Lic. Research Assistant. Radiation genetics of natural populations. Relations in the sibling group *willistoni*.

São PauloUniversidade de São Paulo, Faculdade de Filosofia, Ciências e Letras, Departamento de Biologia Geral, Caixa Postal 8.105

Basile, R. Graduate Student. Cytogenetics.
Breuer, M. E. Technical Assistant. Comparative studies on *Drosophila* genitalia. Radiation genetics.
Cestari, A. N. Lic. Cien., Assistant. Tissue. Culture. Human chromosomes.
Camba, C. S. Graduate Student. Speciation.
da Cunha, A. B. Ph.D. Associate Professor. Population genetics: polymorphism in *Drosophila* and effects of radiation on populations.

Frota-Pessoa, O. Ph.D. Assistant Professor. Human genetics.
Matos, N. S. Graduate Student. Population genetics.
Magalhães, L. E. de. Ph.D. Assistant Professor. Population genetics and speciation.
Oros, J. Lic. Cienc. Fellow in Human Genetics.
Pavan, C. Ph.D. Professor. Head of the Department. Population genetics, radiation genetics and cytogenetics.
Poletto, D. Graduate Student. Human genetics.
Toledo, J. S. Lic. Cienc. Assistant. Population genetics, radiation genetics.
Toledo, S. A. Lic. Cienc. Assistant. Population genetics.
Wajntal, A. Graduate Student. Human genetics.

CANADA

Toronto

University of Toronto, Department of Zoology

Butler, L. Ph.D. Associate Professor. Director of the Laboratory. Population genetics.
Tallan, I. Ph.D. Assistant Professor. Genetics of antigens.
Seiger, M. B. M.A. Graduate Student. Quantitative inheritance.
Mileiko, V. V. B.A. Technical Assistant. Curator of Stocks.

Vancouver, B. C.

The University of British Columbia, Department of Biology and Botany

Cole, Kathleen M. M.A., Ph.D. Assistant Professor. Mutations and cytogenetics.
Cohen, Barrie. Genetics graduate student. A study of aging.
Wills, Christopher. Curator of Stocks. Genetics graduate student. Population studies.

Vancouver, B. C.

The University of British Columbia, Department of Zoology

Band, Henretta T. (Mrs.) Ph.D. Research Associate. Population genetics
Ogawa, Tomoye. Technical Assistant
Tabata, Kazi. Technical Assistant

CHILE

Santiago

Universidad de Chile, Instituto de Biología "Juan Noé," Cátedra de Biología, Zañartu 1042

Brncic, D. Associate Professor. Population genetics.
Casanova, Adriana. Technical Assistant. Curator of Stocks.
Covarrubias, Edmundo. M.D. Research Assistant.
del Solar, Eduardo. Graduate student. Population genetics.
Fernandez, Raúl. Graduate student. Spermatogenesis in *Drosophila*.
Koref-Santibañez, Susi. M.D. Research Associate. Population genetics, isolating mechanisms.
Pellicer, M. Dolores. Technical Assistant.

COLOMBIA

Bogotá

University of the Andes, Department of Biology, Apartado Aereo 4976

Hunter, Alice S. Ph.D. Physiology, taxonomy--*Drosophila*.
Newball, Sarah. Assistant.

Bogotá D. E.

University of the Andes, Department of Genetics

Hoenigsberg, H. F. B.A., D.Sc. Professor of Genetics. Radiation genetics. Sexual isolation in evolution.
Cortés, Yolanda Garcia (Miss). B.Sc. Research Assistant. Mating preferences in mutants. Radiation genetics.

Rubio, Dilia Ortíz (Miss). B.Sc. Research Assistant. Mating preferences and fitness in mutants.
Cortés, Blanca Inés (Miss). Laboratory technician.
Díaz, Napoleón (Mr.). Laboratory technician.
Duplat, Hermán (Mr.). Laboratory technician.
Chejne, Abraham (Mr.). Laboratory technician.
Rios, Cesar (Mr.). Technician's help.

DENMARK

Copenhagen

University of Copenhagen, Institute of Genetics, 2A Øster Farimagsgade

See DIS 34:128.

FINLAND

Helsinki

University of Helsinki, Institute of Genetics, P. Rautatiekatu 13

Halkka, Olli. Ph.D. Assistant. Salivary chromosomes.
Lakovaara, Seppo. Ph.D. cand. Assistant. Eye mutants.
Sammalisto, Lasse. Ph.D. Assistant. Population genetics.
Suomalainen, Esko. Ph.D. Professor. Head of Department.
Tiivola, Airi. (Mrs.) Technical assistant. Curator of Stocks.

Turku

University of Turku, Institute of Genetics

Frost, Justin N. Ph.D. N. I. H. Postdoctoral Fellow. *Melanogaster*: interchromosomal effects.
Hannah-Alava, Aloha (Mrs.). Ph.D. Research Associate. *Melanogaster*: developmental genetics; mutations.
Harmoinen, Liisa (Miss). Research Assistant. *Melanogaster*: mutations.
Heinonen, Pirkko (Miss). Research Assistant. *Melanogaster*: mutations.
Oksala, T. A. Ph.D. Professor. Head of Department. *Melanogaster*: mechanism of segregation; interchromosomal effects.
Puro, J. Ph.D. cand. Assistant Teacher. *Melanogaster*: mutations.
Savolainen, Salme (Mrs.) Technical Assistant.
Wallenius, Marja-Liisa (Miss). Research Assistant. *Melanogaster*: mechanism of segregation; interchromosomal effects.

FRANCE

Gif-sur-Yvette (S. et O.)

Centre National de la Recherche Scientifique, Laboratoire de Génétique évolutive et de Biométrie

Bergerard, J. Professor. Cytogenetics.
Bigler, J. (Miss) Technician.
Bösiger, E. Ph.D. Chargé de recherches. Heterosis, sexual selection.
Laugé, G. (Miss) Assistant. Triploid intersexes of *Drosophila*.
Léon, M. (Miss) Graduate student. Irradiation effects on development.
Louis, M. (Mrs.) Technician.
Piva, A. Graduate student. Quantitative inheritance.
Queiroz, J. (Mrs.) Attachée de recherches. Quantitative inheritance.
Teissier, G. Professor. Head of the department. Population genetics, quantitative inheritance, biometry.

Gif-sur-Yvette (S. et O.)

Centre National de la Recherche Scientifique, Laboratoire de Génétique Formelle

Lestrangé, M.-Th. de (Miss). Attachée de recherches. CO₂ sensitivity in *Drosophila*.
L'Heritier, Ph. Professor. Head of the Department. CO₂ sensitivity in *Drosophila*.

Ohanessian-Guillemain, A. (Mrs.) Chargée de recherches. CO₂ sensitivity in *Drosophila*.
Plus, N. (Mrs.) Chargée de recherches. CO₂ sensitivity in *Drosophila*.
Proust, J. (Mrs.) Attachée de recherches. Quantitative inheritance in *Drosophila*.
Vigier, Ph. Maître-assistant. CO₂ sensitivity in *Drosophila*.

Lyon (Rhône)

Laboratoire de Zoologie Expérimentale, Faculté des Sciences, 16, quai C. Bernard

Brun, J. Maître-Assistant. Cytology and genetics of nematodes.
Daillie, J. Maître-Assistant. Nucleic acid metabolism.
Dalmon, J. Assistant. Nucleic acid metabolism.
David, J. Maître-Assistant. Quantitative inheritance in *Drosophila*.
Fourche, J. Maître-Assistant. Respiratory metabolism in *Drosophila*.
Godet, J. (Mrs.) Assistant. Ovogenesis in *Drosophila*.
Guerrier, P. Cytology of nematodes.
Legay, J. M. Maître de conférences. Physiology and genetics of phytophagous insects.
Neulat, M. M. (Miss) Assistant. Nucleic acid metabolism.
Nigon, V. Professor. Head of the department. Nucleic acid metabolism.
Perdrix, S. (Miss) Assistant. Ovogenesis in *Drosophila*.

Orsay (S. et O.)

Université de Paris, Faculté des Sciences, Biologie Générale

Bernard, J. (Miss) Assistante. CO₂ sensitivity in *Drosophila*.
Bregliano, J-C. Assistant. CO₂ sensitivity in *Drosophila*.
Brun, G. Chef de Travaux Pratiques. CO₂ sensitivity in *Drosophila*.

Paris

Faculté des Sciences, Laboratoire de Zoologie, 1 rue Victor Cousin, Paris 5 ème

See DIS 34:129.

Strasbourg (Bas-Rhin)

Université de Strasbourg, Faculté des Sciences

See DIS 34:129.

GERMANY

Berlin-Buch

Deutsche Akademie der Wissenschaften, Institut für experimentelle Krebsforschung,
Genetische Abteilung, Lindenberger Weg 70

Bender, Erhard. Dr. Microbial genetics: Chemical mutagenesis.
Geissler, Erhard. Dr. Head of Department. Microbial genetics: Lysogeny.
Pasternak, Luise. *Melanogaster*: Chemical mutagenesis.

Berlin-Dahlem

Institut für Genetik der Freien Universität Berlin, Rudeloffweg 9

Bartelt, Jutta. Technical Assistant. *Melanogaster*: radiation genetics.
Belitz, Hans-Joachim (Dr.). Research Assistant. *Melanogaster*: induced mutations.
Bochnig, Veronika (Dr.). Research Assistant. *Melanogaster*: physiological genetics, radiation genetics.
Kromm, Natalie. Technical Assistant. Curator of stocks, chemogenetics.
Lüers, Herbert (Prof. Dr.). Director. Comparative genetics; mutagens.
Lüers, Thea (Mrs., Dr.). Guest Associate. *Drosophila* neurology.
Nöthel, Horst. Graduate student. Radiation genetics.
Pasternak, Luise. Graduate student. *Melanogaster*: DDT-resistance. Absent since 13 August 1961.
Polzin, Walter. Technical Assistant. Radiation genetics.
Ravasani, Chapour. Graduate student. *Melanogaster*: radiation genetics.

Röhrborn, Gunter (Dr.). Research Assistant. *Drosophila* tumors; chemical mutagens.
Rudolph, Edeltraud. Technical Assistant. *Melanogaster*: cytology.
Struck, Eva (Mrs., Dr.). Research Assistant. Insects: cytology.
Winterfeldt, Gisela. Graduate student. *Melanogaster*: radiation genetics. Absent since 13 August 1961.
Wolf, Erich (Dr.). Associate. Insects: cytology.

DarmstadtBotanisches Institut der Technischen Hochschule

Ziegler, Irmgard (Mrs., Dr.). Physiology of pteridines under the influence of genes.

Hamburg 13Zoologisches Staatsinstitut und Zoologisches Museum, von-Melle-Park 10

Koske-Westphal, Thea (Mrs.). Ph.D. Study of hybrids between triploid *melanogaster* females and x-rayed *simulans* males.
Kosswig, Curt. Prof. Dr. Director.

Hamburg-EppendorfUniversitäts-Frauenklinik, Strahlenbiologische Abteilung

See DIS 34:130.

HeidelbergUniversität Heidelberg, Zoologisches Institut, Sofienstr. 6

See DIS 34:130.

KarlsruheInstitut für Strahlenbiologie, Kernforschungszentrum Karlsruhe

Apitzsch, Ursula. Curator of Stocks.
Catsch, Alexander. Prof. Dr. *Drosophila* genetics.
Dittrich, Wolfgang. Prof. Dr. Molecular genetics.
Ebeling, Wolfgang. Graduate student. *Drosophila* genetics.
Hotz, Gerhart. Dr. Bacteriophage genetics.
Kircheisen, Gerda. Dr. *Drosophila* genetics.
Köhnlein, Wolfgang. Dipl. Phys. Radiation biology.
Müller, Adolf. Dr. Radiation biology.
Traut, Horst. Dr. *Drosophila* genetics.
Ufholz, Ilse. Technical Assistant.
Zimmer, Karl Günther. Prof. Dr. Radiation genetics.

Marburg/LahnZoologisches Institut der Phillips-Universität, Ketzerbach 63

Becker, Gweneth L. Ph.D. Independent investigator. Lethals.
Becker, Hans J. Ph.D. Assistant. Puffing; variegation.
Scriba, Martin. Graduate student. Deficiencies and early embryology.
Seidel, Friedrich. Ph.D. Professor. Head of Department. Early embryology of insects.

Mariensee über WunstorfMax-Planck-Institut für Tierzucht und Tierernährung

Gellert, Heidemarie. Technical Assistant.
Gottschewski, G. H. M. Prof. Dr. Head of department. Developmental and physiological genetics.
Querner, Waltraud. Dr. Assistant, Stockkeeper. Tissue culture.
Schwinck, Ilse. Dr. Guest investigator. Physiological genetics.
Zimmermann, Wolfgang. Dr. Assistant. Genetics.

Münster (Westf.)Institut für Humangenetik der Universität Münster

Graebner, Erika. Technical Assistant.

Ostertag, Wolfram. Ph.D. Radiation genetics (somatic damage).

TübingenMax Planck-Institut für Biologie, Spemannstr. 34

Beermann, Wolfgang. Prof. Dr. Director. Physiology of salivary gland chromosomes.

Hess, Oswald. Dr. Research Assistant. Physiology of chromosomes (Y-chromosome).

Joneleit, Christa (Miss). Technical Assistant. Curator of stocks.

Meyer, Günther F. Dr. Research Assistant. Gametogenesis, light and electron microscopy.

Seidel, Sigrid (Miss). Graduate student. Sex determination (tra-mutation).

GHANALegon, AccraUniversity of Ghana, Department of Zoology

See DIS 34:131.

Legon, AccraUniversity of Ghana, Department of Chemistry

Blair, J. A. Ph.D. Lecturer. Origin of pteridine compounds in Drosophila.

GREAT BRITAINAberdeen, ScotlandUniversity of Aberdeen, Department of Zoology

See DIS 34:131.

Bayfordbury, Hertford, Herts, EnglandJohn Innes Horticultural Institution

Harrison, B. S. Multiple insecticide resistance.

Birmingham 15, EnglandThe University, Department of Genetics

See DIS 34:131.

Cambridge, EnglandUniversity of Cambridge, Department of Genetics, Milton Road

Alderson, T. Ph.D. Research worker. Chemical mutagenesis.

Batten, J. L. Research student.

Gibson, J. B. Ph.D. Assistant in Research. Analysis of selected lines.

Nash, D. Research student. Developmental genetics.

Parsons, P. A. Ph.D. Demonstrator. Population genetics.

Pelecanos, M. Research student. Chemical mutagenesis.

Spickett, S. G. Research student. Developmental genetics of quantitative characters.

Thoday, J. M. Ph.D. Professor. Selection, particularly disruptive. Location of polygenes.

Chalfont St. Giles, Bucks, EnglandInstitute of Cancer Research: Royal Cancer Hospital, Pollards Wood

Fahmy, Myrtle J. Ph.D. Mutagenesis.

Fahmy, O. G. M.Sc., Ph.D. Cytogenetics.

Gleaves, C. Technical Assistant.
Gibbons, A. B.Sc. Research Assistant.
Hope, L. Technical Assistant.
Knight, E. B.Sc. Research Assistant.
Sweron, M. Technical Assistant.

