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MATERIALS REQUESTED OR AVAILABLE

Industrial size packages of Grandma's Molasses will undergo a name change. The new name for the product will be HOME MAID MOLASSES. The necessity for the change arose when the rights to the "Grandma's" trademark were sold to Duffy-Mott Company. Home Maid Molasses is not a new brand - just a new name. It will be exactly the same unsulphured, high quality, consistent and dependable molasses as it was when marketed under the Grandma's label, according to The American Molasses Company, 120 Wall Street, New York, N.Y. 10005.

F.v. Schilcher of the Department of Zoology. Jniversity of Edinburgh, Scotland, is involved in genetical research on Drosophila melanogaster courtship song and would like to investigate the effects of relaxed or absent selection pressure for song characteristics. This condition would be fulfilled in all severe wing or antenna mutations. Consequently he is looking for back mutants or revertants in such mutant stocks. He would greatly appreciate receiving any future appearing revertant together with a sample of the original mutant stock. He is interested in the following loci: (any allele is of interest as long as it is not sterile in either sex.) The Zoology Department will be happy to refund any costs of postage.

Chromosome 1

At:Attenuated
Bb:Bubble
Bx:Beadex
crt:crumpled tips
ct:cut
hdp:held up
obl:oblique wings
r:rudimentary
sw:short wing
Tu:Turned up wing
unp:unexpanded
up:upheld
vs:vesiculated
wy:wavy

Chromosome 2

a:arc
al:aristaless
ang:angle wing
ant:antennaless
ap:apterous
arch:arch
ba:baloon
blo:bloated
bs:blistered
c:curved
ck:crinkled
dp:dumpy
el:elbow
hy:humpy
nub:nubbin
ove:overetherized
pa:patulous
pu:pupal
rw:raised wing
U:Upturned
vg:vestigial
wx:waxy

Chromosome 3

aa:anarista
as:ascute
bod:bowed
by:blistery
cmp:crumpled
cp:clipped
D:Dichaete
Rf:Roof
ss:spineless-aristapedia
th:thread
tt:tilt
tx:taxi
W:Wrinkled

A group working on Peruvian Drosophila population genetics has just been created at the Universidad Nacional Mayor de San Marcos, Lima, Perú. They would be very grateful for reprints on Drosophila population genetics mainly of South American or other tropical species.

A new Drosophila genetics laboratory is being established at the University of Campo Grande in Campo Grande, Mato Grosso State, Brazil. Sueli Maria Ferreira Pereira and Eliezer José Marques, Departamento de Morfologia, Universidade Estadual de Mato Grosso, request an exchange of reprints from other laboratories.

D.L. Lindsley, University of California, La Jolla, Calif. 92037, would appreciate receiving a replacement stock of C(1)A. He needs it for balancing T(Y;A)'s. It is the most stable compound X that he knows of and his was lost this year.

D.L. Lindsley would be most appreciative if every Drosophila worker would please notify him of all errors that he has encountered in the book The Genetic Variations of Drosophila melanogaster, by Lindsley and Grell. They are contemplating the preparation of a supplement. Any additions of unpublished material will also be welcome.

Lee Ehrman, Division of Natural Sciences, State University, Purchase, New York 10577, requests reprints, etc. of published and in press descriptions of drosophilid mating behaviors for a chart to be included in the planned chapter, "Sexual Behavior" in The Genetics and Biology of Drosophila, Vol. 2, edited by T.Wright and M. Ashburner.

Mrs. Thea Muller of Bloomington, Indiana, wishes to remind Drosophila workers that sets of Prof. Muller's papers are still available. Send requests to E. Novitski, Eugene, Oregon.

The Fourth European Drosophila Conference will be held June 6-7, 1974, at the University of Umea, Sweden. For more information write Bertil Rasmusmuson or Agneta Södergren, University of Umeå, Department of Genetics, S-901 87 Umeå, Sweden.

Roger Milkman, Department of Zoology, University of Iowa, Iowa City, has prepared a lab exercise for introductory genetics courses which employs enzyme electrophoresis to demonstrate natural variation; a locus is also mapped. The Adamkewicz multiple applicator (DIS 46) permits the analysis of large numbers of flies quickly and rather cheaply. Copies of the instructions are available on request.

The first meeting of Drosophila workers in the USSR took place in Petergoff (near Leningrad) from June 26 to 30, 1973. The meeting was held under the auspices of Leningrad University. More than one hundred people from different scientific organizations in the USSR delivered reports and exchanged views in the field of Drosophila genetics. The meeting lasting five days was divided into six symposia containing 37 reports and 23 demonstrations. Three of these symposia were devoted to phenogenetics of Drosophila. Special attention was paid to the maintaining of Drosophila stocks. It was decided to organize such meetings every two years, according to the report of T.M. Turpaev, Assistant Director of the Institute of Developmental Biology of the Academy of Sciences of the USSR, and M. Evgeniev, member of the organizing committee of the Drosophila meetings.

An International Stock List: Work is being initiated in La Jolla on a computerized listing of Drosophila melanogaster stocks. Each stock will be listed once with indication of the laboratories in which it is carried. As envisioned, stocks will be arranged according to the chromosomes that differ from wild type; each will be cross referenced with regard to its component chromosomes, and each chromosome according to its component rearrangements and mutations. Terminology will be standardized according to conventions established in "Genetic Variations of Drosophila melanogaster". It is anticipated that the initial list will encompass only the two major stock centers in the United States, but as the computer program is proved the remainder of the world's stock lists will be added. Once the list is complete it will be updated annually simply by adding and subtracting stocks. Each laboratory submitting a stock list will receive a sublisting of its own stocks selected from the master list, and the master list will appear in DIS either annually or biannually. D.L. Lindsley, La Jolla.

Populationsgenetik: An introduction to population genetics in German by D. Sperlich has been published by G. Fischer Verlag, Stuttgart 1973 (Germany). Its German title is: Populationsgenetik, Grundlagen und experimentelle Ergebnisse.

Behavioural and Ecological Genetics: A Study in Drosophila by P.A. Parsons, Department of Genetics, La Trobe University, Melbourne, Australia, has been published by Clarendon (dist. Oxford University Press).

Man's Future Birthright: Essays in Science and Humanity by H.J. Muller, edited by Elof A. Carlson, SUNY at Stony Brook, has been published by the State University of New York Press.

A Bibliography of Theoretical Population Genetics, edited by Joseph Felsenstein and Bruce Taylor, has been published by the U.S. Atomic Energy Commission. It contains 3,749 references in theoretical population genetics, quantitative genetics, and statistical human genetics. It is A.E.C. Report No. RLO-2225-5-18 and is available from the National Technical Information Service, U.S. Department of Commerce, Springfield, Virginia 22151 at a price of $10.60 printed or $1.45 in microfiche.

Film on Drosophila behavior: CRM Educational Filsm, Del Mar, California 92014, has produced a 30 minute color film called "The Fruit Fly: A Look at Behavior Biology". E. Novitski has seen an advance copy of this work and feels that each person will react quite differently to its contents, depending on his background, orientation and sophistication, and suggests that it be seen first by any person contemplating acquiring it permanently.

TURKU, FINLAND: UNIVERSITY OF TURKU
Department of Genetics

Wild Stocks

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

Chromosome 1

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

Chromosome Y

--- B^SY (see 138)
151 f.Y^S/Y^L
152 sc^8.Y/y ac sc oc ptg & y f:=
153 v f B XY/y^2 su-w^a w^a bb

Chromosome 2

201 al dp
202 al dp b pr c px sp
203 al dp b pr c px sp/In(2L+2R)Cy, al^2 Cy lt^3 L^4 sp^2
204 ap^4/In(2LR)Rev^B
205 Bl L^2/In(2L+2R)Cy, Cy
206 bw
207 dp b pr
208 In(2L)Cy, al^2 ast^3 b pr (Cy not present)
209 In(2L)Cy, al^2 Cy lt^3/In(2R)Cy, b pr Bl lt^3 cn^2 L^4 sp^2
210 In(2L+2R)Cy, S^2 Cy cn^2/In(2R)Cy, dp^{lv} Sp cn^2
211 In(2L+2R)NS, b mr/In(2L+2R)Cy, Cy
212 In(2L+2R)NS, px sp/In(2R)Cy, cn^2 cg sp^2
213 pr cn ix/SM5, al^2 Cy lt^v sp^2
214 rl
215 stw^2
216 stw^2 vg
217 vg

Chromosome 3

301 $Antp^{49}$/TM1, Me ri pP sbd^1
302 Bd^G/In(3R)C, l(3)a
303 ca bv
304 cu e^s
305 Df(3L)in^{61j1}/TM1, Me ri pP sbd^1
306 Dp(1;3)in^{61j2}/In(3LR)Ubx^{130}, ri Ubx^{130} e^s
307 Dp(1;3)in^{61j2}, th st/In(3LR)Ubx^{130}, ri Ubx^{130} e^s
308 dsx^{60l} cu sr e^s/TM1, Me ri sbd^1
309 e
310 eg rn^3 pP bx sr e^s ca/In(3LR)Ubx^{130}, ri Ubx^{130} e^s ca
311 Gl Sb/LVM
312 h Pc^2 sr e^s/TM1, Me ri sbd^1
313 h th st cp in ri pP sr e^s ca/In(3LR) Ubx^{130}, ri Ubx^{130} e^s ca
314 h th st cp in ri Pc^2 sr^{61j2}/In(3LR) Ubx^{130}, ri Ubx^{130} e^s(ca±)
315 In(3L)P, Me Sb/In(3LR)DcxF, ru h D

316 In(3LR)Ubx^{130}, ri Ubx^{130} e^{s}/TM1, Me ri p^{p} sbd^{1}
317 In(3R)Antp^{B}, ss^{a}/TM1, Me ri sbd^{1}
318 In(3R)Dl^{B}, st Dl^{B}/In(3R)P^{W}, st l(3)W ca
319 In(3R)P^{FLA}
320 k^{D} e^{s}/In(3LR)Ubx^{130}, ri Ubx^{130} e^{s}
321 k^{D} e^{s} Bd^{G}/In(3R)C, l(3)a
322 Ki
323 Ly Sb/LVM
324 Me^{65d} h th/TM3, Sb Ser
325 rn^{3} p^{p} bx sr e^{s}/TM1, Me ri p^{p} sbd^{1}
326 (ru) h th st cp in ri p^{p} ss^{a} bx^{3} sr e^{s}/TM1, Me ri sbd^{1}
327 ru h th st cu sr e^{s} ca
328 Sb Ubx/T(2;3)ap^{Xa}
329 se app
330 se e
331 se rt^{2} th/In(3L)P, Me
332 ss bxd k e^{s}/T(2;3)ap^{Xa}
333 st sr H^{2} ca/In(3R)P^{W}, st l(3)W ca
334 th st cp in ri p^{p}
335 th st cp in ri p^{p} bx sr e^{s}/TM1, Me ri p^{p} sbd^{1}
366 th st cp in ri p^{p} ss bxd sr e^{s}/TM1, Me ri p^{p} sbd^{1}
337 th st cp in ri Scx p^{p} sr^{R947}/In(3LR)Ubx^{130}, ri p^{p} Ubx^{130} e^{s}
338 th st cp Pc^{2}/In(3LR)DcxF, ru h D
339 tra/T(2;3)Me
340 W Sb/In(3LR)DcxF

Chromosome 4

401 ci
402 ci^{W}
403 C(4)RM, ci ey^{R}.gvl sv^{n}
404 spa
405 spa^{pol}

Multichromosomal

501 In(1)w^{m4}, w^{m4};In(2L+2R)Cy, Cy/ap^{4} vg
502 y^{2} su-w^{a} w^{a} bb;dsx^{601}/In(3LR)Ubx^{130}, ri Ubx^{130} e^{s}
503 bw;e
504 bw;st
505 bw^{V1}, dp b/In(3L+2R)Cy, Cy sp^{2};Sb/In(3LR)DcxF (ru h ca?)
506 C(1)DX, y f/B^{S}Y;C(4)RM, spa^{pol} & y Hw w/B^{S}Y;C(4)RM, spa^{pol}

Translocations

701 T(2;3)101, ru h e^{4} ro ca/In(3L+3R)P, Dfd ca
702 T(2;3)A, Bl;ru h D TA ss e^{s}/In(3LR) Ubx^{130}, ri Ubx^{130} e^{s}
--- T(2;3)ap^{Xa} (see 328, 332)
703 T(2;3)B;ru h D TB ss e^{s}/In(3L+3R)P
--- T(2;3)Me (see 339)
704 T(2;3)rn/In(2L+2R)Cy, Cy
705 T(2;3)rn/In(2L+2R)Cy, Cy sp
706 T(2;3)rn + In(2R)M, al/In(2L+2R)Cy, al^{2} Cy lt^{3} L^{4} sp^{2}
707 T(2;3)rn, Sb/In(3L+3R)P
708 T(2;3)spy

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

SYDNEY, AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Animal Husbandry

Wild Stocks

4 strains from N.S.W. and Victoria

Chromosome 1

In rst^{3}
w
w^{bl}
y

y w

Chromosome 2

b j
net

vg

Chromosome 3

e^{ll}

MÜNSTER, GERMANY: INSTITUT FÜR STRAHLENBIOLOGIE DER UNIVERSITÄT

Wild Stocks

Berlin wild

Chromosome 1

B

w
y

Altered Y

y/y^{+Y}

Chromosome 3

e^{ll}

Chromosome 4

ci ey^{R}

Multichromosomal

y sc^{S1} In49 sc^{8};bw;st

Attached 4

C(4)RM, ci ey^{R}/gvl sv^{n}

JERUSALEM, ISRAEL: HEBREW UNIVERSITY
Laboratory of Genetics

W. Wild Stocks

Berlin
Canton S
Qiryat Anavim
Iso Amherst

A. Chromosome X

1 B
2 B & C(1)DX, y
3 br w^e ec rb t^4
4 Bx
5 cv f
6 cx ct v g f & C(1)DX, y f
7 f
9 In(1)dl-49, y Hw m^2 g^4 & C(1)RM, y w f bb
10 In(1)sc^{4L} sc^{8R}, y sc^4 sc^8 Tu w & C(1)DX, y f/B^S Y
11 In(1)sc^{4L} sc^{8R} + S, y sc^4 sc^8 w^a B/y^+ Y & C(1)RM
12 In(1)sc^8, y^{S1} sc^8
13 In(1)sc^8, y^{S1} sc^8 pn1/In(1)FM6, y^{31d} sc^8 dm B
14 In(1)sc^8, y^{S1} sc^8 sn^3 w
15 In(1)sc^{L8L} sc^{8R}, sc^{L8} g^s v lzg sc^8 & C(1)DX, y f
16 In(1)sc^{S1L} sc^{8R} + S, sc^{S1} sc^8 w^a B ("Basc")
17 In(1)w^{m4}, y w^{m4}/y^+ Y
18 1(X)7 w sn/In(1), FM6, y^{31d} sc^8 dm B (Oster, 1965)
19 1(1)X15 y ac sc pn^{59j}/In(1)FM6, y^{31d} sc^8 dm B/y^+ Y
20 1(1)X15 y ac sc pn^{59j} spl sn/In(1)FM6, y^{31d} sc^8 dm B/y^+ Y
21 lz/ClB
22 mal
23 $N^{264-105}$(dm)/In(1)dl-49, y Hw m^2 g^4
24 pn^2
25 pn^2 w
26 pn^{66x2} cv
27 pn^{66x4} cv f & C(1)DX, y f
28 pn* - a series of induced pn - alleles
29 R(1)2, In(1)w^{vC}, w^{vC}/In(1)dl-49, y w lz/y^+ Y
30 R(1)2, y f & C(1)RM, y w f
31 ras v
32 sc pn^2
33 sc t^2 v f & C(1)DX, y f
34 sn
35 spl t v f & C(1)DX, y f
36 t v g
37 v
38 v f
39 v g f
40 v f su(f)
41 v mal^{bz}
42 w
43 w^a
44 w^a f 1(1)3DES $su^{1(1)3DES}$/In(1)FM6, y^{31d} sc^8 dm B 1/Y mal^+
45 w^e
46 w^{mo}
47 y
48 y ac sc 1(1)X6 1(1)3DES/In(1)FM6, y^{31d} sc^8 dm B
49 y ac sc 1(1)X6 v f 1(1)3DES/In(1)Basc, sc^{S1} sc^8 w^a B/Y w^+ Co y^+
50 y ac sc pn sn
51 y ct g f car & C(1)DX, y f
52 y^2 cv
53 y rb cx sn
54 y^{56K} f
55 y pn^{54C} spl
56 y^2 pn^{FG} sc 1(1)B57/In(1)FM6, y^{31d} sc^8 dm B 1/Y mal^+
57 y^2 sc
58 y^2 sc w^a ec & C(1)RM, y^{+60d19} sc w^{aRM} (M. Green)
59 y sn
60 y v
61 y v f
62 y^2 v f car
63 y w
64 y w sn
65 y w^a spl & C(1)DX, y f
66 y^2 cho^2
67 y pn cho
68 Dp(1;1)112, y w f/w^+Y

Y. Chromosome Y
a Y-derivatives

1 B^S Y y^+/C(1)DX, y f
2 1(1)J1$^+$ Y/1(1)J1 $sc^{J1(+)}$ & C(1)DX, y f (Oster, 1965)
3 Y mal^+ #2/y^2 v mal
4 Y pn^- w^+/y w^a/C(1)RM, y pn
5 Y w^+/y w^a(Nicoletti, 1966)
6 Y w^+ Co/y w^a (Nicoletti, 1966)
7 Y w^+ Co y^+/y w^a (Nicoletti, 1966)
8 y^+ Y w^+/y w^a (Nicoletti, 1966)
9 Y mal^+/pn v 1(1)B275/In(1)FM6, y^{31d} sc^8 dm B
10 y^+ Y mal^{106}/1(1)A112/In(1)FM6, y^{31d} sc^8 dm B (Schalet, 1970)
11 T(1;Y B^S)19F#132/In(1)FM4, y^{31d} w dm f
12 *Y 1^+/y 1(1)/In(1)FM6, y^{31d} sc^8 dm B - induced derivatives of various length
13 *Dp(1;f)/XY^L.Y^S(108-9), y^2 su(w^a)w^a 1(1)SH-4/In(1)FM6, y^{31d} sc^8 dm B - induced fragments of different length
14 Y bb$^-$/In(1)dl-49.B^{M1}, y B^{M1} & C(1)RM, y v bb

b X-Y combinations

14 Y^S X·Y^L, In(1)EN, Y^S B y·Y^L & C(1)RM, y^2 su(w^a)w^a bb/O
15 Y^S X·Y^L, In(1)EN, Y^S v cv y·Y^L y^+ & C(1)RM, y^2 su(w^a)w^a bb/O
16 Y^S X·Y^L, In(1)EN, dl-49, Y^S car f v y·Y^L & C(1)RM, y^2 su(w^a)w^a bb/O
17 X Y^L·Y^S(108-9), y^2 su(w^a)w^a Y^L·Y^S & C(1)RM, y v bb/O
18 X Y^L·Y^S(108-9), y^2 su(w^a)w^a 1(1)SH-4 Y^L·Y^S/In(1)FM6, y^{31d} sc^8 dm $\overline{B}$/Y mal^+
19 X Y^L·Y^S(108-9), y^2 su(w^a)w^a 1(1)SH-5 Y^L·Y^S/In(1)FM6, y^{31d} sc^8 dm $\overline{B}$/Y mal^+
20 X Y^L·Y^S(108-9), y^2 su(w^a)w^a 1(1)SH-16 Y^L·Y^S/In(1)FM6, y^{31d} sc^8 dm $\overline{B}$/Y mal^+
21 X Y^L·Y^S(108-9), y^2 su(w^a)w^a 1(1)SH-17 Y^L·Y^S/In(1)FM6, y^{31d} sc^8 dm $\overline{B}$/Y mal^+

B. Chromosome 2

1 b
2 b cn bw
3 Bl L/In(2)Cy
4 bri
5 bs^2
6 bw
7 bw^{V4}/In(2LR)SM1, al^2 Cy sp^2
8 bw^{V5}/In(2LR)SM5, al^2 Cy lt^v sp^2
9 C(2L)RM;C(2R)RM
10 C(2L)RM, b;C(2R)RM, cn
11 C(2L)RM, j63;C(2R)RM, px
12 cl
13 cn
14 cn bw
15 cn bw bs^2
16 dp
17 dp^{lv} Sp cn/In(2L)Cy, S^2 Cy cn
18 fes ms cn bw/In(2L)Cy, Cy cn bw
19 fes ms cn bw/In(2LR)CyO, dp^{lvI} Cy pr cn^2
20 fes ms pr cn/In(2LR)CyO, dp^{lvI} Cy pr cn^2
21 fes ms cn sp/In(2)Cy, net dp^{lvI} Cy b pr Bl lt^3 cn^2 L^4 sp^2
22 In(2L)Cy, Cy L/In(2LR)bw^{Vl}, bw^{Vl} ("Cy L/Pm")
23 In(2LR)CyO, dp^{lvI} Cy pr cn^2/In(2LR)bw^{Vl}, bw^{Vl} ("Improved Cy/Pm")
24 pr vg
25 S fes Sp ms ts cn mr crs/In(2)Misl+Cy, al^2 dp^{lvI} Cy pr Bl cn^2 L^4
26 stw ap^{blt}
27 stw bw bs
28 tu^{ql56} (E. Goldschmidt)
29 vg

C. Chromosome 3

1 $Antp^R$/Sb
2 C(3L)RM;C(3R)RM
3 C(3L)RM, se h^2 rs^2;C(3R)RM, sbd gl e^s
4 ca^{572j} $III a^3$/In(3L)P, Me, ri sb^1
5 ca bv
6 ca Kpn
7 ca^{nd}/In(3LR)TM3, Sb Ser
8 cp
9 cp st th
10 Dr^{Mio} Kpn/In(3LR)TM3, ri p^p bx^{34e} Ser
11 Gl Sb/In(3)LVM
12 e
13 gl^2 e^4
14 gl^{GZ63}
15 h
16 h^2
17 Hn^{r-3}
18 In(3)D/Sb
19 Kpn#1
20 Pyd
21 RK-C8/Sb (Lifschytz)
22 RK-S3/Sb (Lifschytz)
23 ri p^p
24 ru h th st cu sr e^s ca ("rucuca")
25 se
26 se e
27 se ss k e^s ro
28 ss^a
29 st
30 st in ri p^p

D. Chromosome 4

1 bt^D/ci^D
2 ci ey
3 ey^{opt}
4 ey^D/Df(4)M

M. Multichromosomal

1;2

1 T(1;2)Bld, Bld w^a;+/w;In(2)Cy
2 v;bw
4 y;In(2)Cy, Cy L/In(2LR)bw^{Vl}, bw^{Vl}

1;2;3

1 pn^2/In(1)FM6, y^{31d} sc^8 dm B/w^+Y;αGpdh-1^F Adh^F;ca Kpn
2 y;αGpdh-1^S Adh^S;mwh

1;3

1 Hw;ca Kpn & C(1)RM, y
3 pn^{59j}/C(1)RM, y/w^+ Y;ca Kpn
4 pn;In(3)D/Sb
5 pn;RK-C2/Sb (Lifschytz)
6 pn;ry
7 pn;se
8 v g 1(1)AA33 1(1)D13/In(1)FM6, y^{31d} sc^8 dm B/Y mal^+;ca Kpn
9 v g 1(1)B275 1(1)3DES/In(1)FM6, y^{31d} sc^8 dm B 1/Y mal^+;ca Kpn
10 w^a ec/C(1)DX, y;tra
12 y f;mwh
13 y pn;RK-9/Sb (Lifschytz)

2;3

1 bw;e
2 bw;st
3 cn bw;ri e
4 dp;e
5 fes ms cn sp/In(2LR)CyO, dp^{lvI} Cy pr cn^2;h ri e^s/ In(3)Me, Me ri
6 In(2)Cy, Cy/In(2LR)bw^{V1}; Sb/In(3)D
7 pr;st
8 T(2;3)606, In(2)Cy Sb

3;4

1 vli;ey^2

2;3;4

1 bw;e;ci ey^R

L. Lethals

a lethals in proximal segment (covered by Y mal+)

1 l(1)A19/Basc
2 l(1)A33/FM6
4 l(1)A112/FM6/Ymal+
5 l(1)A118/FM6/Ymal+
6 l(1)A122/FM6/Ymal+
7 l(1)A209/FM6
8 l(1)B3/FM6/Ymal+
9 l(1)B12/FM6
10 w^a l(1)B12/FM6l/Ymal+
11 y l(1)B56/FM6/Ymal+
12 l(1)B57/FM6/Ymal+
13 l(1)B96/FM6
14 l(1)B111/FM6/Ymal+
15 l(1)B214/FM6/Ymal+
16 l(1)B220/FM6
17 y^2 v l(1)B264/FM6/ Ymal+
18 y^2 pn^{FG} cv l(1)B275/FM6l/ Ymal+
19 pn^{59j} f os^o l(1)AA33/FM6l/ Ymal+
20 l(1)D13/FM6
21 y^2 v f l(1)3DES/FM6/Ymal+
22 w^a f l(1)3DES/FM6/pn^- w^+Y
22a pn^{MS2} l(1)3DES/FM6/pn^- w^+Y
23 w^a f l(1)3DES/FM6/Ymal+
24 y^2 v f l(1)E54/FM6/Ymal+
25 cv f l(1)E81/FM6
27 l(1)F280/FM6/Ymal+
28 l(1)F319/FM6
29 l(1)F338/FM6
30 l(1)F365/FM6
31 l(1)F422/FM6
32 l(1)F441/FM6/Ymal+
33 l(1)F442/FM6
34 y^2 v f l(1)P19/FM6/Ymal+
35 y^2 v f l(1)P235/FM6
36 f os^o l(1)P253/FM6/Ymal+
37 pn l(1)P425/FM6
38 pn l(1)P431/FM6
39 pn l(1)P464/FM6/Ymal+
40 w l(1)R9-1/FM6
41 w l(1)R9-5/FM6
42 w l(1)R9-6/FM6
43 w l(1)R9-10/FM6
44 w l(1)R9-14/FM6
45 w l(1)R9-15/FM6
46 w l(1)R9-18/FM6
47 w l(1)R9-21/FM6
48 w l(1)R9-22/FM6
49 w l(1)R9-26/FM6
50 w l(1)R9-29/FM6
51 w l(1)R9-31/FM6/Ymal+
52 w l(1)R10-2/FM6
53 w l(1)R10-3/FM6
54 y^2 v f l(1)Q2/FM6/Ymal+
55 y v f l(1)Q56/FM6/Ymal+
56 w^a l(1)Q219/FM6l/Ymal+
57 y^2 v f l(1)Q256/FM6/Ymal+
58 l(1)Q414/FM6/Ymal+
59 y^2 v f l(1)Q456/FM6/Ymal+
60 l(1)Q463/FM6/Ymal+
61 y l(1)Q464/FM6/Ymal+
62 w l(1)W1/FM6/Ymal+
63 w l(1)W2/FM6/Ymal+
64 w l(1)W3/FM6/Ymal+
65 w l(1)W4/FM6/Ymal+
66 w l(1)W5/FM6/Ymal+
67 y l(1)Y1/FM6/Ymal+
68 w l(1)WL2/FM6/Ymal+
69 y ac sc l(1)X1/FM6
70 y ac sc l(1)X2/FM6
71 y ac sc l(1)X3/FM6
72 pn^2 w l(1)X4/FM6

b. lethals in distal segment (covered by Y w^+)

1 y ac sc l(1)X5/FM6
2 y ac sc pn l(1)X6/FM6
3 y ac sc l(1)X7/FM6/Y w^+ Co y^+
4 y ac sc l(1)X9/FM6
5 y ac sc l(1)X10/FM6
6 y ac sc l(1)X10/FM6/y^+ Y w^+
7 y ac sc l(1)X11/FM6
8 y ac sc l(1)X12/FM6
9 l(1)F337/FM6/Y w^+ Co y^+
10 l(1)F362/FM6/Y w^+ Co y^+
11 l(1)F380/FM6
12 l(1)F459/FM6/Y w^+ Co y^+
13 sc rb l(1)K1/FM6/Y w^+ Co y^+
14 y w l(1)K2/FM6/Y w^+ Co y^+
15 sc rb l(1)K5/FM6
16 y^2 cv l(1)K6/FM6
17 y w l(1)K7/FM6
18 y w l(1)K15/FM6
19 y w l(1)K27/FM6/Y w^+ Co y^+
20 y^2 cv l(1)K28/FM6
21 y^2 cv l(1)K49/FM6
22 sc rb l(1)K50/FM6
23 sc rb l(1)K60/FM6
24 sc rb l(1)K74/FM6
25 y^2 cv l(1)K84/FM6
26 y^2 cv l(1)K94/FM6
27 y^2 cv l(1)K95/FM6

HYDERABAD, INDIA: OSMANIA UNIVERSITY
Department of Genetics

Wild Stock

ORK

Chromosome 1

1 Yellow
2 M-5
3 W
4 B

Chromosome 2

1 Vg
2 dp b cn bw
3 Cy Bl/1^2
4 Cy C^4/pm
5 Cy/Bl 1^2/D/LVM
6 g^{137} dp cm^2 cl
7 g^{45} b cn vg B/In nr px sp

Multichromosomal

1 bw;st
2 bw;vg
3 o_1;bw;st
4 vg;f
5 y ct^6 f Sac^3 w^a ct f:=

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Biology

Note: Several hundred additional stocks are maintained.

Wild Stocks

2 Chicago

Chromosome 1

10 Bx^3 mal
28 lix
32 mal^{bz}
171 pn^{51h8} spl
47 sc z^{59d15}/C(1)DX, y f
48 sc z w^{z1}
49 sc z w^{zm}
67 v^{50j}
192 w^{+u} Etd f/FM1-G
159 y z w^{Bwx} w^{crr}/C(1)DX, y f
153 y ac w^{ch} w^{sp2} spl/C(1)RM, y w
150 w^{Bwx} w^a spl
411 y^2 su(w^a)w^a w^{ch} spl/C(1)DX, y f
177 z w^{11e4}

Chromosome 1 Inversions

129 In(1)sc^{4L}, In(1)sc^{8R}, y sc^4 sc^8 cv/B^S Y/C(1)DX, y f
127 In(1)sc^{4L}, In(1)d1-49, In(1)sc^{8R}, y sc^4 sc^8 w^a/C(1)DX, y f
465 In(1)sc^{4L}, In(1)sc^{V2R}, y sc^4 sc^{V2}/y^+ Y/C(1)DX, y f
120 In(1)sc^{8L}, In(1)S, In(1)sc^{4R}, w^a B/C(1)DX, y f;Dp(1;2)sc^{19}/In(2L+2R)Cy, Cy cn^2
406 In(1)sc^{8L}, In(1)sc^{L8R}, w^a/C(1)DX, y f; Dp(1;2)sc^{19}
407 In(1)sc^{8L}, In(1)sc^{S1R}, B/C(1)DX, y f; Dp(1;2)sc^{19}
405 In(1)sc^{8L}, In(1)sc^{V2R}, y^{31d} sc^{V2} f B/C(1)DX, y f
487 In(1)sc^9, sc^9 w^a sn^3 f Bx
437 In(1)sc^{L8}, sc^{L8} w^a sn^3 car/In(1)d1-49, y Hw m^2 g^4
149 In(1)sc^{L8}, sc^{L8} w^a sn^3 car/B^S Y/C(1)DX, y f
416 In(1)sc^{L8}, sc^{L8} w^a cho^2 car/C(1)DX, y f
164 In(1)sc^{L8L}, In(1)sc^{4R}, sc^{L8} sc^4 m car/B^S Y/C(1)DX, y f
174 In(1)sc^{L8L}, In(1)sc^{8R}, sc^{L8} sc^8 car/B^S Y/C(1)DX, y f
87 In(1)sc^{L8L}, In(1)S, In(1)sc^{S1R}, sc^{L8} B/C(1)DX, y f
417 In(1)sc^{L8L}, In(1)sc^{S1R}, sc^{L8} sn^3 car/B^S Y/C(1)DX, y f
203 In(1)sc^{L8L}, In(1)sc^{V2R}, sc^{L8} sc^{V2} m car/C(1)DX, y f
141 In(1)w^{m4L}, In(1)sc^{L8R}, w^a sn^3/In(1)d1-49, y Hw m^2 g^4/B^S Y
414 In(1)sc^{S1L}, In(1)sc^{4R}, y sc^{S1} m/B^S Y/C(1)DX, y f
304 In(1)sc^{S1L}, In(1)sc^{8R}, y sc^{S1} sc^8 cv v f/B^S Y/C(1)DX, y f
84 In(1)sc^{S1L}, In(1)d1-49, In(1)sc^{8R}, y sc^{S1} sc^8 w^a v f/C(1)DX, y f
110 In(1)sc^{S1L}, In(1)S, In(1)sc^{8R}, sc^{S1} sc^8 w^a B bb^{83}/In(1)AM, y/B^S Y
214 In(1)sc^{S1L}, In(1)sc^{L8R}, y sc^{S1} w^a m/B^S Y/C(1)DX, y f
442 In(1)sc^{S1L}, In(1)sc^{L8R}, y sc^{S1} w^a m/In(1)d1-49, y Hw m^2 g^4
212 In(1)sc^{S1L}, In(1)sc^{V2R}, y sc^{S1} sc^{V2}/B^S Y/C(1)DX, y f
409 In(1)sc^{V2L}, In(1)d1-49, In(1)sc^{8R}, sc^8 v f B/C(1)DX, y f
408 In(1)sc^{V2L}, In(1)sc^{L8R}, w^a/B^S Y/C(1)DX, y f;Dp(1;2)sc^{19}
316 In(1)sc^{V2L}, In(1)S, In(1)sc^{S1R}, B/C(1)DX, y f;Dp(1;2)sc^{19}

Chromosome 1 Duplications and Deficiencies

148 Df(1)w^{rG}, w^- spl
138 Df(1)w^{-59k13}, w^{-59k13} spl;Dp(1;3)w^{Vco}
152 y sc z w^{rdf} spl/C(1)DX, y f
154 y^2 w w^{rdf} ec f
412 Dp(1;f)1337/$XY^L.Y^S$, y w f/C(1)RM, y w
114 Dp(1;f)60g, y^{31d}/C(1)RA, In(1)d1-49, y Hw B/X·Y, y v

Ring 1 Chromosomes

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

Reversed Acrocentric Chromosome 1

168 C(1)RA60g, +-In(1)sc^8, In(1)d1-49, y Hw^- y^- sc^8 B/Sp(1;f)60g, y^{31d}/$Y^S X.Y^L$, y v

X-Y Combinations

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

Y Derivatives

198 B^S Y y^+/y v;bw
199 bw^{54k} Y y^+/y v;bw
200 y^{54e} ac^{54e} Y/y v;bw
202 w^+ Y/y ac br pn w^e spl
205 Ybbl (Spofford)
218 $Y^L.sc^{S1}$/X·Y^S, w^a ec sn

221 Y^L13/X·Y^S, y f/C(1)RM, y w
222 Y^L16/X·Y^S, y ct^6 f/C(1)RM, y v bb;bw
223 Y^L sc^{S1}2/X·Y^S, y f/C(1)RM, y w
224 Y^L sc^{S1}/X·Y^S, y ac w^a ct^6 f/C(1)DX, y f
226 R(Y)L15/X·Y^S, y f/C(1)RM, y w
208 sc^{V1}·Y^S/X·Y^L, y v f bb/C(1)DX, y f
209 Y^S y^+5/X·Y^L, B/C(1)RM, y w
211 Y^S y^+7/X·Y^L, B/C(1)RM, y w
213 Y^S·Y^S2/X·Y^L, y v f/C(1)DX, y f
215 Y^S y^+ bb^+8/X·Y^L, y g^2 B/C(1)RM, y v bb;bw
216 Y^S/X·Y^L, y w^a rb
433 Y^S/g^2 B Y^L/C(1)RM, y w

Chromosome 2

225 C(1)RM, y w;abo/In(2L+2R)Cy, Cy
227 abo/In(2L+2R)Cy, Cy
250 In(2LR)lt^{m3}

Chromosome 3

410 c3G

Multichromosomal

310 lix;ry^2
311 lix;sf^2
312 mal;ry^2
313 pn;ry^2
287 y sn;c3G/D
341 T(2;3)lt^{m29}
342 T(2;3)lt^{m100}/lt stw^3 l
54 y w;Su(var)
57 y w;Su(var)$^+$
488 y w/C(1)DX, y f;mwh
434 Y^S w y·Y^L y^+/Y/C(1)RM, y w;Su(var) Dp(1;3)N^{264-58}, $w^{m264-58}$/Su(var)
S1 Y^S X·Y^L, In(1)EN, y w·y^+/C(1)RM, y w; S(var)$^+$ Dp(1;3)N^{264-58}, $w^{m264-58}$/+
S2 y w;Su(var) Dp(1;3)N^{264-58}, $w^{m264-58}$/Su (var)

VARANASI, INDIA: BANARAS HINDU UNIVERSITY
Department of Zoology

Revision of list in DIS 48:14

Chromosome 1: second and ninth stocks deleted; Chromosome 2: fifth and sixth deleted; Multichromosomal: second stock deleted

CATONSVILLE, MARYLAND: UNIVERSITY OF MARYLAND BALTIMORE COUNTY
Department of Biological Sciences

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

Chromosome 1

21 w^{+u} Etd f/In(1)sc^{4L} sc^{8R} + d149, y sc^4 sc^8 w^a
24 w^{+u} w^{sp}
29 w^{Bwx} w^{+u}/FM3
34 w^{udf}/C(1)DX, y f
68 y^2 m B
38 y^2 su(w^a)w^a w^{+u}/Basc, bb^{83}
40 y^2 su(w^a)w^a w^{udf}/C(1)RM, w^a

Chromosome 2

199 S^x Tft bw^D/SM1

Chromosome 3

200 ve R D^3 Ser/Ubx^{130}

Compound-X

131 C(1)RA60g, y/y/Y, su(f)$^+$
132 C(1)RA60g, y Hw B + In(1)d149/$\overline{XY}$, y v/ DP(1;f)60g, y^{31d}
126 C(1)RM, y/$\overline{XY}$, v f B/O;C(4)RM, ci ey^R/O (no free Y)

Compound Autosomes

133 C(2L)RM4, dp;C(2R)RM4, px
23 C(2L)RM, b;C(2R)RM, cn
134 B^SY;C(2L)RM4, dp;C(2R)RM4, px
135 +/C(1)RM, y^2/B^SY;C(2L)RM, +;C(2R)RM, +
124 +/C(1)RM, y^2/B^SY;C(2L)RM4, dp;C(2R)RM, +
174 C(2L)RM4, dp;C(2R)RM4, px;Ubx^{130}/e^s l(3)G58
201 Y^SX·Y^L, In(1)EN, y/C(1)RM, y^2;C(2L)RM4, dp;C(2R)RM, + (no free Y)
138 B^SY;F(2L)2, nub^2 b pr;C(2R)cn
137 C(2L)dp;F(2R)1, bw
56 CyO/F(2L)1, dp;F(2R)1, cn c bw
139 C(3L)RM, se h^2 rs^2;C(3R)RM, sbd gl e^s
54 y^2;C(2L)RM, dp;C(2R)RM, px;C(3L)RM, h rs^2;C(3R)RM, +

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

Wild Stocks

1 Algeria
2 Amherst-3
2A Berlin (Kalisch)
3 Boa Esperance, Minas Gerais, Brazil
4 Canton-S
5 Crimea
6 Curitiba
7 Falsterbo
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 San Miguel, Buenos Aires, Argentina
17 Stäket
18 Tunnelgatan
19 Ultuna
20 Örebro

Chromosome 1

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

Special Stocks

1101 sc z w^{zm+}
1102 sc z w^{zmzrb}
1103 y z w^{zm} sn^3
1104 y w f:= z w^{z1}
1105 y z $w^{zm(z)}$ sn^3
1106 y z w^{zmzw} sn^3
1107 sc In(1)z w^w
1108 w^{62}/FM7
1109 y z^a 48
1110 z w^w spl

Chromosome 2

2001 al b c sp
2002 Cy/bw^{Vl}, ds^{33k}
2003 Cy cn/S
2004 ds dp
2005 l(2)gl a px or/SM5, al^2 Cy lt^v sp^2
* 2006 M(2)1^2/SM1, al^2 Cy sp^2
2007 M(2)S7/SM5, al^2 Cy lt_v sp^2
2008 ms(2)2 b cn sp/dp^{lvl} Cy pr Bl lt^3 cn^2 L^4 sp^2
2009 ms(2)2 cn rm/Cy cn^2 L^4 sp^2
2010 net bw crs/dp^{lvl} Cy pr Bl lt^3 cn^2 L^4 sp^2
2011 nw^2/In(2L)Cy, In(2R)NS
2012 S^2 Cy pr Bl cn^2 L^4 bw sp/ In(2L)NS, In(2R)NS px sp
2013 S Sp Bl bw^D/al^2 Cy lt^3 L^4 sp^2
2014 shr bw^{2b} abb sp/SM5, al^2 Cy lt^v sp^2
2015 ta cn rk^5 bw/al^2 Cy pr Bl cn^2 L^4 sp^2

* M(2)21C1-2 (associated with Df(2L)al, stock 6021)

2016 vg
2017 vg bw

Chromosome 3

3001 c(3)G
3002 ca K-pn
3003 D^3/In(3L)P
3004 Est-6^S Est-C^F
3005 Est-6^S Est-C^S
3006 Est-6^F Est-C^F
3007 Est-6^S Lap-A^0
3008 Est-6^F
3009 Est-6^S
3010 Est-6^S/6^F
3011 Gl Sb/LVM
3012 gl
3013 In(3LR)D cx F/Sb
3014 jv Hn^r h
3015 Lap-A^0
3016 M(3)h^y/In(3L)P, Me
3017 M(3)w^{124}/In(3R)C, e l(3)e
3018 ri ss
3019 ru h st p^p ss e^s
3020 ru h th st p^p H e^s ro/TM6, ss$^-$ bx^{34e} Ubx^{P15} e
3021 ru h th st cu sr e^s ca/TM3, ru Sb Ser
3022 ry cd
3023 st
3024 st c(3)G ca/ve h th c(3)G Sb Ubx^{130}
3025 st c(3)G ca/TM1, Me ri sbd^1 (sp^2)
3026 st p e
3027 st ry
3028 st ss e^{11}

Chromosome 4

4001 ci gvl bt
4002 ci^D spa^{pol}/spa^{cat}
4003 sv^n
4004 spa^{pol}

Multichromosomal

5001 FMA3 y^2;net;sbd^2;spa^{pol}
5002 f^{36a};mwh jv
5003 sc z is;Cy;Ubx^{130}/Xa
5004 sc z is;jv Hn^r h
5005 sc z is;spa^{pol}/spa^{cat}
5006 sc^{S1}InS w^a sc^8;Cy; Ubx^{130}/Xa
5007 $su(s)^2$ v;vw
5008 w^{ch};Su-w^{ch}/Cy cn
5009 w^{m4} y^{511};E(var)7/Cy
5010 y f:=;bw;e;spa^{pol}
5011 y^2 sc w^a w^{ch} fa;Cy; Ubx^{130}/Xa
5012 ♀ z+ sn:= net;spd^2;spa^{pol} ♂ sc z is;net;spd^2;spa^{pol}
5013 SM1:Ubx (Kalisch)
5014 bw;st
5015 cn bw;e^{11}
5016 Cy cn/S;D/In(3L)P
5017 L sp;th
5018 L^2/+ sp;th
5019 sp;th
5020 Pod-R

Deficiencies

Deficiencies - X

6001 Df(1)bb, y sl^2 bb$^-$/FM4, y^{31d} sc^8 dm B
6002 Df(1)w^{258-11}, y/In(1) dl-49, y Hw m^2 g^4
6003 Df(1)w^{258-42}/FM4, y^{31d} sc^8 dm B
6004 Df(1)w^{258-45}, sc z/FM4
6005 Df(1)w^{258-45}, y/FM4, y^{31d} sc^8 dm B
6006 Df(1)w^{258-45}, y w spl dm; Dp(1;3)w^{vco}/y w f:=
6007 Df(1)w^{258-48}, y sc^5 spl; Dp(1;3)w^{vco}/y f:=
6008 Df(1)w-N(EMS Kalisch)/FM7

Deficiencies - Y

6011 Df(Y)Y^{bb-}, y^2 eq
6012 Df(Y)Y^{bb-}, y^2 sc z w$^-$ spl/y pn:=

Deficiencies - 2

6021 Df(2L)al/In(2L+2R)Cy, Cy E(S)
6022 Df(2L)S2/In(2L+2R)Cy;Cy E(S)
6023 Df(2L)S3/SM1, al^2 Cy sp^2

Deficiencies - 4

6031 Df(4)M/ey^D

Duplications

6041 Dp(1;1)z^{59d15}, sc z^{59d15}/y f:=
6042 Dp(1;1), z(w^{a4}/w^a)/y f:=
6043 Dp(1;1)w^a, (w^a/w^a)/y f:=
6044 Dp(1;1), (w^{bf}/w^a)ec
6045 Dp(1;1)112, y f
6046 Dp(1;Y)y w^a/Y·w^+ Co

Translocations

7001 ♀ C(1)DX yf/B^S·Y C(4)pol ♂ yHw w/B^S·Y C(4)pol
7002 T(1;4)w^{258-18}/y Hw dl-49 m^2 g^4
7003 T(1;4)yw^{258-18}/y w dm
7004 T(1;4)w^{m5}/w;cy^D
7005 T(2;3)bw^{VDe4}/Cy
7006 T(2;3)Sb^V, Sb^V, In(3R) Mo, In(3LR)P35/SM1, al^2 Cy sp^2;In(3LR)Ubx^{130}, Ubx^{130} e^S
7007 T(1;Y)y/y^+Y

Triploid

8001

Extra Y

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

Closed-X

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

X with Y fragments attached

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

LYON, FRANCE: UNIVERSITÉ CLAUDE-BERNARD
Faculte des Sciences, Biologie Générale et Appliquée

Wild Stocks	Chromosome 1	Chromosome 2	Multichromosomal
Algerie	w	b cn vg bw	st;bw v;bw cn;bw

PASADENA, CALIFORNIA: CALIFORNIA INSTITUTE OF TECHNOLOGY
Division of Biology

Note: For a complete listing of stocks see DIS 47:7

Additions:

153a w^a mw mit
185a y z^a w^{65a25} spl sn^3/y w f:=
185b y z^a w^{cn}/y w f:=
223a ap^{wow}
671a y ac;esc^D/+ (1;2)
680a FMA3, y^2/z w^{11E4};In(2LR)O, dp^{lvl} Cy pr cn^2/Sp Bl L^{rm} (1;2)
709a $XY^L.Y^S$, y^2 su(w^a)w^a $Y^L.Y^S$
710a $Y^SX.Y^L$, In(1)EN/y^2 su(w^a)w^a bb
715a y w f/y w^a/w^+.Y
718a R(1)5A, y w f B/In(1)dl-49, y v f car/ B^S.Y (Pasztor's unstable ring)
755a kar^2, Df(3R)ry^{75}/In(3LR), M(3)S34 kar^4 ry^2 Sb
758a Df(4)M4/ey^D

820
*
* In(2LR)O 680a

After 851, under new heading:
Translocations - 1;3;4

851a Df(1)sc^8, w^a/y f:=;T(1;3;4)sc^{J4} ey^D

Corrections:

270 cn 1(2)crc/SM5, al^2 Cy lt^v sp^2
425 Su(H)/In(2L+2R)Cy, Cy pr
759 Dp(1;f)101 Dp(1;f)101;In(1)sc^8, Df(1)sc^8, w^a
760 Dp(1;f)107 Dp(1;f)107;In(1)sc^8, Df(1)sc^8, w^a
761 Dp(1;f)118 Dp(1;f)118;In(1)sc^8, Df(1)sc^8, w^a
762 Dp(1;f)135 Dp(1;f)135;In(1)sc^8, Df(1)sc^8, w^a
824 In(3L)P In(3L)P, mot-36e/R

The following stock

227 Ata . . . . . .868 should simply read:
* Ata 868

Deletions: 199, 274, 661, 677, 692b, 700, 708, 715, 728, 756, 758, 766, 768 and 856

BOCHUM, GERMANY: RUHR-UNIVERSITÄT
Institut für Genetik

Chromosome 1

Bx
ec ct^6 lz^g v f/y w f:=
f Bx car/y w f:=
sc z ec cv/y w f:=
sc z w^{11E4} spl sn^3
sc z w^{17G2} ec
sc z w^{z1}/y w f:=
sn^3 lz^{y4} v/y f:=
v
v f^{3n} car/y f:=
w sn^3
w sn^3 m
w sn^3 lz^{46}
w^a
w^{co} spl cv f
y ac z w^{zmz}/y f:=
y v f
y w f
y w sn^3 lz^{46}
y z w^{zm} sn^3
y z^a
y^2 sc w^i
z spl cv f

Chromosome 2

al dp b pr c px sp
cn bw
vg
vg bw

Chromosome 3

cu
mal
ru h th st cu sv e^S ca
ry
se cu
se h
ve h th

Multichromosomal

sc z is;Su(z)^1/Cy (1;2)
w;ve h (1;3)
w;ey (1;4)
z spl cv f;Cy;Ubx^{130}/Xa (1;2;3)
bw;st (2;3)
Cy/Pm;CxD/Sb (2;3)
Cy;Ubx^{130}/Xa (2;3)
SM1, al^2 Cy sp^2/In(2LR) 102 ds^w sp^2;In(3LP,3RC) Sb e^s/Ubx^{130} e^s (2;3)

Closed-X

R(1)2, w^8 sn^3 f/y w f:=

Duplications

Dp(1;1)B
Dp(1;1)B + Bx^r, car/ y f:=
Dp(1;1)Bx^{r49k}, v f car/y f:=
Dp(1;1)Gr, y^2 (w^- spl sn^3) (w^c sn^3)/y Hw dl-49 w m^2 g^4
Dp(1;1)lz-1, (lz^{50e}) (lz^{y4})v/y f:=
Dp(1;1)lz-2, (lz^{50e}) (lz^{y4})v/y f:=

Inversions

In(1)sc^8
In(1)w^w,sc z

CLEVELAND, OHIO: CLEVELAND STATE UNIVERSITY
Department of Biolcgy and Health Science

Wild Stocks

Oregon-R (Texas)
Oregon-R (Lewis)
Lausaunne-S

Chromosome 1

y^{68c}
br
pn^2
w
w^{Bwx}
w^{co}
w^{e}
w^{sat}
Ax
Co
fa
fa^{no}
nd
ec
rb
bo
cx
rux^2
vs
cm
lz^K
ras^2
v
dy
s
f
os

Bx^3
Bx^J
car
sw
mal^{bz}
$y^2\ w^a\ ct^6$
y cv v f
$y^2\ w^a\ ct^6$ m f
w m f
fa fa^{no} sn
ec dx
cv f
g^1 cv net^{one}
f B
dor/ClB
N^8/y Hw In49 m^2 g^4
fa Nj^{24c} sn3 B & y f:=
sc ct^6 car/y f:=
sc^{51} B InS w^a sc^8
("Basc")

Chromosome 2

net
ho
dp
cl
J^{34e}
ab
nub^2
rk^4
b
Coi
el
hk

pr
Alu
stw
ad
sca
L
L^2
c
bw^{2b}
bw^D
Pin
Hx
al b c sp
b vg
Bl/esc
Cy/Pm (dp;b)
dp^{02} dp^{lvl} b/Cy Bl L
ds^{38k}/Cy(2L)dp^2 b pr
U/cgC
Px^2 bw sp/SM1 al^2 Cy sp^2
Bl L^2/Cy dp^2
b Go/Gla
Pu^2/SM1 al^2 Cy sp^2
Ruf/ds^{38k} Pm

Chromosome 3

ve
se
eyg
app
th
st
cp

W
Ki
drb
bx^{34e}
Cbx
e^{ll}
cd
bar-3
rsd
ca
bv
ca K-pn
ve h th
se ss k e^s ro
D/Gl
Gl Sb H/Payne
Bd^g/In3R C, 1(3)a
Mc/ap^{Xa}

Chromosome 4

bt
ey^2
spa^{pol}
sv^{35a}
gvl
ar/ey^D
ci^D/4-sim

Multichromosomal

$b;e^{ll}$
bw;e
Cy/Pm(dp b);st
Cy/Pm(dp b);D/Sb

BALTIMORE, MARYLAND: JOHNS HOPKINS UNIVERSITY
Department of Biology

Chromosome 2

b, ADH^{n2}, cn, pr
b, ADH^{n3}, cn
b, ADH^{n4} ADH^{n5}, pr
b, ADH^{n6}, cn, vg
b, ADH^{n7}, cn vg
b, ADH^{n8}, cn, vg
b, ADH^{n9}, cn, vg
b, ADH^{n10}, cn, vg
Df(2L)64j/CyO, Adh neg^A
Df(2L)64j/CyO, Adh neg^B

Chromosome 3

Gl/TM1, Me ri sbd^e
Gl/TM1, Me ri sbd^e e
Gl Sb Ser/TM2, Ubx^{130} e^s
Gl Sb Ser/TM3 y^+ ri p^p Sep bx^{34e} e^s
mwh cp in ri p^p red e
mwh e
mwh red e
mwh red e Pr^L
mwh red e Ser/TM1, Me ri sbd
red

Multichromosomal

w;ADH^{n1}
mal;Df(2L)64j/CyO
mal;b, cn, vg

VIENNA, AUSTRIA: UNIVERSITY OF VIENNA
Institut für allgemeine Biologie

Revision of list in DIS 46:22
Wild Stocks add: St. Pölten
Kairo
delete: Norwegen

LONDON, ENGLAND: BIRKBECK COLLEGE
Department of Zoology

Wild Stocks

Oregon R
Oregon +

Chromosome 1

ClB/w m f
Muller-5
N^8/dl-49 y Hw m^2 g^4
w
w m f
w^a
v
y
y w m f

Chromosome 2

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

Chromosome 3

e
e ss^a se
Sb/+
ss
ss^a
ve

Chromosome 4

ey^2

Multichromosomal

bw;e
bw;ss^a
w;ss^a

Inversions

In(1)sc^{S1L} sc^{4R} sc^{S1} sc^4
cv v B/C(1)DX y f/B^S Y
In(1)sc^{4L} sc^{8R} y sc^4 sc^8
cv v B/C(1)DX y f/B^S Y

Attached X

y/w^{bl} wy f

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

Wild Stocks

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

Chromosome 1

101 B
102 car
103 cv
104 f
105 m
106 sc ec ct v g f
107 $su(s)^2$ w^a cv t
108 v
109 w
110 w^a
111 w^{bf}
112 w^e
113 w sn^3
114 w^{co} sn^2
115 w^{ch} wy
116 wy
117 y ac sc pn
118 y cv v f
119 y w
120 z w^{l1E4}
121 z
122 y
123 sn^3
124 w^{bf2}
125 mal

Chromosome 2

201 dp
202 b cn vg
203 bw
204 vg

Chromosome 3

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

Chromosome 4

401 ar/ey^D
402 bt ey^R sv^n
403 ci ey^R
404 ci gvl bt
405 ey^2
406 Ce^2/spa^{Cat}
407 ci gvl ey^R sv^n
408 spa^{Cat}/ci^D
409 spa
410 sv^{de}/ey^D
411 Df (4) M^4/ey^D
412 gvl ey^R sv^n
413 gvl ey^R
414 bt^D/ci^D

Multichromosomal

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

Inversions

601 oc ptg^3/In(1)ClB
602 In(1)dl-49, tyl bb^1/C(1)RM, y v car
603 In(1)sc^8 + dl-49, y^{SL} v f B
604 In(1)sc^8 + dl-49, y^{SL} v f B;e;spa^{pol}
605 In(1)sc^{S1} L sc^{8R} + S, sc^{S1} sc^8 w^a B (=M-5)
606 In(2L)Cy/L^2
607 In(2LR)SM5, al^2 Cy lt^v cn^2 sp^2/Bl L^2
608 In(3L)D^3/Ly
609 In(3LR)TM3, y^+ ri p^p sep Sb bx^{34e} e^s Ser/Me^{65d} h th
610 In(1)sc^{SiL} sc^{8R} + S, sc^{S1} sc^8 w^a B; In (2LR)SM1, al^2 Cy cn^2 sp^2/In(2LR)bw^{V1}, dp b bw^{V1} ds^{33k}; In(3LR)Ubx^{130}, Ubx^{130} e^s/ C Sb;spa^{pol}
611 M(3)h^v/In(3L)P, Me
612 M(3)h^{S37}/In(3L)P, Me
613 In(3LR)TM3, y^+ ni p^p sep Sb bx^{34e} e^s Ser/In(3L)D^3

Attached XX's and XY's

701 C(1)RM, y/+
702 C(1)RM, y f/+;bw;e;spa^{pol}
703 $Y^SX.Y^L$, In(1)EN + dl-49, Y^S car f v
$y.y^L$/C(1)RM, y^2 $su(w^a)w^a$ bb/0
704 B^SY y^+/y v;bw

Selected Stocks

801 Berlin wild, DDT-resistant 1

MÜNCHEN, GERMANY: ZOOLOGISCHES INSTITUT DER UNIVERSITÄT MÜNCHEN

Wild Stocks

Berlin
Oregon
Sevelen

Chromosome 1

ct^{6}
cv
pn^{2}
rux^{2}/y
sc z w^{117G2} ec ct^{6}
sc z w^{zm}
sc z w^{z1}
scp t
t
v
w
w^{a}

w^{co} sn^{2}
w^{co} spl cv f
w spl cv f
y
y w
y sc z w$^{r,def.}$ spl f/y f:=
y v f
y w spl cv f
y z^{a}
z
z w^{11E4}

Chromosome 2

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

Chromosome 3

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

Multichromosomal

y v;bw (1;2)
v;bw (1;2)
w;se h (1;3)
w^{co} sn^{2};ru^{g} (1;3)
w^{co} sn^{2};ru^{g} jv se (1;3)
w^{co} sn^{2};se h (1;3)
y;se h (1;3)
z;ru^{g} (1;3)
y;D/Sb (1;3)
y/y;D/Sb (1;3)
y/w^{m4};se h (1;3)
w;ey (1;4)
b;se (2;3)
bw;e (2;3)
bw;st (2;3)
c;e (2;3)
cn;st (2;3)
Cy/Pm;CxD/Sb (2;3)
SM1, al^{2} Cy sp^{2}/In(2LR) 102 ds^{w} sp^{2};In(3LP, 3RC)SB e^{s}/Ubx^{130} e^{s} (2;3)
ri;ci ey^{R} (3;4)

X with Y Arm Attached

X·Y^{S}(A-3), y w·Y^{S}/y v f/R(Y)L
X·Y^{L}(A-2), y w·Y^{L}/y v bb/Y"
X·Y^{L}(U-8e), y w·Y^{L}/y v bb/Y"

Attached XY

C(1)RM, yv/XY^{L}Y^{S}, y w spl
XY^{S}·Y^{L}(115-9 Parker), y^{2} su-w^{a} w^{a} Y^{S}·Y^{L} y^{+}/y v bb/O
XY^{L}·Y^{S}(108-9 Parker), y^{2} su-w^{a} w^{a} Y^{L}·Y^{S}/y v bb/O
XY^{L}·Y^{S}(2-10 T 13 Parker), y^{2} su-w^{a} Y^{L}·Y^{S}/y/Y
X·Y, v f B·Y/y^{2} su-w^{a} w^{a}/O
Y^{S} X·Y^{L}, Y^{S} y w^{a} rb f·Y^{L}/y w/O

Closed X

R(1)2, In(1)w^{vc}, w^{vc}/In(1) dl-49, y w lz/y^{+} Y

Altered Y's

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

Deficiencies

Df(1)N^{8}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
Df(1)N$^{264-105}$/FM1, y^{31d} sc^{8} w^{a} lz^{s} B

Duplications

Dp(1;3)w$^{m264-58a}$/Y^{S} w y·Y^{L} y^{+}/Y/y w;+$^{Su-V}$
y^{2sc} car·Dp(1;1)sc^{V1}, y^{+}

Inversions

In(1)ClB/w
y w In(1)dl-49 f
In(1)dl-49, pn^{v} B^{M1} bb
In(1)N$^{264-84}$, y/FM6, y^{31d} sc^{8} dm B
In(1)rst^{3}
In(1)rst^{3}, w m v f/M-5
In(1)rst^{3}, y w^{60i29} car bb
In(1)sc^{4}, y
In(1)sc^{4}, y InS w^{a};S sc^{19i} Bl/Cy L^{4} sp;se h
In(1)sc^{7}, w^{a};se h
In(1)sc^{8}
In(1)sc^{8}, w^{a};se h
In(1)sc^{9}, w^{a};se h
In(1)sc^{28}, w^{a};se h
In(1)sc^{29}, w^{a}/y;se h
In(1)sc^{S1} f In dl-49 v w & y f:=;se h
In(1)sc^{L8}, car m w^{a}/y w In dl-49 lz^{s};se h
In(1)w^{m4}
In(1)w^{m4}, ct^{6} f
In(1)y^{4}, w^{a}
In(1)y^{4}, w^{a};se h
In(2)Cy

Translocations

T(1;3)2B7;84A5 y/sc^{S1} dl-49, sc v f car/B^{s} Y
T(1;3)N$^{264-6}$, y/y w dm
T(1;3)N^{65}/w
T(1;3)w^{vco}, v f/Cl B^{36d}
T(1;4)w$^{258-18}$(3C4), y/dl-49, y Hw m^{2} g^{4}
T(1;4)w$^{258-18}$(3C4), y/y w dm
T(1;4)w$^{258-21}$/w;ey
T(1;4)w^{m5}/w;ey

ST. CHRISTOL LES ALES, FRANCE: CNRS ET INRA
Station de Recherches Cypathologiques

endemic strains propagating Picorna viruses
stabilized strains for sigma virus

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

Wild Stock

a^2 - Oregon-R

Chromosome 1

B
cm
cm ct^6
ct
ras^4 m/ClB
rb
sn^3
svr
w
w^{ch} wy
y
y ac sn^3 sx vb^2 sy/y sc^1 In d149 B v w^a sc^8
y w sn^3
z w^{11E4}

Chromosome 2

b
b cn beta
b vg
bw
cg c/SM5, al^2 Cy lt^v sp^2
cg c/U
cn bw
Cy/Pm
dp^T Sp cn InNSR mr/Cy
fj wt/SM5, al^2 Cy lt^v sp^2
fs(2)B Alu lt/SM5, al^2 Cy lt^v sp^2
ft
Gla, InLR/S^2 Cy cn^2 bw sp
InNSL InNSR/al^2 Cy, InL lt^3 L^2 net
pr en
vg

Chromosome 3

e^s
H^2/Xa
red
red e
ru e ca
st

Chromosome 4

ci^w

Multichromosomal

cn;st
dp^T Sp cn InNSR mr/Cy;red e
vg;e^s
y ac sn^3;Cy/Pm

Attached X

br ec/y^{3d} (from Pasadena)
f B/su(s)^S v (from Pasadena)
f^{36a} odsy f^{+ih} & y f:=
w^{bf3}/sn^{36a} (from Pasadena)
y/g^2 ty (from Pasadena)

Altered Y & Attached X·Y

(Maxy)1^{J1+} Y/1^{J1} $sc^{JL(+)}$ In49 ptg oc B^{M1}/y sc^{S1} car odsy f g^2 dy v ras^2 sn^3 ct^6 cm rb ec w pn 1 sc^8
Y^S·X InEN v cv y·Y^L y^+ & y^2 su-w^a w^a bb:= (no free Y)
Y^S X·Y^L(FR-1^L, U-$8d^R$)Y^S y w cv v f·Y^L & y^2 su-w^a w^a bb:= (no free Y)

Translocations (from Pasadena)

T(1;3)O5, D/y^f f:=
T(1;3)ras^v/y f:=
T(1;3)sc^2/y f:=
T(1;3)sc^{260-15}/FM6, y^{31d} sc^8 dm B
T(1;3)sta/FM3, y^{31d} sc^8 dm B 1
T(1;3)sta/y f:=
T(1;3)W^{vco}, vf/ClB^{36d}

Duplications (from Pasadena)

Dp(1;1)112, y f (homozygous)
Dp(1;3)sc^{J4}/Df(1)sc^8, w^a

Triploid (from Pasadena)

C(1)RM, In(1)d149, v^{Of} f/FM7
C(1)RM, y w fa^{no}/FM6 ♀ & FM6, y^{31d} sc^8 dm B/B^S Y^+ ♂

LIMA, PERU: UNIVERSIDAD NACIONAL MAYOR DE SAN MARCOS
Departamento de Ciencias Biológicas, Sección de Biología y Genética

Chromosome 1

1 cy
2 f
3 g
4 w
5 sc cv v f
6 v f

Chromosome 2

7 bw
8 vg

Chromosome 3

9 e
10 st
11 tx

Multichromosome

12 bw;st
13 dp;e
14 e;v
15 e;vg

STOCKHOLM, SWEDEN: UNIVERSITY OF STOCKHOLM
Institute of Genetics

Revision of DIS 48 (1972):3-4
Deletions: 122, 218, 403

PRINCETON, NEW JERSEY: PRINCETON UNIVERSITY
Department of Biology

Wild Stocks

Oregon R

Chromosome 1

B
pn^2
w
w^a
w^e
y
y cv v f
z^a w^{Bwx}/C(1)DX, y w f

Chromosome 2

b
bw

Chromosome 3

ca K-pn
e
e^s
gl
gl^3
ss^{a40a}
st
tra/In(3LR)TM3, ri p^p sep bx^{34e} e^s
Sb/In(3LR)Ubx^{130}, Ubx^{130} e^s

Chromosome 4

ci^D/ey^D

Multichromosomal

tuh-1;tuh-3(1;3)
bw;st(2;3)
In(2)Cy, Cy cn^2/In(2LR) bw^{V1};In(3LR)CxD/In(3LR) TM3, ri p^p sep bx^{34e} e^s

Deficiencies

$Df(4)M/ey^D$
$Df(4)G/ci^D$

Inversions

In(1)dl-49, y w f
In(1)FM7a, y^{31d} sc^8 w^a v^{0f} B
In(1)sc^8, y^{31d} sc^8 w^a
In(1)sc^{S1L} sc^{8R} + S, sc^{S1} sc^8 w^a B

In(2)Cy, Cy cn^2/In(2LR)bw^{V1}
In(3LR)CxD, D/In(3LR)TM3, ri p^p sep bx^{34e} e^s
In(3R)$Antp^B$, $Antp^B$/In(3LR)TM1, Me ri sbd^1

Translocations

T(1;3)OR60/In(3LR)Ubx^{130}, Ubx^{130} e^s ♀;tra^D Sb e/In(3LR)Ubx^{130}, Ubx^{130} e^s ♂
T(1;4)B^S/C(1)DX, y f

Compounds, Y-derivatives, Rings, etc.