Edinburgh 9, Scotland

Agricultural Research Council Poultry Research Centre, King's Buildings

Burnet, B. Ph.D. Physiological genetics.
Pratt, G. Stock keeper.
Sang, J. H. Ph.D., F. R. S. E. Drosophila nutrition and physiological genetics.
Strachan, I. Technical assistant.

Edinburgh 9, Scotland

University of Edinburgh, Institute of Animal Genetics

Auerbach, C. A. D.Sc., F. R. S. Reader, Chemical and induced mutagenesis.
Allan, J. Graduate student. Selection.
Baden, E. B. Research assistant. Wild species.
Clayton, G. Lecturer. Selection.
Kelsall, P. J. Graduate student. Spontaneous and induced nondisjunction.
Khishin, A. Guest investigator. Formaldehyde and radiation induced mutagenesis.
Knight, G. R. Research assistant. Subobscura salivaries.
Leigh, B. Graduate student. Chemical and induced mutagenesis. On leave with Professor Sobels.
Mostafa, A. Graduate student. Selection.
Nafei, H. Graduate student. Formaldehyde induced mutagenesis.
Osman, H. Graduate student. Selection.
Perry, M. Research assistant. Autoradiography.
Reeve, E. C. R. D.Phil. Quantitative inheritance.
Robertson, A. D.Sc. Quantitative genetics.
Robertson, F. W. D.Sc. Population and physiological genetics.
Royes, V. Graduate student. Drosophila nutrition.
Scharloo, W. Ph.D. Guest investigator. Quantitative inheritance.
Sen, B. K. Quantitative genetics.
Slizynska, H. (Mrs.) Ph.D. Cytological analysis.
Slizynski, B. M. Ph.D. Salivaries.
Snyder, L. A. Ph.D. Guest investigator. Chemical and induced mutagenesis.
Strachan, K. (Miss) Stock-keeper.
Waddington, C. H. Sc.D., F. R. S. Professor. General genetics.

Glasgow, Scotland

University of Glasgow, Department of Genetics

Pontecorvo, G. F. R. S. Professor.
Forbes, E. C. Chief Technician.
Dorn, G. L. Ph.D. Research Associate.

Harwell, Didcot, Berks, England

Medical Research Council, Radiobiological Research Unit

Gale, C. Technical Assistant.
Jempson, J. Technical Assistant.
Lamerton, M. Technical Assistant.
McSheehy, T. W. B.Sc. Radiation Genetics.
Purdum, C. E. Ph.D. Radiation Genetics.

London, E. C. 1, England

St. Bartholomew's Hospital Medical College, Department of Zoology and Comparative Anatomy

Hollingsworth, M. Ph.D. Lecturer. Inbreeding and infertility. Bristle patterns in intersexes.

London, W. C. 1. EnglandUniversity College, Department of Biometry, Eugenics and Genetics

Grüneberg, H. Professor.

London, W. C. 1. EnglandUniversity College, Department of ZoologyClarke, Jean M. Research Assistant. Ageing in *Drosophila*.Lamb, Marion J. Research student. Radiation and ageing in *Drosophila*.

Maynard Smith, J. Lecturer. Genetics of pattern formation.

Manchester, EnglandChristie Hospital and Holt Radium Institute, Cytogenetics Department

See DIS 34:133.

Manchester 13, EnglandThe University, Departments of Botany and Zoology

Dearden, Michael. Research student. Development of eye mutants.

Hartshorne, John N. Lecturer in Genetics.

Nottingham, EnglandThe University, School of Agriculture, Department of Agricultural Science

See DIS 34:133.

Sheffield 10, EnglandThe University, Department of Genetics

Boam, T. B. Chief Technician. Stockkeeper.

Roper, J. A. Professor. Microbial Genetics.

INDIACalcutta 19Calcutta University, Department of Zoology, Cytogenetics Laboratory

See DIS 34:133.

Calcutta 35Indian Statistical Institute, 203, Barrackpore Trunk Road

See DIS 34:133.

HyderabadOsmania University, Radiation Genetics Project, aided by Department of Atomic Energy
(Government of India)

Reddi, O. S. Dr. Investigator.

Mathew, C. (Mr.) Senior Scientific Assistant.

Prabhakara Rao (Mr.). Junior Scientific Assistant.

Research problems under investigation:

1. Induction of translocations in the spermatogonia of *Drosophila melanogaster* by CB 1506.
2. Studies on the specific effect of phenylalanine mustard on the II chromosomal lethals of *Drosophila melanogaster*.
3. Induction of translocations in the spermatogonia of *Drosophila melanogaster* by X-rays.
4. Genetic recovery in the spermatozoa of *Drosophila melanogaster* by Fast Neutrons.

Madras 7Veterinary College, Department of Animal Genetics, Vepery

Dharmarajan, M. M.A., M.Sc., Ph.D. Head of Department. *Drosophila* species.
Narayana Rao, N. M.A. Assistant. *Melanogaster*: species, ecology.
Suguna, S. G. (Miss) M.Sc. Advanced student. *Drosophila* mutagenesis.

New Delhi 12Institute of Agricultural Research Statistics (I. C. A. R.), Library Avenue

Narain, Prem. Professor.

ISRAELJerusalemHebrew University, Department of Zoology

Barak, Elisheva. Research student. Induced chromosome breaks.
Blum, Sonya. Laboratory Assistant.
Cividalli, Lia. Research student. Population genetics.
Fattal, S. Laboratory Assistant.
Falk, R. Ph.D. Instructor. Induced mutations: viability effects and mechanisms.
Freund, Ora. M.Sc. Cytogenetics.
Friedlaender, M. Research student. Cytogenetics.
Goldschmidt, Elisabeth. Ph.D. Associate Professor. Pteridines.
Himel, Nechama. Laboratory Assistant. Chromosome breakage.
Hurvitz, Dalia. Research student. Pteridines.
Horowitz, Aviva. Research student. Population genetics.
Lederman-Klein, Ada. M.Sc. Assistant. Homeotic mutant.
Rahat, Ana. M.Sc. Assistant. Induced viability mutations.
Rappaport, Sarah. Research student.
Ritte, U. Research student. Population dynamics.
Ronen, Amiram. M.Sc. Assistant. X-ray induced crossing-over.
Wahrman, J. Ph.D. Lecturer. Cytogenetics.

ITALYMilanoUniversità di Milano, Istituto di Genetica, Via Celoria 10

Barigozzi, C. D.Sc. Professor of Genetics. Director. Genetics of melanotic tumors of *Drosophila* (effect of cytoplasm).
Bairati, A. M.D. Research Fellow. Electron microscopy of *Drosophila* cells.
Castiglioni, M. C. D.Sc. Assistant. Developmental genetics of *Drosophila*. Tissue culture of *Drosophila*.
Di Pasquale, A. D.Sc. Assistant. Genetics of "brown spots."
Gallucci, E. M.D. Research Fellow. Induced mutations in *Drosophila*.
Giavelli, S. M.D. Research Fellow. Induced mutations in *Drosophila*.
Halfer, C. D.Sc. Assistant. Effect of internal environment on gene manifestations.
Kravina, A. M. D.Sc. Assistant. Genetics of melanotic tumors of *Drosophila*.
Locatelli, F. (Miss) Technician. Curator of Stocks.
Pozzi, L. D.Sc. Research Fellow. Induced mutations in *Drosophila*.
Rezzonico, Raimondi G. D.Sc. Assistant. Experimental cytology of *Drosophila*.
Sironi, G. P. Student Assistant. Induced mutations in *Drosophila*.
Zambruni, L. D.Sc. Assistant. Genetics of "brown spots."

NapoliDell Università, Istituto di Genetica

See DIS 34:134.

PaviaUniversità di Pavia, Istituto di Genetica

See DIS 34:134.

RomaIstituto di Genetica, Città Universitaria

Arabia, Lilliana. Research Assistant. *Melanogaster* cytogenetics.
Canuti, Nella. Research Assistant. *Melanogaster* translocation.
Micheli, Aldo. Curator of stocks.
Montalenti, Giuseppe. Professor of Genetics. General genetics.
Nicoletti, Benedetto. Assistant Professor. *Melanogaster* cytogenetics, mutagenesis.
Olivieri, Gregorio. Research Assistant. *Melanogaster* C.O. induced in males.
Olivieri, Mancini Angela. Research Fellow. *Melanogaster*.

JAPANAnzō, AichiNagoya University, Faculty of Agriculture, Department of Animal Breeding

Bitō, J. Graduate student. *Melanogaster*; mutation.
Esaki, K. Instructor. *Melanogaster*; mutation.
Hayakawa, J. Graduate student. *Melanogaster*; mutation.
Kondo, K. (Dr.) Professor. General genetic problems.
Nozawa, K. (Dr.) Assistant Professor. *Melanogaster*, other species; population genetics and mutation.
Ota, N. (Miss) Technical Assistant. Curator of stocks.

Chiba-shiNational Institute of Radiological Sciences, Biology Division

See DIS 34:135.

HiroshimaHiroshima University, Faculty of Science, Zoological Laboratory

Minamori, Sumio. Dr. Assistant Professor (on leave, 1961-1962, National Institute of Genetics, Mishima). *Melanogaster*: population genetics.

KobeKobe University, Biological Laboratory

Fujii, S. Dr. Professor. Chromosomal aberrations; salivary chromosomes; developmental genetics.
Kanehisa, T. Dr. Research Assistant. Biochemical genetics of tumor.
Kawabe, M. Dr. Assistant Professor. Developmental genetics; variations; human genetics.
Kitazume, Y. Research Assistant. Cytochemical studies of lethal mutations.
Maeda, Y. Assistant in Research. *Melanogaster*; mutation.
Magaribuchi, K. Technical Assistant. Curator of stock.

KyotoKyoto University, Faculty of Science, Department of Zoology

Imaizumi, Tadashi. Assistant. Physiological genetics and embryology.
Kato, Masaru. Dr. Assistant Professor. Biochemical genetics and embryology.
Kato, Mikio. Dr. Research associate. Biochemical genetics. (Present address: Department of Zoology, University of Ottawa, Ottawa 2, Canada.)
Miyoshi, Yasuhiro. Graduate student. Physiological genetics.
Nakamura, Kenji. Dr. Professor. Cytogenetics and physiology.
Okuda, Chizuko (Miss). Technical Assistant. Curator of stocks.

Misima, Sizuoka-kenNational Institute of Genetics

- Chigusa, S. Research Assistant. Population genetics; mutation and selection.
Fuwa, K. Research Assistant. Population genetics; deleterious genes in natural populations.
Hiraizumi, Y. Dr. Research Member. Population genetics.
Imai, Y. (Miss) Technical Assistant.
Iyama, S. Dr. Research Member. Population genetics; competition and migration (in University of Minnesota, Department of Agronomy, St. Paul).
Kimura, M. Ph.D. Research Member. Population genetics; theoretical (in University of Wisconsin, Madison).
Masuda, H. (Miss) Technical Assistant.
Minamori, S. Dr. (Assistant Professor of Hiroshima University, Visiting Researcher) Population genetics; deleterious genes in natural populations.
Mukai, T. Ph.D. Research Member. Population genetics; radiation and polygene.
Nakamura, K. (Miss) Technical Assistant.
Narise, T. Dr. Research Member. Population genetics; competition and migration.
Nawa, S. Dr. Research Member. Biochemical genetics; pteridine and nucleic acid (in University of Texas, Austin).
Oshima, C. Dr. Head of Department. Population genetics; resistance, radiation and deleterious genes in natural populations.
Sakaguchi, B. Dr. Research Member. Biochemical genetics; enzymes.
Sakai, K. Dr. Head of Department. Population genetics; competition and migration.
Taira, T. Dr. Research Member. Biochemical genetics; eye pigment formation and metamorphosis.
Toyofuku, Y. (Mrs. Tonomura) Dr. Research Member. Cytogenetics.
Yamada, Y. Dr. Research Member. Population genetics; mutation and selection (in Purdue University, Population Genetics Institute, Lafayette).

Mitaka, TokyoInternational Christian University, Biology Department

- Sinoto, Y. Professor. Salivary chromosomes.
Shoji, T. Instructor. Salivary chromosomes.
Kaminishi, H. (Mrs.) Research fellow. Salivary chromosomes.

Okamoto, KobeKonan University, Biological Laboratory

- Inouye, I. Research Associate. *Melanogaster*, selection.
Kaji, S. Dr. Assistant Professor. *Melanogaster*, selection, physiological genetics.
Takaya, H. Dr. Professor. *Melanogaster*, selection.

OsakaOsaka University, Faculty of Medicine, Department of Genetics; andOsaka University, Faculty of Science, Biological Institute

- Fujio, Y. Graduate Student. *Drosophila*: embryological genetics.
Hiraga, S. Graduate Student. *Musca*: biochemical genetics.
Hiroyoshi, T. Assistant. *Musca*: mutations and sex-determination.
Ichioka, S. (Miss) Technical Assistant. Curator of stocks.
Kikkawa, H. Dr. Professor. *Drosophila* and *Musca*: chemical genetics and resistance to insecticides.
Kuroda, Y. Dr. Lecturer. *Drosophila*: embryological genetics. (Present address: Department of Zoology, University of Chicago, Chicago, Illinois, U. S. A.)
Nobuki, R. (Miss) Technical Assistant. Curator of stocks.
Ogita, Z. Dr. Assistant. *Drosophila* and *Musca*: chemical genetics and resistance to insecticides.
Otuji, Y. (Mrs.) Graduate Student. *Musca*: cytogenetics and sex-determination.
Seki, T. Dr. Lecturer. *Drosophila*, *Musca* and *Bombyx*: chemical genetics.
Tsukamoto, M. Dr. Assistant. *Drosophila* and *Musca*: mutations, chemical genetics and resistance to insecticides.
Watanabe, H. (Mrs.) Graduate Student. *Musca*: chemical genetics.

Sakai, OsakaUniversity of Osaka Prefecture, Department of Biology

Ogaki, M. Dr. Assistant Professor. *Melanogaster*: genetics of physiological character.

Tanaka, E. Assistant. *Melanogaster*: physiological genetics.

SapporoHokkaido University, Faculty of Science, Department of Zoology

Kaneko, A. Research assistant. Geographical distribution; cytogenetics.

Makino, S. Dr. Professor. Cytogenetics; population genetics.

Momma, E. Dr. Assistant Professor. Geographical distribution; cytogenetics; population genetics.

Shima, T. Research assistant. Geographical distribution; cytogenetics.

Takada, H. Dr. Research assistant. Taxonomy; ecology.

TokyoTokyo Metropolitan University, Faculty of Science, Department of Biology, Setagaya-ku

Akita, Y. K. Dr. Professor. *Melanogaster*: gene action, biochemistry, radiation biology.

Fuyama, Y. Undergraduate. *Melanogaster*: selection.

Ichida, H. (Miss) Graduate student. *Melanogaster*: biochemical genetics, tumor.

Ikeda, H. Graduate student. Bifasciata, other species; population genetics, cytoplasmic sex-ratio.

Kitagawa, O. Dr. Research Assistant. Bifasciata, *Melanogaster*: population genetics, radiation genetics.

Kurokawa, H. Dr. Lecturer. *Auraria*, other species; population genetics, taxonomy.

Moriwaki, D. Dr. Professor. *Melanogaster*, bifasciata, other species: population genetics, gene analysis, radiation genetics.

Ohba, S. Assistant Professor. *Melanogaster*, other species: population genetics, ecology, tumor.