C(1)RM, y^2 su(w^a)w^a bb/$\overline{XY}$, v f B
C(1)RM, y pn/In(1)FM6, y^{31d} sc^8 dm B
Y^S/g^2 B·Y^L and C(1)DX, y f
Y^{Lc}/y w Y^S and C(1)M3, y^2
In(X^{c2})w^{vc}/In(1)dl-49, y w lz^s ♀;In(1)dl-49, y w lz^s/sc^8·Y ♂

PURCHASE, NEW YORK: STATE UNIVERSITY OF NEW YORK AT PURCHASE
Division of Natural Sciences

The only D. melanogaster stocks we keep are those behaviorally isolated compound chromosome lines (and their controls) described fully in the American Naturalist 105:479 (1972) and 107:318 (1973). Mrs. Pruzan and L. Ehrman are working with them.

MARBURG, GERMANY: ZOOLOGISCHES INSTITUT DER UNIVERSITÄT

Wild Stock

Berlin

Chromosome 1

w
w^a
v
w^{co} sn^2
y v f

Chromosome 2

cn
vg
bw
cn bw

Chromosome 3

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

Chromosome 4

ey

Multichromosomal

v;bw
cn;se
bw;st
cn;e
vg;e
b;se;ey

Inversions etc.

ClB/+
ClB/w
Cy/+
Cy/Pm
CxD/Sb
Cy/Pm;CxD/Sb
Pm/+;st

y f Y

y^2 su-w^a w^a/v f B, $\overline{XY}$

X^{c2} In(1)w^{vC}/In(1)dl-49, y w lz/y^+Y

XY^L·Y^S(108-9 Parker)

C(2L)RM4, dp;C(2R)RM4, px

CLAYTON, VICTORIA, AUSTRALIA: MONASH UNIVERSITY
Department of Genetics

Wild Stocks

Oregon R
Riverside
East African
Bermuda
Hikone
Canton S

Chromosome 1

v^1
v^{36f}
v^{sp}
y w v f car
ras^2 v m f
w su bb
y pn v
y^2 v ma-1
gt w^a
sc cv v f
$su(5)^2$ w^a cv t
b
w
y

t
mal
v

Chromosome 2

b cn c bw
bw
al dp b pr c px sp
Bl stw^{47} ap tuf sp/
SM5 - Cy al sp
cn
vg
pr
dp stw bw
dp
Pmy/Cy

Chromosome 3

tr a/TM3
ru h st p^p
ss e^s
e^s ca^{nd}
ry
e^{11}

e
se
ve

Multichromosomal

$su(5)^2$ v/y;bw/bw
v;bw
y/y^+·y;SM1/cn mei^{332}

Balancers

SM1, Cy/Pm;Ubx/Sb
FMA3, y^2
$In(1)dl49 + B^{M1}$, sc v B^{M1}
Muller-5
CXD/TM3
Cy/BlL

Marked Y

y^+·y
B^5·y
y^+ v^+·y
y-bb
y·mal^+

Translocations

X·Y^L Y^S
$T(1;3)ras^v$
$T(1;2)v^{65b}$
$T(1;4)$, B^5 y^2 cv v car

Deficiencies

$Df(1)v^{L1}$/FM6
$D(1)v^{L3}$/FM6
$Df(1)v^{64f29}$
$Cf(1)v^{L2}$/FM6

Duplications

1^{12} m^D/Y;Dp v^{63i}

Compounds

C(1)RM, y v/O
C(1)RM, y w f/y
C(1)RM, y v bb/O
C(2), bw
C(3), ri

SYDNEY, N.S.W., AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Agricultural Botany

Wild Stock

Oregon-R-C

Chromosome 1

B
y

y w^e cv
B y w^e cv

Chromosome 2

b
dp
vg

bw
b vg

Chromosome 3

e^{11}
st

Chromosome 4

ci ey

Multichromosomal

bw st
e dp

Special Stocks

Muller 5

Attached-X

y/B
f/B $su\text{-}s^2$ v pr

OXFORD, ENGLAND: UNIVERSITY OF OXFORD
Department of Biochemistry, Genetics Laboratory

Wild Stocks

Oregon-R
Ashwood
Bacup
Bannerdale
Canton-S
Strömsvreten 1
" 2
" 6
" 7
" 8
" 10
P_2

Chromosome 1

ClB/w m f
dor/ClB
dor/y f:=
gr
lz^3/y f:=
M-5
mal/y f:=
r^c/ClB

Chromosome 2

Adh^{n2}/CyO
Adh^{n3}/CyO
Adh^{n5}
Adh^D pr cn
Cy L^4/Pm
l(2)gl B/SM5

Chromosome 3

$Aldox^{nl}$
l(3)tr
pb/In 3LR Cx
Pr Dr/TM3
red
Ubx^{61d}/H^{57c}

Chromosome 4

ey^2
ey^{opt}

OULU, FINLAND: UNIVERSITY OF OULU
Department of Genetics

Wild Stocks

Some fresh wild stocks of Scandinavian origin available

Chromosome 1

M-5
w
w^{ch}
y v f

Chromosome 2

rl
vg
Df(2R)M-S2^{10}/S^{M}, al^{2} Cy sp^{2}
Df(2R)rl^{10a} lt cn/Cy
Df(2R)rl^{10a} lt cn/Pm, al^{4} ds^{33k} lt bw^{V1}

Chromosome 3

e

DÜSSELDORF, GERMANY: INSTITUT FÜR ALLGEMEINE BIOLOGIE DER UNIVERSITÄT

For complete list see DIS 46:38

Changes: Stocks number 3, 14 and 15 discarded.

NORWICH, ENGLAND: JOHN INNES INSTITUTE

Wild Stocks

1 Bayfordbury
2 Hampton Hill
3 Oregon-K
4 Samarkand
5 Teddington

Inbred Lines

6 Bayfordbury (A)
7 Bayfordbury (B)
8 b pr

Chromosome 1

9 B
10 car
11 sc w ec cv ct
12 v
13 w sn B
14 y w

Chromosome 2

15 al b c sp^{2}
16 b pr
17 b pr vg
18 bw
19 cn
20 dp b cn bw
21 vg

Chromosome 3

22 e
23 st

Multichromosomal

24 Cy L^{4}/Pm;H/Sb
25 bw;e

Inversions

25 Muller-5

BRIGHTON, ENGLAND: UNIVERSITY OF SUSSEX
Biology School

REVISION of list in DIS 46:29

Deletions

Chromosome 1

C(1)RM y pn x dor/y
C(1)RM y pn x v
N^{8} Head L-2
N^{45e}/d149
r-9
r I
rI-IV
rII-III
rIV-V
w m f

Chromosome 2

1(2)gd/dp^{tx-1} Cy Ins O pr cn^{2}
1(2)gl b/SM5, al^{2} Cy lt^{v} sp^{2}
tu 36e

Chromosome 3

p^{p}
ry^{2}

Multichromosomal

SM5/Bl L^{2};st

Additions

Chromosome 1

Ms(1)7/FM4

Chromosome 3

tu^{C4} (Edinburgh)

Multichromosomal

bw;e

TERHAN, IRAN: TEACHERS TRAINING COLLEGE
Department of Biology

Wild Stocks

101 Gayaneh
102 Java
103 Oregon-R
104 Tehran

Chromosome 1

111 B
112 Basc
113 ClB/y cv v f
114 m
115 m (of Isfahan)
116 v
117 w
118 w^a
119 w^{vr}
120 y sc

Chromosome 2

131 b
132 Bl L^2/al^2 Cy lt^v sp
113 bw
134 cn
135 cn (Tehran)
136 Cy cn C/cn Df
137 dp
138 L^2
139 vg

Chromosome 3

151 e
152 e^{11}
153 fs(3)/LVM
154 Gl Sb/LVM
155 se
156 se e

Chromosome 4

171 ey
172 spa

Multichromosomal

191 Cy/Pm;D/Sb
192 Cy/Pm;H/Sb

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

Revision of list in DIS 48:13
Wild Stocks: delete: Abeele, Canton-S, Swedish-B and Watou

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

The list of stocks is the same as in DIS 48:6 except for the following stocks that have been discarded: 23, 27, 38, 45, 47 and 52.

NORTH RYDE, N.S.W., AUSTRALIA: MACQUARIE UNIVERSITY
School of Biological Sciences

Chromosome 1

1 B
2 Bx^3 mal
3 $l(1)E6^{ts}$ B/C(1)RM y/w
4 mal/y f:=
5 $para^{ts}$
6 v
7 w
8 y cv v m f
9 y cv v wy car/C(1)RM y/w

Chromosome 2

10 al dp b pr Bl c px sp/SM1, Cy
11 al b c sp
12 b
13 Bl L^2/SM5 Cy
14 bw
15 cn
16 cn bw
17 L^2/Cy
18 vg
19 sca
20 ss/ss^a

Chromosome 3

21 e
22 Ly Sb/LVM
23 st
24 ve se cp e ca
25 ve se cp e Pr ca/TM2, Ubx

Multichromosomal

26 al dp b Bl c px sp/In(2L+2R)Cy; D/In(3L+3R)P
27 Basc; SM1 Cy/In(2LR)bw^{V1};TM2, Ubx^{130}/ In(3LR)C, Sb;spa^{pol}
28 bw;st
29 $In(1)y^{3P}$;In(2L+2R)Cy;In(3LR)Ubx^{130}/ $T(2,3)ap^{Xa}$
30 v;bw

Inversions

31 In(1)FM7a, y^{31d} sc^8 w^a v^{0f} B
32 In(1)dl-49, v^{0f} f
33 N^{105}/In(1)dl-49, y m^2 g^4

Attached Autosomal Arms

34 C(2L)P3, +;C(2R)P3, +
35 C(2L)P4, dp;C(2R)P4, px

BRUSSELS, BELGIUM: UNIVERSITÉ LIBRE DE BRUXELLES
Département de Biologie moléculaire

Wild Stock

1 normal

Chromosome 1

2 v
3 w
4 w^a
5 w^e

6 y v m f

Chromosome 2

7 bw
8 cn
9 cn bw
10 dp
11 vg

Chromosome 3

12 se
13 ss
14 se ss

Chromosome 4

15 ci^W

Multichromosomal

16 Cy/Pm;H/Sb C
17 M(1)o f/FM6
18 M(2)173/SM5
19 $M(2)S2^3$/SM2
20 M(2)S7/SM5
21 $M(3)h^y$/In(3L)P, Me
22 M(3)w/In(3R)C, e l(3)e

OSAKA, JAPAN: OSAKA UNIVERSITY
Medical School, Department of Genetics

Revision of list in DIS 47:32

Chromosome 1
add: cm
delete: sn^3

Chromosome 2
delete: ry^1

Multichromosomal 2;3
delete: b;se
cl^{57j};ss^a
cn;st
vg;e^{11}

Special Stocks (B) Amylase
delete: cn L^2 $Amy^{1.3}$
add: cn L^2 $Amy^{1.3}$ bw

SEOUL, KOREA: EWHA WOMANS UNIVERSITY
Department of Science Education

Revision of list in DIS 46:31

Wild Stocks add: Jinjoo (Korea)
Sinchon-Seoul-3 (Korea)

SD
delete: $SD^{NH}12$
add: SD^{NH}-2

TÜBINGEN, GERMANY: INSTITUT FÜR BIOLOGY
Lehrstuhl für Genetik

Revision of list in DIS 48:14

Wild Stocks
add: Tübingen 1972

Chromosome 2
delete: L/Cy

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

Revision of list in DIS 47:31

Wild Stocks
add: South Orange-A
South Orange-G1
South Orange-G2
South Orange-P

delete: Canton-S
Urbana-S
Swedish-c

add: Chromosome 4
ey

ARLINGTON, TEXAS: UNIVERSITY OF TEXAS AT ARLINGTON
Department of Biology

CO_2 Sensitive: PO♂, TDR-Pittsburg
Delayed-recovery: TDR-1a, TDR-3, TDR orange, TDR orange-yellow, TDR-BC_3, TDR-B

PATRAS, GREECE: UNIVERSITY OF PATRAS
Department of Genetics

Wild Stocks

Oregon-K

Chromosome 1

y
pn^2
w
w^e
sn
f
M-5

Chromosome 2

dp
b
cn
vg
bw
Cl^4/Pm
dp b cn bw
dp cn
b vg

b cn

Chromosome 3

ss
e

BUENOS AIRES, ARGENTINA: COMISIÓN NACIONAL DE ENERGÍA ATÓMICA
Departamento de Radiobiología

Note: Many of our stocks were lost on account of a damaged incubator. A new list will be sent to DIS as soon as possible.

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Zoology

None (No longer supplying stocks)

NEW MUTANTS

Report of L. Søndergaard
University of Copenhagen, Denmark

Ocd^{ts}: $Out\text{-}cold^{ts}$ 1-55.2 ± 0.2. An EMS-induced cold sensitive paralytic behavioural mutant. Ocd^{ts}/+ females and males are paralysed when shifted from 25°C to 19°C. At 25°C females behave normally, while males walk in a reeling manner, fall over frequently, and are unable to fly. Attempts to obtain homozygotic females failed, probably because the males are not able to mate. For further information see the research note in this issue.

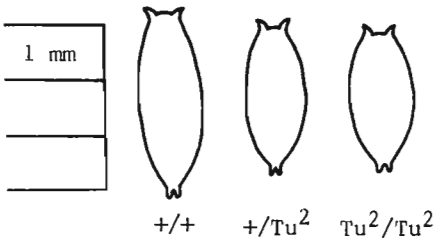
Report of R.D. Snyder
Emory University, Atlanta, Georgia

lz^{71a} Phenotype, eye narrow and ovoid, marked reduction of pteridines (11% wild-type) with only slight spectacled effect around periphery; fusion of facets giving eye a relatively smooth appearance, female sterile (lacking pars ovaria); reduction of tarsal claws both in size and melanin amount, phenol oxidase activity - 5% wild-type activity; phenotype enhanced with addition of su-f.

lz^{71b} Phenotype, eye narrow and ovoid, slight reduction of pteridines (70 - 75% wild-type); irregular fusion of facets giving rise to both smooth and rough areas of the eye; female sterile (lacking pars ovaria); tarsal claws nearly normal with only slight reduction in color; phenol oxidase activity nearly 15% that of wild-type; interaction between allele and su-f not checked.

Report of D.L. Lindsley
University of California, La Jolla

Tu^2: Tubby-2 3-90.6. Gamma ray induced. Lindsley 73a. Pupae shorter and slightly thicker than normal. Pupal dimensions as follows: +/+ = 3.21 ± 0.06 x 1.03 ± 0.02. $+/Tu^2$ = 2.63 ± 0.04 x 1.19 ± 0.02. Tu^2/Tu^2 = 2.34 ± 0.03 x 1.11 ± 0.01. Tu^2 larvae also distinguishable on the basis of reduced length and tortuous tracheal trunks. Adults appear slightly more squat than normal. Classification reliable in larvae and pupae but not adults. RK2. Polytene chromosomes and third-chromosome recombination normal.



Report of D. Anxolabehere and G. Periquet
Université of Paris, France

All the mutants listed here appeared spontaneously in a wild polymorphic population (see Research Notes, this issue) captured in October 1971 in the south of France (Mèze).

vi: vin 3-36.3 ± 1.0 G. Periquet, 72c. eye colour reddish brown, paler in young flies, going to dark brown with age. Generally eye black spotting is absent. Ocelli adult testis sheaths and Malpighian tubules colored.

Linkage studies made with Glued and Wrinkled (Gl:3-41.4, W:3-46.0 Lindsley and Grell 1968) localise vin at 36.3 (s = 0.5) but allelic test shows that it is not an allele of $rose^2$ (rs^2: 3-35.0).

In combination with vermilion, (v;vi) flies have orange eyes. Chromatography on cellulose plate in propanol-amonia shows the presence of isoxantopterine when compared to wild type and rosy mutant.

Viability and fertility of vin are excellent. RK1.

chi: chistera D. Anxolabehere 72g. Wings about two thirds normal size and greatly crumpled and blistered, legs generally crooked. Homozygote female sterile. Viability low in both sexes.

ic: incurvé Periquet 72g. Wings slightly bent upward. Viability and fertility good.

$se^{72\ 1}$: $sepia^{72\ 1}$ 3-26.0 Anxolabehere, 79.1. New allele, phenotype like sepia. RK1.

m^{73a}: miniature 73a 1-36.1 Anxolabehere 73a. New allele, phenotype like miniature.

fri: frisé 1- ?. Anxolabehere 73b. Bristles shortened and bent. Not yet located but it is not an allele of forked.

st^{73b}: $scarlet^{73b}$ 3.44.0 Anxolabehere 73b. New allele, phenotype like scarlet. RK1.

cv-e: crossveinless-e Anxolabehere 73b. Posterior crossvein detached from longitudinals at one or both ends and greatly reduced, often entirely absent. Anterior cross vein usually detached and sometimes punctiform. Viability and fertility good.

rge: rouge Periquet 73b. Eye color bright red, ocelli colorless. Eye color darkens with age but ocelli remain colorless. Viability and fertility good. RK1.

y^{73d}: $yellow^{73d}$ 1-0.0 Periquet 73d. New allele, phenotype like yellow.

ect: écartées Anxolabehere 73d. Wings extended at 45° from body axis, but less divergent on etherized flies. Alulae present. Viability and fertility good.

w^{73d}: white 73d Periquet 73d. New allele, phenotype like white.

Report of S. Gassparian
University of Isfahan, Iran

Populations of Drosophila melanogaster were captured from the south and the southwest of Iran and their genetic structure were studied. Collections were set by vinegar yeast bait. To obtain F_2, mass mating method was used. For all experiments, Mostashfi medium was preferred as the culturing media. Changes in the following traits were studied: Compound eyes - color and shape; ocelli - color; bristles and hairs (scutellars, supra alars, presuturals) - shape and number; wings - length, width and position; abdomen - shape, color and position. Results obtained from the city of Hassein Abad (south of Isfahan) were as follows:

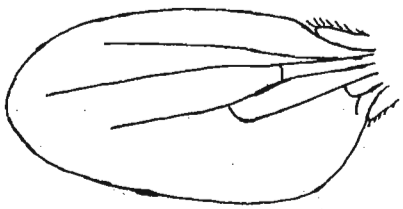
Code	Description
H 1	Wings development normal to basal cell.
H 2	Compound eyes oval, abdomen larger, left wing narrow, absence of wing veination.
H 3	No cross between wing vein L5 and anal cell.
H 4	Compound eyes light red, ocelli colorless.
H 5	Compound eyes red surrounded by white margin, ocelli colorless.
H 6	Undeveloped body, longevity two days from eclosion.
H 7	Compound eyes with light red color, ocelli colorless.
H 8	Body hairs and wings short
H 9	Abdomen larger and wings short (vestigial-like).
H 10	Red circle in abdomen between third and fourth segments.
H 11	Red circle on labium.
H 12	Extra wing vein between L3 and L4 veins.
H 13	White dot on right compound eye.
H 14	Hairs longer and absence of tips of wing veins.
H 15	Three light red lines drawn over the compound eyes
H 16	Eyes divided by a light red line.
H 17	Abdomen red, absence of posterior crossvein.
H 18	Head honey yellow, a gap between L4 and L5 veins.
H 19	Broken anal crossvein in left wing.
H 20	Eyes oval, hairs longer.
H 21	Abdomen cylindrical.
H 22	Gap in dorsal part of abdominal tergites.
H 23	Abnormality in anal part of both wings.
H 24	Eyes circular.
H 25	Body color honey yellow, ocelli colorless, wing vein tip absent.
H 26	Left wing shorter than right.
H 27	Head gray color.
H 28	Abdomen blood red color, marginal gap between L4 and L5 wing veins.
H 29	Eyes white and oval shaped, hairs longer and larger angle between wings.
H 30	Right wing tip curled-like, abdomen shorter.
H 31	Eyes circular and smaller, with a white margin.
H 32	Eyes oval, hairs shorter.
H 33	Eyes surrounded by white margin.
H 34	Dark red eye color, curled wings.
H 35	Eyes oval and light red, halteres darker and wings short.
H 36	Eyes oval and light red, wings shorter.
H 37	Eyes divided by a light red line.
H 38	Eyes kidney-shaped, wings shorter.
H 39	Eyes kidney-shaped, wing tips narrow.
H 40	Eyes with pinky margin.
H 41	Body smaller with light yellow coloration, eyes white.
H 42	Body color honey yellow.
H 43	Eyes circular, ocelli colorless.
H 44	Eyes circular and light yellow.
H 45	Body color darker, hairs longer.
H 46	Eyes kidney-shaped, ocelli colorless.
H 47	Hair absent between abdominal tergites.
H 48	Eyes white and oval, ocelli colorless.

H 49 Wings shorter, third and fourth veins closer.
H 50 Body color red, abdomen long.
H 51 Body color gray, but abdomen yellow.
H 52 Thorax colorless.
H 53 Gap in submarginal part.
H 54 Body color dark apricot, bright red eye color.
H 56 Crown-like dot on thorax.
H 57 Wings short with narrow tips (observed only in males).

A total of 57 mutations and modifications were detected from the combined populations of Hossein Abad and Isfahan region, which was kept for more than 44 generations under artificial selection. The number of mutations detected from the city of Isfahan is 30; the rarest mutation is an allele of miniature, called miniature of Isfahan. One allele, dark red eye color, is a new mutation. This is a single gene mutation on the third chromosome at 108 ± 1 on the genetic map.

The pure lines of both stocks and some unlocalized mutations are available.

Report of F.R. Waddle and I.I. Oster
Bowling Green State University, Bowling Green, Ohio



ve^4: veinlet^4 Found in Y^S.X InEN y.Y^L;bw;e;ci ey^R stock of the Mid-America Drosophila Stock Center (j421 of the 1972 stock list). Phenotype as drawn. Expressivity nearly constant. ve/ve^4 easily distinguishable from both ve and ve^4.

Report of P.A. Roberts
Oregon State University, Corvallis, Oregon

Df(1)y^{13}. X-ray induced in wild type sperm. Cytological appearance is that of a terminal deletion "mimic" (Roberts, P.A., Genetics 74:s231) with material distal to 1B1-3 deleted (traces of what may be capping material can occasionally be seen). X-chromosome deficient for 1J1$^+$, y$^+$, and ac$^+$. Deficiency bearing males survive in presence of y$^+$ Y and are fertile or in presence of 1J1$^+$ Y but are weak and sterile.

Df(1)y^{14}. X-ray induced in wild type sperm. Cytology: Df(1)1A4-6;1A8-B1. X-chromosome deficient for 1J1$^+$, y$^+$, and ac$^+$. Deficiency bearing males survive in the presence of y$^+$ Y (vigorous but sterile) or in presence of 1J1$^+$ Y (weak and sterile). A translocation with heterochromatic breakpoints may be present.

Df(1)y^{15}. X-ray induced in wild type sperm. Cytology: Df(1)1A4-5;1A8-B1. X-chromosome deficient for 1J1$^+$ and y$^+$ but, in contrast with Df(1)y^{13} and Df(1)y^{14} which are classifiable as amorphic for achaete, Df(1)y^{15} appears hypomorphic. Deficiency bearing males are fertile in the presence of either y$^+$ Y or 1J1$^+$ Y (see report of A. Schalet and P.A. Roberts).

Report of A. Schalet and P.A. Roberts
University of Leiden, The Netherlands and Oregon State University, Corvallis, Oregon

arth: arthritic 1-0. Legs weak with pigmented joints; tarsal segments frequently askew with claws fused; movements somewhat uncoordinated. Brownish-black pigment present at joints of over 90% of males, most frequently in posterior and middle legs between femur and tibia, but sometimes between coxa and trochanter or proximal to coxa. Phenotype expressed in males carrying 1(1)J1$^+$ Y and any one of three deficiencies, Df(1)y^{13}, Df(1)y^{14} or Df(1)y^{15} (see separate report of P. Roberts), but only Df(1)y^{15}/1(1)J1$^+$ Y males are fertile.

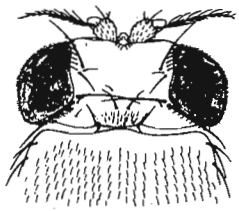
Report of D.T. Kuhn
Florida Technological University, Orlando, Florida

f^{tuh}: forked from tumorous-head 1-56.7. A spontaneous mutation observed in several males from a tumorous-head strain. Tests for allelism place the mutation at the forked locus. The site within the pseudoallelic locus was not determined. Viability and fertility are good.

Bx^{tuh}: Beadex from tumorous-head 1-59.4. A spontaneous mutation observed among both males and females. Flies with Bx^{tuh} were isolated simultaneously with f^{tuh} flies from the same strain. Tests for allelism place the mutation at the Beadex locus.

Report of A. Robertson
University of Edinburgh, Scotland.

bh: baldhead, 3:81 ± 1. Spontaneous in Kaduna cage population. Ocelli and associated bristles absent (see diagram). Wings shortened. Sterile in both sexes.



Report of L.T. Douglas and T.M. Douglas
University of Nijmegen, The Netherlands

v^t: vermillion-tan Discoverer: T.M. Douglas. A spontaneous mutant in F_2's of the cross sc cv v f/sc cv v f ♀♀ x st/st ♂♂ obtained from Carolina Biological Supply Co., Burlington, N.C. 27215. Always more lemon yellow than v^{36f}, becoming yellowish-tan in older flies. Interacts with bw to give a complete white, in contrast with the light pink of v/v;bw/bw (our v/v; bw/bw ♀♀ have eyes with a pale pinkish hue). v^t/v ♀♀ appear vermillion but v^t/v;bw/bw ♀♀ show distinctly less pink than v/v;bw/bw. Brown-eyed offspring are not recovered from the cross v^t/v^t;bw/bw ♀♀ x v/-;bw/bw ♂♂.

Report of R.P. Sharma
Indian Agricultural Research Institute, New Delhi, India

wg^1: wingless 2-30.0, X-ray induced in Oregon-K wild stock. Fertility and viability good. (Detailed description appearing in this number of DIS).

LINKAGE DATA

Report of D.L. Lindsley
University of California, La Jolla

Map position of Tu^2. Progeny from crosses of Tu^2/ru h th st cu sr e^s ca ♀ x ru h th st cu sr e^s ca ♂ were recorded, crossed to wild type, and the resulting progenies examined as pupae to determine whether $Tubby^2$ was segregating. As soon as it became clear that Tu^2 lies between e and ca and that recombination in the e-ca region is not reduced (31.5% versus standard distance of 30.0%), attention was confined to recombinants between e and ca with the following results:

	Tu^2	Tu^+
e +	30	20
+ ca	23	55

Thus the position of Tu is 85/128 the distance from e to ca or at 90.6.

SYDNEY, N.S.W., AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Animal Husbandry

D. simulans

net
jv se
jv st pe
H^h pe
st e
y w
f^2
v

bw
dh b pm
net b py sd pm
py^2 up
stw
rd
st Ubx pe/st pe

Other Species

D. aldrichi
D. ananassae — Wild stock (Rockhampton)
D. brunneipennis
D. busckii
D. buzzatii
D. fumida
D. hydei
D. immigrans
D. lativittata — Wild stocks (Sydney)
D. simulans

TÜBINGEN, GERMANY: INSTITUT FÜR BIOLOGIE
Lehrstuhl für Genetik

Revision of list in DIS 48:25
add: Wild Stocks Tübingen 1972

OSAKA, JAPAN: OSAKA UNIVERSITY
Medical School, Department of Genetics

Revision of list in DIS 47:40
delete: D. virilis 16
D. funebris wild
change: D. simulans wild to 4 strains

VIENNA, AUSTRIA: UNIVERSITY OF VIENNA
Institut für allgemeine Biologie

D. ambigua
Wild stock

one stock

Chromosome 1

or
y
v

Autosomes

pn Bd

D. subobscura
Wild Stocks

Belgrad, Jugoslavia
Drobak, Norway
Küsnacht, Switzerland
Ponza, Italy
St. Pölten, Austria
Wien, Austria

Chromosome A

pm ct sn
y
v m ct bnt sc

Chromosome E

pp pl pt
pp sj pl otp

Chromosome I

cn
ma int
ey
ni
r

Chromosome O

ch cn
Va ch cn
Va/Ba
Va
Ba

Chromosome U

ltr
ho
fd^{Mi} nt

Other Species

D. funebris
D. mercatorum
D. pseudoobscura
D. simulans

JERUSALEM, ISRAEL: HEBREW UNIVERSITY
Laboratory of Genetics

D. hydei D. immigrans D. simulans D. funebris

TURKU, FINLAND: UNIVERSITY OF TURKU
Department of Genetics

Revision of list in DIS 47:40 delete: simulans wild
add: Chromosome 3 first & last stocks listed "to be selected"

CALCUTTA, INDIA: UNIVERSITY OF CALCUTTA
Department of Zoology

Revision of list in DIS 48:24 delete: Chromosome 1: w^a, w^{ml} add: insularis
Translocation melarkotliana
simulans and tropicalis

VARANASI, INDIA: BANARAS HINDU UNIVERSITY
Department of Zoology

Revision of list in DIS 45:46
Wild Stocks: delete: latifshahi
add: andamansis
Unlocated mutants: delete: dct

ananassae: Chromosome 1: delete: y w^a vs
add: y, vs
Chromosome 3: delete: px pc, stw px

SEOUL, KOREA: CHUNGANG UNIVERSITY
Department of Biology

Revision of list in DIS 47:43
change: D. suzukii (3 strains)

add: D. biauraria (4 strains)
D. triauraria (10 strains)

SEOUL, KOREA: SEOUL NATIONAL UNIVERSITY
Department of Zoology

D. immigrans (Jeju Island)

D. virilis (Seoul)

STOCKHOLM, SWEDEN: INSTITUTE OF GENETICS

Revision of list in DIS 48:27 delete: 701

CARACAS, VENEZUELA: UNIVERSIDAD CENTRAL DE VENEZUELA
Instituto de Zoologia Tropical

Combined and single female strains collected at several localities throughout Venezuela and some neighbouring countries:

willistoni group	capricorni	nebulosa	pavlovskiana	tropicalis
	equinoxialis	paulistorum	sucinea	willistoni

SÃO PAULO, BRAZIL: UNIVERSIDADE DE SÃO PAULO
Instituto de Biociências

Stocks from: * Campo Grande, State of Mato Grosso, Brazil; ** São Paulo, State of São Paulo, Brazil; *** Ubatuba, State of São Paulo, Brazil.

ananassae *	kikawai *	paulistorum *	prosaltans *	sturtevanti *, ***
hydei **	nebulosa *	polymorpha *	simultans *, ***	willistoni *, ***

SAN DIEGO, CALIFORNIA: SAN DIEGO STATE UNIVERSITY
Department of Biology

D. ananassae
Wild Stocks

Hawaii (Honolulu)
Palmyra
Samoa (Taputimu, Tutuila)
Samoa (Nafanua, Upolu)
Tonga
Rarotonga
Fiji
Majuro
Rongerik
Mexico (Merida, Yucatan)
New Guinea (Port Moresby)
New Guinea (Popondetta)

Chromosome 1

w^H
y^d
w^H f y^d

Chromosome 2

e^H
cd^A j^A ca^s (Hinton)

Chromosome 3

dp^{ov}
bri pe stw pc ru^2 (Hinton)

Multichromosomal

$w^H;e^H$)1;2)
f;cd;px (1;2;3)

Parthenogenetic (unisexual)

Samoa (Taputimu, Tutila)
ana selected

D. pallidosa
Wild Stocks

Samoa (Pago Pago, Tutuila)
Samoa (Taputimu, Tutuila)
Samoa (Nafanua, Upolu)
Samoa (Aopo, Savaii)

Chromosome 1

w
sn

Chromosome 3

abt

Multichromosomal

sn;abt (1;3)
w sn;abt (1;3)

Parthenogenetic (unisexual)

Samoa (Taputimu, Tutuila)
palli selected
sn
sn;abt

CAMBRIDGE, MASSACHUSETTS: HARVARD UNIVERSITY
Museum of Comparative Zoology

D. pseudoobscura Strains homozygous for electrophoretic allozyme alleles at various loci. Strains from 10 localities in western US, Guatemala and Colombia.

MONTREAL, CANADA: LOYOLA OF MONTREAL
Department of Biology

D. simulans	Chromosome 1	Chromosome 2	Chromosome 3
Wild stocks from various localities	y w	b bw	rd jv se st Dl^2 pe st pe

DÜSSELDORF, GERMANY: INSTITUT FÜR ALLGEMEINE BIOLOGIE DER UNIVERSITÄT

For complete list see DIS 46:46
Changes: D. fulvimacula, y/Y and +/Y lost
D. nigrohydei, discarded

PURCHASE, NEW YORK: STATE UNIVERSITY OF NEW YORK AT PURCHASE
Division of Natural Sciences

We keep all those strains, including mutants, of paulistorum formerly kept in the laboratory of Professor Th. Dobzhansky at the Rockefeller University. We also maintain a few pseudoobscura lines for pheromonal and behavioral analyses.

MISIMA, JAPAN: NATIONAL INSTITUTE OF GENETICS

ananassae
Wild Stocks

a7 (Panama)
Cuba
Yakatan
Hawaii
Hawaii-W
Truk (very light)
IM-2 (Madras, India)
IM-4 " "
F2 (Peng-Hu Is., Formosa)
F5 " "
F8 " "
Ph5 (Malaybalay, Philippines)
Ph15 " "
Tonga (dark)
T_{13-6} (Tonga, inbred line)
T_{15-4} " "
Okinawa (3 lines)
Taiwan (2 lines)
Borneo (7 lines)
Singapore (1 line)
Luala Lumpur (6 lines)
Thailand (2 lines)
Calcutta (2 lines)
Sri Lanka (Ceylon)(4 lines)
Mysore, India (7 lines)

Chromosome 1

amb ecv
ct^6
f^{72}
kk
kk^c
w^{65}
w^{65} f^{49} y^{51}
w^{65} sn^{65} y^{51}
w^{65} ty y^{51}
w^{65} y^{51}
sn^{65} y^{51} ct^6
w^{65} y^{51} kk
scar
w^m

Chromosome 2

Arc
Arc bw
Arc se^T
b
b bn-b^{67}
b eyg
b ma
b pea
b se^T
bw
bw ba-b
bw^{71}
cd bw
cd bw L
cd L
Dl
Dl^{71}/+Truk
Dl^{71}/cd bw
Dl^{71} pea
eyg
j b
Ir-a
L
ma ba-a
pea
rus
se^T
se^T ba-b

Chromosome 3

bri pc
bri px
bri ru
bs
M(3)65 px
M(3)b67 mot bn-c
M(3)c68 px
M(3)d70
M(3)d70 bs
M(3)d70 Rf
M(3)d70 ru
mot
pc
px
px^{66}
px^{66} ru Tr
Rf
Rf mot
ri
ri Tr
ru
Snp
Snp bri ru

Chromosome 4

bb
bb^{67}

Multichromosomal

f;cd
w^{65};bb
bw;bri
bw;Ms ru
bw;ru
bw^{71};px^{66} ru Tr
bw^{71};ri^{71}
Ir-a;M(3)b67 mot
Ir-a;mot
Ir-a;px
se^T;ru

OXFORD, ENGLAND: UNIVERSITY OF OXFORD
Department of Biochemistry, Genetics Laboratory

arizonensis
buzzattii
funebris
mulleri
saltans
simulans
virilis

LONDON, ENGLAND: BIRKBECK COLLEGE
Department of Zoology

simulans

wild type

subobscura

D inbred
K inbred
) both lines inbred for over 250 generations

MATO GROSSO, BRAZIL: UNIVERSIDADE ESTADUAL DE MATO GROSSO
Departamento de Morfologia

All stocks from Campo Grande, State of Mato Grosso, Brazil

D. ananassae
D. Kikawai
D. nebulosa
D. paulistorum
D. polymorpha
D. prosaltans
D. simulans
D. sturtevanti
D. willistoni

OTTAWA, ONTARIO: CARLETON UNIVERSITY
Department of Biology

Wild strains from single females collected in Ontario and Quebec: algonquin, athabasca

ST. CHRISTOL LES ALES, FRANCE: CNRS ET INRA
Station de recherches cytopathologiques

immigrans: endemic strains propagating Picorns viruses

OULU, FINLAND: UNIVERSITY OF OULU
Department of Genetics

simulans	littoralis	limbata
ambigua	lummei (virilis group)	phalerata
obscura	ovivororum (virilis group)	transversa
subobscura	new species (virilis group)	testacea

LINCOLN, NEBRASKA: UNIVERSITY OF NEBRASKA
Department of Zoology

Wild strains of D. affinis, algonquin, athabasca, azteca, narragansett and tolteca.

MILANO, ITALY: UNIVERSITA DI MILANO
Istituto di Genetica

hydei
pseudoobscura
virilis

simulans
Wild Stocks

1 Aspra
2 Giannutri

Chromosome 3

3 st

Stocks selected for tumor manifestation

4 tu Bl
5 tu Aspra

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Biology

Revision of list in DIS 47:41

delete: Chromosome 3 S/+

Multichromosomal "scute"(II);pe^{m3}

add: Multichromosomal gp^{2} S/gp^{2} +;pe

peach mottled

32 b;T(3;5)pe^{m4} cn B^{3}
33 b;T(3;5)pe^{m4} cn ru
34 b;T(3;5)pe^{m51} cn A strain
35 b;T(3;5)pe^{m51} cn B strain
36 b;T(3;5)pe^{m51} cn B^{3}
37 T(Y;5)pe^{m1}
38 T(Y;5)pe x/y:pe;pe/pe strain 19A
39 T(Y;5)pe^{m15}/pe
40 T(2;5)pe^{m39}
41 T(3;5)pe^{m39}
42 T(3;5)pe^{m51} strain 7A
43 T(3;5)pe^{m51} strain 10B
44 T(3;5)pe^{m51}
45 T(3;5)pe^{m51} R(pe^{+} L sv)pe;sv^{+}
46 T(3;5)pe^{m51} R(pe)#2;t tb gp^{2}
47 T(3;5)pe^{m51} R(ru st B^{3} pe^{+} sv)
48 T(4;5)pe^{m3} homozygous
49 Texmelucan 17-2 (v-5)Y-5 mottled

ananassae

Report of D. Moriwaki
National Institute of Genetics, Misima, Japan

X-chromosome

f72: forked72 Moriwaki 72c23. Appeared in se;ru stock. Allelic to f.

l(1)M5: lethal 5 of Moriwaki Moriwaki 72d. Spontaneous in w sn y ct stock. Semi-lethal (ca. 70%). Seemingly located near the end of either arm of XL or XR.

ecv: extra crossvein Moriwaki 72d14. Recovered as two males accompanied with amb in a cross f^2/amb;se/+;ru/+ x f^2. Less viable, though amb-gene seems to improve the viability.

Chromosome 2

L: Lobe T. Oishi 71d28. Spontaneous as a single ♂ in a cross referring to Dl bw;M-d ru. Dominant, less viable. Homo. lethal. Heterozygous L eyes smaller. Expression ranges from bar-like to kidney-shaped depending on genetic backgrounds; sometimes nearly overlaps wild type.

eyg: eye gone Fuyama 72d. Spontaneous in b124 stock. Eyes and head much smaller than normal. Percent emergence low.

rus: russet Moriwaki 72j25. Arose spontaneously in a wild stock, KL-1-4 (isofemale line from Kuala Lumpur). Reddish brown eye color. Female sterile.

Chromosome 3

Tr: Trident Moriwaki 72c15. Spontaneous in bw^{71};ri^{71} stock. Posterior crossvein branched with trident shape: extra veins in marginal and submarginal cells. Dominant. Homo. lethal. Expression variable according to combined mutant genes. For example, combined with homozygous ri, tip of L3 thickened as delta.

Revisions

Some of the descriptions in the previous issues will be revised as follows:

(DIS 43:79) ba^{65} ⟶ ba-b, bw^R ⟶ bw, l-l ⟶ l(1)M2, M^{65} ⟶ M(3)65, px^2 ⟶ px^{66}

(DIS 45:58) Bd ⟶ Bd^3, bn^{67} ⟶ bn-b^{67}, M-b ⟶ M(3)b67, M-c ⟶ M(3)c68

(DIS 46:49) M-d ⟶ M(3)d70, pe ⟶ pea

(DIS 48:27) ct ⟶ ct^6,
Pt ⟶ Dl^{71}. Allelic to Dl. The manifestation of Dl^{71} varies depending on genetic backgrounds.
The linkage group of bs is misprinted. Read 3- for 2-.
Description of w^m is duplicated. Delete the upper one.

auraria

Report of T.J. Lee
Chungang University, Seoul, Korea

tx: taxi Wings held out at 75° - 90° from body axis, curved downward. Autosomal recessive. Spontaneous in wild stock. Good viability.

ananassae

Report of D.G. Futch
San Diego State University, California

X- chromosome

w^H: white Spontaneous in stock from Hawaii (single male).

y^d: yellow Spontaneous in stock from Taputimu, Samoa (single male).

Chromosome 2

e^H: ebony Spontaneous in stock from Hawaii (single female).

Chromosome 3

dp^{ov}: dumpy Spontaneous in a single female from a backcross to f, cd ♀♀ of F_1 from a cross f, cd ♀♀ x px ♂♂. Truncation of wings expressed phenotypically only in females and is variable, overlapping wild-type. Vortices occur in both sexes but this phenotype is also variable and overlaps wild-type.

nigromaculata

Report of J.W. Smith
Purdue University, Lafayette, Indiana

yellow body mutation. This is a report of a spontaneous mutation discovered by the author in a stock bottle of D. nigromaculata, a member of the quinaria species group. The mutation produces a yellow body color similar to the yellow body color mutation of D. melanogaster (Lindsley and Grell 1968).

Isolation of the mutant: Two males exhibiting the yellow body color were removed from a stock bottle and mated to virgin wild females in vials. The F_1 female offspring (which were phenotypically wild) were then backcrossed to the yellow parent males. The F_2 progeny consisted of yellow males, yellow females, wild males and wild females. The yellow bodied F_2 flies were then isolated as virgins and allowed to mate. All F_3 offspring were phenotypically yellow bodied.

Genetics of the mutant: The mutant is sex-linked as indicated by crossing yellow females to wild males; all F_1 males were yellow bodied as would be expected if the gene controlling the yellow body were in the hemizygous condition on the X-chromosome. Since no other mutants of D. nigromaculata have been identified and isolated, no mapping data is available.

Description of the mutant phenotype: The body color is yellow. The wings are yellow with yellow veins and hairs. The bristles are brown with light brown tips. The distinctive triangular black patches on the lateral margins of tergites 2-6 of the wild stock are light brown with indistinct margins on the mutant.

Behavior of the mutant: Observation of 10 courtships of virgin yellow males paired with virgin yellow females shows a lethargic courtship consisting of long bouts of orientation followed by long bouts of stroking with flicking occurring only near the termination of courtship. This is in marked contrast to the wild courtship in which stroking reciprocates rapidly with flicking so that just prior to acceptance it is difficult to temporally separate the two components (Smith, 1972).

Culture methods for the mutant: At present the mutation is being cultured on a high protein media in vials (25mm x 95mm). Several attempts to maintain cultures in large 1/2 pint milk bottles have proved unsuccessful.

References: Lindsley, D.L. and E.H. Grell, 1968 Genetic Variations of D. melanogaster, Carn. Inst. Wash. Pub. No. 627; Smith, J.W. 1972, DIS 49:117-118.

pallidosa

Report of D.G. Futch
San Diego State University, California

X-chromosome

w: white Spontaneous in parthenogenetic line from Taputimu, Samoa (single female).

sn: singed Spontaneous in parthenogenetic line from Taputimu, Samoa (single female).

Chromosome 3

abt: abrupt Spontaneous in parthenogenetic line from Taputimu, Samoa (single female). Fifth longitudinal wing vein short, terminating distally just beyond its intersection with the posterior crossvein. Heterozygotes occasionally show slight shortening of L5. Probably homologous with abrupt in D. melanogaster.

paulistorum

Report of A. Pruzan
State University of New York, Purchase, N.Y.

w: white in C-2 Lancetilla, Honduras strain, Centroamerican semispecies. Sex linked and apparently homologous to melanogaster white.

Report of L. Ehrman
State University of New York, Purchase, N.Y.

px: plexus in the same paulistorum material as above. Autosomal recessive, may be homologous to melanogaster plexus.

hydei

Report of K.H. Glätzer
Universität Düsseldorf, Germany

Ba: Bandless abdomen Glätzer 73f. X-ray induced in 1 ♀. Characteristics: The abdomen is dark yellow and without any black bands except for the faint dark brown colouring of the markings on the abdominal tergites. The bands begin at the tergite-sternite boundary as black flecks visible only from the side. The wings are uniformly extended to an angle of about 45° relative to the body axis and slightly elevated, and is, therefore, suggestive of the D. melanogaster mutant, Dichaete. Because of this trait none of the flies except young ones are able to cover the abdomen with their wings and then only for a moment or two. Fertility and penetrance is good. Homo- and hemizygous lethal.
Localization: X, preliminary estimation between 1-80 and 1-85. RK 1.

Report of P.T. Wong, W.D. Kaplan, W.E. Trout, III, and M. Colin
City of Hope National Medical Center, Duarte, California

sh^1: shaker-1 1-112.4. EMS induced. Legs shake vigorously when etherized. Some flies also exhibit wing scissoring.

sh^2: shaker-2 1-112.2. EMS induced. Same phenotype as sh^1. Allelic to sh^1.

Paik, Y.K. and K.C. Sung. University of Hawaii, Honolulu. Inversion frequencies in D. immigrans populations from the Island of Kauai.

The data presented in this communication is a continuation of the work reported by Paik and Sung (1972) on the inversion frequency of the species from the two major islands of Hawaii. In January 1972, a collection was made on Kauai. Populations were sampled at two sites, i.e., Halemanu (at 3800') and Berry Flat Trail (at 4100') on Mt. Kokee. Another collection was conducted at the same test sites in February 1973. The samples of 1972 are referred to as KH-72j and KT-72j respectively; those of 1973 as KH-73f and KT-73f respectively.

The majority of our results were obtained by performing cytological studies on the F_1 larvae of the wild-caught females ("egg samples") with some results in Table 2 coming from the examination of the chromosomal constitution of the wild males ("male samples"). The smear preparations of salivary gland chromosomes were fully examined for the presence of both hetero- and homokaryotypes. Table 1 summarizes the data on the observed frequencies of inversion heterozygotes. The inversions designated as "A", "B" and "C" in the tables are identical to those used by Brncic (1955). From Table 1 we have compiled the separate frequencies of individuals which are heterozygous for a single inversion. These are shown in Table 2 together with the frequencies of separate gene-arrangements. The inversion frequency variations between the four samples in terms of both the heterokaryotypes and gene-arrangements was investigated. The chi-square tests based on the G statistic indicated no statistical significance bweteen the four samples with one possible exception. In the between populations analysis for 1973, inversion B had a significance level of P = 0.047. It is interesting to note that in all Kauai samples the most frequent arrangements are the same, i.e., inversion C. A more detailed paper discussing the differences in the relative distribution of the inversion frequencies between the Hawaiian Islands is in preparation.

Table 1. Frequency of inversion heterozygotes and mean inversion heterozygosity per larva, measured by the "egg sample" technique.

Collection	Larvae tested	Percent inversion heterozygotes						% homozygotes*	Mean/larva (S.E.)
		A	B	C	A+B	A+C	B+C		
KH-72j	53	5.7	11.3	13.2	-	-	1.9	67.9	0.34 ± 0.07
KT-72j	37	13.5	2.7	13.5	2.7	-	-	67.6	0.35 ± 0.09
KH-73f	201	7.4	3.5	20.0	-	1.0	0.5	67.7	0.34 ± 0.02
KT-73f	187	11.2	5.3	16.6	0.5	0.5	3.2	62.6	0.42 ± 0.04
Pooled	478	9.2	5.0	17.4	0.4	0.6	1.7	65.7	0.37 ± 0.02

* Indicates percentage frequency of individuals which are not heterozygous for inversion(s).

Table 2. Frequency of separate karyotypes and gene-arrangements.

Collection	N	Karyotype			Arrangement		Karyotype			Arrangement		Karyotype			Arrangement	
		A/A	A/+	+/+	A	+	B/B	B/+	+/+	B	+	C/C	C/+	+/+	C	+
KH-72j	88*	.02	.10	.88	.074	.926	.00	.14	.86	.068	.932	.00	.19	.81	.097	.903
KT-72j	45*	.00	.13	.87	.067	.933	.00	.09	.91	.044	.956	.00	.16	.84	.078	.922
KH-73f	201	.00	.08	.92	.042	.958	.00	.04	.96	.020	.980	.01	.21	.78	.112	.888
KT-73f	187	.00	.12	.88	.061	.939	.00	.09	.91	.045	.955	.02	.20	.78	.118	.882
Pooled	521	.004	.11	.89	.057	.943	.00	.08	.92	.039	.961	.01	.20	.79	.108	.892

N Indicates the number of flies tested.
* Includes "male samples".

Acknowledgement: We express appreciation to Mr. W.W.M. Steiner for useful comments on the manuscript.

References: Brncic, D. 1955, J. Hered. 46:59-63; Paik, Y.K. and K.C. Sung 1972, DIS 48:115-116.

Gvozdev, V.A., S.A. Gostimsky, T.I. Gerasimova and E.M. Gavrina. Kurchatov Institute of Atomic Energy, Moscow, and Department of Genetics of Moscow State University. Complementation and fine structure analysis at the 2D3-2F5 region of the X-chromosome of D. melanogaster.

The 2D3 region of the X-chromosome was saturated with the lethals induced by the treatment of males with ethyl methane-sulphonate (EMS) or nitrosomethylurea (NMU). This region contains the pn and kz loci and is deleted in the deficiency Df(1)Pgd-kz[1]. The Df(1)Pgd-kz and the 2F5-3C5 deficiency, obtained from Prof. M. Green complement each other, thus indicating that the 2F6 band is possibly included in the former chromosome.

The lethals in the 2D3-2F5 region were located by mating 1/Muller-5 females to Df(1)Pgd-kz/w$^+$Y males. In this region, which includes no more than 12 bands of the nearly 1000 bands of the whole X-chromosome 0.5-1.2% (independent series of expts.) of the induced X-linked lethals were mapped.

64 lethals located in 2D3-2F5 region were divided into two groups by deletion mapping with the use of Df(1)64c18, 2E1.2-3C2, obtained from Dr. G. Lefevre: 1) lethals located to the left of the 2E1.2 doublet, in the 2D3-2D5.6 region, containing 3-4 bands; 2) lethals located in the 2E1.2-2F3.5 region, containing 7-10 bands.

The Pgd locus coding for 6-phosphogluconate dehydrogenase (6PGD) was mapped in the 2D3-6 region (1). The Pgd^A and Pgd^B loci determine the electrophoretically fast and slow forms of 6PGD respectively (2). Seven lethal and semilethal mutations in the Pgd^A locus were selected by the absence of the fast (A) and hybrid (AB) or only the fast PGD isozymes in the extract of the Pgd^B/l females produced from a cross of ♀ Pgd^B/Pgd^B x ♂ 1/w$^+$Y.

To detect the complementation between the lethals, matings of ♀ l_1/FM4 x ♂ l_2/w$^+$Y were performed (see Table, asterisks indicate the lethals induced with NMU). Two vital loci, in addition to the well known non-vital pn locus, were revealed in the 2D3-6 region: the Pgd and the locus mapping between the Pgd and pn (lethals 3, 25, 29 etc.) whose function is unknown.

Table. Saturation of the 2D3-2F5 region with lethals.

Complementation groups	Bands	Number of bands in region	Lethals	Number of lethals
1 (Pgd)			11,35,39,45,50,71,109	7
2	2D3-5.6	3-4	3,25,29,33,37,38,40,44,48,49,52,56	12
3 (pn)			-	-
4			1*	1
5			20	1
6			70	1
7			69*	1
8	2D6-2F3.5	7-10	2,8,30,51,59	5
9			14,15,19,42,64	5
10			23,26,31,53,60,105*	6
11			16,18,22,24,28,57,58*,64,102,108	10
12			4,5,17,21,32,34,36,41,47,55,61,63,67,71	15
			Total	64

Nine complementation groups were revealed in the 2E1.2-2F3.6 region containing 9 bands. Thus a good correlation between the number of bands and functional units was demonstrated for the whole 2D3-2F5 region as well as for the adjacent 3A1-3C3 region (3).

This type of investigation allows us to determine whether lethal mutations are situated in the structural region of Pgd locus coding for its product or are scattered in a nearby, possibly regulatory, region.

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Kaji, S. Konan University, Kobe, Japan.
Incorporation of the tritiated acetamide into DNA in Drosophila melanogaster.

It was found that acid amides have a strong action in accelerating the facet-formation of the mutant Bar eye. After acetamide treatment, the Bar eye became larger than that of the wild type in extreme cases (Kaji, 1954, 1960). The tritiated acetamide was mainly incorporated into the nuclei in the facet-forming region of the Bar eye discs by means of autoradiographic analysis. The mode of incorporation of tritiated acetamide resembled that of tritiated thymidine. These results suggested that the action of acetamide has a relation to the metabolism of DNA synthesis in the development of the eye discs (Hirose and Kaji, 1968, 1969; Hirose, 1968).

In the present work attempts were made to study the incorporation of the tritiated acetamide into DNA of the Bar larvae. The 70 hour larvae after hatching were exposed to ^{3}H-acetamide (348 μC/ml) or ^{3}H-thymidine (333 μC/ml) for one hour, and then transferred to the condition medium (yeast 1.5%, molasses 17%, agar 1.5%) for growth until they reached the end of the larval stage. Thereafter, the materials were kept in 85% ethyl alcohol at -20°C. They were then transferred to a cold glass homogenizer and broken with cold 85% ethyl alcohol. The Schmidt-Thannhauser method (1945) was used to extract DNA from homogenized tissue.

In the process of extraction of DNA, homogenized extracts can be fractionated into the following fractions: alcohol soluble, hot alcohol·ether soluble, cold PCA soluble, RNA and DNA fractions. The radioactivity of these fractions was assayed by the liquid scintillation counter (Ten Nucleonics, GSL-163). C.P.M. of the fractions are given in Table 1.

Table 1. The radioactivity of the various fractions in the process of DNA extraction.

Fractions	^{3}H-acetamide		^{3}H-thymidine	
	C.P.M.	%	C.P.M.	%
Alcohol soluble	963,588	95.28	408.922	74.62
Hot alcohol·ether soluble	19,520	1.93	20,640	3.77
Cold PCA soluble	3,124	0.31	13,520	2.47
RNA	6,960	0.69	6,880	1.26
DNA	18,105	1.73	98,072	17.90

The ^{3}H-acetamide was apparently incorporated into DNA fraction. However, both of the ^{3}H-acetamide and ^{3}H-thymidine were marked incorporated into the alcohol soluble fraction. Incorporation into the other fractions was much less than that of the alcohol soluble fraction. Incorporation into DNA fraction was varied between the ^{3}H-acetamide and the ^{3}H-thymidine. In this respect, the ^{3}H-thymidine incorporation was 17.90%, while the ^{3}H-acetamide incorporation was only 1.73% of the total value of incorporated isotopes.

After purification of DNA, it was destroyed by 12 N PCA and was neutralized by KOH. Decomposed DNA was separated into base compositions by thin layer chromatography. The radioactivity of the base compositions, that is, thymine, adenine, cytosine and guanine, was determined by the liquid scintillation counter. The results are shown by C.P.M. in Table 2.

Table 2. The radioactivity of the base components of DNA.

Bases	^{3}H-acetamide		^{3}H-thymidine	
	C.P.M.	%	C.P.M.	%
Thymine	526	73.6	7196	96.2
Adenine	58	8.1	103	1.4
Cytosine	62	8.7	119	1.7
Guanine	68	9.5	49	0.6
(Background)	50		50	

As is apparent in the Table, the ^{3}H-acetamide was incorporated mainly into thymine among the base components of DNA. The mode of incorporation of ^{3}H-thymidine showed a similar tendency. These facts indicate that the acetamide has a close connection with the metabolism of DNA during larval development.

References: Kaji, S. 1954, Annot. Zool. Japon. 27:194-200; ____ 1960, Mem. Konan Univ., Sci. Ser. 4:1-17; DeMarinis, F. 1966 DIS 41:149-150; Hirose, Y. and S. Kaji 1968, Proc. Japan. Acad. 44:363-368; ____ 1969, Experientia 25:199-200; Hirose, Y. 1968, Mem. Konan Univ., Sci. Ser. 11:29-41; Schmidt, S. and S.J. Thannhauser 1945, J. Biol. Chem. 161:83.

De la Rosa, M.E. and R. Félix. National Institute of Nuclear Energy, Mexico City, Mexico. Chromosome X loss and non-disjunction in oocytes of D. melanogaster, induced by treatment with actinomycin D.

Actinomycin D in minute concentration is toxic to mammalian cells (Reich et al., 1961) and microorganisms (Kirk, 1960). The antibiotic selectivity inhibits RNA synthesis also in microorganisms and mammalian cells (Harbers and Muller, 1962). It seems likely that the antibiotic is primarily toxic to only those cellular activities which require the direct participation of DNA itself. The results with normal or virus infected cells indicate that actinomycin inhibits selectively the DNA-directed synthesis of RNA. The binding of actinomycin to DNA, which requires the presence of guanine in a helical configuration, is responsible for the inhibition of DNA-dependent RNA snythesis by RNA polymerase and is considered to account for the biological properties of actinomycin.

The present experiment was designed to evaluate the effects of actinomycin-D on chromosome X loss and non-disjunction, when injected to adult females of Drosophila melanogaster.

Actinomycin D dissolved in 0.7N NaCl at concentrations of 1 microg./ml., 10 microg./ml. or 100 microg./ml. were injected into localized regions of gonadal tissue, with a micropipette made at the laboratory. The physiolcgical 0.7N NaCl solution was used instead of distilled water to obviate the problem of induced sterility and possible cell selection by osmotic shock. The actinomycin D solutions were prepared less than 1 hour before each series of injections.

Since Carlson and Oster (1962) have shown that the amount of liquid expelled after injection varies from fly to fly, estimates of the amounts injected were not made. Solutions were simply injected at the ventrolateral region of the fourth or fifth abdominal segment, until the abdomen was noticeably distended. 90 to 95% of the adults treated in this way survived.

Bridges (1913) identified non-disjunction by the recovery of exceptional females and males among the progeny, by their being matroclinous and patroclinous, respectively, in phenotype for sex-linked characters. In the present work an improved method for detecting non-disjunction that gives particularly reliable evidence concerning the origin of each exceptional female and male makes use of a tester male stock with attached XY chromosomes. This stock was derived from translocations between X and Y chromosomes and has the markers y (yellow) and B (Bar). The treated female stock had the markers y^2 (yellow-2) and w^a (white apricot) in the X chromosomes, and ebony (e) in the third chromosome; this marker insures that the isolated females are virgin, when the phenotype is examined before the mating.

P	$y^2\ w^a/y^?\ w^a;e/e$	x	$In(1)EN,\ Y^S{\cdot}B\ y{\cdot}Y^L;+/+$
F_1 regular:	$y^?\ w^a/In(1)EN,\ Y^S{\cdot}B\ y{\cdot}Y^L;e/+$		$(B\ y/y^2)$
	$y^2\ w^a;e/+$		$(y^?\ w^a)$
exceptional:	$y^2\ w^a/y^?\ w^a;e/+$		$(y^?\ w^a)$
	$In(1)EN,\ Y^S{\cdot}B\ y{\cdot}Y^L;e/+$		$(B\ y)$

The computation of X-loss and non-disjunction frequencies is based on the definitions given by Traut (1964) which consider either the number of regular males (definition 1) or the number of regular females (definition 2) counted in the F_1 (in this experiment X·Y males and XX females are scored as exceptional progenie).

	X-loss frequency	non-disjunction frequency
Definition 1	$\frac{\text{X·Y males}}{\text{X/O males+ X·Y males}}$	$\frac{\text{X/X females}}{\text{X/O males+ X/X females}}$
Definition 2	$\frac{\text{X·Y males}}{\text{X/X·Y females+ X·Y males}}$	$\frac{\text{X/X females}}{\text{X/X·Y females+ X/X females}}$

The process of oogenesis in adult Drosophila has been revised in detail (King, 1970) and in so far as studies on germ cells stage sensitivity are concerned, the essential features are as follows: an ovary of an adult female Drosophila melanogaster consists of a parallel cluster of ovarioles, each of which is differentiated into an anterior germarium and a

posterior vitellarium (Koch, Smith and King, 1967). The development of the egg chamber has been subdivided into a series of consecutive stages, ending with stage 14, the mature primary oocyte (King, Rubinson and Smith, 1956). At eclosion all oocytes are in previtellogenic stages, and the most posterior egg chamber in each ovariole is generally in stage 7. During the first day of adult life vitellogenesis begins, and stage 14 oocytes appear during the second day.

The differences in response of stages 7 and 14 to irradiation are both qualitative and quantitative. Stage 14 shows a higher incidence of all types of genetic damage which have been looked for, with break-rejoining delayed until fertilization, while breaks induced in stage 7 rejoin in about 10-15 minutes (Parker, 1955; Parker and Hammond, 1958; King, Darrow and Kaye, 1956).