Ohnishi, E. Dr. Research Assistant. *Melanogaster*, virilis: biochemistry.

Okada, T. Dr. Professor. Various species: variations, taxonomy, ecology.

Tobari, I. Research Assistant. *Melanogaster*: radiation genetics.

Tobari (Nakajima), Y. (Mrs.) Research Assistant. *Ananassae*, other species: population genetics, heterosis, gene analysis.

Tsukamoto, H. (Miss) Technical assistant.

KOREAKongju, Chung Cheong Nam DoKongju National Teachers' College, Department of Biology

See DIS 34:137.

Kwangju, ChunnamNational Chunnam University, College of Liberal Arts and Sciences, Department of Biology

Kim, D. U. Assistant Professor. Microbial genetics.

Kim, K. W. Assistant Professor. *Drosophila* taxonomy.

Park, M. S. Instructor.

Wui, I. S. Instructor.

SeoulChung-Ang University, College of Liberal Arts and Sciences, Department of Biology

Chun, W. S. Graduate student. Geographical survey.

Chung, J. Y. Graduate student. Geographical survey.

Lee, C. S. Graduate Research Assistant. Cytology.

Lee, T. J. Assistant Professor. Population genetics, geographical distribution.

SeoulSeoul National University, Department of Zoology

Kang, Yung Sun. Dr. Professor. Cytology.
Chung, Ok Ki. Instructor. Cytogenetics.
Lee, Hei Yung. Instructor. Cytogenetics.
Choi, Jung Ji. Assistant. Genetics.

SeoulSung Kyun-Kwan University, College of Arts and Science, Department of Biology

Kim, D. S. Graduate student. Migration and competition.
Paik, Y. K. Dr. Consultant. (Permanent address: Yonsei University, Department of Biology, Seoul)
Sung, K. C. Graduate student. Migration and competition.

SeoulYonsei University, College of Science and Engineering, Department of Biology

Kim, D. S. Graduate student. (Permanent address: Sung Kyun-Kwan University)
Migration and competition.
Paik, Y. K. Dr. Associate Professor. Chairman. Population genetics.
Sung, K. C. Graduate student. (Permanent address: Sung Kyun-Kwan University)
Migration and competition.
Yoo, C. S. Undergraduate assistant.
Youn, J. S. Graduate Research Assistant. Radiation genetics.

NETHERLANDSGroningenState University, Genetical Institute, Haren (Gr)

Beardmore, J. A. Professor. Population studies.
Bult, P. Graduate student. Competition.
du Pui, L. (Miss) Technical assistant.
Fockens, W. (Miss) Technical assistant.

LeidenGenetisch Laboratorium der Rijksuniversiteit

Bentvelzen, P. A. J. Assistant. Population genetics.
Berendes, H. D. Assistant. Salivaries D. hydei.
Gloor, H. J. Professor. Developmental genetics.
Heerkens, C. M. (Miss) Technical Assistant.
Jacobs, A. A. C. M. (Miss) Assistant. Localization mutants D. hydei.
Scharloo, W. Assistant. On leave at the Inst. of Animal Genetics, Edinburgh, Scotland.
Schepers, A. M. (Miss) Assistant. Eye pigments.
Schulten, G. G. M. Research student. "Sex-ratio."
Volkers, W. S. Assistant.

LeidenState University, Department of Radiation Genetics, Wassenaarseweg 62

Goedhart, A. (Miss) Technical assistant.
van Hooft, J. I. M. Technical assistant.
den Hollander, C. J. M. (Miss) Technical assistant.
de Klerk, T. H. (Miss) Technical assistant.
Leigh, Barry. B.Sc. Radiation mutagenesis.
Lommerse, M. A. H. (Miss) Technical assistant.
de Ruiter, F. J. (Miss) Technical assistant.
Sobels, F. H. Ph.D. Professor. Radiation mutagenesis, repair mechanism.
Tates, A. D. M.Sc. Research Assistant. Radiation mutagenesis and electronic microscopy.

UtrechtState University, Institute of Genetics, Opaalweg 20

Dykstra, W. T. (Mrs.) Technical assistant.
Hemel, J. O. van. Demonstrator.
Rünke, C. L. Professor. Director.
Schouten, S. C. M. Assistant. Radiation mutagenesis.
Tuinstra, E. J. (Mrs.) Stockkeeper.

NORWAYBergenUniversity of Bergen, Zoological Laboratory

Abro, Arnold. c.r. *Melanogaster*, radiation effects.
Brinkmann, Aug. Jr. Professor of Zoology, Director of the Laboratory.

BlindernUniversity of Oslo, Institute of Genetics

Hansteen, Inger-Lise. Graduate student. Cytology.
Kiil, Wilhelm. Ph.D. *Funebris*.
Kvelland, Ingerid. cand. real. Research Assistant. Radiation genetics.
Mohr, Otto Lous. Dr.Med., L.L.D. Professor Emeritus.
Smith, Edna W. (Miss) B.Sc. Curator of Stocks.
Sollunn, Frank-Jörgen. Graduate student. Radiation genetics.
Strömnaes, Øistein. Ph.D. Assistant Professor. Radiation genetics.
Wedvik, Hans. Graduate student. Radiation genetics.

OsloNorsk Hydro's Institute for Cancer Research, The Norwegian Radium Hospital

Johansen, Ivar. cand. real. Research Fellow. Oxygen effect on radiosensitivity of early embryos.
Mossige, Jeanne Coyne. Research Fellow. Radiosensitivity in sperm.
Ofteidal, Per. dr. philos. Research Fellow. Radiosensitivity of spermatogonia.
Isotope distribution.

PANAMAPanamaThe Gorgas Memorial Laboratory, Balboa Heights Post Office Box 65, Canal Zone

Pipkin, Sarah Bedichek.

SOUTH AFRICAJohannesburgSouth African Institute for Medical Research

See DIS 34:139.

JohannesburgUniversity of the Witwatersrand, Department of Zoology

Hartmann, Ingeborg J. Ph.D. Lecturer. *Zaprionus*: cytogenetics.
Nolte, D. J. D.Sc. Senior Lecturer. Eye pigmentary system; polygenes in geographic strains.
Pillmann, Loré. Curator of Stocks.

PretoriaUniversity of Pretoria, Department of Genetics

Geerthsen, J. M. P. B.S. Senior lecturer.
Hofmeyr, J. D. J. M.S., Ph.D., D.Phil. Professor.
Nel, P. M. B.S., B.S.(For.) Graduate student. Chromosomal polymorphism.
Van Niekerk, Brenda. Technician.
Van Schaik, Nancy W. M.S., Ph.D. Lecturer.

SPAINBarcelonaUniversidad, Centro de Genética Animal y Humana del C. S. I. C.

Alcobé, S. (Mr.) Dr. Director of the Centro. Professor of Anthropology.
Cama, J. (Mr.) Technical Assistant. Curator of Stocks.
Fusté, M. (Miss) Graduate student. D. subobscura populations.
Monclús, M. (Mrs.) Research Assistant. Population genetics.
Nadal, A. (Miss) Graduate student. Lethals in natural populations.
Pons, J. (Mr.) Dr. Research worker. Human genetics.
Prevosti, A. (Mr.) Dr. Head of Drosophila Department. Population genetics.

Madrid 6Centro de Investigaciones Biológicas, Laboratorio de Genética, Velazquez 138

García-Bellido, A. Research Assistant. Developmental genetics.
Miralles, L. Graduate student. Cytogenetics.
Morey, M. Research Assistant. Mutagenesis.
Ortiz, E. Dr. Head of Department. Mutagenesis.
Ramírez, P. Technical Assistant.
Rodríguez, C. Graduate student. Cytogenetics.
Solana, I. Technical Assistant.
Torroja, E. Research Assistant. Mutagenesis.

SWEDENStockholmUniversity of Stockholm, Institute of Genetics

Eiche, A. Ph.K. Research Assistant. *Melanogaster*: population genetics and mutations.
Lüning, K. G. Ph.D. Professor. Director of the Institute. *Melanogaster*: population genetics.
Montelius, I. Ph.K. Research Assistant. *Melanogaster*: population genetics.
Ramel, C. Ph.D. Research Associate. *Melanogaster*: interchromosomal effects, viability mutations.
Sävthagen, Ruth. Ph.D. Research Associate. *Melanogaster*: mutations.
Sheridan, B. B.A. Research Assistant. *Melanogaster*: population genetics.
Ytterborn, K. Ph.Lic. Research Assistant. *Melanogaster*: population genetics.

Uppsala 7University of Uppsala, Institute of Genetics

Gidholm, Kerstin. Ph. M. Research Assistant. *Melanogaster*: developmental disturbances.
Johansson, K. Ph. M. Research Assistant. *Melanogaster*: selective mating.
Lund, B. Curator of Stocks.
Ohlendorff, Helga. Ph.D. Research Assistant.
Rasmuson, B. Ph.D. Research Associate. *Melanogaster*: physiological genetics.
Rasmuson, Marianne. Ph.D. Research Associate. *Melanogaster*: population genetics.
Svensson, Margit. Agr. Research Assistant. *Melanogaster*: physiological genetics.

SWITZERLANDBernZoologisches Institut der Universität

Rosin, Siegfried. Ph.D. Professor. Developmental genetics.
Tschumi, Pierre. Ph.D. Developmental genetics.

ZürichRöntgeninstitut der Universität, Strahlenbiologisches Laboratorium

See DIS 34:140.

ZürichZoologisches Institut der Eidgenössischen Technischen Hochschule

Kroeger, Heinrich. Dr. Research assistant. Chromosome metabolism; pattern formation.
Lezzi, Markus. Graduate student. Chromosome metabolism.
Müller, Melanie (Miss). Technical assistant.
Schneider, Annemarie (Mrs.). Graduate student. Cytology.
Würgler, Fritz. Graduate student. Radiation effects; mutation. Oxygen effect.
Ulrich, Hans. Dr. Professor. Differential radiation effects on nucleus and cytoplasm; oxygen effect.

ZürichZoologisches Institut der Universität

Aeppli, Lislott. Graduate student. Drosophila simulans.
Altmann, Jacques. Graduate student. Salivary glands.
Buck, Dieter. Graduate student. Imaginal discs.
Burla, Hans. Ph.D. Professor. Taxonomy, population genetics.
Chen, Pei Shen. Ph.D. Professor. Physiology and development.
Cohen, Judith. M.S. Guest. Graduate student. Developmental genetics.
Diem, Claudia. Graduate student. Enzymes.
Gloor, Regula. Ph.D. Assistant. Lethals.
Goetz, Walter. Graduate student. Inversion frequencies in Drosophila subobscura.
Grassmann, Anneliese. Graduate student. Wasp parasites of Drosophila.
Greuter, Mark. Graduate student. Release experiments with Drosophila species.
Hadorn, Ernst. Ph.D. Professor. Developmental and biochemical genetics; lethals.
Hanly, E. William. Ph.D. Research guest. Developmental and physiological genetics.
Heinsoo, Maili. M.A. Research guest. Physiological genetics.
Koch, Rudolf. Student. Dispersal rates in Drosophila species.
Laird, Charles. B.S. Guest. Graduate student. Chromosome behaviour.
Munz, Peter. Graduate student. Enzymes.
Nöthiger, Rolf. Assistant. Imaginal discs.
Novitski, Edward. Ph.D. Professor. Research guest. Chromosome mechanics; physiological genetics.
Schlöpfer, Theo. Graduate student. Imaginal discs.
Schneider, Imogene. Ph.D. Research guest. Position effect.
Weinmann, Hanspeter. Graduate student. Metabolism.
Zürcher, Christian. Graduate student. Wild type allele of ebony (e).

UNITED ARAB REPUBLICAlexandria, EgyptUniversity of Alexandria, Faculty of Agriculture

Dawood, M. M. Ph.D. Lecturer. Lethals in natural populations of Drosophila. On study leave at the Department of Genetics, University of California, Berkeley 4, U. S. A.
El-Helw, M. R. B.Sc. Graduate student. Selection, egg production and size in D. melanogaster.

Emara, R. M. B.Sc. Graduate student. Radiation and dominant lethals in natural populations of *Drosophila*.
Ibrahim, S. R. B.Sc. Graduate student. Heterosis in natural populations of *Drosophila*.
Moawad, H. B.Sc. Graduate student. Heritability under severe conditions.
Mourad, A. M. M.Sc. Graduate student. Population genetics. On study leave at the Department of Zoology, Columbia University, New York, N. Y., U. S. A.)
Rakha, F. A. B.Sc. Graduate student. Genetic variance.
Shoeb, Y. Z. Dipl. Agric. Technical assistant.
Soliman, G. A. B.Sc. Graduate student. Lethals in natural populations.
Soliman, M. H. B.Sc. Graduate student. Competition.
Tantawy, A. O. Ph.D. Associate professor and acting head of the division. Population genetics; radiation genetics and physiological genetics, studies on natural populations of *Drosophila melanogaster* and *D. simulans*.

AssuitUniversity of Assuit, Department of Genetics

See DIS 34:141.

UNITED STATESAlliance, OhioMount Union College, Department of Biology

Blount, Jerry L. Ph.D. Associate Professor. Chairman of Department. Chemical mutagenesis; longevity factors.
Savage, Ellery. Technical Assistant.

Ames, IowaIowa State University, Genetics Department

Gowen, John W. Ph.D. Professor. *Melanogaster*: crossing over, gene structure and physiological action; heterosis.
Hollander, W. F. Ph.D. Professor. General genetics.
Kloos, Wesley E. Graduate student. *Simulans*.
Stadler, Janice (Miss). Ph.D. Assistant Professor. *Melanogaster*: agents for mutations, heterosis.
Thompson, Peter E. Ph.D. Assistant Professor. *Melanogaster*: mutation.

Amherst, MassachusettsAmherst College, Department of Biology

Casey, Lucy (Mrs.). Curator of Stocks, Research Assistant.
Hexter, W. M. Ph.D. Associate Professor. Genetic fine structure and crossing over.
Ives, P. T. Ph.D. Research Associate. Radiation and population genetics.
Plough, H. H. Ph.D. Professor Emeritus. Mutation and environmental effects.
Russell, Phyllis (Mrs.). Research Assistant.
Tiffany, Barbara (Miss). Technical Assistant.
Yost, H. T. Jr. Ph.D. Associate Professor. Cell particulates and radiation effects.

Ann Arbor, MichiganThe University of Michigan, Department of Zoology

File, Sharon. Undergraduate student.
Randerson, Sherman. Graduate student.
Rizki, Rose M. Research.
Rizki, T. M. Associate Professor.