In the case of chemical treatments administered by injection, a longer period of treatment is required than with irradiation, and the responsible mutagenic reaction(s) is expectedly prolonged after the period of treatment. Thus, if newly emerged adult females are treated by injection, the most advanced germ cell stages present during the 24 hours following the treatment (unless the treatment itself delays development), is the stage(s) immediately preceeding stage 7, stage 7 itself, and stages 7-13 which have developed during the 24 hr period after the treatment. The sensitivity of the stage 14 oocyte to adult injection can be studied with more confidence, since no further development of this cell takes place during or after treatment. Consequently, although an accurate study of the mutational response of the premeiotic stage 7 primary oocyte can not be made after chemical treatment, a comparison can be made of the mutational responses covering approximately stages 7-13 with the mutational response of stage 14.

The treatment to the oocyte stages with actinomycin D at the concentrations named above, was parallel to a control group, without treatment, and to a group injected with 0.7N physiological saline solution. The actinomycin D solution was prepared half an hour before the injection dissolving this substance in a drop of 25 per cent ethanol, and diluting in the saline physiological solution to the concentration desired.

In order to treat oocytes of the 7-13 stages, virgin females were isolated from 0 to 24 hours after eclosion; they were injected and then maintained in cultures without males during 24 hours before transferring them for breeding, to vials, each with 2 males.

So that the oocytes would be in the 14 stage in the moment of being treated, the virgin females were isolated and allowed to age during 4 days, after which they were injected and immediately bred. In each case, only the eggs deposited during the first 24 hours in every individual culture, corresponding to the treated oocytes, were collected.

The scoring of first generation descendants was made 13 days after the date of treatment of the P females, so that it might include all the progeny, classifying them in four phenotypes that correspond to "exceptional" individuals and "normal" individuals, following the scheme previously described.

The exceptional females originate from non-disjunction and the exceptional males from the loss of the X-chromosome which takes place during oogenesis. The individuals originating from non-disjunction, of the constitution $y^2\ w^a/y^2\ w^a/In(1)EN,\ Y^S \cdot B\ y \cdot Y^L$, are metafemales with a very low viability. In the present experiment not one individual whose phenotype corresponded to such genetic constitution was found.

Effect of actinomycin D upon fertility. The administration of actinomycin D in different concentrations, during the 7-13 and 14 stages of oogenesis of D. melanogaster, produced a diminution in the fertility of the females injected. The data contained in Table 1 illustrates the dose-effect relation which is most noticeable in the oocytes of stage 14, corresponding to the stage of oogenesis with the greatest sensitivity to the antibiotic. The number of descendants originating from the 7-13 stages of treatment, isolated during the first 24 hours of oviposition, is greater than the number of descendants originating from the 14 stage, isolated during the same period. The effect that actinomycin D had upon the fertility of the oocytes can be related to the inhibitory activity of genetic transcription which shows this compound. The intensity of the effect seems to depend upon the doses administered, within the limits of concentration applied in this experiment. It is necessary to take also into consideration the notable differences in metabolic activity that exist between the two stages, in order to explain this reduction resulting from sterility or from induced meiotic delay.

Table 1. Total progeny and average per vial from each treatment of 7-13 and 14 oocyte stages.

Group	Total progeny/number of vials		Average progeny per vial	
	Stages 7-1	Stage 14	Stages 7 13	Stage 14
Control	819/29	1276/23	28	42
Saline solution	934/27	519/17	31	17
Actinomycin 1 µg/ml	1125/26	589/14	36	19
Actinomycin 10 µg/ml	1441/75	843/48	19	17
Actinomycin 100 µg/ml	332/17	604/40	19	15

The frequency of non-disjunction and loss of X-chromosome. Tables 2 and 3 contain data on the numbers of normal and exceptional offspring that were obtained from oocytes treated during the 7-13 and 14 stages of oogenesis.

Table 2. Normal and exceptional progenies from each of the groups treated during 7-13 oocyte stages.

Group	Normal progeny		Exceptional progeny	
	females	males	females	males
Control	346	483	0	0
Saline solution	400	532	1	0
Actinomycin 1 µg/ml	493	686	0	0
Actinomycin 10 µg/ml	606	835	0	0
Actinomycin 100 µg/ml	111	219	0	2

Table 3. Normal and exceptional progenies from each of the groups treated during 14 oocyte stage.

Group	Normal progeny		Exceptional progeny	
	females	males	females	males
Control	530	742	0	1
Saline solution	262	256	1	0
Actinomycin 1 µg/ml	307	281	1	0
Actinomycin 10 µg/ml	319	513	9	2
Actinomycin 100 µg/ml	264	340	4	4

The percentages of exceptional offspring obtained among the descendency of the oocytes of D. melanogaster treated with different concentrations of actinomycin D during the stages of oogenesis studied, are contained in Tables 4 and 5, following the definitions given by Traut (1964).

Table 4. Chromosome X loss and non-disjunction from each of the groups treated during 7-13 oocyte stages.

Group	Chromosome X loss (%)		Non-disjunction (%)	
	def.1	def.2	def.1	def.2
Control*	0	0	0	0
Saline solution	0	0	0.18 ± 0.42	0.24 ± 0.48
Actinomycin 1 µg/ml	0	0	0	0
Actinomycin 10 µg/ml	0	0	0	0
Actinomycin 100 µg/ml	0.90 ± 0.94	1.76 ± 1.32	0	0

* The spontaneous frequencies for non-disjunction and X chromosome loss for this stock amount to about 0.11% and 0.07% respectively.

The administration of actinomycin D in a 100 µg/ml concentration during the 7-13 stages increases the frequency of the loss of the X chromosome, which is statistically significant. The group which was injected with physiological serum, has, likewise, a significant number of individuals originating from non-disjunction.

Table 5. Chromosome X loss and non-disjunction from each of the groups treated during 14 oocyte stage.

Group	Chromosome X loss (%)		Non-disjunction (%)	
	def.1	def.2	def.1	def.2
Control*	0	0	0	0
Saline solution	0	0	0.38 ± 0.61	0.38 ± 0.61
Actinomycin 1 µg/ml	0	0	0	0
Actinomycin 10 µg/ml	0.38 ± 0.61	0.62 ± 0.78	1.72 ± 1.31	2.74 ± 1.65
Actinomycin 100 µg/ml	1.17 ± 1.08	1.49 ± 1.22	1.17 ± 1.08	1.49 ± 1.22

* The spontaneous frequencies for non-disjunction and X-chromosome loss for this stock, amount to about 0.11% and 0.07%, respectively.

The frequency of the loss of the X-chromosome during the 14 stage (Table 5) because of the treatment with actinomycin D, increases significantly in the individuals of the groups injected with physiological serum and actinomycin D in the concentrations of 10 µg/ml, and 100 µg/ml.

The frequency of non-disjunction, as the frequency of the X-chromosome loss, resulting from the inhibition of protein synthesis, is greater when the treated oocytes are found in stage 14. The oocytes at this stage are in a very intense stage of metabolism, in its proximity to fecundation.

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Belyaeva, E.S. Institute of Cytology and Genetics, Novosibirsk, U.S.S.R. Asynapis of homologues in Drosophila melanogaster salivary chromosomes.

The asynapsis of homologues is unevenly distributed along the chromosomes of D. melanogaster (Figure 1). The occurrence rate of chromosomes with conjugation impairment and the distribution of asynapsis does not depend upon the conditions in which the culture was maintained (different food, temperature variations from 12-25°C) and upon how the preparations were obtained (staining for 2 min, squashed preparations in acetocarmine or staining for 12 hours in HCl containing orcein and squashing in lactic acid). The occurrence rate and distribution of asynapsis are the same in different D. melanogaster stocks. In all the wild stocks asynapsis is much more frequent at the base of the X-chromosome. The analysis of the X-chromosome with scute-inversions suggest that asynapsis is not due to the properties of chromosome regions of the X, but rather to close localization with respect to heterochromatin and to the amount of this chromatin (Figures 2 and 3). An exception is X with sc^{v2} inversion, on whose tip a heterochromatic region is transferred, smaller than that in sc^{8} X, but with much more frequent asynapses. If the position of the right break point of the inversion in stock sc^{v2} actually conforms to the generally accepted scheme (Figure 2), then this may be indicative of a qualitative heterogeneity of heterochromatic blocks in the heterochromatin of the X-chromosome and of their specific influence on asynapsis.

The author is indebted to Prof. I.I. Kiknadze for valuable discussions.

Reference: Baker, W.K. 1971, Proc. Nat. Acad. Sci. USA 68:2472-2476.

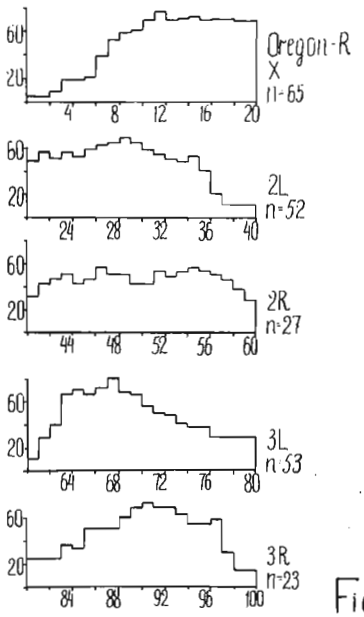


Fig.1

Figure 1. Asynapsis of homologues along chromosome in D. melanogaster (Oregon-R); abscissa - chromosome regions; ordinate - asynapsis frequency in chromosomes with impaired conjugation; n - number of chromosomes studied.

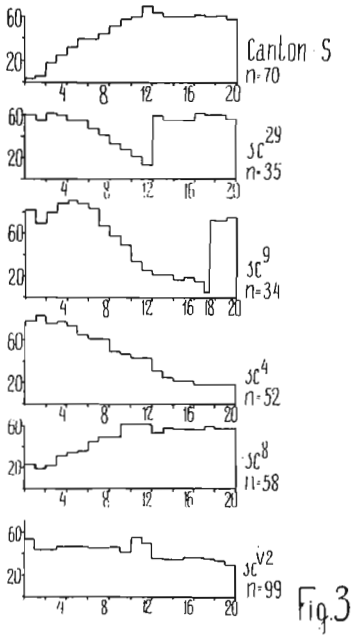


Fig.3

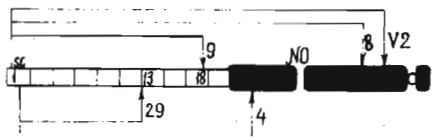


Fig.2

Figure 2. Scheme of the X-chromosome scute inversions. Euchromatic regions are open, heterochromatic regions are closed. NO - nucleolus organizer. (Baker, 1971)

Figure 3. Asynapsis of homologues in X-chromosomes in stock Canton-S and stocks with sc inversions.

Peacock, W.J. and G.L.G. Miklos. CSIRO, Canberra, Australia and Australian National University, Canberra, Australia.

Light microscope analysis of spermiogenesis in Drosophila melanogaster males exhibiting meitoic drive.

The electron microscope studies of Tokuyasu et al. (1972a, b) have revealed many of the processes involved in normal spermiogenesis in D. melanogaster. These authors also described the modifications of some of these processes which occur in Segregation Distorter heterozygotes and homozygotes and we have recently described similar events in the sc^4sc^8 meiotic drive system (Peacock et al. 1973). We have now found that it is possible to detect a number of these normal and abnormal spermiogenic processes in light microscope preparations. Since the time involved in slide preparation for light microscopy is small relative to that needed for electron microscope analysis, we consider that this technique may be useful in characterising spermiogenic lesions in Drosophila stocks.

Methods: Testes of males (usually 0-24 hours old) are dissected in an aqueous solution of 0.7 percent NaCl, the accessory glands removed and the testes transferred to freshly mixed glacial acetic acid:absolute ethanol (1:3). Testes can be stored at -20°C in fixative for extended periods if necessary. The fixed testes are then placed in IN HCl at 60°C for 5 minutes, stained for 30 minutes with feulgen, and examined in 45 percent acetic acid with phase optics. For most observations very little pressure need be applied to the coverslip. All of the solution transfers may be conveniently carried out with a hypodermic syringe to avoid mechanical damage to the testes.

Results: The arrangement of the various developmental stages within the testis is readily seen in a favourable squash. Gonial mitoses in the apical region are sometimes visible and meiotic cysts are frequently observed in the mid-region of the testis. The head regions of the majority of spermatid cysts are grouped in the basal portion of the testis with the tail regions of the cysts extending the full length almost to the apex. Relatively few groups of heads are seen in the mid-region and the ones that do occur there are in early stages of spermatid development. It is thus possible to quantitate the developmental and spatial distribution of cysts in the testis. All these observations are in accord with the descriptions from the electron microscope studies.

Tokuyasu et al. (1972a) described the process of individualisation whereby each of the 64 spermatids, which have developed in the germinal syncytium, are enclosed with a membrane. This process occurs after spermatid elongation. Individualisation is initiated in the head region of the cyst and proceeds caudally. The diameter of the spindle-shaped individualisation bulge (Figure 1) increases as the bulge proceeds along the cyst (Figure 2), the increase being due to the accumulation of cytoplasmic debris. Under the light microscope the individualisation bulge is clearly discernible, and the individualised spermatids (basal to the bulge) and syncytial spermatids (apical to the bulge) have distinctive appearances. The bulge eventually forms a "waste bag" in the apical region of the testis. This too is visible in the light microscope as a patchy, refractile structure near the gonial cysts (Figure 3).

One of the more remarkable processes of spermiogenesis is the entrapment of the head region of each cyst by the cells of the terminal epithelium in the base of the testis (Tokuyasu et al. 1972b). This head trap is not often obvious under the light microscope with the present technique, but can occasionally be seen (Figure 4). More frequently, in developing cysts, the relation of the head cyst cell to the spermatid heads is very clear (Figure 5).

Once the bundle of 64 individualised spermatids is firmly anchored in the terminal epithelium, the entire linear bundle is "reeled in" during the process of coiling. As coiling proceeds, the cyst is distended in the basal region and the waste bag is withdrawn from the apex down the full length of the testis. It is at this stage of spermiogenesis in the SD/SD^+ and sc^4sc^8 meiotic drive systems that developmental lesions are first obvious under the light microscope. Spermatids which have failed to be individualised are not coiled in the regular manner of individualised spermatids but are loosely folded into the descending waste bag (Figure 6). This figure also shows that non-individualised, or syncytial, spermatids are also visible in the linear cyst because of their increased refractivity and granular appearance. In control males where there are no abnormal spermatids, waste bags are very difficult to see at the base of the testis (Figure 7), and in fact there is a progressive loss of the patchy appearance as it moves down the length of the testis. However in both SD heterozygotes and sc^4sc^8 males waste bags containing abnormal spermatids in various stages of breakdown can be seen in the base of the testis (Figures 8 and 9). Although we have not been able to quantitate this breakdown in respect to the extent of meiotic drive in a particular male, there is a clear distinction between meiotic drive and control males.

Many modifications can be made to this technique. We have found it convenient to make an initial analysis as to the presence or absence of syncytial spermatids, inclusion of syncytial spermatids into descending waste bags, presence or absence of degenerating spermatids in the base of the testis and then to squash more heavily in order to do a detailed analysis of meiotic cysts in the same testis.

This technique should prove useful in surveys for cases of abnormal spermatid development and for characterising stocks showing meiotic drive and partial or complete sterility.

References: Peacock, W.J., G.L.G. Miklos and D.G. Goodchild 1973, Sex chromosome meiotic drive systems in Drosophila melanogaster. I. Abnormal spermatid development in males with a heterochromatin deficient X chromosome (sc^4sc^8). Submitted to Genetics; Tokuyasu, K.T., W.J. Peacock and R.W. Hardy 1972a, Z. Zellforschung Mikroskopische Anatomie 124:479-506; _____ 1972b, Z. Zellforschung Mikroskopische Anatomie 127:492-525.

Figures 1-5 on Facing Page

Fig. 1. Individualisation bulge (IB) in the lower one third of the testis proceeding along a cyst toward the apex of the testis and leaving behind it individualised spermatids (IS) whilst accumulating cytoplasmic and nucleoplasmic debris.

Fig. 2. A larger individualisation bulge in the upper one third of the testis showing a more granular appearance of the contents.

Fig. 3. A waste bag (WB) of debris of the apex at the testis. Although now separated from the spermatid tails, the waste bag is still within the cyst lumen.

Fig. 4. Basal region of the testis showing coiled spermatid bundles (CB) with the head region of one cyst embedded in the terminal epithelium, or head trap (HT).

Fig. 5. Mid region of the testis showing an immature cyst of spermatids with syncytial tails (ST) with the heads embedded in the head cyst cell (HC). The head cyst cell-spermatid head region of the cyst will subsequently move basally and be involved in the head trap with terminal epithelial cells.

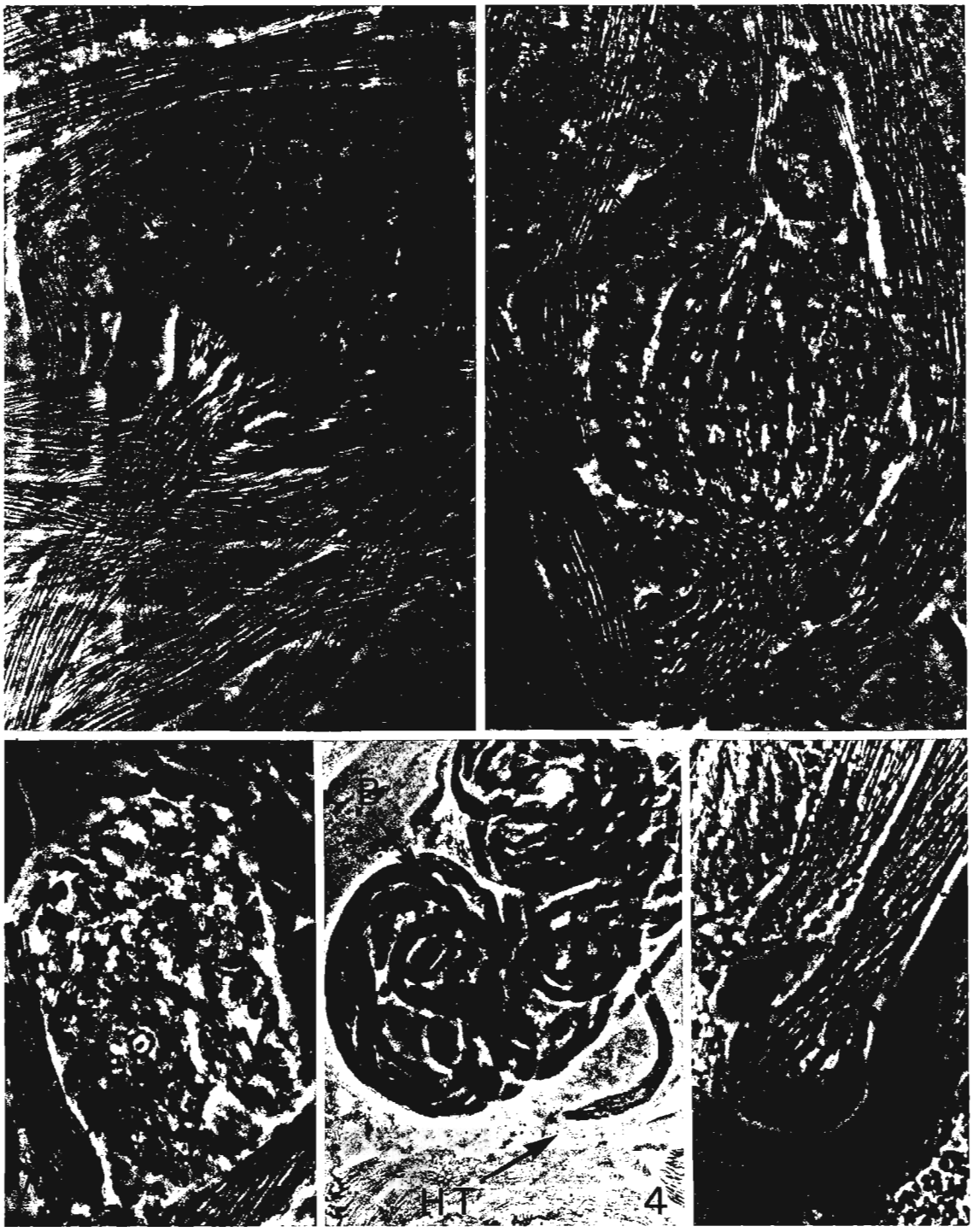
Figures 6-9 on Following Page

Fig. 6. Mid region of the testis of a sc^4sc^8/y^+Y male exhibiting meiotic drive. A spermatid bundle is in the process of coiling. The individualised spermatids (IS) are distinguishable from the abnormal syncytial spermatids (SS) which have a granular and irregular appearance. The syncytial spermatids terminate in the waste bag (WB) and are folded irregularly into the waste bag as it is drawn basally down the testis during coiling of the normal spermatids.

Fig. 7. Portion of the reproductive apparatus of a control male (y/y^+Y) showing coiled bundles of sperm (CB) in the base of the testis, the testicular duct (TD) and the seminal vesicle (SV) packed with masses of mature sperm (MS). It should be noted that no refractile bodies are visible near the coiled sperm bundles in the base of the testis.

Fig. 8. Portion of the reproductive apparatus of a Segregation-Distorter heterozygote. Coiled bundles of sperm (CB) are seen in the base of the testis along with degenerating spermatids (DS). These refractile bodies represent waste bags containing abnormal nonindividualised spermatids.

Fig. 9. High power shot of the basal testicular region of a sc^4sc^8/y^+Y male exhibiting meiotic drive. The degenerating spermatids (DS) are easily recognizable and are an unmistakable indication of abnormal spermatid development. A coiled bundle of sperm (CB) has its heads clearly visible (SH).


HT
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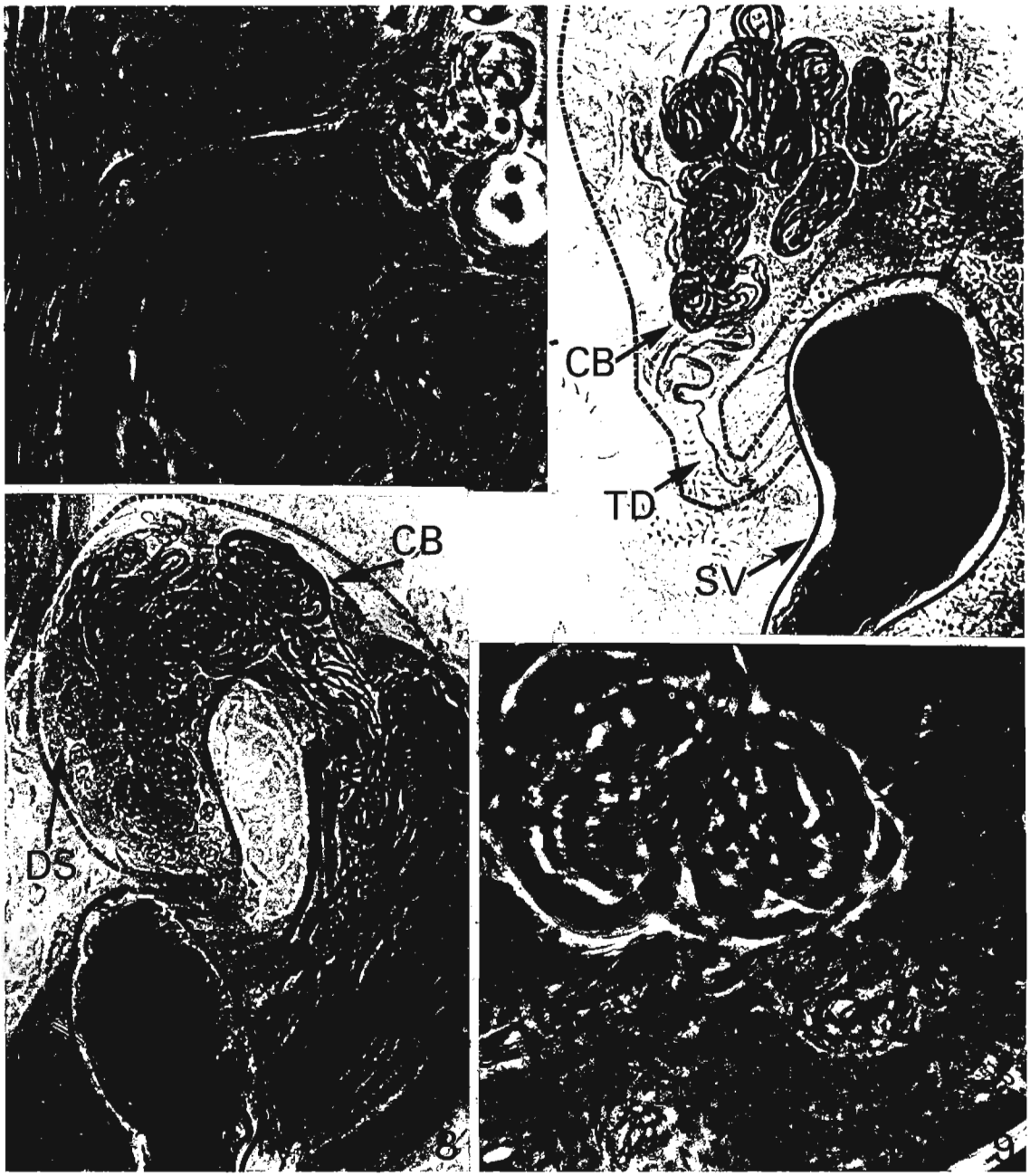
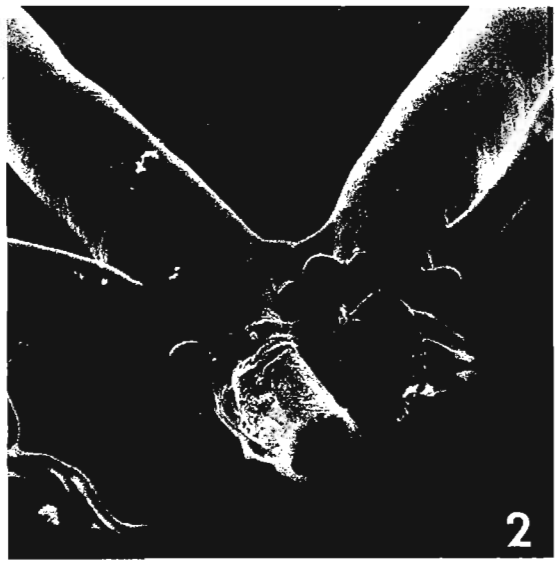
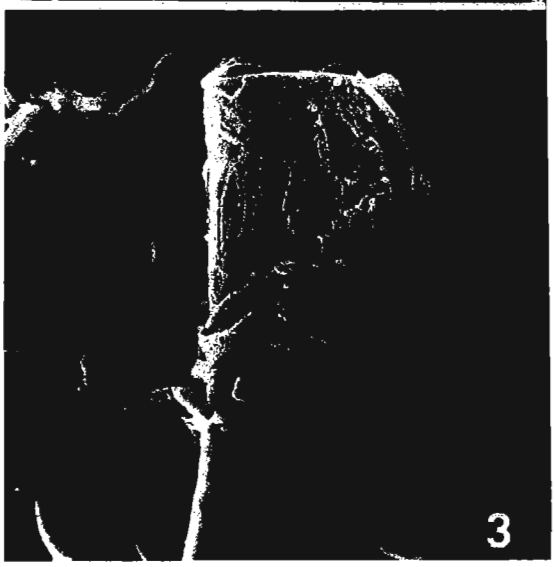

CB
TD
SV
CB
DS

Rizki, T.M. and R.M. Rizki. University of Michigan, Ann Arbor. The micro-topography of the posterior spiracles of D. melanogaster larvae.

The posterior spiracles and the spiracular glands of third instar larvae of D. melanogaster were examined with the scanning electron microscope. Figure 1 (1000X) illustrates the structures associated with the posterior spiracles. The four peristigmatic tufts of bristles are unusual since unlike the ordinary bristles of Drosophila, they are palmate, branched and ribbonlike or lamellar in appearance, and hinged at the base. This structural feature together with a lipid coating provides increased hydrophobic surface area to keep the larva afloat in semi-liquid medium as well as for trapping of air bubbles over the spiracular slits while the larva is tunneling through the medium. Some of these palmate lamellar bristles as well as the accumulations of lipid globules from the openings of the peristigmatic glands can be seen in lateral profile in the lower spiracle in the photograph. Three slit-like spiracular clefts can be visualized on each spiracle. At the end of each cleft proximal to the basal region of the bristles is the opening of the peristigmatic gland. The two main dorsal tracheal trunks, each with three peristigmatic glands, are pictured in Figure 2 (250X) and the duct of one of these unicellular glands can be seen along the surface of the tracheal trunk in Figure 3 (750X). Cytological details of these glands and their secretory function have been presented previously (Rizki, 1956, J. Morphology 98:497).

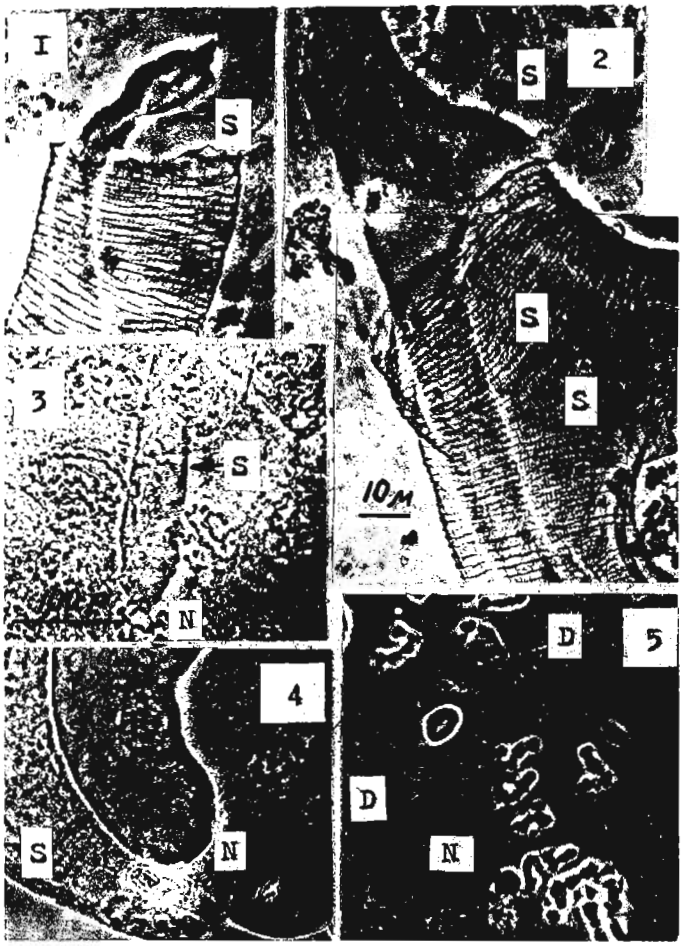




We are using the technique of critical point drying for external morphology of Drosophila as well as for examining the internal anatomy of larvae with particular emphasis on the topology of various cell types. Photographs of fat body cells and hemocytes in normal and tumorous strains will be published elsewhere. (Supported by NIH Grant CA12600.)

Zhimulev, I.F. Institute of Cytology and Genetics, Novosibirsk, U.S.S.R. Description of a new type of secretion in the larval salivary gland of D. melanogaster.

The mucoprotein secretion which appears in the salivary glands of Drosophila larvae shortly before puparium formation has been described in a number of papers (reviewed by Lane et al. 1972). The secretion has a rough granular appearance under phase contrast microscope (Zhimulev, Kolesnikov, in press). This paper describes new type of secretion, the so-called "silk-secretion" which differes sharply from the mucoprotein. Patches of this secretion have been found in the ducts of all the Batumi-L larvae studied (Figure 1) and in $l_{(2)}gl$ larvae. In the $l_{(3)}tl$ larvae the silk-secretion is easily seen because there is no mucoproteine secretion in the salivary gland (Zhimulev, Kolesnikov, in press). The silk-secretion has been observed both in the duct



Figures 1. End of a salivary gland duct with a patch of the silk-secretion; Batumi-L stock, 115 hours, phase contrast. 2. The same, $l_{(3)}tl$ stock, 144 hours. 3. Secretion in the distal part of the gland; $l_{(3)}tl$ stock, 168 hours. 4. The same, 264 hours. 5. Droplets of the silk-secretion in the cytoplasm of salivary gland; $l_{(3)}tl$ stock, 408 hours. S-secretion, N-nucleus, D-droplet of secretion.

(Figure 2) and in the distal part (Figures 3, 4). The mass of the secretion increases with larval age (Figure 4). Information of the silk-secretion is represented in the Table. In 264-408 hour old $l_{(3)}tl$ larvae droplets of a substance reminiscent of silk-secretion are observed in the cytoplasm (Figure 5). It may be suggested that the silk-secretion possesses a digestive function or it is a predecessor of the mucoprotein secretion.

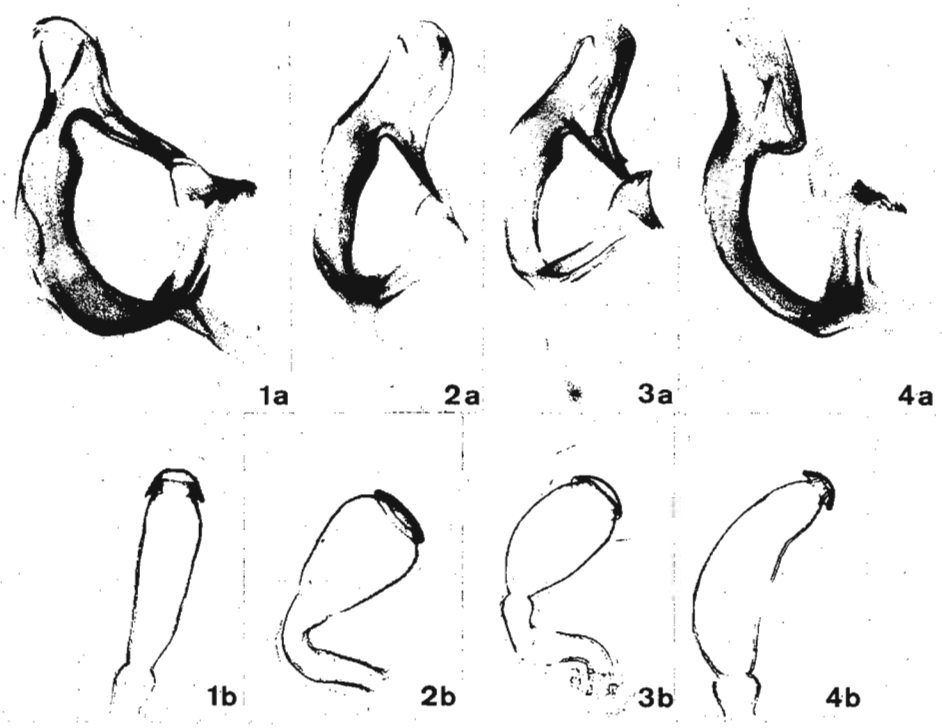
Reference cited: Lane, N.J., J.R. Carter and M. Ashburner 1972, Wilhelm Roux' Archiv 169:216-238.

Presence of silk-secretion in salivary glands

Larvae	Hours after oviposition	Total number of glands	Glands with silk-secretion
$l_{(2)}gl$	168	10	10
$l_{(3)}tl$	120	33	26
(lethal tumorous larvae)	132	21	18
	144	30	21
	168	30	18
	264	23	9
	408	34	9
Laboratory stocks:			
Batumi-L	90-120	20	20
Oregon-R	90-120	8	6

Glätzer, K.H. Universität Düsseldorf, Germany. The status of Drosophila "pseudoneohydei."

In 1962 the Max-Planck-Institut für Biologie in Tübingen, Germany, received D. neohydei WASSERMAN from Austin, Texas, carrying the stock number H 207.26. Since this stock was difficult to maintain in the laboratory, a second, supposedly better breeding one, was subsequently ordered (Number H 186.58). An investigation of the nuclear structures in the spermatocytes of this stock carried out by O. Hess did not, however, support the classification as D. neohydei and the stock was, therefore, given the provisional name D. "pseudoneohydei." In spite of the International Rules of Zoological Nomenclature which prevent the use of species designations not yet officially published, this name, designated for internal laboratory use only, has already appeared in the literature (Hennig et al., 1970). In early 1973 this species was newly investigated in this laboratory and has now finally been conclusively identified as being D. eohydei WASSERMAN. The identification was carried out by the appearance of the reproductive organs, the penes and the spermathecae, organs which are very characteristic for these species of the repleta group (Wasserman, 1962). The photos clearly show the equality between D. eohydei (Figs. 2a, b) and D. "pseudoneohydei" (Figs. 3a, b) as well as the structural characteristics of the penes and spermathecae of D.



hydei (Figs. 1a, b) and D. neohydei (Figs. 4a, b). The conclusions drawn from these morphological criteria are in full agreement with breeding data. For instance, progeny can be obtained from both reciprocal crosses between D. eohydei and D. "pseudoneohydei," whereas crosses between D. neohydei and D. "pseudoneohydei" in either direction are always unsuccessful. On the basis of these investigations, as well as those carried out by Wasserman in 1962 (in which stock number H 186.58 was already listed under D. eohydei!), it is unequivocally clear that D. "pseudoneohydei" is, in reality, a stock of D. eohydei. With this clarification it is hoped that the "remarkable status" of D. "pseudoneohydei" (Wheeler, 1972) can now be assumed to represent a closed case.

References: Hennig, W., I. Hennig and H. Stein 1970, Chromosoma (Berlin) 32:31-63; Wasserman, M. 1962, Univ. Texas Publ. 6205:73-83; Wheeler, M.R. 1972, DIS 48:154.

Katoh, S. Josai Dental University, Sakado-machi, Saitama-ken, Japan. Two yellow eye pigments of D. melanogaster catalyzed by sepiapterin reductase.

Two yellow pigments have been isolated and identified from the eye of mutant sepia of D. melanogaster: sepiapterin (2-amino-4hydroxy-6-lactyl-7,8-dihydropteridine) and isosepiapterin (2-amino-4hydroxy-6-propionyl-7,8-dihydropteridine). Sepiapterin, the main yellow pigment in the eye, was found to be catalyzed to a non-color substance with the aid of NADPH by an extract of the fat body of adult Drosophila or by a homogenate of mammal liver of several species. This catalyzation of sepiapterin is, in fact, mediated by an enzyme named "sepiapterin reductase" as the following equation:

$$\text{sepiapterin} + \text{NADPH} + \text{H}^+ \rightleftharpoons \text{dihydrobiopterin} + \text{NADP}^+$$

Sepiapterin is reduced to dihydrobiopterin (2-amino-4-hydroxy-6-1',2'-dihydroxypropyonyl-7,8-dihydropteridine) by the enzyme, the C^6-substituted lactyl group of sepiapterin is converted to the 1',2'-dihydroxypropyl group[1].

Sepiapterin reductase has recently been purified 5000-fold from an extract of horse liver by protamine sulfate treatment, ammonium sulfate fractionation and column chromatography of hydroxylapatite and of DEAE-cellulose[2]. The pH optimum of the purified enzyme is 5.5 and Km values of sepiapterin and NADPH are 2.1×10^{-5} M and 1.4×10^{-5} M, respectively, at pH 6.4.

Isosepiapterin and xanthopterin B_2 (2,4-dihydroxy-6-lactyl-7,8-dihydropteridine, isolated from mutant lemon of silkworm) are also reduced by the purified enzyme at a very slow rate (2 and 3% of that of sepiapterin, respectively). All these yellow pteridines have a carbonyl group in $C^{1'}$ in C^6 - side chain and are 7,8-dihydroform.

When isosepiapterin is added to the reaction mixture of sepiapterin reductase with sepiapterin, the reduction of sepiapterin by the enzyme is strongly inhibited. Besides isosepiapterin, many other eye pigment pteridines such as 6-carboxypterin, biopterin, leucopterin and pterin also inhibit the enzyme reaction more or less. % inhibitions are 83, 77, 24, 21 and 18, respectively, when 5×10^{-5} M of each pteridine is added to the same concentration of sepiapterin in the reaction mixture.

The enzyme reaction is reversible as shown in the equation. The pH optimum of this reverse reaction is 10.5. Both reactions of reduction and oxidation are possible to proceed significantly at neutral pH, although the equilibrium lies in favor of reduction (formation of dihydrobiopterin) ($-\Delta G = -12$ Kcal per mole).

These above natures of sepiapterin reductase suggest the complexity of biosynthesis of eye pigment pteridine in Drosophila.

In mammal liver, sepiapterin is reduced to tetrahydrobiopterin by the functions of NADPH-dependent sepiapterin reductase and dihydrofolate reductase as below[3]:

$$\text{sepiapterin} \xrightarrow[\text{(sepiapterin reductase)}]{\text{NADPH} + \text{H}^+} \text{dihydrobiopterin} \xrightarrow[\text{(dihydrofolate reductase)}]{\text{NADPH} + \text{H}^+} \text{tetrahydrobiopterin}$$

Dihydrobioterin has been isolated from rat liver[4]. Tetrahydrobiopterin, thus reduced from sepiapterin, serves as the indispensable natural cofactor of liver phenylalanine hydroxylase, brain and adrenal medulla tyrosine hydroxylase and brain tryptphan 5-hydroxylase, and then, it controls the synthesis of noradrenaline, adrenaline, serotonine and melatonine.

Isosepiapterin can also be converted to an active cofactor of this system but with a very slow rate.

Therefore, sepiapterin and isosepiapterin are not only eye pigments of Drosophila but also very active substances in animals.

References: 1) Nagai, M. 1968, Archi. Biochem. Biophys. 126:426; 2) Katoh, S. 1971, Arch. Biochem. Biophys. 146:426; 3) Matsubara, M., S. Katoh, M. Akino and S. Kaufman 1966, Biochem. Biophys. Acta 122:202; 4) Kaufman, S. 1963, Proc. Natl. Acad. Sci. USA 50:1085.

Siddaveere Gowda, L., H.A. Ranganath and N.B. Krishnamurthy. University of Mysore, Manasagangothri, India. Virginity and longevity in Drosophila.

The viability, longevity, fecundity and fertility are some of the adaptive components of a population. Population density and its regulation depends on these characteristics. The present report deals with the longevity of virgin and non-virgin flies of D. nasuta. Virgins were isolated from the Coorg strain of D. nasuta. 30 males and 30 females were placed separately in milk bottles. Here the mating activity and hence reproduction is avoided. In another series, 15 males and 15 females were kept together thus allowing them to participate in copulation and reproduction. Once in 10 days, the flies were transferred to fresh bottles and the number of dead flies was recorded. Table 1 gives the mean longevity in days of the two sexes both in independent and mixed cultures. The summary of the student t-test is given

Table 1. Mean longevity in days of males and females in independent and mixed cultures

	Independent		Mixed	
Replicates	Males	Females	Males	Females
1	61.66 ± 3.25	60.33 ± 3.33	34.33 ± 3.70	42.33 ± 6.56
2	65.33 ± 3.97	55.83 ± 4.83	41.00 ± 4.86	23.00 ± 2.42
3	60.33 ± 2.58	62.00 ± 2.82	39.66 ± 5.47	33.00 ± 5.59
4	60.00 ± 3.87	63.00 ± 3.34	51.60 ± 4.79	33.00 ± 5.74
5	-	-	46.33 ± 4.48	33.00 5.47
Average	61.83 ± 1.66	61.50 ± 1.47	42.60 ± 2.10	32.86 ± 2.47

in the Table 2. In brief, the unmated males and females have more life span over the corresponding mated sexes. This confirms the concept of Dobzhansky (1968) that virgin females of Drosophila live longer on the average than those inseminated and actively ovipositing.

Table 2. Summary of the student t-test computed to compare the mean longevities of the two sexes.

1.	Independent males	= Independent females	t = 0.37	p > 0.50
2.	Independent males	> Mixed males	t = 2.56	p = 0.025-0.010
3.	Independent females	> Mixed females	t = 4.08	p = < 0.001
4.	Mixed males	> Mixed females	t = 7.10	p = < 0.001

This situation may not simulate the conditions in a natural population. Mating and actively reproducing individuals are the positive contributors to the population structure and growth. Stern (1970) has opined that the relative number of progeny produced by individuals is controlled by some systematic factors which are equally scrutinised by natural selection. One of them is the 'death of the organisms'. The indefinite prolongation of life and reproductive capacity would lead to overpopulation. It would interfere with further evolution since evolution involves replacement of older forms of life by newer ones and presumably better adapted ones (Dobzhansky, 1968). Thus, at the population level, natural selection puts an embargo on the higher longevity of the reproducing individuals, thus regulating the population density.

Relatively, the organisms involved in reproduction may wear out more with age than the non-reproducing ones. Thus, the physiological senescence may set-in at an earlier date and curtail the life span of reproducing individuals.

Acknowledgments: Authors wish to express their most sincere gratitude to Professor M.R. Rajasekarasetty for his valuable suggestions and criticisms. This work is financially supported by the Mysore University Research Grants.

References: Dobzhansky, Th. 1968, In: Evolutionary Biology Vol. II pp 1-34; Stern, J. T. 1970, In: Evolutionary Biology Vol. IV pp 39-66.

Doane, W.W., M.M. Kolar and P.M. Smith.
Yale University, New Haven, Connecticut.

Purified α-amylase from D. hydei.

Anylase was extracted from adults of the Zurich strain of D. hydei, which is homozygous for the electrophoretic variant Amy^7. Flies were mass reared (Doane, DIS 45:189, 1970) and aged one week on a starch-yeast diet to increase enzyme production.

Partial purification was achieved through modification of the method Loyter and Schramm (Biochim. Biophys. Acta 65:200, 1962). By this method α-amylases with two or more binding sites per molecule are specifically precipitated from crude extracts as a glycogen-enzyme complex insoluble in 40% ethanol. However, some unidentified contaminating proteins were also precipitated by this means from the Drosophila material. These were eliminated by concentrating the proteins in the partially purified extract through lyophilization and then subjecting it to slab acrylamide gel electrophoresis. The apparatus of Roberts and Jones (Analyt. Biochem. 49:592, 1972) was used in a preparative manner, with 5% acrylamide, 0.1 M Tris-borate buffer, pH 9.4, and a constant voltage of 450 V for 1 1/2 hours at 7°C. Amylase migrated as a single band to a position relatively distant from contaminants so that it could be easily cut out and eluted. The eluted enzyme was dialyzed against deminized water for 16 hrs and lyophilized to dryness. Samples of purified amylase were tested for protein impurities by running them through the disc electrophoresis procedure and staining the gels with Coomassie Blue, a very sensitive protein stain. No contaminants were detected by this means.

Data on the partially purified extract from a sample of 71.3 g of flies (Doane, Kolar and Smith, Genetics 74:s64, 1973, and in prep.) are given in the table below. Two different assays were used to determine specific activities (Doane, J. Exp. Zool. 171:321, 1969); the amylase yield and purification factor for each are in fair agreement. Lowry assays for total protein indicated that only 0.3% of the soluble protein in the crude extract was present in the partially purified S_7 extract. Further analysis showed that approximately one-third of this protein was α-amylase. Hence, this enzyme makes up about 0.1% of the total soluble protein in the original crude extract, S_1.

Partial purification of amylase
Source: D. hydei, 71.3 g of flies

Extract	Protein[1]		Reduction Assay[2]			Starch-Iodine Assay[3]		
	Total	Yield	Spec. Activ.	Enzyme Yield	Purif. Factor	Spec. Activ.	Enzyme Yield	Purif. Factor
	mg	%	MU/μg	%		SU/μg	%	
Crude (S_1)	4,118.00	100.0	4.28	100.0		2.16	100.0	
Partially purified (S_7)	12.18	0.3	975.52	67.5	227.9	453.45	62.2	209.9

[1] Method of Lowry et al. (J. Biol. Chem. 193:265, 1951).
[2] 3,5-Dinitrosalicylic acid reduction assay: 1 MU = 10^{-4} μmoles maltose equiv./min. at 25°C.
[3] Starch-Iodine assay: 1 SU = 10^{-4} mg starch hydrolyzed/min. at 25°C.

The Molecular Weight of purified "α-amylase-7" was determined in SDS gels by the method of Weber and Osborn (J. Biol. Chem. 244:4406, 1969; also, Dunker and Rueckert, Ibid.: 5074). The average of four independent estimates was 54,500 daltons, with a deviation from this in any given run of < 5% (Doane and Kolar, Isoz. Bull. 7:in press). Reduction of the amylase in 1% solutions of SDS containing 1% 2-Mercaptoethanol, ± 4 M urea, gave no indication of dissociable subunits. Neither did treatment with 0.0015 M Dithiothreitol, a more powerful reducing agent than 2-Me. It is concluded that the amylase molecule in Drosophila is a monomer, as suggested by genetic analyses. (Supported by NSF grant GB 29276 and USPHS grant GM 18-72901A1).

Agnew, J.D. University of the Witwatersrand, Johannesburg, South Africa. Morphological differences in larval D. melanogaster and simulans.

Third instar larvae (♂♂ and ♀♀) of wild-type strains of melanogaster (Cape Town) and simulans (Zoutpansberg) were fixed in the extended condition in warm (45-50°) Bouin, washed and dehydrated in an ascending ethanol series. After mounting ventral side up, larvae were coated first with silver and then gold and examined under scanning electron microscopy (Cambridge 'Stereoscan').

Attention was concentrated on the ventral bands of minute teeth and hooks ornamenting the integument. Larvae have paired rows of teeth above the mouth (head segment), and eleven bands of teeth, one on each segment, i.e. three on the thoracic segments and eight on the abdominal segments. The postoral bands are weakly developed dorsally and laterally and strongly developed ventrally. The oral and thoracic bands are scarcely visible under the highest power of the (light) stereo microscope, being colourless, whereas the abdominal bands are black and thus are more readily seen. A well-developed band, such as occurs in the middle region of the larva, consists of 6-8 irregularly developed rows of teeth.

Some typical results are shown in the photomicrographs. The number of oral rows was impossible to determine because in most specimens the head was not fully extended; better fixation should rectify this. Eight pairs of oral rows are seen in the photo of simulans. The thoracic bands consist of rows of flat triangular teeth, the free apex of each tooth making an angle of about 30°. Abdominal bands consist of rows of varying sizes of conical teeth with the ends directed cephalad (anterior rows) and caudad (posterior rows). Sex differences were not observed within a species. Some differences between melanogaster and simulans are: (1) the oral teeth are relatively more slender in melanogaster (see Figs. 1, 2, 6); (2) in the second thoracic band, the teeth are set closer together in simulans than in melanogaster (Figs. 3, 7). Teeth in this band were counted from the photographs, per units of 50 μ with the following results from three specimens: (Figures on following pages)

Numbers of teeth/50 μ, second thoracic band, ventral

	mean ± std. dev.	coëff. variation
(1) melanogaster ♀	10.30 ± 0.62	6.02
(2) melanogaster ♂	11.25 ± 1.25	11.11
(3) simulans (sex ?)	16.50 ± 2.07	12.54

Variance-ratio test between (1) & (2): $F = 4.05$, $P > 0.10$
" " " " (1) & (3): $F = 11.17$, $P > 0.05$

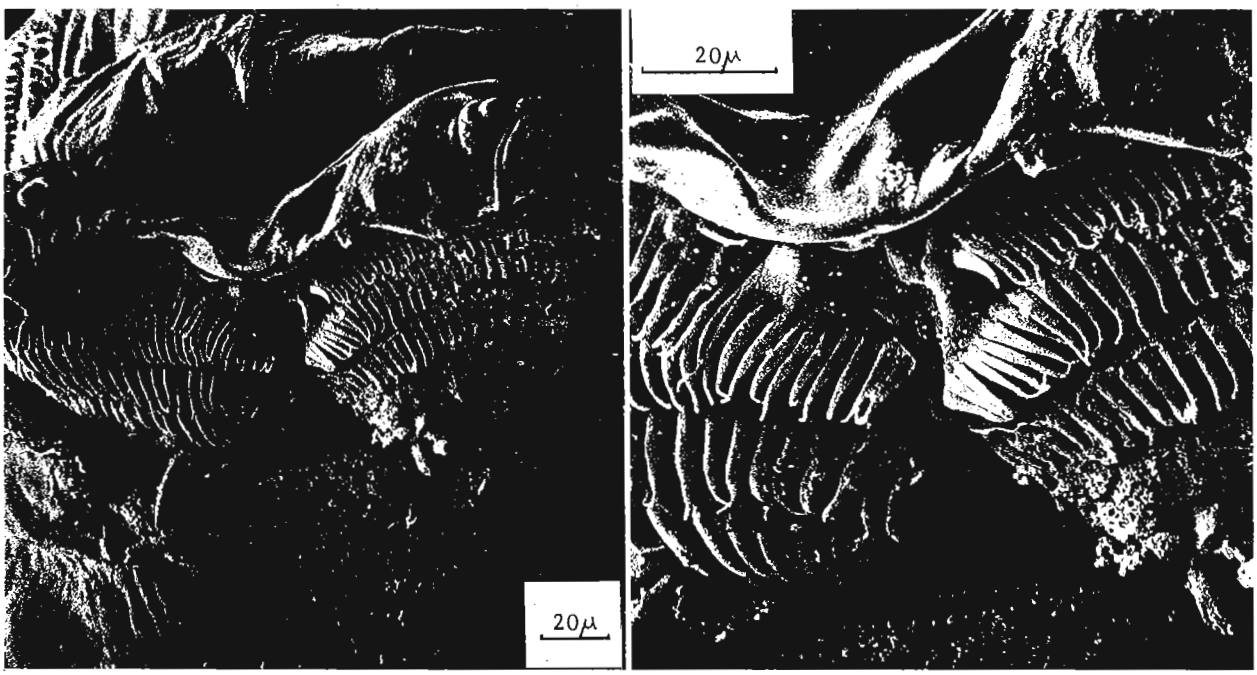
t test for difference between means:
between (1) & (2): $t = 1.16$, d.f. = 5, $P > 0.10$
" (1) & (3): $t = 2.99$, d.f. = 5, $P < 0.05$*
" (2) & (3): $t = 2.51$, d.f. = 6, $P < 0.05$*

* significant at 5 percent level.

The conclusion is that, in the specimens studied, there is no sex difference in mean tooth number, in melanogaster at least; but that simulans has a significantly higher mean number than melanogaster.

A fuller study is planned. The teeth must play a role in the movement of the larva through the food medium, and it may later be possible to correlate the differences noted above with the consistency of the food under natural conditions. The denser packing of teeth in simulans may indicate an adaptation to a firmer or drier substrate offering more resistance to the passage of the larva. Evidence supporting this: simulans becomes more abundant relative to melanogaster during hot dry summer months (1); and in the lab pupates mainly on the food surface (2). These observations appear to indicate an adaptation to a drier medium. Also, Hoenigsberg (3) has described how a change in the biotic environment (elimination of soft fruits) led to an immediate change in the melanogaster/simulans ratio favouring the latter species.

References: (1) Patterson, J.T. 1943, Univ. Texas Publ. 4313; (2) Sameoto, D.D. and R.S. Miller 1968, Ecology 49:407; (3) Hoenigsberg, H.F. 1968, Amer. Nat. 102:389.



Figures 1 and 2: D. melanogaster ♂. oral region.

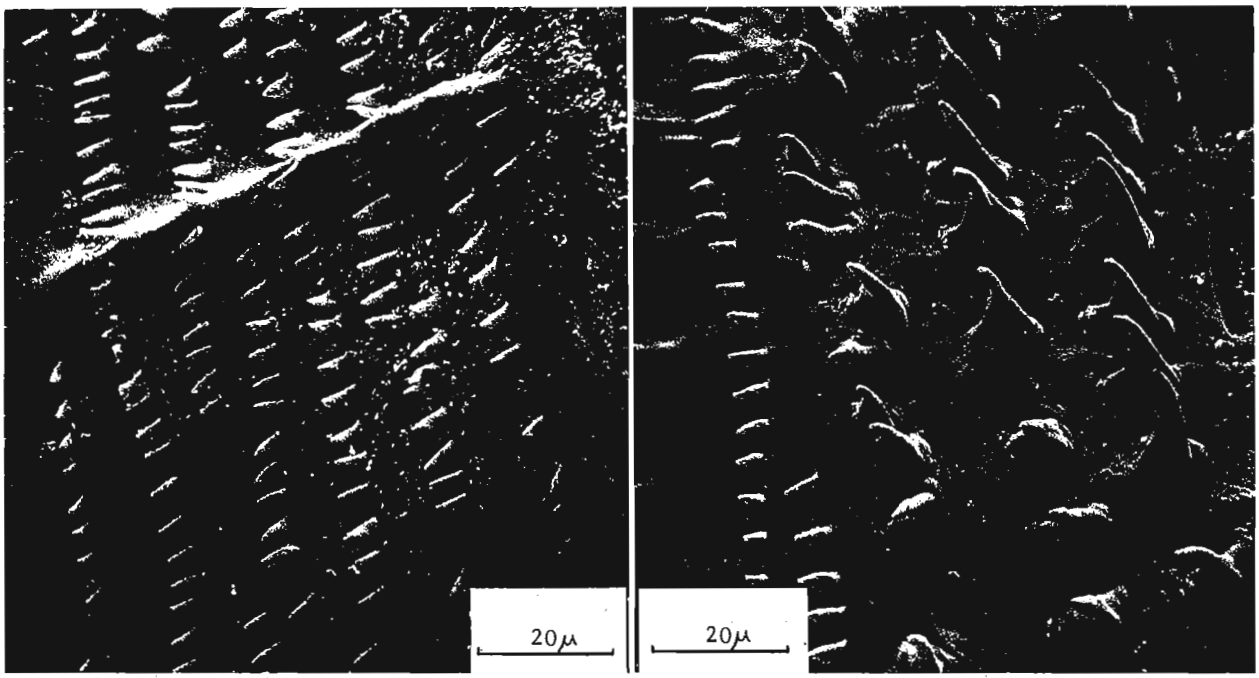


Figure 3: D. mel. ♂, second thoracic band

Figure 4: D. mel. ♂, first abdominal band

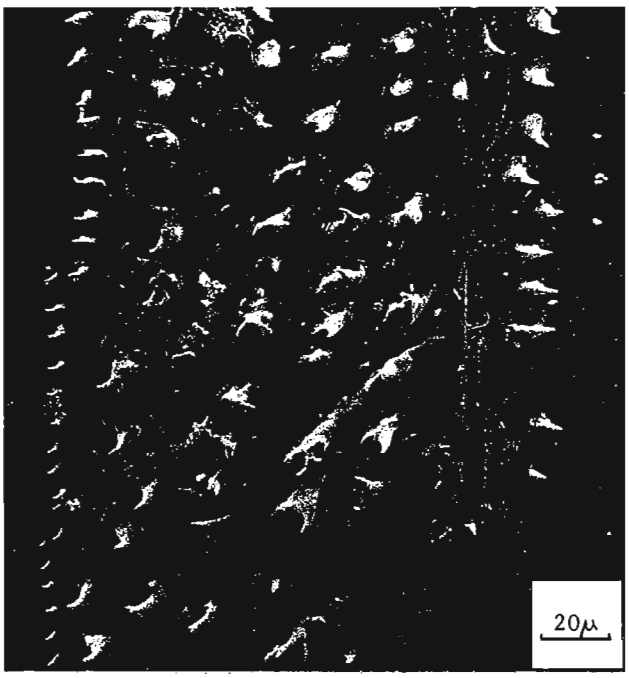


Figure 5: D. mel. ♂, third abdominal band

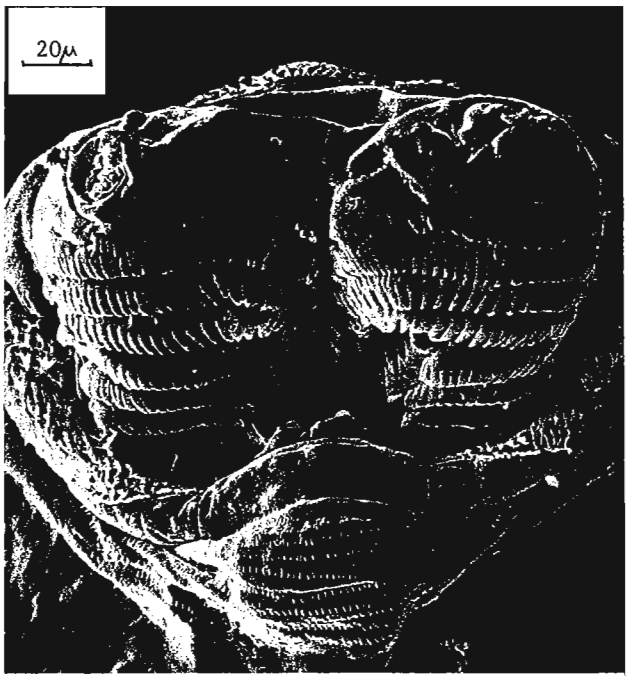
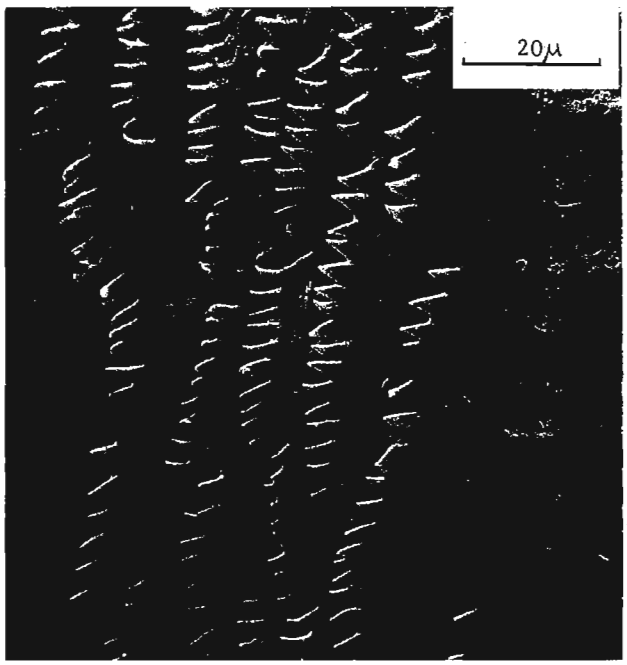
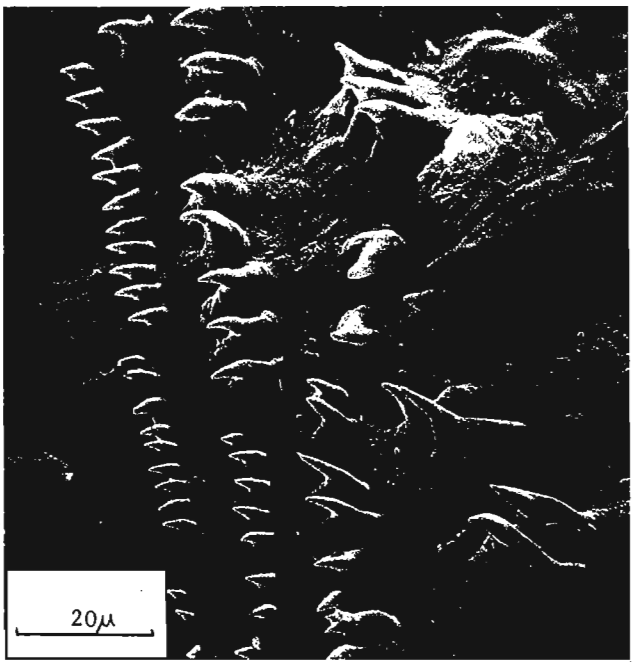


Figure 6: D. simulans (? sex), oral region





Figures 7 and 8: D. simulans (? sex), second thoracic band (7) and first abdominal band (8)

De la Rosa, M.E. and R. Félix. National Institute of Nuclear Energy, Mexico City, Mexico. Cytogenetic effects of cycloheximide during spermatogenesis of D. melanogaster.

Cycloheximide inhibits protein synthesis in vivo in yeast (Siegel and Sisler, 1964a) and fungi (Sussman, 1965), as well as in mammalian cells (Ennis and Lubin, 1964). This antibiotic prevents the transfer of amino acid from aminoacyl-tRNA to protein (Siegel and Sisler, 1964b; Bennett et al., 1965). Whereas cycloheximide inhibits protein synthesis without interfering with RNA synthesis, DNA synthesis is also inhibited (Kerridge, 1958; Young et al., 1964). In certain animal cells DNA synthesis appears to be as sensitive or more sensitive to cycloheximide than protein synthesis (Cooney and Bradly, 1961; Bennett et al., 1965).