Argonne, IllinoisArgonne National Laboratory, Division of Biological and Medical Research

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Athens, GeorgiaUniversity of Georgia, Department of Zoology

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Austin 12, TexasUniversity of Texas, Department of Zoology, Genetics Foundation

Allen, Archie C. Ph.D. N. I. H. Postdoctoral Fellow. Population genetics.
Bunde, Daryl. N. I. H. Predoctoral Fellow.
Burmesiter, Maritha (Mrs.). Welch Foundation Predoctoral Fellow.
Chertkoff, Lynn (Mrs.). Research Assistant. Position effect; pseudoalleles.
Dickerman, Richard C. N. I. H. Training Grant Predoctoral Fellow.
Elequin, Flora T. M.A. N. I. H. Training Grant Predoctoral Fellow.
Fabergé, A. C. Ph.D. Research Associate. General genetics.
Forrest, H. S. Ph.D. Associate Professor. Biochemical genetics.
Futch, David G. M.A. N. I. H. Training Grant Predoctoral Fellow.
Gerstenberg, Virginia L. (Mrs.). Technical Assistant.
Judd, Burke H. Ph.D. Associate Professor. Position effect; pseudoalleles.
Lagowski, Jeanne M. (Mrs.). Ph.D. Research Associate. Biochemical genetics.
Norwood, Sharon. Technical Assistant.
Oliver, C. P. Ph.D. Professor. Gene action; human genetics.
Resch, Kathleen. Technical Assistant.
Rinehart, Robert R. N. I. H. Training Grant Predoctoral Fellow.
Stone, Wilson S. Ph.D. Professor. Evolution, gene action, radiation genetics.
Schmid, Werner. M.D. Research Associate (Switzerland). Radiation genetics, general genetics.
Wagner, Robert P. Ph.D. Professor. Gene action; biochemical genetics.
Welch, Robert M. Ph.D. Research Associate. Cytochemistry.
Wheeler, Marshall R. Ph.D. Professor. Taxonomy, evolution.
Wilson, Florence D. (Mrs.). Research Assistant. Radiation effects.

Baltimore 18, MarylandJohns Hopkins University, Department of Biology

Caples, Susan W. (Mrs.) B.A. Research Assistant. *Melanogaster*; comparative study of induced mutation in males and females.
Glass, H. Bentley. Ph.D. Professor. *Melanogaster*; population genetics of suppressor systems (erupt and tumor); gene action of su-er and su-tu; tryptophan metabolism in *Drosophila*; radiation and oxygen effects; comparative effects of mutagens on males and females at different ages.
Glass, Suzanne S. (Mrs.) M.A. Research Assistant. *Melanogaster*; genetic control of tryptophan metabolism in *Drosophila* and its relation to abnormal growth.
Laufer, Hans. Ph.D. Assistant Professor. Differential gene action during development.
Mahowald, Anthony P. B.S. Graduate student. Electron microscopy of early embryogenesis; developmental cytology of early embryonic lethals.
Marzluf, George S. B.S., M.S. Graduate student. Nature of gene action and interactions with specific suppressors; tryptophan metabolism in *D. melanogaster*.
Ritterhoff, Rebecca K. (Mrs.) B.S. Research Staff Assistant. *Melanogaster*: comparative study of induced mutation in males and females; effect of very low doses of ionizing radiation; Minutes: recessive lethals and spontaneous visibles in males and females; effects of oxygen concentration.
Ursprung, Heinrich. Ph.D. Research Associate. Imaginal discs; xanthine dehydrogenase.
Wright, Eileen Y. (Mrs.) B.A. Research Assistant. Ontogeny of gene-enzyme systems; phenogenetics of embryonic lethals.
Wright, Theodore R. F. Ph.D. Assistant Professor. Ontogeny of gene-enzyme systems; esterases and xanthine dehydrogenase; phenogenetics of embryonic lethals.

Bar Harbor, MaineJackson Memorial Laboratory

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Baton Rouge 3, LouisianaLouisiana State University, Department of Zoology

Brannon, James R. M.S. Graduate student.
Courreges, Eleanor Jane. Technical assistant.
Iyengar, Shanta V. Ph.D. Assistant Professor.
Prestridge, Martha Ann. Undergraduate research worker.

Berea, KentuckyBerea College, Department of Biology

McCune, Thomas. Undergraduate technical assistant.
Seto, Frank. Ph.D. Developmental genetics.

Berkeley, CaliforniaUniversity of California, College of Agriculture, Department of Genetics

Brown, Spencer W. Ph.D. Cytogenetics.
Dawood, M. M. Ph.D. Population genetics.
Dempster, Everett R. Ph.D. Population genetics.
Sokoloff, Alexander. Ph.D. Population genetics. Comparative genetics of Coleoptera.
Walen, Kirsten H. Ph.D. Cytogenetics.

Berkeley, CaliforniaUniversity of California, Department of Zoology

Brunt, Cole M. A.B. Laboratory Technician.
Gottlieb, Frederick. M.A. N. I. H. Predoctoral Trainee. Developmental genetics.
Hildreth, Philip. Ph.D. Research Associate. Mutation, mating behavior.
Horn, Selina. M.A. Graduate student. Sex ratio.
King, Jack. M.A. Graduate student. Developmental genetics.
Lucchesi, John. M.S. N. I. H. Predoctoral Fellow. Dosage compensation.
Mukherjee, A. M.Sc. Graduate student. Curator of stocks. Developmental genetics.
Sherwood, Eva. A.B. Research Assistant. General.
Stern, Curt. Ph.D. Professor. General.
Tokunaga, Chiyoko. Ph.D. Visiting investigator. Developmental genetics.

Bloomington, IndianaIndiana University, Department of Zoology

Barbour, Evelyn. M.A. Research Assistant.
Bart, Carol. B.S. Research Assistant.
Edmondson, Margaret (Mrs.). M.A. Graduate Investigator.
Meyer, Helen Unger (Mrs.). Ph.D. Research Associate.
Muller, H. J. D.Sc. Professor.
Oster, Irwin I. Ph.D. Consultant. (Permanent address: Institute for Cancer Research, Philadelphia, Pennsylvania)
Thomas, Sandra (Mrs.). A.B. Research Assistant.
Trout, William E. A.B. Predoctoral N. I. H. Fellow.
Wagoner, Dale E. A.B. Predoctoral N. I. H. Fellow.
Zimmering, Stanley. Ph.D. Research Executive.

Buffalo 14, New YorkUniversity of Buffalo, Department of Biology

Farnsworth, Marjorie W. Ph.D. Lecturer and Research Associate. Melanogaster developmental genetics and biochemistry.
Goldin, Herbert. A.B. Graduate student.
Luchowski, Elizabeth (Mrs.). A.B. Research technician.
Trenor, Katherine. A.B. Graduate student.

Cambridge 38, Massachusetts
Harvard University, The Biological Laboratories

Emrich, Nancy (Mrs.). Research Assistant. *Melanogaster*.
Jonsson, Ulla-Britt (Miss). Senior Research Assistant. Curator of Stocks. Mutation and fertility in *melanogaster*.
Lefevre, George. Ph.D. Radiation genetics; mating behavior in *melanogaster*.
Levine, R. Paul. Ph.D. Mutation and gene action.
Rose, Barbara (Miss). Research Assistant. *Melanogaster*.

Cambridge, Massachusetts
Massachusetts Institute of Technology

See DIS 34:144.

Chambersburg, Pennsylvania
Wilson College

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Chapel Hill, North Carolina
University of North Carolina, Medical School, Department of Biochemistry

Glassman, Edward. Ph.D. Biochemical genetics.
Hodge, Lon. D.V.M. Biochemical genetics.
Karam, J. A.B. Biochemical genetics.
Keller, E. C. Jr. Ph.D. Biochemical genetics.
McLean, Janice. B.S. Research Laboratory Supervisor.
Moore, R. Laboratory Assistant.
Parish, J. Laboratory Assistant.
Yen, Terrence T. A.B. Biochemical genetics.

Chapel Hill, North Carolina
University of North Carolina, Department of Zoology

Henderson, Ann S. Graduate Assistant.
Hubbard, William B. M.Ed. Predoctoral Fellow.
Kiesselbach, Theodore H. Honors Student.
James, Judy McNease (Mrs. Wm. S.). A.B. Research Assistant.
Price, Mary Jane (Mrs. Robt. E., Jr.). Research Assistant.
Wall, Lynn. A.B. Research Assistant.
Whittinghill, Maurice. Ph.D. Professor. Irradiation; chemical mutagens; crossing over.

Chicago 11, Illinois
Loyola University, College of Arts and Sciences

Arnold, Lloyd L. Ph.D. Aging.
Peters, Walter, Rev. S. J. Ph.D. Population studies.

Chicago 37, Illinois
University of Chicago, Department of Zoology

Baker, William K. Ph.D. Professor. Position effect, developmental genetics, mutation.
Batt, Murray. Research Assistant.
Gersh, Eileen Sutton. Ph.D. Research Associate. Cytogenetics of *melanogaster*.
Hubby, Jack L. Ph.D. Instructor. Biochemical genetics.
Roberts, Paul A. M.D. Graduate student. Nondisjunction, developmental genetics.
Sieux, Mrs. Joseph. M.Ed. Curator of stocks.
Sims, Maureen (Miss). B.S. Graduate student.
Spieler, Richard A. B.A. Graduate student. Nondisjunction, evolution.
Spofford, Janice B. Ph.D. Research Associate. Parental effects on phenotype.
Throckmorton, Lynn H. (Mr.) Ph.D. Research Associate. Pteridine metabolism and protein differences in *Drosophila*, general Dipteran and *Drosophila* taxonomy.

Cleveland 15, Ohio
Fenn College, Department of Biology

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Cleveland 15, Ohio
Western Reserve University, Biological Laboratory

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Cold Spring Harbor, New York
Carnegie Institution of Washington, Department of Genetics

Buchanan, Jennie (Mrs. Paul). Research Assistant, Curator of Stocks.
Das, C. C. Ph.D. Guest Investigator (on leave from Allahabad University, Allahabad, India). Cytogenetics.
Gay, Helen. Ph.D. Associate Cytogeneticist. *Melanogaster*: chromosome organization, nuclear-cytoplasmic relations; electron microscopy, histochemistry.
Gillies, Gloria (Mrs.). Research Assistant.
Kaufmann, B. P. Ph.D. Acting Director. *Melanogaster*: cytology, cytochemistry.
Weingart, E. Ann. B.A. Research Assistant.

Cold Spring Harbor, New York
Long Island Biological Association, Biological Laboratory

Chovnick, Arthur. Ph.D. Laboratory Director. Gene structure and function.
Kernaghan, R. Peter. M.A. Research Assistant. Graduate student. Gene structure and function.
Krauss, Marian (Miss). B.S. Research Assistant.
Prokop, Barbara (Miss). B.S. Research Assistant. Curator of Stocks.
Schalet, Abraham. Ph.D. Investigator. Mutation and gene structure.
Talsma, Joy (Mrs.) M.A. Research Assistant.
Taylor, Albert. Technical Assistant.

Columbus, Ohio
Ohio State University, Department of Zoology and Entomology

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Corvallis, Oregon
Oregon State University, Department of Zoology

Heath, Gloria (Mrs.) Student.
Mohler, J. D. Ph.D. Associate Professor.
Neeley, John R. B.S. Graduate student.
Smith, Sheila (Mrs.). Assistant in Zoology.
Thompson, Steven R. B.S. Graduate student.

Davis, California
University of California, Department of Genetics

Bowman, J. T. B.S. N. I. H. Predoctoral Fellow.
Eggert, J. B.S. Laboratory Technician.
Geer, B. W. M.S. Research Fellow.
Green, M. M. Ph.D. Professor.

Dayton 9, Ohio
University of Dayton, Department of Biology

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DeKalb, IllinoisNorthern Illinois University, Department of Biological Sciences

Bennett, Jack. Ph.D. Assistant Professor. Selection, insecticide resistance, populations.
Bennett, Katherine Wilson. B.A. Cytogenetics.
Capek, Ronald. B.S. Graduate student. Selection, behavior.
Gianopoulos, Harold W. B.S. Graduate student. Selection, insecticide resistance.
Hampel, Arnold. Research Assistant. Radiation.
Harmon, Charles E. Research Assistant. Radiation.
Landy, Ronald. B.S. Graduate student. Wild population.
Le Blanc, Wayne. B.A. Graduate student. Research Assistant. Mutagenesis, radiation.
Monkman, Marie (Mrs.). B.S. Graduate student. Research Technician.
Martin, Robert J. B.S. Graduate student. Research Assistant. Radiation.
Mittler, Sidney. Ph.D. Professor. Mutagenesis.
Weideman, Jeannine. Research Assistant.
Wei, Irene Y. L. Undergraduate student. (Summer N. S. F. Undergraduate Research Participant) Research Technician.
Wu, Ching-kuei. B.S. Graduate student. Populations.

Duarte, CaliforniaCity of Hope Medical Center, Department of Genetics

Brawley, Mary Anne. Stockkeeper.
Gugler, David H. Research Technician.
Kaplan, William D. Ph.D. Mutagenesis, cytology.
Tanaka, Tatsuya. Ph.D. Cytology.

Durham, North CarolinaDuke University, Department of Zoology

Bird, Margaret Ann (Miss). B.A. Research Assistant.
Burnham, Deborah (Miss). Research Assistant.
Ward, Calvin L. Ph.D. Associate Professor.

East Lansing, MichiganMichigan State University, Department of Biochemistry

Burnett, Jean B. Ph.D. Research Associate.
Bernhard, Karen L. Technician.
Fox, Allen S. Ph.D. Professor.
Fuchs, Morton S. M.S. Graduate Research Assistant.
Kan, James L. Ph.D. N. I. H. Postdoctoral Fellow.
Kang, Suk Hee. B.S. Graduate Research Assistant.
Kapetan, Anne S. Technician.
Parzen, Sheldon D. B.S. Graduate Research Assistant.
Yoon, Sei Byung. Ph.D. Research Associate.

East Lansing, MichiganMichigan State University, Department of Zoology

Camp, Herbert L. Technician.
DeVries, JoAnne K. Graduate student.
Myszewski, Michael E. Graduate student.
Nugent, Karen L. Technician.
Seaton, Robert K. N. S. F. Research Participant.
Stanich, Gloria J. N. S. F. Research Participant.
Trosko, James E. N. D. E. A. Predoctoral Fellow.
Yanders, Armon F. Associate Professor. Radiation effects; mutagenesis; fertilization.

Eugene, OregonUniversity of Oregon, Department of Biology

Clancy, C. W. Ph.D. Professor. Developmental genetics.
Dorsey, R. Graduate student. Statistician.
Ehrlich, Elizabeth (Mrs.). Research Assistant Adj. Characteristics of sex-linked lethals.
Erickson, J. M.S. Instructor. Meiotic drive.
Farhang, M. Helper.
Foster, T. B.S. Graduate student. Mutations and chromosomal aberrations.
Hamilton, J. (Miss) Technician. Tandem metacentrics.
Johnson, R. B.S. Graduate student. X-linked non-autonomous lethals.
Landenberger, M. (Mrs.) B.S. Research Assistant.
Masterson, J. M.S. Research Assistant. Developmental genetics.
Mickel, S. (Miss) Undergraduate Research Participant. Statistician.
Novitski, E. Ph.D. Professor. (On leave at Zoologisches der Universität, Zürich, Switzerland)
Parker, D. M. (Mrs.) Research Assistant Adj. D. simulans.
Teviotdale, F. (Miss) B.S. Graduate student.

Evanston, IllinoisNorthwestern University

Brown, Edward H. B.A. N. I. H. Predoctoral Fellow. *Melanogaster*, sex determination and differentiation.
Butterworth, Francis M. B.A. Graduate student. *Melanogaster*, cytochemistry and ultrastructure of the fat body.
Falk, Gretchen J. B.A. Graduate student. Autoradiography.
Green, Christopher C. Undergraduate research student.
King, Robert C. Ph.D. Associate Professor. *Melanogaster* oögenesis.
Koch, Elizabeth A. B.S. Graduate student. *Melanogaster*, ultrastructure of fcs ovaries.
Mills, Richard P. Undergraduate. *Willistoni*, ultrastructure.
Pakeltis, Helen. B.S. Curator of stocks.
Smith, Patricia A. B.S. Graduate student.