Ring chromosomes are potentially useful for studying the process of rejoining after chromosome breakage, since a certain portion of the restitutions should produce dicentric ring chromosomes or interlocked rings rather than structurally normal chromosomes. The relevance of ring chromosome studies to problems of induced chromosome breakage in Drosophila melanogaster sperm has been recognized for some time. In this experiment the effect of a physiological saline solution, and of cycloheximide on chromosome X or Y loss was measured in the progeny of pre-treated and irradiated "Oster males" by means of the genetic scheme designed by Oster (1958). Male flies from a stock containing a marked sc^8Y chromosome and the closed X, X^{c2} marked with the mutants yellow (y) and Bar (B) in the males, and yellow, forked (f), attached X chromosomes in the female (y f:=) were aged for 72 hours before the injection of the solutions, irradiation, or both, and mated to virgin "Oster females" with markers in the I, II and III chromosomes (y sc^{S1} In49 sc^8;bw;st p^p). The markers B and y, to identify the treated sex chromosomes of the males, makes the detection of X or Y chromosome loss fairly easy. The frequency of exceptional (X/O) males among the F_1 flies is determined by counting the yellow males, which represent cases of loss of the whole or part of the X or Y chromosomes. Normal males have non-yellow bodies, since they carry the normal dominant allelomorph of yellow in the sc^8 insertion of their Y chromosome.

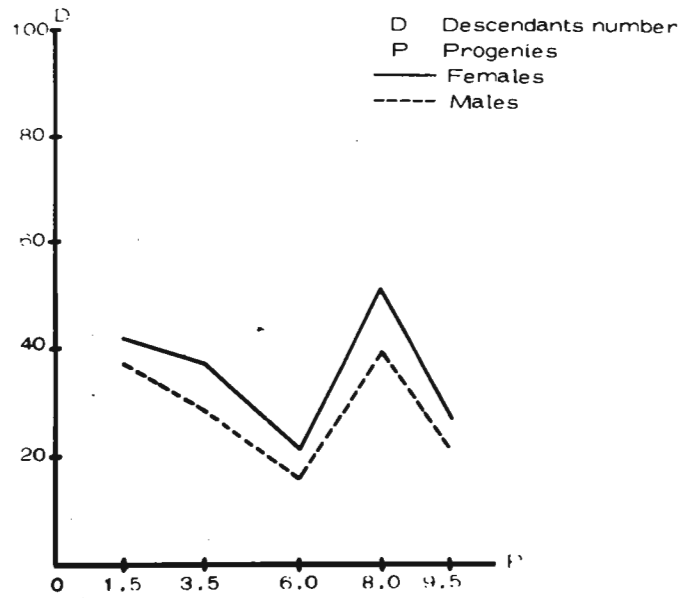


Figure 1. Progenies (average per culture/total) from the group treated with cycloheximide.

Several concentrations of cycloheximide (Schuchardt, München) dissolved in 0.7N NaCl solution were administered by injection in the gonadal area of aged "Oster males" in order to determine the concentration to be used without interfering with their viability or fertility. Since Carlson and Oster (1962) have shown that the amount of liquid expelled after injection varies from fly to fly, estimates of the amount of solution injected into each fly were not attempted. A concentration of 50 µg/ml was used; at this level no mortality was recorded among the injected adults within fifteen days. A physiological 0.7N NaCl solution was injected to male controls, instead of using distilled water which obviates the problem of induced sterility and possible cell selection by osmotic shock. The source of radiation was a "Gamma-Cell 200" apparatus.

All the flies used throughout the experiment were reared in the agar-cornmeal medium regularly employed at the laboratory. The cultures were kept at 25 ± 1°C before and after the treatments.

Three day old non-bred males were treated, placing them in separate vials (individual cultures) containing an aged male and five virgin females, which were renewed on the 2nd, 4th, 6th, 8th and 10th days; thereby five broods were obtained from gametes treated during specific stages of spermatogenesis. The fertilized females of each brood oviposited during 3 days in

the same vial, after which they were eliminated. The counting of the progenies to determine percentage of exceptional XO individuals was made 15 days after starting the cultures, before the following generation emerged.

According to Auerbach (1954) consecutive broods from males mated sequentially represent successively younger germ cells at the time of irradiation. Thus the spatial pattern of spermatogenesis in the treated testes becomes translated into a temporal pattern of genetical effects in successive broods which in turn "can be re-translated into the underlying sensitivity pattern of spermatogenesis".

In the mating procedure mentioned above the first cross among the treated or non-treated males and the five females in the same vial was designated the First Brood (0 - 1.5 days). These males were then mated in fresh cultures on the fourth day (1.5 - 3.5 days), or Second Brood. The following cultures correspond to the Third Brood, Fourth Brood and Fifth Brood. An approximate correspondence between the stages of spermatogenesis and the five cultures in which each male was successively bred is presented below.

1st. Brood - Days 0 to 1.5 post-treatment. Stage at time of treatment: mature sperm.

2nd. Brood - Days 1.5 to 3.5 post-treatment. Stage at time of treatment: spermiogenesis.

3rd. Brood - Days 3.5 to 6 post-treatment. Stage at time of treatment: second meiotic division and spermiogenesis.

4th. Brood - Days 6 to 8 post-treatment. Stage at time of treatment: first meiotic division and probably a few early second meiotic stages and late gonial cells.

5th. Brood - Days 8 to 9.5 post-treatment. Stage at time of treatment: spermatogonial cells and early first meiotic cells.

The following groups were conducted in parallel:

Group I. Injection of 0.7N NaCl solution.
Group II. Injection of 50 µg/ml cycloheximide solution.
Group III. Irradiation with 2,500 rads.
Group IV. Injection of 0.7N NaCl solution and irradiation with 2,500 rads.
Group V. Injection with 50 µg/ml cycloheximide solution and irradiation with 2,500 rads.

Effects of cycloheximide upon fertility. The administration of physiological serum (Table 1) to the males constitutes the test group which shows the fertility characteristic of each stage present during spermatogenesis, after injection of saline solution.

Table 1. Progenies (average per culture/total) from the group treated with 0.7N NaCl solution.

Progenies	0-1.5 days	1.5-3.5 days	3.5-6 days	6-8 days	8-9.5 days	Total
XX females	28/700	77/856	16/96	44/176	54/162	219/1990
XY males	26/639	64/708	17/104	39/155	53/160	199/1766
XO males	2	1	0	0	0	3

Table 2 sumarizes the data concerning the number of individuals obtained apart from specific stages of spermatogenesis, when physiological serum was administered before the irradiation with 2,500 r.

The differences between Tables 1a and 2 result from the modifications in the specific survival of successive stages of spermatogenesis, owing to its intrinsic radiosensibility.

Table 2. Progenies (average per culture/total) from the group treated with 0.7N NaCl solution and 2,500 r.

Progenies	0-1.5 days	1.5-3.5 days	3.5-6 days	6-8 days	8-9.5 days	Total
XX females	28/702	23/522	8/198	4/50	6/64	69/1536
XY males	24/592	16/505	7/167	5/60	4/44	56/1340
XO males	9	7	1	3	0	20

It is well established that the most radiosensitive stages of spermatogenesis are the

spermatid, and the spermatocites, which correspond in the tables to the progenies recovered between 3.5 - 6 days and between 6 - 8 days after treatment, respectively.

Table 3. Progenies (average per culture/total) from the group treated with 50 µg/ml cycloheximide.

Progenies	0-1.5 days	1.5-3.5 days	3.5-6 days	6-8 days	8-9.5 days	Total
XX females	42/1043	37/1343	21/451	51/672	27/288	178/3797
XY males	37/925	28/1039	16/345	39/507	21/230	141/3046
XO males	2	1	2	1	3	9

The data contained in Table 3 and Fig. 1 indicate that fertility diminishes when the administration of cycloheximide takes place during the stages of spermatid, and spermatogonia.

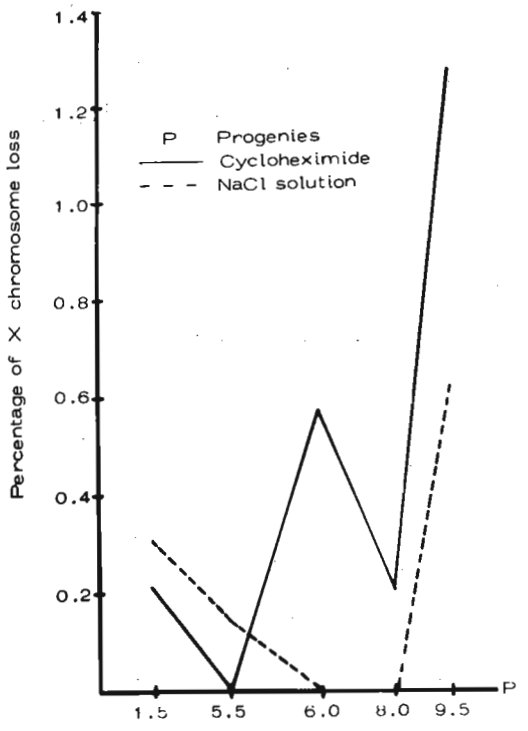


Figure 2. Percentage of X chromosome loss during spermatogenesis.

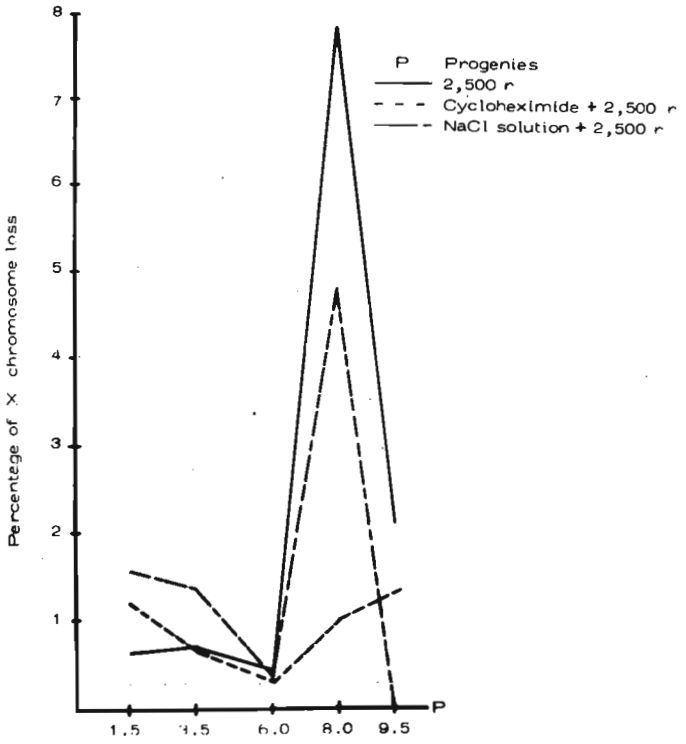


Figure 3. Percentage of X chromosome loss during spermatogenesis.

Radiation produced a diminution in fertility during all stages of gametogenesis preceding spermatozoa (Table 4).

Table 4. Progenies (average per culture/total) from the group treated with 2,500 r.

Progenies	0-1.5 days	1.5-3.5 days	3.5-6 days	6-8 days	8-9.5 days	Total
XX females	29/1354	23/1044	8/312	5/126	9/225	74/3061
XY males	26/1200	19/859	6/235	2/91	6/139	59/2524
XO males	8	6	1	6	3	24

Table 5 presents the progenies obtained from spermatozoa treated during such stage, and previous stages of spermiogenesis and spermatogenesis, with cycloheximide and gamma radiation.

A radioprotective effect of cycloheximide is noticeable from 3.5 to 9.5 days progenies, which includes stages from spermatogonia to spermiogenesis. Spermatids and late spermatocytes are the most affected cells in which a three-fold increase in the size of the progenies is obtained.

Table 5. Progenies (average per culture/total) from the group treated with 50 µg/ml cycloheximide and 2,500 r.

Progenies	0-1.5 days	1.5-3.5 days	3.5-6 days	6-8 days	8-9.5 days	Total
XX females	28/1031	29/1266	29/1020	11/375	12/343	109/4035
XY males	22/797	20/876	21/732	9/299	11/301	83/3005
XO males	10	6	3	3	4	26

The effects of cycloheximide upon X or Y chromosome loss. The data on exceptional individuals resulting from chromosome X or Y loss at successive stages of spermatogenesis following different treatments are contained in Table 6 and Fig. 2 and 3. It is observed that cycloheximide diminishes the frequency of the chromosome X or Y loss during non-irradiated states of spermatozoa and spermatid, increasing such losses during the stages of spermatocyte and spermatogonia.

Table 6. Percentage of X or Y chromosome loss during spermatogenesis (Definition 1, Traut, H., 1964).

Treatment	Spermatozoa	Spermatid	Spermatocyte		Spermatogonia
NaCl sol.	0.31 ± 0.55	0.14 ± 0.37	0.0 ± 0.0	0.0 ± 0.0	0.62 ± 0.78
NaCl sol.+2,500 r	1.56 ± 1.24	1.36 ± 1.16	0.59 ± 0.76	4.76 ± 2.18	0.0 ± 0.0
2,500 r	0.66 ± 0.81	0.69 ± 0.83	0.42 ± 0.64	7.79 ± 2.79	2.11 ± 1.45
cyloheximide sol.	0.21 ± 0.45	0.09 ± 0.30	0.57 ± 0.75	0.21 ± 0.45	1.28 ± 1.13
cycloheximide sol+2,500 r	1.23 ± 1.10	0.68 ± 0.82	0.37 ± 0.60	1.0 ± 1.0	1.34 ± 1.15

In the irradiated groups, there is a radioprotective effect of cycloheximide during spermatogonial stages and during meiosis, while an enhancement of chromosome X or Y loss is shown in spermatozoa. Such results are not compatible with the data obtained from experiments in which other protein-synthesis inhibitors, such as actinomycin D have been assayed (Félix and Rodriguez, 1968; Proust et al., 1972).

References: Auerbach, C. 1954 Z. Induk. Abst.- Vererb. 86:113-125; Bennett, L.L., V.L. Ward and R.W. Brockman 1965 Biochim. et biophys. acta 103:478; Carlson, E.A. and I.I. Oster 1962 Genetics 47:561-576; Cooney, W.J.B. 1961 In: Antimicrobial Agents and Chemotherapy, p. 237, Ed. by Finland, M. and G. Savage, Detroit 1962 American Society for Microbiology; Félix, R. and R. Rodríguez 1968 An. Inst. Biol. Univ. Autón. México 39, Ser. Biol. Exp. (1):13-21; Kerridge, D. 1958 J. Gen. Microbiol. 19:497; Oster, I.I. 1958 Rad. Biol. Proc. Sec. Austr. Conf. Rad. Biol. :253-267; Proust, J.P., K. Sankaranarayanan and F.H. Sobels 1972 Mutation Res. 16:65-76; Siegel, M.R. and H.D. Sisler 1964a Biochim. et biophys. acta 87:70; Siegel, M.R. and H.D. Sisler 1964b Biochim. et biophys. acta 87:83; Sussman, M. 1965 Biochem. and Biophys. Res. Commun. 18:763; Young, C.W., S. Hodas and J.J. Fennelly 1964 Proc. Am. A. Cancer Res. 5:279.

Gingeras, T.R., H. Gelti-Douka and M.P. Kambysellis. New York University, New York. Yolk proteins in Drosophila.

Yolk proteins (vitellogenic proteins) have been extensively studied in many insects with emphasis on their identification and isolation (Telfer, 1953), hormonal regulation (Pan, M.L. and G.R. Wyatt, 1971) and site of synthesis (Hagedorn, H.H. and L.C. Judson, 1972).

We have initiated a project to study the genetic mechanisms regulating the synthesis and uptake of yolk proteins in Drosophila. We have been able to demonstrate by means of polyacrylamide gel electrophoresis, the presence of numerous proteins in the crude yolk extract of mature oocytes from several Drosophila species (Table 1). One of these proteins is predominant and represents 60-80% of the total egg proteins. We have found that the predominant yolk protein is also present in large quantities in the hemolymph of mature females, and is totally absent or present in very small quantities in males and newly emerged females (Figure 1). Partial purification of Drosophila yolk protein can be achieved by ammonium sulfate fractionation. For precipitation, the protein of D. virilis and D. hydei required 60% saturation, while 55% was necessary for D. mulleri. The molecular weight of the yolk protein (determined on 10% SDS polyacrylamide gels) was found to be in the range of 190,000 and appears to be identical for all the species analyzed. However, the electrophoretic mobility on 7.5% urea gels or 7.5% non-urea gels, pH 8.3, shows significant variability among the species, suggesting differences in the charge of the molecule. Furthermore, the yolk proteins from only some of the species gave immunoprecipitin lines (Table 1) when tested on Ouchterlony double diffusion immunoplates against antibodies prepared from D. virilis crude yolk extracts. The reaction appeared to vary among species, both qualitatively and quantitatively, and to parallel the presumed phylogenetic kinship of the species. Comparable results were obtained when yolk proteins were tested against antibodies prepared from D. melanogaster and D. silvestris crude yolk extracts. These results suggest that although the yolk proteins in Drosophila (at least in the species analyzed) are constant in MW, they have significant structural substitutions in different species. Such interspecific differences parallel the phylogenetic distance. (Supported by NSF Research Grant GB-34168.)

Table 1. Immunochemical reactions of Drosophila antibodies against crude yolk extracts from various Drosophila species.

Antibody yolk extract	virilis	melanogaster	silvestris
D. virilis	+	-	-
D. cardini	+	-	-
D. robusta	+	-	-
D. hydei	+	+	-
D. wheeleri	+	-	-
D. altrichi	+	-	-
D. melanogaster	-	+	-
D. subobscura	-	-	-
D. silvestris	-?	-	+
D. mimica	-	-	+
D. hamifera	-	-	+
A. aduncus	-	-	+

Figure 1. SDS polyacrylamide electrophoresis of D. silvestris.
a) crude yolk extract
b) female hemolymph (30 day old)
c) male hemolymph (30 day old)
d) female hemolymph (one day old)
e) male hemolymph (one day old)
(Arrow points at the yolk protein.)

References: Telfer, W.H. 1953, J. Gen. Physiol. 37:539; Pan, M.L. and G.R. Wyatt 1971, Science 174:503; Hagedorn, H.H. and C.L. Judson 1972, J. Exp. Zool. 182:367.

Thompson, J.N., Jr. University of Cambridge, England. A new suppressor of veinlet and comments on two other vein mutants.

In a veinlet selection line, a mutation occurred which tended to suppress the expression of ve by making the L2, L3, and usually the L4 veins complete. The mutation is located at about 3-28.5, and is named gravel (gra) after a pleiotropic roughened eye effect resulting from irregularly arranged and enlarged facets in the compound eyes. When the gra mutation was removed from the homozygous ve selection line, it was found that the wings (Figure 1) often have fragments of extra venation in the marginal, discal, and second and third posterior cells. This is interesting in view of the fact that plexus, characterised by an extensive network of extra veins, also suppresses ve, and the fact that Waddington (1940) reported that the

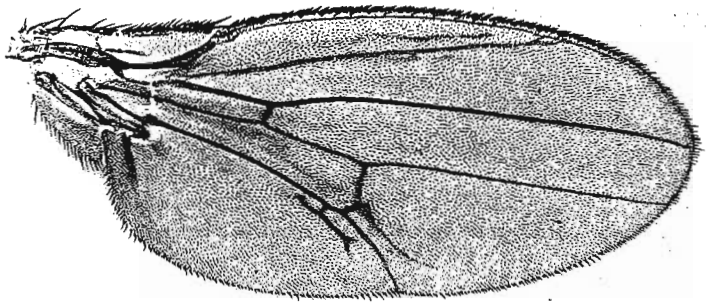


Figure 1

formation of both vein gaps and vein fragments appear, histologically, to be closely related phenomena. The suppression may be due simply to mutual compensation of mutant effects upon a single developmental process.

In another line, the expression of short vein (shv, 2-3.8) was enhanced by selecting for shorter veins. After several generations, many flies had terminal gaps in the L2, L3, L4, and L5 veins (Figure 2). Lindsley and

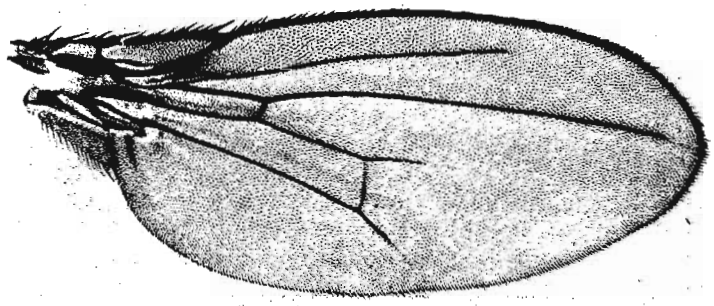


Figure 2

Grell (1967) described shv as producing gaps in only the L2 and L4 veins, but it now appears that shv is similar in effect to ve, though less extreme.

Carlson (1966, 1970) reported experiments using a new mutant with gaps in the L2 vein. Chromosome locations had been made using dominant markers and indicated that phenotypic effects were associated with both the second and third chromosomes. He has kindly provided me with cultures of this mutant, and I have repeated the chromosome location experiments using recessive chromosome markers. Although there is a modifier of rather large effect on the third chromosome in one of his selection lines, the primary mutant is located on chromosome II at about 0.0cM. An associated character, the post-scutellar bristles being erect, suggests that Carlson's mutant may be an allele of the lost telegraph (tg) mutant described by Bridges, and it is proposed to call the new mutant telegraph of Carlson (tg^C).

I am very grateful to Mr. Brian Curtis for the photography.

References: Carlson, J.H. 1966, Ohio J. Sci. 66:340-346; Carlson, J.H. 1970, Ohio J. Sci. 70:365-371; Lindsley, D.L. and E.H. Grell 1967, Carn. Inst. Pub. 627; Waddington, C. H. 1940, J. Genet. 41:75-139.

Mather, W.B. and P. Thongmeearkom.
University of Queensland, Brisbane, Australia. The nasuta complex in Luzon.

In January 1972, sixty one iso-lines were established from a collection from Luzon, Philippines. Twenty seven heterozygous inversions were detected in variable frequency (see Table). Previous studies in Cebu, Philippines and Taiwan (Mather and Thongmeearkom 1972a, 1972b) had detected 20 inversions and of these 10 are present in Luzon but 17 are new.

Morphological study of a sample of 32 iso-lines allowed the separation of four taxons. Three of these had already been detected previously, Taxon B with white orbits in Cebu and

Inversion	Type	Position	Heterozygosity Frequency
B	Sim	III P	1.6
C	Sim	III D	39.3
D	Sim	III P	1.6
E	Sim	II L C	68.8
G	Sim	I P	11.4
H	Com	III P	1.6
I	Sim	III P	4.9
K	Sim	III D	1.6
L	Sim	III P	1.6
P	Sim	III P	3.3
T	Sim	I C	1.6
U	Com	III P	9.8
V	Com	III C	9.8
W	Com	III D	9.8
X	Com	III C	1.6
Y	Sim	III P	1.6
Z	Com	III C	1.6
$\overline{AA}$	Sim	III P	1.6
$\overline{BB}$	Com	I D	1.6
$\overline{CC}$	Sim	I P	1.6
$\overline{DD}$	Com	III C	1.6
$\overline{EE}$	Com	III P	1.6
$\overline{FF}$	Sim	II R P	6.6
$\overline{GG}$	Sim	II R D	4.9
$\overline{HH}$	Sim	II R P	1.6
$\overline{II}$	Sim	II L C	1.6
$\overline{JJ}$	Com	II L C	1.6

Sim = Simple, Com = Complex, D = Distal, C = Central and P = proximal to centromere.

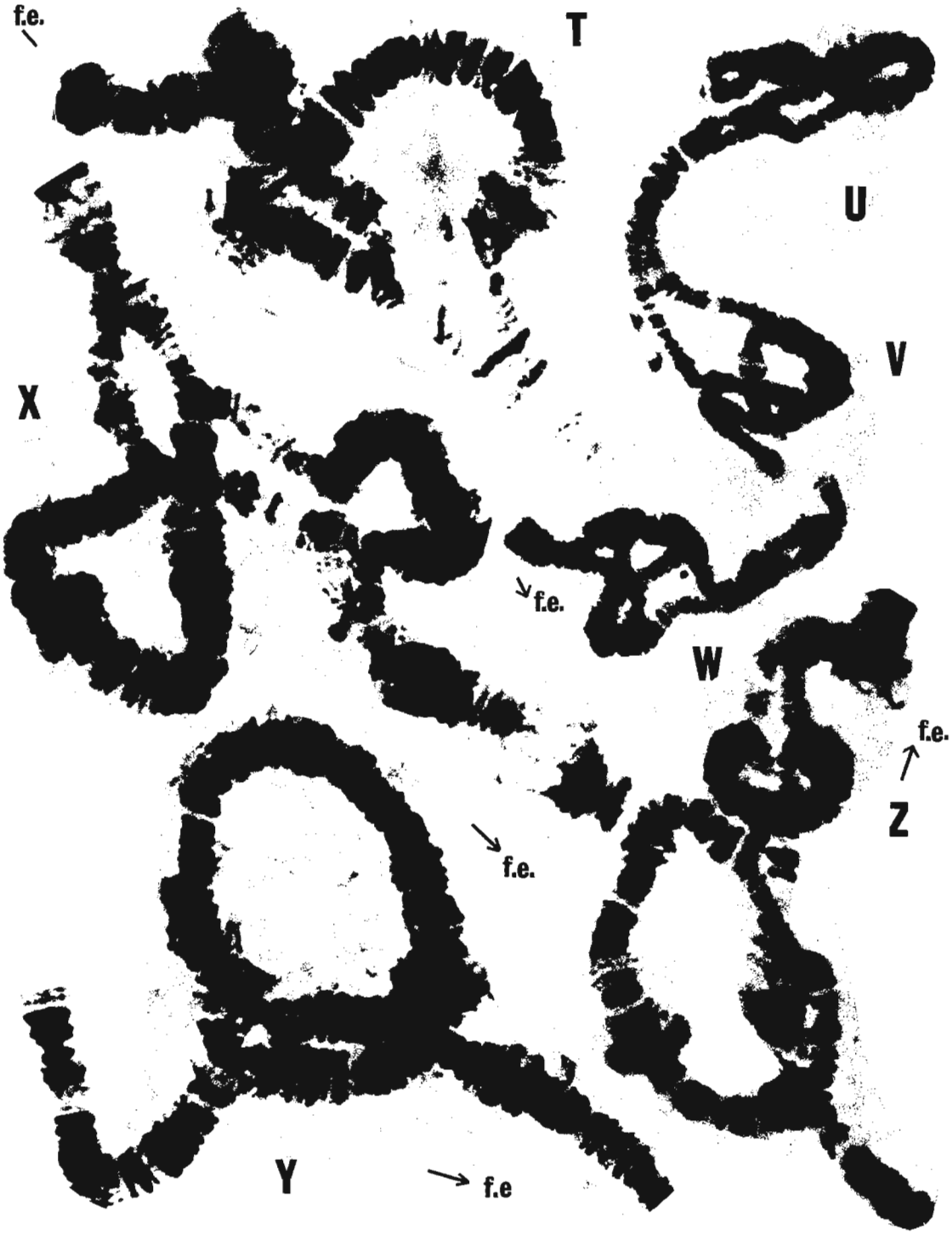
Taiwan, Taxon C with no white on the face in Taiwan and Taxon D with a white face in Cebu. In addition a new taxon - E with no white on the face but consisting of much smaller flies than Taxon C was distinguished.

The distribution of the inversions between the taxons are: Taxon B: C, E, G, $\overline{AA}$, $\overline{BB}$, $\overline{CC}$, $\overline{DD}$, $\overline{HH}$, $\overline{II}$, $\overline{JJ}$; Taxon C: none; Taxon D: B, D, E, H, I, K, L, P, T, X, Y, Z, $\overline{EE}$; Taxon E: U, V, W, $\overline{FF}$, $\overline{GG}$. Thus only inversion E is common between Taxon B and D and Taxon E appears to have unique inversions. The six lines examined of Taxon E all had the inversion U, V, W, associated (see Figure).

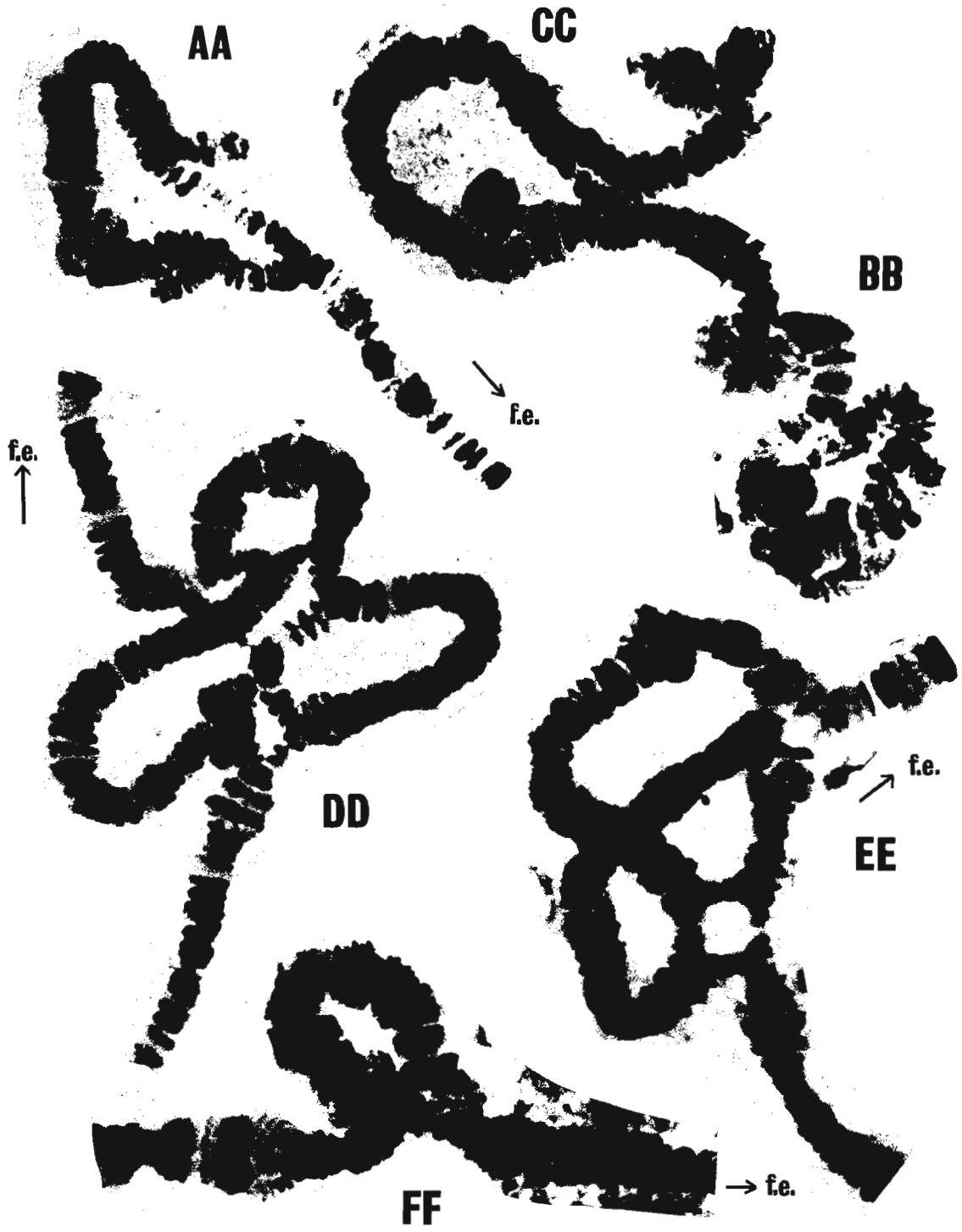
The hybridisation relationships between the postulated taxons which will throw light on their taxonomic status will be reported on in a later communication.