Fayetteville, ArkansasUniversity of Arkansas, Department of Zoology

Bryniarski, Teresa. Research assistant.
Clayton, Frances E. Ph.D. Associate Professor. Radiation effects; development.
Halpern, Lynda S. (Mrs.) B.S. Graduate assistant. Radiation effects.

Gainesville, FloridaUniversity of Florida, Department of Biology

Wallbrunn, Henry M. Mutation rates, population genetics.

Harrisonburg, VirginiaEastern Mennonite College, Department of Biology

Jacobs, M. E. Ph.D. Professor. Melanism.

Hiram, OhioHiram College, Department of Biology

Friedman, Lawrence D. Ph.D. Assistant Professor. General genetics.

Houston, TexasRice University, Department of Biology

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Houston, TexasUniversity of Texas, M. D. Anderson Hospital and Tumor Institute, Department of Biology

Alexander, Mary L. Ph.D. Radiation; population genetics.
Bergendahl, Janet (Mrs.). M.A. Research Assistant.
Duval, Donya (Miss). B.A. Research Technician II.
Haas, Felix L. Ph.D. Radiation; biochemical genetics.
McKinley, Kay (Miss). B.A. Research Technician II.

Iowa City, IowaUniversity of Iowa, Department of Zoology

Brosseau, George E., Jr. Ph.D. Assistant Professor. *Melanogaster*: genetics of the Y chromosome.
LeVier, Robert L. Undergraduate student assistant.
Gilmore, G. Thomas. Undergraduate student assistant.

Ithaca, New YorkCornell University, Plant Breeding Department

Baumann, James L. Graduate Research Assistant.
Everett, Herbert L. Ph.D. Associate Professor. General genetics.
Gutenmann, Hilda (Mrs.). Research Assistant.
Loomis, Margaret (Mrs.). Technical Assistant.
Myers, Oval. Graduate Teaching Assistant.
Sanderson, K. E. Research Associate. General genetics.
Schafrik, Carol. Graduate Teaching Assistant.
Silberman, June (Mrs.). Research Assistant.
Suska, Jadwiga (Mrs.). Research Assistant.
Thompson, Margaret Emmerling. Ph.D. Assistant Professor. General genetics.
Vanoucek, E. G. Graduate Research Assistant.
Wallace, Bruce. Ph.D. Professor. Population studies.

Jamaica, New YorkSt. John's University, Department of Biology, Graduate School

Fuscaldo, Kathryn E. Ph.D. Assistant Professor. Biochemical genetics.
Siracusano, Vincent C. Graduate Research Assistant. Biochemical genetics.
Gonnella, Victoria M. Graduate Research Assistant. Immunogenetics.

Johnson City, TennesseeEast Tennessee State College, Department of Biology

Perry, Thomas L. M.A. Research associate. Biotic potential.
Stevenson, Richard. M.A. Professor. Population genetics, speciation.

Lafayette, IndianaPurdue University, Department of Biological Sciences

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Lafayette, IndianaPurdue University, Population Genetics Institute

Bartlett, A. C. M.S. Instructor. Radiation genetics.
Bell, A. E. Ph.D. Professor. Population genetics, selection, G x E interactions.
Bhat, P. N. M.S. Graduate Assistant. Population genetics.
Englert, D. C. M.S. Graduate fellow. Population genetics.
Hardin, R. T. M.S. Graduate Research Assistant. G x E interactions.
Krause, Eliot. B.S. Graduate Assistant. Population genetics.
Pare, J. P. M.S. Graduate fellow. Selection methods.

Shideler, Doris (Mrs.). Research assistant.
Yamada, Yukio. Ph.D. Assistant Professor. Population genetics. G x E interactions.

Lawrence, Kansas

University of Kansas, Department of Entomology

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Le Mars, Iowa

Westmar College, Department of Biology

Divelbiss, J. E. Ph.D. Assistant Professor. Complex loci, red eye pigments.

Lexington, Kentucky

University of Kentucky, Department of Zoology

Carpenter, John M. Ph.D. Professor and Department Head. Seasonal fluctuations of *Drosophila* in relation to wild yeast populations, reproductive potential, gene ecology.

Gilliland, Karen P. Student Assistant.

Semp, Bernard A. Graduate Assistant.

Stewart, Walter H. Graduate Assistant.

Lincoln, Nebraska

The University of Nebraska, Department of Zoology

Lund, Douglas E. Graduate student. *D. obscura* group CO₂ sensitivity

Miller, Dwight D. Professor.

Stone, Larrie E. Graduate student. *D. affinis* cytology.

Sulerud, Ralph L. Graduate student. *D. melanogaster* CO₂ sensitivity.

Logan, Utah

Utah State University, Department of Zoology

Barber, Richard T. B.S. Research Assistant. *Melanogaster*: development of head abnormalities.

Edwards, James W. M.S. N. D. E. A. Fellow. *Melanogaster*: eye mutations.

Egbert, Larre N. B.S. N. I. H. Fellow. Biometrical genetics.

Gardner, Eldon J. Ph.D. Professor. *Melanogaster*: mutants of the head region.

Hansen, Afton M. Ph.D. N. S. F. Fellow. *Melanogaster*: eye mutants (after January 1, 1962, Snow College, Ephraim, Utah).

Hawkes, N. Roger. B.S. Research Assistant. *Melanogaster*: influence of nutrients and drugs on head abnormalities.

Johnson, George R. M.A. Research Assistant. *Melanogaster*: population studies on genes related to maternal effects.

Simmons, John R. Ph.D. Assistant Professor. *Melanogaster*: biochemical genetics.

Sorensen, William K. B.S. Graduate student. *Melanogaster*: development of head abnormalities.

Los Angeles, California

University of California, Department of Botany

Ball, Francis M. B.S. Technical Assistant. *Pseudoobscura*: mutants.

De Young, Patricia. Laboratory Assistant.

Epling, Carl C. Ph.D. Professor. *Pseudoobscura*: population genetics.

Kato, Stephen. Undergraduate Technical Assistant.

Mayhew, Stephen. Undergraduate Technical Assistant.

McCullough, Marilyn. Undergraduate Technical Assistant.

Whitesel, Barbara. Laboratory Assistant.

Los Angeles, California

University of California, Department of Zoology

Carlson, Elof A. Ph.D. Assistant Professor. The dumpy locus; comparative mutagenesis.

Corwin, Harry. B.A. Graduate student.
Falk, Peter. Student assistant, University High School.
Hawkins, Evelyn. Technical assistant.
Hendrickson, Robert. B.A. Graduate student.
Phillips, Barry. B.A. Summer investigator from Queen's University, Canada.
Phillips, Claire. B.A. Stock-keeper and research assistant.
Sederoff, Ronald. B.A. Graduate student.
Southin, John. B.Sc. Graduate student. Jane Selby Jacobson Fellow.

Madison, Wisconsin

University of Wisconsin, Departments of Genetics and Medical Genetics and Zoology

Abrahamson, Seymour. Ph.D. Assistant Professor. Radiation genetics.
Baumiller, Robert. S.J., Ph.D. Post-doctoral Fellow of the National Foundation.
Chung, Yong Jai. B.S. Graduate student.
Coifman, Robert. B.E.P. Graduate student.
Crow, James F. Ph.D. Professor.
Davis, Brian. B.A. Research Assistant.
Greenberg, Rayla (Miss). M.S. Graduate student.
Lux, Edith (Mrs.). Project Assistant.
Maruyama, Takeo. M.S. Graduate student.
Mattson, Thomas. B.A. Graduate student.
Rosenfeld, Averil (Mrs.). B.S. Project Assistant.
Sandler, L. Ph.D. Assistant Professor.
Thomas, Constance (Miss). M.S. Project Assistant.
Voynow, Nancy (Mrs.). B.A. Research Assistant.

Minneapolis 14, Minnesota

University of Minnesota, Departments of Zoology and Animal Husbandry

See DIS 34:150.

Moscow, Idaho

University of Idaho, Department of Biological Sciences

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Newark, New Jersey

Rutgers, The State University, 40 Rector Street

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New Haven 11, Connecticut

Albertus Magnus College, Department of Biology

Cullen, Sister Mary Urban. O.P., Ph.D. Professor. Developmental genetics.

New Haven, Connecticut

Yale University, Department of Zoology

Counce, Sheila J. (Mrs. R. Bruce Nicklas) Ph.D. Research Associate. Developmental genetics, experimental embryology.
Doane, Winifred W. (Mrs.) Ph.D. N. I. H. Post-doctoral Trainee. Developmental genetics, insect physiology.
Gill, Kulbir Singh. Ph.D. N. I. H. Post-doctoral Trainee. Developmental genetics.
Grabicki, Eugenia (Mrs.). Curator of Stocks and Technician.
Hadler, Norton. Undergraduate. Population genetics of phototaxes.
Jura, Czeslaw. Ph.D. Fellow of the Rockefeller Foundation. Insect embryology.
(On leave from Department of Zoology, Jagiellonian University, Cracow, Poland, until September, 1962.)
Leventhal, Elaine (Mrs.). M.S. N. I. H. Pre-doctoral Trainee. Developmental genetics and cytology.

Maxim, Peter. Undergraduate. N. S. F. Undergraduate Research Program. Population genetics.
Mills, Richard P. Undergraduate. Developmental genetics, heritable infections.
Nicklas, R. Bruce. Ph.D. Assistant Professor. Cytology of Diptera.
Passano, Kari Nordback (Mrs.). Cand. Real. Guest.
Poulson, D. F. Ph.D. Professor. Physiological and developmental genetics, hereditary infection.
Rosner, J. L. B.S. Graduate Teaching Assistant. Microbial genetics.
Williamson, D. L. Ph.D. N. I. H. Post-doctoral Trainee. CO₂-sensitivity, hereditary infections.

New York 27, New York
Columbia University, Department of Zoology

Barker, J. S. F. Ph.D. (University of Sydney, Australia) Fullbright Fellow. Interspecific competition.
Carmody, George. Graduate student. Reproductive isolating mechanisms.
Dobzhansky, Th. Professor. Population genetics: pseudoobscura, persimilis, willistoni, prosaltans, and other species.
Ehrman, Lee. Ph.D. Paulistorum: population genetics.
Kessler, Sydney. Graduate student. Reproductive isolating mechanisms.
King, James C. Ph.D. Research Associate. Population genetics: developmental aspects.
Levine, Louis. Ph.D. Research Fellow. Pseudoobscura: laboratory populations, heterosis.
Malogolowkin, Chana. Ph.D. (Universidade Nacional do Brasil) Paulistorum, cytoplasmic sex-ratio.
Mishara, Joan. Graduate student. Population genetics.
Mourad, Abd el Khalik. Graduate student. (University of Alexandria, Egypt) Population genetics and radiation.
Pavlovsky, O. A. Research Assistant. Cytology; population genetics.
Polivanov, Sergei. Graduate student. Population genetics.
Sankaranarayan, Krishna. Graduate student. (Annamalai University, India) Population genetics.
Solima, Angela. Ph.D. (University of Naples, Italy) Population genetics.
Spassky, Boris. Research Associate. Comparative genetics of species.
Spassky, N. P. (Mrs.) Research Assistant. Population genetics.
Strickberger, Monroe. Graduate student. Population genetics.
Tidwell, Thomas. Graduate student. Reproductive isolating mechanisms.
Van Valen, Leigh. Ph.D. Population genetics; Drosophila and fossils.
Weisbrot, David. Graduate student. Melanogaster, simulans.

New York 21, New York
The Rockefeller Institute

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Norman, Oklahoma
University of Oklahoma, Department of Zoology

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Notre Dame, Indiana
University of Notre Dame, Department of Biology

Bender, Harvey A. Ph.D. Assistant Professor. Developmental genetics.
Craig, George B. Ph.D. Associate Professor. Population genetics (Aedes).
Moskewski, Theresa (Miss). Curator of Stocks, Technician.
Rai, Karamjit S. Ph.D. Postdoctoral Fellow. Cytogenetics (Aedes).

Oak Ridge, TennesseeOak Ridge National Laboratory, Biology Division, P. O. Box Y

Grell, E. H. Ph.D. Chromosome behavior and biochemical genetics.
Grell, Rhoda F. Ph.D. Chromosome behavior.
Lindsley, Dan L. Ph.D. Chromosome behavior and radiation genetics.
Mead, Charles G. Ph.D. Biochemistry of *Drosophila* nucleic acids.
Petty, John. B.S. Research assistant.
Pratt, Guthrie T. (Mrs.). M.S. Research assistant.
Scandlyn, Bobbie J. (Miss) B.S. Research assistant.
Suzuki, David T. Ph.D. Chromosome behavior.
von Borstel, R. C. Ph.D. Radiation genetics.
Von Halle, Elizabeth S. (Mrs.) B.A. Research consultant.
Welshons, William J. Ph.D. Pseudoallelism.
Wilkerson, Ruby D. (Mrs.) Curator of stocks.
Wolff, Sheldon. Ph.D. Radiation genetics.

Pasadena, CaliforniaCalifornia Institute of Technology, Division of Biology

Del Campo, Gladys. B.S. Research Assistant.
Kiger, John. Student.
Lewis, E. B. Ph.D. Professor.
Markowitz, E. Student.
Mitchell, Annamarie (Mrs.) Dipl. Lab.
Mitchell, H. K. Ph.D. Professor.
Mora, Sergio. M.S. Curator of Stocks.
Seecof, R. L. Ph.D. Research Fellow.
Sturtevant, A. H. Ph.D. Professor.

Philadelphia 11, PennsylvaniaThe Institute for Cancer Research, Fox Chase, Division of Chemotherapy

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Philadelphia 11, PennsylvaniaThe Institute for Cancer Research, Fox Chase, Department of Genetics and Cytochemistry

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Philadelphia 22, PennsylvaniaTemple University, Department of Biology

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Philadelphia 29, PennsylvaniaWoman's Medical College, Department of Anatomy

Campbell, Shirley. Cytology Assistant.
Levitan, Max. Ph.D. Associate Professor. Population genetics.
Rybachock, Rosemary. Cytology Assistant.
Schiller, Ruth. Research Assistant.
White, Susan. Technical Assistant.

Pittsburgh 13, PennsylvaniaUniversity of Pittsburgh, Department of Biological Sciences

Carver, James E., Jr. M.S. Graduate student. Research Assistant. Lethals in *melanogaster* populations.
Langer, Bozena (Mrs.). Ph.D. Research Associate. Mating propensity, persimilis.
Spiess, Eliot B. Ph.D. Associate Professor. Population genetics.
Spiess, Loretta D. (Mrs.) Ph.D. Research Associate. Population genetics.
Sweet, Edward E. Ph.D. Research Associate. Sterility in populations.

Portland, OregonReed College, Department of Biology

See DIS 34:152.

Pullman, WashingtonWashington State University, Department of Zoology

Moree, Ray. Ph.D. Associate Professor. Population genetics.

Hudson, James E. Student.

Raleigh, North CarolinaNorth Carolina State College, Department of Genetics

Brown, J. C. (Mrs.) Research Assistant.

Bruck, David. Graduate Research Assistant.

Collins, F. P. (Mrs.) Research Assistant.

Council, S. B. (Mrs.) Research Assistant.

Dobie, N. B. (Mrs.) Research Assistant (Stockkeeper).

Dyson, J. G. (Mrs.) Graduate Research Assistant.

Kojima, Ken-ichi. Ph.D. Experimental and theoretical population genetics; quantitative genetics.

Mettler, Lawrence E. Ph.D. Experimental population genetics; cytogenetics.

Richardson, R. H. N. S. F. Cooperative Fellow (Graduate Research Assistant).

Schaffer, H. E. N. D. E. A. Predoctoral Fellow.

Wing, M. S. (Mrs.) Research Assistant.

Richmond 19, VirginiaMedical College of Virginia, Department of Biology and Genetics

Bridges, Elizabeth P. B.S. Research Assistant.

Hughes, Roscoe D. Ph.D. Professor. Cytogenetics.

Townsend, J. Ives. Ph.D. Assistant Professor. Population genetics; marginal populations.

Ridgefield, ConnecticutNew England Institute for Medical Research

Freeborn, John. Technical Assistant.

Mahler, Marilyn (Mrs.). B.A. Research Assistant.

Mickey, George H. Ph.D. Cytogeneticist. Mutations.

Sondhi, K. C. Ph.D. Geneticist. Developmental and quantitative genetics.

Sondhi, Gunthild (Mrs.). Technician.

Riverside, CaliforniaUniversity of California, Department of Biology

See DIS 34:153.

Rochester 20, New YorkUniversity of Rochester, Department of Biology

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St. Louis, MissouriSaint Louis University, Department of Biology

See DIS 34:153.

St. Louis, MissouriWashington University, Department of Zoology

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Salt Lake City, UtahUniversity of Utah, Department of Genetics

Hanks, George D. Ph.D. Population genetics; meiotic drive.
Hochman, Benjamin. Ph.D. Population genetics; isoalleles, lethals.
Prows, Ronald. B.S. Technician.
Wrathall, C. Richard. Student.

Salt Lake City, UtahUniversity of Utah, College of Medicine, Department of Surgery

Burdette, Walter J. Ph.D., M.D. Professor and Head, Department of Surgery.
Anderson, Ruth. Ph.D. Research Associate.
Mukherjee, Barid B. Ph.D. Research Associate.
Pilgrim, H. Ira. Ph.D. Research Assistant Professor.
Anderson, Betty. Laboratory technician.
Baumgart, Gerda Isolde. Laboratory technician.
Bigelow, Robert R. Laboratory technician.
Hegewald, Eva. Laboratory technician.
Hegewald, Rudolph J. Laboratory technician.
Janke, Hannelore. Laboratory technician.
Nomura, Koji. Laboratory technician.
Paul, Lloyd A. Laboratory technician.
Pilar, Beatriz M. M. Laboratory technician.
Steinitz, John. Diener.
Stratopoulos, George. Laboratory technician.
Thomas, Carol B. Research Assistant.

San Diego, CaliforniaSan Diego State College, Department of Zoology

Johns, Ruth E. B.A. Graduate student.
Lovellette, Edward J. B.A. Graduate student.
Ratty, Frank J. Ph.D. Associate Professor.

Staten Island 1, New YorkWagner College

Annan, Murvel E. Ph.D.
Reitan, Phillip J. Ph.D. Drosophila development.

Storrs, ConnecticutUniversity of Connecticut, Department of Zoology and Entomology

Brown, William P. Ph.D. Population genetics.

Syracuse 10, New YorkSyracuse University, Department of Zoology and Division of Science Teaching

Milkman, Roger D. Ph.D. Associate Professor. Drosophila genetics: population, developmental, and physiological.
Phillips, Donald. B.A. Research Assistant.
Petersen, Kathleen L. (Mrs.) B.A. Graduate student. Funebris.
Druger, Marvin. Ph.D. Assistant Professor. Population genetics.
Collette, Alfred T. Ph.D. Professor. Virilis.

Tallahassee, FloridaFlorida State University, Department of Biological Sciences

Edington, C. W. Ph.D. Radiation genetics.
Epler, J. L. M.S. Radiation genetics and chemical mutagenesis.

Tucson, ArizonaUniversity of Arizona, Department of Zoology

See DIS 34:154.

University, AlabamaUniversity of Alabama, College of Arts and Sciences, Department of Biology

Guest, William C. Ph.D. Assistant Professor. Cytogenetics.

University Park, PennsylvaniaPennsylvania State University, Buckhout Laboratory

Come, Thomas V. M.A. Graduate student.

Grun, Paul. Ph.D. Associate Professor. Cytogenetics.

Nash, Donald J. Ph.D. Assistant Professor. Population genetics.

Upton, New YorkBrookhaven National Laboratory, Department of Biology

See DIS 34:155.

Urbana, IllinoisUniversity of Illinois, Department of Psychology, Behavior Genetics Laboratory

Hirsch, Jerry. Ph.D. Associate Professor. Behavior genetics.

Hosteller, Roy C. B.A. Research Assistant.

Urbana, IllinoisUniversity of Illinois, Department of Zoology

Luce, Wilbur M. Ph.D. Professor. Bar series; effect of environmental agents; radiation; effect of chemicals; physiological genetics.

Olson, John B. B.S. Research Assistant. Curator of stocks. Location of mutants.

Tanaka, Eiji. New address: Department of Biology, University of Oraka Prefecture, Sakai, Japan.

Washington 25, D. C.National Science Foundation, Genetic Biology Program

See DIS 34:155.

Wellesley 81, MassachusettsWellesley College, Department of Zoology and Physiology

Bull, Alice Louise. Ph.D. Assistant Professor. Developmental genetics.

Wilson, Louise Palmer. Ph.D. Professor. Melanogaster: physiology of growth; emphasis on tumors.

Alphabetical

Abrahamson, S. Madison, Wis.
Abro, A. Norway, Bergen
Aeppli, L. Switzerland, Zürich
Akita, Y. Japan, Tokyo
Alcobé, S. Spain, Barcelona
Alderson, T. Gr. Britain, Cambridge
Alexander, M. Houston, Texas
Allan, J. Gr. Britain, Edinburgh
Allen, A.C. Austin, Texas
Altenburg, E. see DIS 34:147
Altmann, J. Switzerland, Zürich
Anderson, B. see DIS 34:128
Anderson, Betty Salt Lake City, Utah
Anderson, F. see DIS 34:151
Anderson, J. see DIS 34:150
Anderson, R. Salt Lake City, Utah
Anderson, W. see DIS 34:142
Angus, D. Australia, Brisbane
Annan, M. Staten Island, New York
Antochevizky, N. Brazil, Porto Alegre
Apitzsch, U. Germany, Karlsruhe
Arabia, L. Italy, Rome
Arnold, L. Chicago, Illinois
Aronson, M. see DIS 34:153
Auerbach, C. Gr. Britain, Edinburgh

Bairati, A. Italy, Milano
Baker, W. Chicago, Illinois
Ball, F. Los Angeles, California
Band, H. Canada, Vancouver
Banerjee, S. see DIS 34:133
Barak, E. Isreal, Jerusalem
Barber, R. Logan, Utah
Barbour, E. Bloomington, Indiana
Barigozzi, C. Italy, Milano
Barker, J. New York, New York
Bart, C. Bloomington, Indiana
Bartelt, J. Germany, Berlin-Dahlem
Bartlett, A. Lafayette, Indiana
Basden, E. Gr. Britain, Edinburgh
Basile, R. Brazil, São Paulo
Bateman, A. see DIS 34:133
Batt, M. Chicago, Illinois
Batten, J. Gr. Britain, Cambridge
Baumann, J. Ithaca, New York
Baumgart, G. Salt Lake City, Utah
Baumiller, R. Madison, Wisconsin
Beardmore, J. Netherlands, Groningen
Becker, G. Germany, Marbugh/Lahn
Becker, H. Germany, Marbugh/Lahn
Beermann, W. Germany, Tübingen
Beggs, C. see DIS 34:131
Belitz, H. Germany, Berlin-Dahlem
Bell, A. Lafayette, Indiana
Bender, E. Germany, Berlin-Buch
Bender, H. Notre Dame, Indiana
Bennett, J. Dekalb, Illinois
Bennett, K. Dekalb, Illinois
Bentvelzen, P. Netherlands, Leiden
Berendes, H. Netherlands, Leiden
Bergendahl, J. Houston, Texas
Bergerard, J. France, Gif-sur-Yvette
Bernard, J. France, Gif-sur-Yvette
Bernhard, K. East Lansing, Michigan
Bert, G. see DIS 34:153
Bhat, P. Lafayette, Indiana
Bigelow, R. Salt Lake City, Utah
Bigler, J. France, Gif-sur-Yvette
Bird, M. Durham, North Carolina
Bito, J. Japan, Anzyo-Shi
Blair, J. Ghana, Legon
Blair, P. see DIS 34:148
Blake, P. see DIS 34:154
Blout, J. Alliance, Ohio
Blum, S. Isreal, Jerusalem
Boam, T. Gr. Britain, Sheffield
Bochnig, V. Germany, Berlin-Dahlem
Bondreu, C. see DIS 34:153
Bösiger, E. France, Gif-sur-Yvette
Bowman, J. Davis, California
Brannon, J. Baton Rouge, La.
Braver, G. see DIS 34:150
Braver, N. see DIS 34:150
Brawley, M. Duarte, California
Bregliano, J. France, Gif-sur-Yvette
Breuer, M. Brazil, São Paulo
Bridges, E. Richmond, Virginia
Brink, N. Australia, Hobart
Brinkmann, A. Norway, Bergen
Brncic, D. Chile, Santiago
Brosseau, G. Iowa City, Iowa
Brown, E. Evanston, Illinois
Brown, J. Raleigh, North Carolina
Brown, S. Berkeley, California
Brown, W. Storrs, Connecticut
Browning, L. see DIS 34:147
Bruck, D. Raleigh, North Carolina
Brun, G. France, Gif-sur-Yvette
Brun, J. France, Lyon
Brunt, C. Berkeley, California
Bryniarski, T. Fayetteville, Arkansas
Buchanan, J. Cold Spring Harbor, N.Y.
Buck, D. Switzerland, Zürich
Bull, A. Wellesley, Massachusetts
Bull, S. Australia, Sydney
Bult, P. Netherlands, Groningen
Bunch, A. see DIS 34:148
Bunde, D. Austin, Texas
Bunker, M. see DIS 34:143
Burdette, W. Salt Lake City, Utah
Burdick, A. see DIS 34:148
Burger, C. see DIS 34:144
Burla, H. Switzerland, Zürich
Burmester, M. Austin, Texas
Burnet, B. Gr. Britain, Edinburgh
Burnett, J. East Lansing, Michigan
Burnham, D. Durham, North Carolina
Butler, L. Canada, Toronto
Butterworth, F. Evanston, Illinois
Buzzati-Traverso, A. see DIS 34:134

Cacheiro, N. Argentina, Buenos Aires
Cama, J. Spain, Barcelona
Camba, C. Brazil, São Paulo
Camp, H. East Lansing, Michigan
Campbell, R. Australia, Hobart

- Campbell, S. Philadelphia, Penn.
Canuti, N. Italy, Rome
Capek, R. Dekalb, Illinois
Caples, S. Baltimore, Maryland
Carfagna, M. see DIS 34:134
Carlson, E. Los Angeles, California
Carlson, J. see DIS 34:145
Carmody, G. New York, New York
Carpenter, J. Lexington, Kentucky
Carson, H. see DIS 34:153
Carver, J. Pittsburgh, Pennsylvania
Casanova, A. Chile, Santiago
Casey, L. Amherst, Massachusetts
Caster, J. see DIS 34:153
Castiglioni, M. Italy, Milano
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Catsch, A. Germany, Karlsruhe
Cestari, A. Brazil, São Paulo
Chandley, A. see DIS 34:133
Chang, S. see DIS 34:137
Chejne, A. Colombia, Bogotá
Chen, P. Switzerland, Zürich
Chertkoff, L. Austin, Texas
Chiquisa, S. Japan, Misima
Chiscon, J. see DIS 34:148
Choi, J. Korea, Seoul
Chovnick, A. Cold Spring Harbor, N.Y.
Chun, W. Korea, Seoul
Chung, J. Korea, Seoul
Chung, O. Korea, Seoul
Chung, Y. Madison, Wisconsin
Gioffi, E. see DIS 34:134
Gividalli, L. Israel, Jerusalem
Glancy, C. Eugene, Oregon
Clark, A. Australia, Hobart
Clark, E. Australia, Hobart
Clarke, J. Gr. Britain, London
Clayton, F. Fayetteville, Arkansas
Clayton, G. Gr. Britain, Edinburgh
Clise, R. see DIS 34:145
Cohen, B. Canada, Vancouver
Cohen, J. Switzerland, Zürich
Coifman, R. Madison, Wisconsin
Collins, F. Raleigh, North Carolina
Cole, K. Canada, Vancouver
Collette, A. Syracuse, New York
Come, T. University Park, Penn.
Comstock, R. see DIS 34:150
Cooke, P. see DIS 34:131
Goon, H. see DIS 34:153
Cordeiro, A. Brazil, Porto Alegre
Cordeiro, E. Brazil, Porto Alegre
Corn, J. see DIS 34:153
Cortés, B. Colombia, Bogotá
Cortés, Y. Colombia, Bogotá
Corwin, H. Los Angeles, California
Coughlin, A. see DIS 34:152
Counce, S. New Haven, Connecticut
Council, S. Raleigh, North Carolina
Covarrubias, E. Chile, Santiago
Courreges, E. Baton Rouge, La
Coyle, M. see DIS 34:153
Craig, E. see DIS 34:151
Craig, G. Notre Dame, Indiana
Crow, J. F. Madison, Wisconsin
Cruickshank, W. see DIS 34:131
Cullen, M. New Haven, Connecticut
Cummins, E. see DIS 34:151
da Cunha, A. Brazil, São Paulo
Daillie, J. France, Lyon
Dalmon, France, Lyon
Das, C. Cold Spring Harbor, New York
David, J. France, Lyon
Davis, B. Madison, Wisconsin
Davis, D. see DIS 34:142
Dawood, M. United Arab Rep., Alexandria
Dearden, M. Gr. Britain, Manchester
de Capoa, A. see DIS 34:134
Deconinck, C. see DIS 34:127
de Fincati, W. Argentina, Buenos Aires
de Frescheville, J. see DIS 34:129
de Klerk, T. Netherlands, Leiden
del Campo, G. Pasadena, California
del Solar, E. Chile, Santiago
de Marinic, S. Argentina, Buenos Aires
de Marinis, F. see DIS 34:145
Dempster, E. Berkeley, California
den Hollander, C. Netherlands, Leiden
de Ruiter, F. Netherlands, Leiden
de Vries, J. East Lansing, Michigan
de Young, P. Los Angeles, California
Dharmarajan, M. India, Madras
Diamantis, B. see DIS 34:150
Diáz, N. Colombia, Bogotá
Dickerman, R. Austin, Texas
Diem, C. Switzerland, Zürich
Diez, J. Argentina, Buenos Aires
Diener, E. see DIS 34:140
Digot, A. see DIS 34:129
Dildy, E. see DIS 34:147
Di Pasquale, A. Italy, Milano
Ditadi, T. Brazil, Porto Alegre
Dittrich, W. Germany, Karlsruhe
Divelbiss, J. Le Mars, Iowa
Doane, W. New Haven, Connecticut
Dobie, N. Raleigh, North Carolina
Dobzhansky, Th. New York, New York
Dodds, J. see DIS 34:150
Dorn, G. Gr. Britain, Glasgow
Dorsey, R. Eugene, Oregon
Druger, M. Syracuse, New York
Duplat, H. Colombia, Bogotá
du Pui, L. Netherlands, Groningen
Duspiva, F. see DIS 34:130
Duval, D. Houston, Texas
Dyer, W. see DIS 34:144
Dykstra, W. Netherlands, Utrecht
Dyson, J. Raleigh, North Carolina
Ebeling, W. Germany, Karlsruhe
Edington, C. Tallahassee, Florida
Edmondson, M. Bloomington, Indiana
Edwards, J. Logan, Utah
Egbert, L. Logan, Utah
Eggert, J. Davis, California
Ehrlich, E. Eugene, Oregon
Ehrman, L. New York, New York

Eiche, A. Sweden, Stockholm
 Elens, A. see DIS 34:127
 Elequin, F. Austin, Texas
 El-Helw, M. United Arab Rep., Alexandria
 Emara, R. United Arab Rep., Alexandria
 Emrich, N. Cambridge, Massachusetts
 Englert, D. Lafayette, Indiana
 Epler, J. Tallahassee, Florida
 Epling, C. Los Angeles, California
 Erickson, J. Eugene, Oregon
 Esaki, K. Japan, Anzjo-Shi
 Everett, H. Ithaca, New York

Fabergé, A. Austin, Texas
 Fahmy, M. Gr. Britain, Chalfont
 Fahmy, O. Gr. Britain, Chalfont
 Falk, G. Evanston, Illinois
 Falk, P. Los Angeles, California
 Falk, R. Isreal, Jerusalem
 Fanale, L. see DIS 34:150
 Farham, M. Eugene, Oregon
 Farnsworth, M. Buffalo, New York
 Fattal, S. Isreal, Jerusalem
 Fernandez, R. Chile, Santiago
 File, S. Ann Arbor, Michigan
 Finlay, D. Australia, Sydney
 Fletcher, M. see DIS 34:148
 Fockens, W. Netherlands, Groningen
 Forbes, C. see DIS 34:150
 Forbes, E. Gr. Britain, Glasgow
 Forrest, H. Austin, Texas
 Foster, T. Eugene, Oregon
 Fourche, J. France, Lyon
 Fox, A. East Lansing, Michigan
 Freeborn, J. Ridgefield, Connecticut
 Freed, J. see DIS 34:152
 Freund, O. Isreal, Jerusalem
 Friedlaender, M. Isreal, Jerusalem
 Friedman, L. Hiram, Ohio
 Fristrom, J. see DIS 34:150
 Fritz-Niggli, H. see DIS 34:140
 Frost, J. Finland, Turku
 Frota-Pessoa, O. Brazil, São Paulo
 Frydenberg, O. see DIS 34:128
 Fuchs, M. East Lansing, Michigan
 Fujii, S. Japan, Kobe
 Fujio, Y. Japan, Osaka
 Fuscaldo, K. Jamaica, New York
 Fusté, M. Spain, Barcelona
 Futch, D. Austin, Texas
 Fuwa, K. Japan, Misima
 Fuyama, Y. Japan, Tokyo

Gale, C. Gr. Britain, Harwell
 Gallucci, E. Italy, Milano
 García-Bellido, A. Sapin, Madrid
 Gardner, E. Logan, Utah
 Gay, H. Cold Spring Harbor, N. Y.
 Geer, B. Davis, California
 Geerthsen, J. South Africa, Pretoria
 Geissler, E. Germany, Berlin-Buch
 Gellert, H. Germany, Mariensee
 Gerletti, M. see DIS 34:134
 Gersh, E. Chicago, Illinois
 Gerstenber, V. Austin, Texas

Ghini, C. see DIS 34:134
 Ghosh, H. see DIS 34:133
 Gianopulos, H. Dekalb, Illinois
 Giavelli, S. Italy, Milano
 Gibbons, A. Gr. Britain, Chalfont
 Gibson, J. Gr. Britain, Cambridge
 Gidholm, K. Sweden, Uppsala
 Gill, K. New Haven, Connecticut
 Gillies, G. Cold Spring Harbor, N.Y.
 Gilliland, K. Lexington, Kentucky
 Gilmore, G. Iowa City, Iowa
 Glass, H. B. Baltimore, Maryland
 Glass, S. S. Baltimore, Maryland
 Glassman, E. Chapel Hill, N. Carolina
 Gleaves, C. Gr. Britain, Chalfont
 Gloor, H. Netherlands, Leiden
 Gloor, R. Switzerland, Zürich
 Godet, J. France, Lyon
 Goedhart, A. Netherlands, Leiden
 Goetz, W. Switzerland, Zürich
 Goldin, H. Buffalo, New York
 Goldschmidt, E. Isreal, Jerusalem
 Gonnella, V. Jamaica, New York
 Gottlieb, F. Berkeley, California
 Gottschewski, G. Germany, Mariensee
 Gowen, J. Ames, Iowa
 Grabicki, E. New Haven, Connecticut
 Graener, E. Germany, Münster
 Grassmann, A. Switzerland, Zürich
 Green, C. Evanston, Illinois
 Green, M. Davis, California
 Greenberg, R. Madison, Wisconsin
 Grell, E. Oak Ridge, Tennessee
 Grell, R. Oak Ridge, Tennessee
 Greuter, M. Switzerland, Zürich
 Griech, H. see DIS 34:152
 Griffen, A. see DIS 34:143
 Grisseau, C. see DIS 34:145
 Grun, P. University Park, Pennsylvania
 Grüneberg, H. Gr. Britain, London
 Guerrier, P. France, Lyon
 Guest, W. University, Alabama
 Gugler, D. Duarte, California
 Guglielmi, A. see DIS 34:134
 Guillaumin, M. see DIS 34:129
 Gunson, M. Australia, Melbourne
 Gutenmann, H. Ithaca, New York

Hass, F. Houston, Texas
 Hadler, N. New Haven, Connecticut
 Hadorn, E. Switzerland, Zürich
 Hagens, H. see DIS 34:130
 Haldane, J. see DIS 34:133
 Halfer, C. Italy, Milano
 Halkka, O. Finland, Helsinki
 Halpern, L. Fayetteville, Arkansas
 Hamilton, J. Eugene, Oregon
 Hampel, A. Dekalb, Illinois
 Hanks, G. Salt Lake City, Utah
 Hanly, E. Switzerland, Zürich
 Hannah-Alava, A. Finland, Turku
 Hansen, A. Logan, Utah
 Hansteen, I. Norway, Blindern
 Hardin, R. Lafayette, Indiana
 Harlock, R. Australia, Brisbane

- Harmoinen, L. Finland, Turku
Harmon, C. Dekalb, Illinois
Harrington, D. see DIS 34:154
Harrison, B. Gr. Britain, Bayfordbury
Hartmann, I. S. Africa, Johannesburg
Hartshorne, J. Gr. Britain, Manchester
Hauser, A. see DIS 34:152
Hawkes, N. Logan, Utah
Hawkins, E. Los Angeles, California
Hayakawa, J. Japan, Anzjo-shi
Hayman, D. Australia, Adelaide
Heath, G. Corvallis, Oregon
Heed, W. see DIS 34:154
Heerkens, C. Netherlands, Leiden
Hegewald, E. Salt Lake City, Utah
Hegewald, R. Salt Lake City, Utah
Heinonen, P. Finland, Turku
Heinsoo, M. Switzerland, Zürich
Hemel, J. Netherlands, Utrecht
Henderickson, R. Los Angeles, Calif.
Henderson, A. Chapel Hill, North Carolina
Henke, H. see DIS 34:130
Henningsen, K. see DIS 34:128
Herskowitz, I. see DIS 34:153
Hess, O. Germany, Tübingen
Heuts, M. see DIS 34:127
Hexter, W. Amherst, Massachusetts
Higgins, W. see DIS 34:143
Hildreth, P. Berkeley, California
Hillman, R. see DIS 34:152
Himel, N. Isreal, Jerusalem
Hinton, C. see DIS 34:142
Hiraga, S. Japan, Osaka
Hiraizumi, Y. Japan, Misima
Hiroyoshi, T. Japan, Osaka
Hirsch, J. Urbana, Illinois
Hirsh, D. see DIS 34:152
Hochmann, B. Salt Lake City, Utah
Hodge, L. Chapel Hill, North Carolina
Hoenigsberg, H. Colombia, Bogotá
Hofmeyer, J. S. Africa, Pretoria
Höhne, G. see DIS 34:130
Hollander, W. Ames, Iowa
Holler, A. see DIS 34:152
Hollingsworth, M. Gr. Britain, London
Hope, L. Gr. Britain, Chalfont
Horn, S. Berkeley, California
Horowitz, A. Isreal, Jerusalem
Hosteller, R. Urbana, Illinois
Hotz, G. Germany, Karlsruhe
House, M. see DIS 34:145
House, V. see DIS 34:145
Howe, M. see DIS 34:149
Hubbard, W. Chapel Hill, N. Carolina
Hubby, J. Chicago, Illinois
Hudson, J. Pullman, Washington
Hughes, R. Richmond, Virginia
Hungerford, D. see DIS 34:152
Hunter, A. Colombia, Bogotá
Hurvitz, D. Isreal, Jerusalem

Ibrahim, S. United Arab Rep., Alexandria
Ichida, H. Japan, Tokyo
Ichioka, S. Japan, Osaka
Ikeda, H. Japan, Tokyo

Imai, Y. Japan, Misima
Imaizumi, T. Japan, Kyoto
Inagaki, E. see DIS 34:135
Inouye, I. Japan, Okamoto
Ives, P. Amherst, Massachusetts
Iyama, S. Japan, Misima
Iyengar, S. Baton Rouge, La.

Jacobs, A. Netherlands, Leiden
Jacobs, M. Harrisonburg, Virginia
James, J. Chapel Hill, N. Carolina
Janke, H. Salt Lake City, Utah
Jayakar, S. see DIS 34:133
Jempson, J. Gr. Britain, Harwell
Johansen, I. Norway, Oslo
Johansson, K. Sweden, Uppsala
Johns, R. San Diego, California
Johnsen, R. Eugene, Oregon
Johnson, G. Logan, Utah
Johnson, W. see DIS 34:150
Joneleit, C. Germany, Tübingen
Jonsson, U. Cambridge, Massachusetts
Judd, B. Austin, Texas
Jura, C. New Haven, Connecticut

Kaji, S. Japan, Okamoto
Kaminishi, H. Japan, Mitaka
Kan, J. East Lansing, Michigan
Kanehisa, T. Japan, Kobe
Kaneko, A. Japan, Sapporo
Kang, S. East Lansing, Michigan
Kang, Y. Korea, Seoul
Kapetan, A. East Lansing, Michigan
Kaplan, W. Duarte, California
Karam, J. Chapel Hill, N. Carolina
Karlik, A. Austria, Vienna
Kasai, I. see DIS 34:135
Kato, M. Japan, Kyoto
Kato, Masaru, Japan, Kyoto
Kato, S. Los Angeles, California
Kaufman, B. Argentina, Buenos Aires
Kaufmann, B. Cold Spring Harbor, N.Y.
Kawabe, M. Japan, Kobe
Kaye, R. see DIS 34:153
Keller, E. Chapel Hill, N. Carolina
Kelsall, P. Gr. Britain, Edinburgh
Kernaghan, R. Cold Spring Harbor, N.Y.
Kessler, S. New York, New York
Kheiralla, A. see DIS 34:133
Khishin, A. Gr. Britain, Edinburgh
Kiesselbach, T. Chapel Hill, N. Carolina
Kiger, J. Pasadena, California
Kiil, W. Norway, Blindern
Kikkawa, H. Japan, Osaka
Kim, D.S. Korea, Seoul
Kim, D. V. Korea, Kwangju
Kim, K. Korea, Kwangju
Kim, O. see DIS 34:137
Kimura, M. Japan, Misima
King, Jack, Berkeley, California
King, James. New York, New York
King, R. Evanston, Illinois
Kirsehtbaum, W. Argentina, Buenos Aires
Kirchseisen, G. Germany, Karlsruhe
Kitagawa, O. Japan, Tokyo

Kitazuma, Y. Japan, Kobe
 Kloos, W. Ames, Iowa
 Knight, E. Gr. Britain, Chalfont
 Knight, G. Gr. Britain, Edinburgh
 Knott, B. Australia, Hobart
 Koch, E. Evanston, Illinois
 Koch, R. Switzerland, Zürich
 Köhnlein, W. Germany, Karlsruhe
 Kojima, K. Raleigh, North Carolina
 Konarski, M. see DIS 34:152
 Kondo, K. Japan, Anzō-Shi
 Koref-Santibanez, S. Chile, Santiago
 Koske-Westphal, T. Germany, Hamburg
 Koswig, C. Germany, Hamburg
 Krause, E. Lafayette, Indiana
 Krauss, M. Cold Spring Harbor, N. Y.
 Kravina, A. Italy, Milano
 Krawinkel, M. see DIS 34:148
 Krebs, J. see DIS 34:153
 Krivshenko, E. see DIS 34:153
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 Kroeger, H. Switzerland, Zürich
 Kromm, N. Germany, Berlin-Dahlem
 Kvelland, I. Norway, Blindern
 Künkel, H. see DIS 34:130
 Kunze-Mühl, Austria, Vienna
 Kuroda, Y. Japan, Osaka
 Kurokawa, H. Japan, Tokyo
 Kyle, W. see DIS 34:148

Lagowski, J. Austin, Texas
 Laird, C. Switzerland, Zürich
 Lakovaara, S. Finland, Helsinki
 Lamb, M. Gr. Britain, London
 Lamerton, M. Gr. Britain, Harwell
 Landenberger, M. Eugene, Oregon
 Landy, R. Dekalb, Illinois
 Langer, B. Pittsburgh, Penn.
 Laugé, G. France, Gif-sur-Yvette
 Laufer, H. Baltimore, Maryland
 Laureys, see DIS 34:127
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 Le Blang, W. Dekalb, Illinois
 Lederman-Klein, A. Israel, Jerusalem
 Lee, C. Korea, Seoul
 Lee, H. Korea, Seoul
 Lee, T. Korea, Seoul
 Lefevre, G. Cambridge, Massachusetts
 Legay, J. France, Lyon
 Leigh, B. Gr. Britain, Edinburgh
 Leigh, Berry, Netherlands, Leiden
 Léon, M. France, Gif-sur-Yvette
 Leon, W. Argentina, Buenos Aires
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 Leventhal, E. New Haven, Connecticut
 LeVier, R. Iowa City, Iowa
 Levine, L. New York, New York
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 Lewis, E. Pasadena, California
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 Loomis, M. Ithaca, New York
 Louis, M. France, Gif-sur-Yvette
 Lovellette, E. San Diego, California
 Lucchesi, J. Berkeley, California
 Luce, W. Urbana, Illinois
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 Lund, B. Sweden, Uppsala
 Lund, D. Lincoln, Nebraska
 Luning, K. Sweden, Stockholm
 Lux, E. Madison, Wisconsin

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 McLean, J. Chapel Hill, N. Carolina
 McSheehy, T. Gr. Britain, Harwell
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 Magalhaes, E. Brazil, Sao Paulo
 Magaribuchi, K. Japan, Kobe
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 Mahowald, A. Baltimore, Maryland
 Mainx, F. Austria, Vienna
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 Markowitz, E. Pasadena, California
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 Mathew, C. India, Hyderabad
 Matos, N. Brazil, São Paulo
 Mattson, T. Madison, Wisconsin
 Maxim, P. New Haven, Connecticut
 Mayhew, S. Los Angeles, California
 Maynard Smith, J. Gr. Britain, London
 Mayo, M. Australia, Adelaide
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 Meyer, G. Germany, Tübingen
 Meyer, H. Bloomington, Indiana
 Micheli, A. Italy, Rome
 Mickel, S. Eugene, Oregon
 Mickey, G. Ridgefield, Connecticut
 Mileiko, V. Canada, Toronto
 Milkman, R. Syracuse, New York
 Miller, D. Lincoln, Nebraska
 Mills, Richard Evanston, Illinois
 Mills, R.P. New Haven, Connecticut
 Minamori, S. Japan, Misima
 Miralles, L. Spain, Madrid
 Mishara, J. New York, New York
 Mitchell, A. Pasadena, California
 Mitchell, H. Pasadena, California
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 Miyoshi, Y. Japan, Kyoto
 Moawad, H. United Arab Rep., Alexandria
 Mohler, J. Corvallis, Oregon
 Mohr, O. Norway, Blindern
 Momma, E. Japan, Sapporo
 Monclús, M. Spain, Barcelona
 Monkman, M. Dekalb, Illinois
 Montalenti, G. Italy, Rome
 Montelius, I. Sweden, Stockholm
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 Mora, S. Pasadena, California
 Morales, N. Brazil, Pôrto Alegre
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 Morey, M. Spain, Madrid
 Moriwaki, D. Japan, Tokyo
 Moskwinski, T. Notre Dame, Indiana
 Mossige, J. Norway, Oslo
 Mostafa, A. Gr. Britain, Edinburgh
 Mourad, A. United Arab Rep., Alexandria
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 Muller, H. J. Bloomington, Indiana
 Müller, M. Switzerland, Zürich
 Mundt, C. Brazil, Pôrto Alegre
 Munoz, E. Argentina, Buenos Aires
 Munz, P. Switzerland, Zürich
 Myers, O. Ithaca, New York
 Myszevske, M. East Lansing, Michigan
 Nadal, A. Spain, Barcelona
 Nafei, H. Gr. Britain, Edinburgh
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 Narain, P. India, New Delhi
 Narayana Rao, N. India, Madras
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 Nash, D. Gr. Britain, Cambridge
 Nash, Donald J. University Park, Penn.

Nawa, S. Japan, Misima
 Neeley, J. Corvallis, Oregon
 Nel, P. South Africa, Pretoria
 Neulat, M. France, Lyon
 Newball, S. Colombia, Bogotá
 Nicklas, R. New Haven, Connecticut
 Nicoletti, B. Italy, Rome
 Nigon, V. France, Lyon
 Nobuki, R. Japan, Osaka
 Nolte, D. S. Africa, Johannesburg
 Nomura, K. Salt Lake City, Utah
 Norwood, S. Austin, Texas
 Nöthel, H. Germany, Berlin-Dahlem
 Nöthiger, R. Switzerland, Zürich
 Novitski, E. Eugene, Oregon
 Nozawa, K. Japan, Anzyo-Shi
 Nugent, K. East Lansing, Michigan
 Oftedal, P. Norway, Oslo
 Ogaki, M. Japan, Sakai
 Ogita, Z. Japan, Osaka
 Ogawa, T. Canada, Vancouver
 Ochanessian-Guillemain, A. France, Gif-sur-Yvette
 Ohba, S. Japan, Tokyo
 Ohlendorff, H. Sweden, Uppsala
 Ohnishi, E. Japan, Tokyo
 Okada, T. Japan, Tokyo
 Oksala, T. Finland, Turku
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 Otuji, Y. Japan, Osaka
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 Parish, J. Chapel Hill, N. Carolina
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Perdrix, S. France, Lyon
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Phillips, D. Syracuse, New York
Pilar, B. Salt Lake City, Utah
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Plus, N. France, Gif-sur-Yvette
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Pozzi, L. Italy, Milano
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Pratt, Guthrie, Oak Ridge, Tennessee
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Price, M. Chapel Hill, North Carolina
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Puro J. Finland, Turku

Queiroz, J. France, Gif-sur-Yvette
Querner, W. Germany, Mariensee

Rahat, A. Isreal, Jerusalem
Rai, K. Notre Dame, Indiana
Rakha, F. United Arab Rep., Alexandria
Ramel, C. Sweden, Stockholm
Ramila, D. Brazil, Pôrto Alegre
Ramírez, P. Spain, Madrid
Randerson, S. Ann Arbor, Michigan
Rao, K. India, Hyderabad
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Rasmuson, B. Sweden, Uppsala
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Reguly, M. Brazil, Pôrto Alegre
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Resch, K. Austin, Texas

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Rios, C. Colombia, Bogotá
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Rizki, R. Ann Arbor, Michigan
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Robertson, F. Gr. Britain, Edinburgh
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Roper, J. Gr. Britain, Sheffield
Rose, B. Cambridge, Massachusetts
Rosenfeld, A. Madison, Wisconsin
Rosin, S. Switzerland, Bern
Rosner, J. New Haven, Connecticut
Royers, V. Gr. Britain, Edinburgh
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Sammalisto, L. Finland, Helsinki
Sanderson, K. Ithaca, New York
Sandler, L. Madison, Wisconsin
Sang, J. Gr. Britain, Edinburgh
Sankaranayan, K. New York, New York
Santos, A. Brazil, Pôrto Alegre
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Sävhaugen, R. Sweden, Stockholm
Savolainen, S. Finland, Turku
Scandlyn, B. Oak Ridge, Tennessee
Schaffer, H. Raleigh, North Carolina
Schafrik, C. Ithaca, New York
Schallet, A. Cold Spring Harbor, N.Y.
Scharloo, W. Gr. Britain, Edinburgh
Schepers, A. Netherlands, Leiden
Scheren, A. Philadelphia, Penn.
Schideler, D. Lafayette, Indiana
Schiller, R. Philadelphia, Penn.
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Schmid, W. Austin, Texas
Schneider, A. Switzerland, Zürich

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Seaton, R. East Lansing, Michigan
Sederoff, R. Los Angeles, California
Seecof, R. Pasadena, California
Seidel, F. Germany, Marbugh/Lahn
Seidel, S. Germany, Tübingen
Seiger, M. Canada, Toronto
Seki, T. Japan, Osaka
Semp, B. Lexington, Kentucky
Sen, B. Gr. Britain, Edinburgh
Seto, F. Berea, Kentucky
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Sheldon, B. Australia, Sydney
Sheridan, B. Sweden, Stockholm
Sherwood, E. Berkeley, California
Shideler, D. Lafayette, Indiana
Shima, T. Japan, Sapporo
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Shoji, T. Japan, Mitaka
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Sieux, J. Chicago, Illinois
Silberman, J. Ithaca, New York
Silva, L. Brazil, Pôrto Alegre
Silva, T. Brazil, Pôrto Alegre
Simmons, J. Logan, Utah
Simões, G. Brazil, Pôrto Alegre
Sims, M. Chicago, Illinois
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Sinoto, Y. Japan, Mitaka
Siracusano, V. Jamaica, New York
Sironi, G. Italy, Milano
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Slizynski, H. Gr. Britain, Edinburgh
Smith, E. Norway, Blindern
Smith, P. Evanston, Illinois
Smith, S. Corvallis, Oregon
Snyder, L. Gr. Britain, Edinburgh
Sobels, F. Netherlands, Leiden
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Solana, I. Spain, Madrid
Solima, A. New York, New York
Soliman, G. United Arab Rep., Alexandria
Soliman, M. United Arab Rep., Alexandria
Sollum, F. Norway, Blindern
Sondhi, G. Ridgefield, Connecticut
Sondhi, K. Ridgefield, Connecticut
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Sorensen, W. Logan, Utah
Southin, J. Los Angeles, California
Spassky, B. New York, New York
Spassky, N. New York, New York
Sperlich, D. Austria, Vienna
Spickett, S. Gr. Britain, Cambridge
Spieler, R. Chicago, Illinois
Spiess, E. Pittsburgh, Pennsylvania
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Springer, R. Austria, Vienna
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Stern, Curt Berkeley, California
Stevenson, R. Johnson City, Tennessee
Stewart, W. Lexington, Kentucky
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Stone, W. Austin, Texas
Strangio, V. Australia, Melbourne
Strachan, I. Gr. Britain, Edinburgh
Strachan, K. Gr. Britain, Edinburgh
Stratopoulos, G. Salt Lake City, Utah
Strickberger, M. New York, New York
Strömnaes, O. Norway, Blindern
Struck, E. Germany, Berlin-Dahlem
Sturtevant, A. Pasadena, California
Suguna, S. India, Madras
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Suomalainen, E. Finland, Helsinki
Suska, J. Ithaca, New York
Suzuki, D. Oak Ridge, Tennessee
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Sweet, E. Pittsburgh, Pennsylvania
Sweron, M. Gr. Britain, Chalfont
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Talsma, J. Cold Spring Harbor, New York
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Tates, A. Netherlands, Leiden
Taylor, A. Cold Spring Harbor, New York
Teissier, G. France, Gif-sur-Yvette
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Teviotdale, F. Eugene, Oregon
Thedy, O. Brazil, Pôrto Alegre
Thoday, J. Gr. Britain, Cambridge
Thomas, Carol Madison, Wisconsin
Thomas, Constance, Salt Lake City, Utah
Thomas, S. Bloomington, Indiana
Thompson, M. Ithaca, New York
Thompson, P. Ames, Iowa

- Thompson, S. Corvallis, Oregon
Thomson, J. Australia, Melbourne
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Tidwell, T. New York, New York
Tiffany, B. Amherst, Massachusetts
Tiivola, A. Finland, Helsinki
Tobari, I. Japan, Tokyo
Tobari, Y. Japan, Tokyo
Tokunaga, C. Berkeley, California
Toledo, J. Brazil, São Paulo
Toledo, S. Brazil, São Paulo
Tondo, C. Brazil, Porto Alegre
Torroja, E. Spain, Madrid
Townsend, J. Richmond, Virginia
Toyofuku, Y. Japan, Misima
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Treator, K. Buffalo, New York
Trogildo, D. Brazil, Porto Alegre
Trosko, J. East Lansing, Michigan
Trout, W. Bloomington, Indiana
Tschumi, P. Switzerland, Bern
Tsukamoto, H. Japan, Tokyo
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Urspring, H. Baltimore, Maryland
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Valencia, R. Argentina, Buenos Aires
van Hooft, J. Netherlands, Leiden
Van Niskerk, B. South Africa, Pretoria
Vanoucek, E. Ithaca, New York
Van Schaik, N. South Africa, Pretoria
Van Valen, L. New York, New York
Vigier, P. France, Gif-sur-Yvette
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Volkers, W. Netherlands, Leiden
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von Borstel, R. Oak Ridge, Tennessee
von Halle, E. Oak Ridge, Tennessee
Voynow, N. Madison, Wisconsin
- Waddington, C. Gr. Britain, Edinburgh
Wagner, R. Austin, Texas
Wagoner, D. Bloomington, Indiana
Wahrman, J. Israel, Jerusalem
Wajntal, A. Brazil, São Paulo
Walen, K. Berkeley, California
Wall, L. Chapel Hill, North Carolina
Wallace, B. Ithaca, New York
Wallbrunn, H. Gainesville, Florida
Wallenius, M. Finland, Turku
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Ward, C. Durham, North Carolina
- Wasserman, M. Australia, Melbourne
Watanabe, H. Japan, Osaka
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Wedvik, Hans Norway, Blindern
Wei, I. Dekalb, Illinois
Weideman, J. Dekalb, Illinois
Weingart, E. Cold Spring Harbor, N.Y.
Weinmann, H. Switzerland, Zürich
Weisbrot, D. New York, New York
Welch, R. Austin, Texas
Welshons, W. Oak Ridge, Tennessee
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Whitesel, B. Los Angeles, California
Whittinghill, M. Chapel Hill, N. Carolina
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Wills, C. Canada, Vancouver
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Wilson, F. Austin, Texas
Wilson, L. Wellesley, Massachusetts
Wing, M. Raleigh, North Carolina
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Winterfeldt, G. Germany, Berlin-Dahlem
Wolf, E. Germany, Berlin-Dahlem
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Wrathall, C. Salt Lake City, Utah
Wright, E. Baltimore, Maryland
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Wu, C. Dekalb, Illinois
Wui, I. Korea, Kwangju
Würzler, F. Switzerland, Zürich
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Yanders, A. East Lansing, Michigan
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Yoo, C. Korea, Seoul
Yoon, S. East Lansing, Michigan
Yost, H. Amherst, Massachusetts
Youn, J. Korea, Seoul
Ytterborn, K. Sweden, Stockholm
- Zambruni, L. Italy, Milano
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Zimmer, K. Germany, Karlsruhe
Zimmering, S. Bloomington, Indiana
Zimmermann, W. Germany, Mariensee
Zürcher, C. Switzerland, Zürich

(Editor's Comment--continued from page 8)

In any case, one must at the present time regard DIS primarily as a medium for the dissemination of stock lists and related technical data. However, I would regard the strictest adherence to this as unnecessarily stultifying, even if it were possible to define the above terms unambiguously. If one regards DIS instead as an informal means of promoting and facilitating the work of Drosophila geneticists, filling a niche not occupied by any other means of communication, then one can readily justify a broader interpretation of its function.

We are now including a section in which workers are given the opportunity to give approval to the quotation of specific notes of theirs by others. This may eliminate a good deal of unnecessary letter writing. I hope that the notes are mentioned specifically by DIS number and page number, for the following reason: when casual informal notes are written, sometimes covering work not yet completed, there are bound to be errors and it would help greatly if each worker would look closely at his past contributions and indicate (in the form suggested in the call) where further information about a preliminary note might be found. It is not really a question of whether the worker is willing to stand by any criticism for an incomplete or inaccurate note, as it is a matter of offering positive help to workers who want to get the maximum amount of correct information possible. Similarly I wonder about the desirability of the flat statement made by a number of workers that "all my notes, past present and future, may be quoted." The first presumes an infallibility that is more appropriate for publications rather than DIS notes and it is my considered judgment that this infallibility can be rightfully claimed by no more than three workers in the entire field. The second statement wills the infallibility to the second or third scientific generation and may be even more suspect. For these same reasons, I would, for the present at least, like to defer granting permission to quote current notes, in order to give each worker some time to consider carefully his own judgment of the quotability of that note.

Finally let me extend my thanks to the staff in Eugene, Oregon, who have put out this issue while I have been sojourning in Switzerland. These include: Mrs. Dorothy Parker, who has had over-all responsibility for the operation, Mrs. Elizabeth Ehrlich, Miss Jan Hamilton, Mrs. Mary Helen Landenberger, and Miss Hermina Ehrlich.

E. Novitski