References: Mather, W.B. and P. Thongmeearkom 1972a, DIS 48:40; Mather, W.B. and P. Thongmeearkom 1972b, DIS 49:109

The material was collected and maintained and the larvae reared for dissection by W.B.M. The chromosomes were prepared, analysed and photographed by P.T.



Nasuta complex inversions. f.e., free end.



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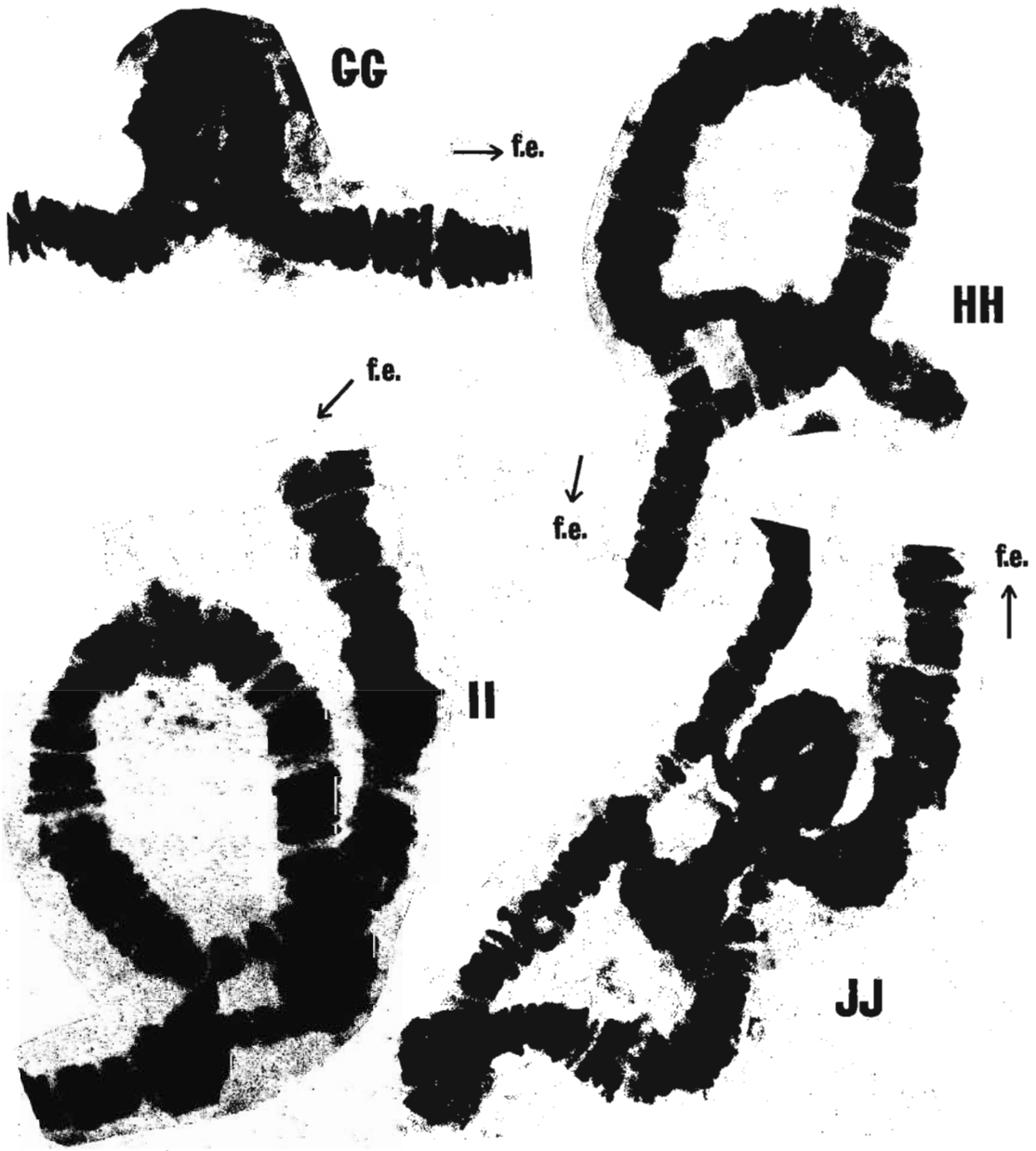
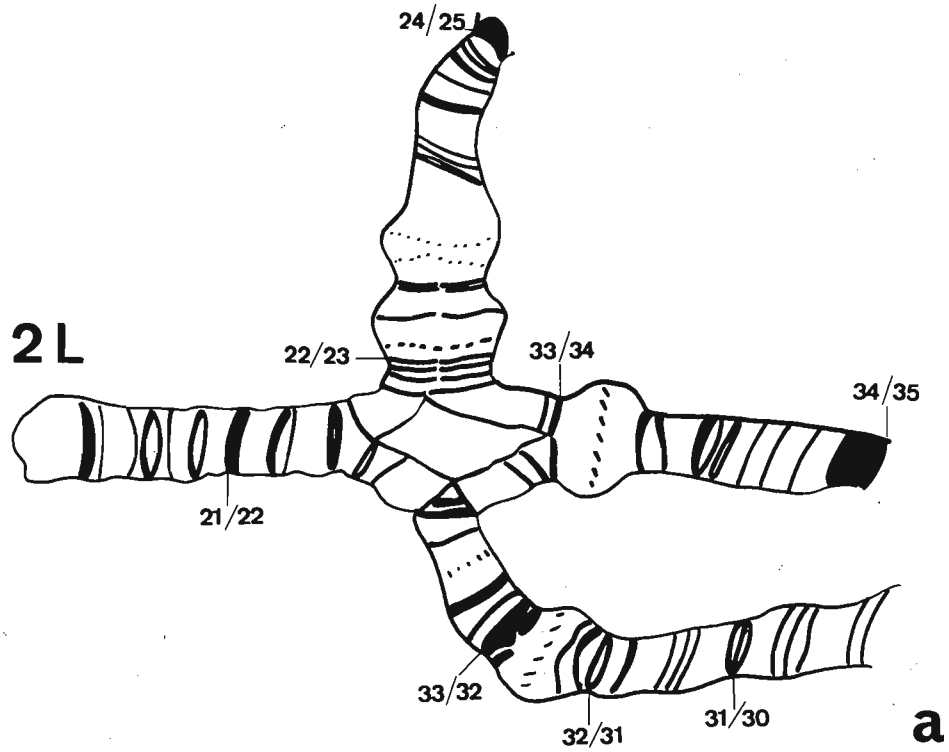


Nasuta complex inversions. f.e., free end.

Pfeifer, S. and M. Sorsa. University of Helsinki, Finland. A report on inversion polymorphism in a local population of Drosophila melanogaster (Porvoo wild).

The existence of chromosomal inversion polymorphism in natural populations of Drosophila melanogaster has recently been reported to be rather high even in this species (e.g. Watanabe 1967; Mukai and Yamaguchi, 1972). The existence of an inversion and the analysis of its frequency in the wild strain "Porvoo" of Drosophila melanogaster is reported here. The strain



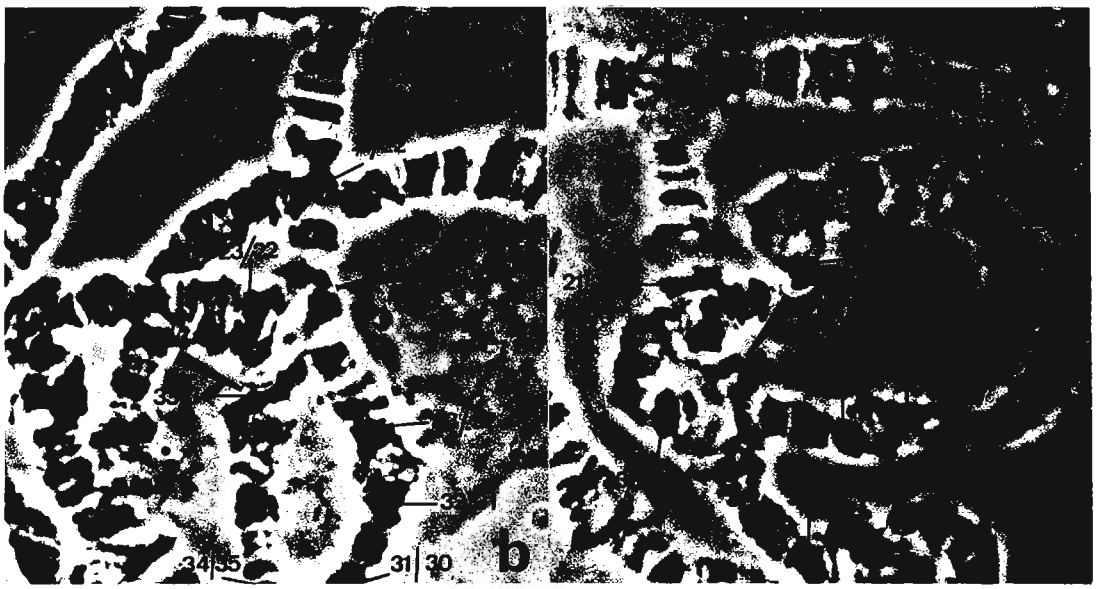


Fig. 1. Inversion heterozygotes in D. melanogaster strain Porvoo wild. a) interpretation of inversion 2L extending from 22D1-2 to 33F5-34A1. b) and c) photographs of inversion heterozygotes. Magnification x 2500.

was captured nearly 40 years ago in the southern coastal part of Finland (60° 20'; 25° 30') in Porvoo, Åminsby, and since then has been cultured in normal laboratory conditions. Inbreeding has been efficient due to small population size and several "bottlenecks" in the cultured history of the strain.

The inversion quite evidently corresponds to In(2L)Cy = In(2L)22D 1-2;33F 5 - 34A 1 (Lindsley and Grell 1968, cf. Fig. 1). In the strain Porvoo wild, the inversion exists without Curly phenotype and the homozygotes are thus viable. However, the frequency of homozygous individuals was significantly smaller than theoretically expected. For the determination of the frequency of the inversion genotypes, the chromosomes of 130 individuals were analyzed from squash preparations of salivary glands (cf. Sorsa and Pfeifer 1972). The frequencies of +/+ individuals, In(2L)/+ heterozygotes and In(2L)/In(2L) homozygotes were 40.0%, 58.5% and 1.5% respectively, thus equalling chromosome frequencies p=0.69, q=0.31. Inversion heterozygotes thus possess a positive selective value. At this point it is not known whether the disproportionate frequencies would be due to meiotic drive (Sandler and Novitski, 1957) type of mechanism resulting in an increased proportion of heterozygotes. The permanence of the inversion types in the stock has now been followed for over 10 generations.

References: Lindsley, D.L. and E.H. Grell 1968, Carn. Inst. Wash. Publ. 627; Mukai, T. and O. Yamaguchi 1972, DIS 48:43; Sandler, L. and E. Novitski 1957, Amer. Nat. 91:105-110; Sorsa, M. and S. Pfeifer 1972, Hereditas 71:119-130; Watanabe, T.K. 1967, Japan. J. Genet. 42:375-386.

Subbarao, S.K., P. Szabo and O. Uhlenbeck
University of Illinois, Urbana. Visualization of poly rA:dT hybrids in the mitochondrial genome of D. melanogaster.

There is good evidence that long AT rich stretches exist in mitochondrial (Mt) DNA of Drosophila melanogaster. Polan et al. (1973) report that the thermal denaturation profile of purified MtDNA has three discernable transitions. The first transition has a T_m of 71°C in SSC, implying that a portion (about 20%) of MtDNA contains few if any GC base pairs. Denaturation mapping of MtDNA by electron microscopy shows that the early melting portion of MtDNA is contiguous (Peacock et al. 1973). We have found that this segment and therefore MtDNA can be visualized by in situ RNA-DNA hybridization with ^3H polyriboadenylate, implying that at least a portion of MtDNA is dA:dT. Testicular tissue was chosen for preliminary experiments because mitochondria (Nebenkern) of sperm and spermatids are localized in well defined positions, along the entire length of the tail.

The standard in situ procedures of Steffensen and Wimber (1971) were used with the following modifications: 1) the slides were denatured with 0.2 N HCl for 20 minutes at 25°C; 2) the hybridization conditions were 17°C in 50% formamide: 2xSSC for 15 hours, which should optimize the rate and specificity for rA:dT hybridization; 3) T_2 ribonulease was used as the post treatment to remove excess poly A. ^3H polyribonuleotides were synthesized from their corresponding ^3H nucleotide diphosphates with E. coli polynucleotide phosphorylase.

Figure 1a shows heavy poly A labelling over tails of late spermatids whereas the heads are poorly labelled. To verify that the DNA in the heads is accessible for hybridization, a mixture of random polynucleotides, ^3H poly (AUGC) was used on the same tissue. In this case (Figure 1c) very heavy labelling of the heads and only light labelling of the tails was observed in accordance with the DNA concentrations in the two regions. In an additional control experiment, ^3H poly U was, as expected, also shown to hybridize to the spermatid tails (not shown).

The density of grains observed after poly A hybridization varies with the stage of the developing sperm. Figure 1b shows that while early spermatid tails label heavily, the narrower mature sperm tails do not label at all. The inability of mature sperm tails to label with poly A may either be due to the elimination of MtDNA or to interference with the hybridization reaction by proteins deposited in the Nebenkern of mature sperm. Currently we are using in situ hybridization with poly A to follow MtDNA during all stages of spermatogenesis.

References: Polan, M.L., S. Friedman, J.G. Gall and W. Gehring 1973, J. Cell. Biol. 56: 580; Peacock, W.J., D. Brutlag, E. Goldring, R. Appels, C.W. Hinton and D.L. Lindsley 1973, C.S.H.S.Q.B. 38:in press; Steffensen, D.M. and D.E. Wimber 1971, Genetics 69:163.

(See Figure next page)

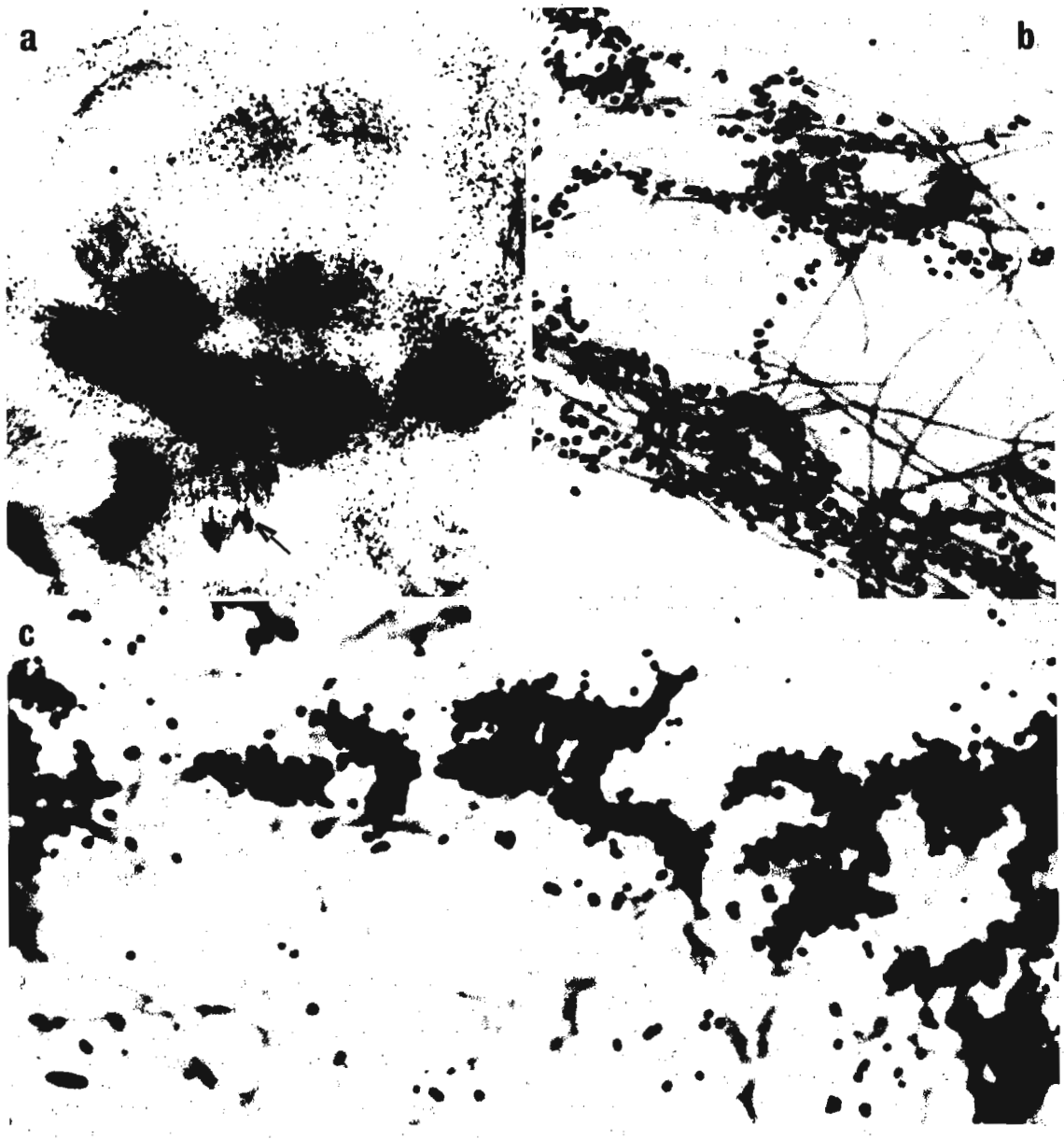


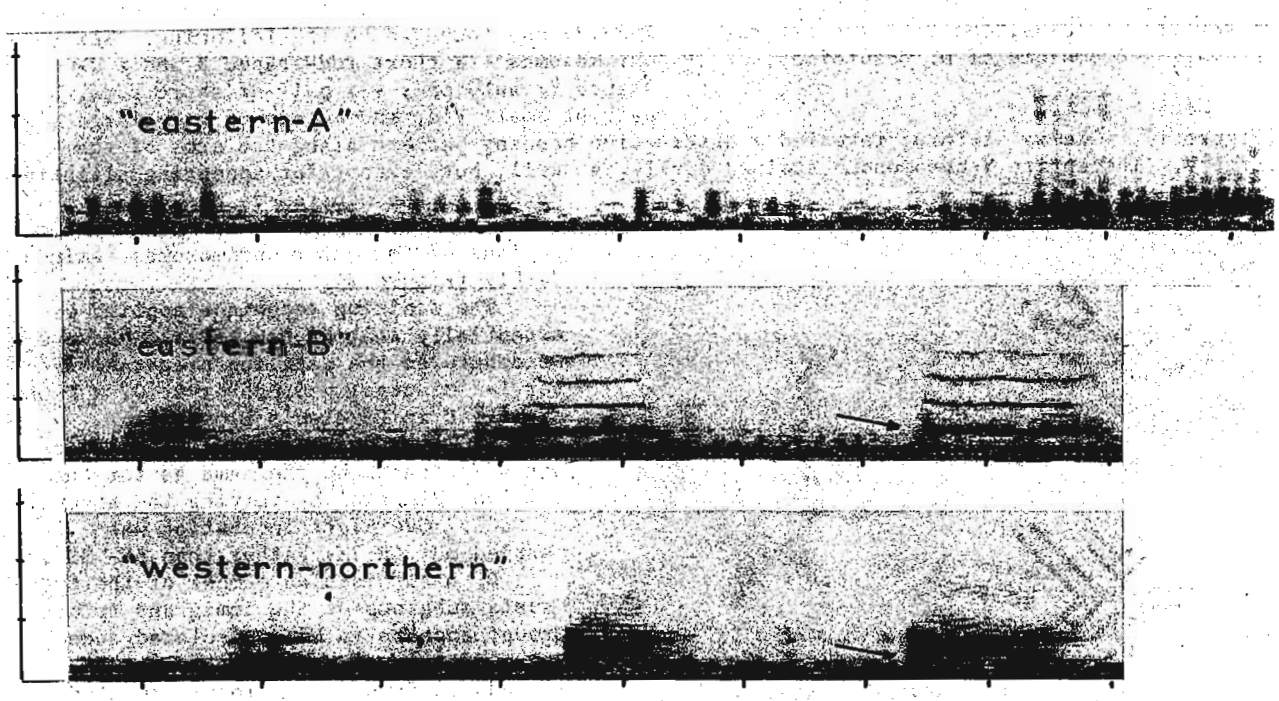
Figure 1

a) A late spermatid bundle, hybridized with poly A (s.a. 18 Ci/mmole) and exposed for twelve days, shows label over the tails. The arrow marks the position of the spermatid heads. b) A similar preparation at a higher magnification, the tails of early spermatids are heavily labeled with poly A. The label is lost during maturation; the mature sperm tails in the figure show no label above background. c) Spermatid heads from a slide hybridized with poly AUGC (s.a. 1 Ci/mmole) show dense label over the heads and a few grains over tails. The conditions of this hybridization were incubation in 50% formamide: 2xSSC at 40°C for fifteen hours.

Patty, R.A.[1], R.B. Goldstein[2] and D.D. Miller[2]. (1) Wake Forest University, Winston-Salem, North Carolina, and (2) University of Nebraska, Lincoln. Sonagrams prepared from D. athabasca male courtship sounds.

The Figure shows sonagrams made from recordings of amplified sounds of courting D. athabasca males. As previously stated (e.g. Miller and Voelker, Journ. Hered. 63:2, 1972), there appear to be two kinds of "eastern" athabasca, now designated "eastern A" and eastern B". The sonagrams so designated are those of males of two strains from Netcong, New Jersey (kindly provided by Dr. Max Levitan). The "western-northern" sonagram was derived from a Hallock, Minnesota, strain. Patterns similar to each of these have been found in sonagrams from a total of 17 "eastern A", 10 "eastern B", and 13 "western-northern" strains from widely scattered geographical sources in the ranges of these kinds of athabasca and will be presented and discussed elsewhere. The divisions of the vertical coordinates in the figure represent 1000 Hz (Hertz, or cycles per second) each, those in the horizontal coordinates 200 msec (milliseconds) each, 6-8 day old adults that had been isolated by sex soon after eclosion were put together (usually 1 female, 2 or more males) in a cylindrical clear plastic chamber (ca. 17 mm in diameter with a glass "ceiling" ca. 4 mm high) erected on the exposed Mylar membrane of a Wollensak dynamic microphone, which was fastened in an upright position under a dissecting microscope so the flies' activity could be watched. When courtship took place, the sound vibrations were fed into a Webcor tape recorder set at maximum volume and recording at 7.5 rpm. Sonagrams were prepared by one of us (R.B.G.) with a Kay Electric Company 6061-B sound spectrograph.

Our work began as an attempt to determine significant differences of spacing of vibration pulses between the different partially sexually isolated kinds of D. athabasca. The earliest



observations (by R.A.P.) employed a crystal microphone with a specially constructed band-pass filter to reduce noise by restricting transmitted frequencies mainly to the 200 to 800 Hz range (since a male courtship wing vibration frequency of about 444 Hz had been reported for D. athabasca by Ewing and Bennet-Clark, Behaviour XXXI:288, 1968). It became apparent from these observations that the intra-pulse frequencies of "eastern A" (e.g. Carbon County, Pennsylvania; Miller and Westphal, Evolution 21:479, 1967) were much lower than those of "eastern

B" and "western-northern" athabasca, the fundamental frequency of "eastern A" being about 204 Hz while those of "eastern B" and "western-northern" averaged about 462 and 407 Hz respectively. To improve reception, especially at the low frequencies, the simpler system described in the first paragraph was adopted.

The difference between the courtship sounds of "eastern A" and the other two is clearly audible in the amplified recordings. Males of "eastern A" produce a low-pitched "grunting, croaking" sound that does not appear to be limited to discrete pulses but which, as the sonagram shows, consists of elements (i.e. short bursts of sound consisting of fundamental and harmonics) spaced about 25 msec and produced in rather irregular "runs". Because of unavoidable background noise that accompanied all recordings it was often difficult to determine the fundamental frequency of "eastern A" by measuring the elevation of the lowest marking in the sonagram; however, this value appears regularly to be in the neighborhood of 200 Hz. Both "eastern B" and "western-northern" athabasca males produce discrete pulses of sound with a "buzzing, whining" quality. The level of the fundamental frequency, indicated by arrows in the figure, seems similar in these two kinds, usually in the 450-500 range, and their values overlap. Although the spacing of pulses appears different in the examples in the figure (about 450 msec for "eastern B", 400 msec for "western-northern"), other sonagrams show overlapping of these values too. As shown in the figure, these pulses may be produced in groups of three, the last a little longer than the others. Although these two songs are not easily distinguishable by ear (though some persons might be able to do so), the sonagrams clearly show different patterns of harmonics, fairly widely spaced ones (about 500 Hz intervals) for "eastern B" and closely spaced ones (about 125 Hz intervals) for "western-northern".

Wheeler, L.L., A.S. Capps and F.D. Wilson
University of Texas, Austin. The heterochromatic chromosome of D. nasutoides.

The karyotype of D. nasutoides Okada (UT stock 3035.2) was found to be the following: sex chromosomes - a short rod-shaped X and a J-shaped Y; autosomes - a pair of short rods, a pair of small V's, and a pair of very large V's (Figure 1). Giemsa staining revealed a distinctive banding pattern along the arms of the large V. The entire Y-chromosome stained darkly, as well, but, except for appearing slightly darker in the centromere region, it showed no banding pattern. Centromeric regions of the other chromosomes stained darkly (Figure 2).

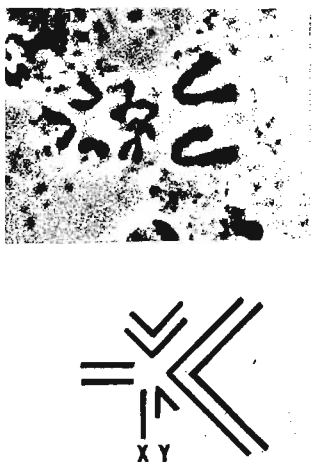


Fig. 1.

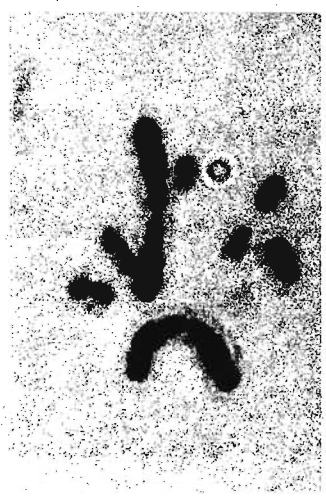


Fig. 2.

The staining technique applied was essentially that of Hsu (1971). However, we denatured the ganglion preparations for 1-2 minutes in 0.035 or 0.007 M NaOH dissolved in Demerec's Drosophila Ringers'; the concentration of Na^+ ions was adjusted to be the same as for the original Ringers' by varying the amount of NaCl. Overnight incubation was carried out in 2X- rather than 6X- SSC.

D. nasutoides, a member of the hypocausta subgroup of the immigrans species group, appears to be an isolated endemic whose distribution is limited to the islands of Samoa. No closely related species have been recognized (M.R. Wheeler, personal communication). However, the possibility of variation among local populations should be investigated.

The molecular structure of the D. nasutoides chromosomes is currently being investigated by C.S. Lee and M. Cordeiro in this department.

This work was supported by NSF Grant GB 22770 to R.H. Richardson.

References: Hsu, T.C. 1971, J. Hered. 62:285-287.

Belyaeva, E.S. and I.F. Zhimulev.
Institute of Cytology and Genetics, Novosibirsk, U.S.S.R. Puff size variability in D. melanogaster.

Variability in puff size was studied in D. melanogaster salivary chromosomes in normal and experimental conditions. To rule out the influence of sex and asynchronous development of single individuals, only females at the stage of spiracle eversion (0 hour prepupae) were used.

Puff studies included the visual estimation of the puffing patterns of the X-chromosome in prepupae from a natural population of Drosophila (Alma-Ata), laboratory stocks (Batumi-L,

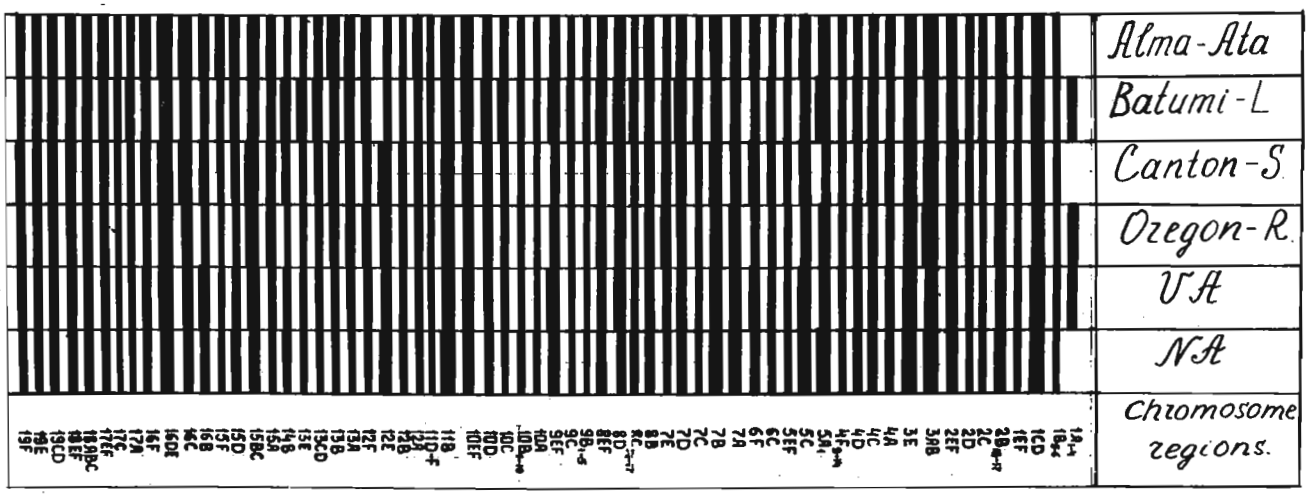


Figure 1. Puff size in the X-chromosome in a natural population and different D. melanogaster stocks. Width of bar corresponds to average puff size estimated visually in 50 chromosomes of each stock.

Oregon-R, Canton-S) and stocks NA and VA inbred for 100 generations (brother x sister). As a result, no significant differences were established in puffing patterns, nor in puff size (Figure 1). Puff $5A_1$ is an exception because stocks Canton-S and VA were found to differ in size. The differences are statistically significant. Thus, this study did not confirm the

Table 1. Variability in the size of puffs $63E_{1-5}$ and 71CE in 0 hour prepupae of Oregon-R stock.

Experimental conditions		Average ratio of diameters $\frac{63E_{1-5}}{63A}$	$\frac{71CE*}{72A}$	Variation range in size	Standard square deviation	Occurrence rate of puff	No. of puffs and individuals studied	
Normal culture		2.0		1.3-3.0	0.34	100%	100	10
		1.8*		1.3-2.7	0.28*	100%	100	10
Overcrowded culture		2.0		1.2-3.1	0.37	100%	100	10
		1.9*		1.3-3.0	0.34*	100%	100	10
Parts of one gland	proximal	1.7		1.3-2.1		100%	24	
		1.6*		1.3-2.0		100%	20	
	intermediate	1.7		1.3-2.1	0.27	100%	22	
		1.7*		1.3-2.2	0.24*	100%	13	
	distal	1.8		1.3-2.5		100%	15	
		1.7*		1.3-2.3		100%	8	

* Data for puff 71CE are given in last line.

results of Lychev (1965) according to which inbreeding decreases puff activity.

In the other experiments puffs $63E_{1-5}$ and 71CE and neighboring unpuffed bands (63A and 72A) were measured with an ocular micrometer. Average ratios puff diameters to band diameters were calculated for 0 hour prepupae developed in normal culture (yeast medium, 75 larvae per bottle) and overcrowded culture (75 larvae developed in a bottle to imago hatching. Their progeny were grown on stale food in the same bottle and subsequently was used for chromosome preparations). Average ratios in these puffs are similar in prepupae samples grown in normal conditions and very crowded conditions (Table 1). Therefore, the conditions under which the culture is maintained do not influence puff size, nor its variability. To analyze puff variability in a gland, aceto-orcein-stained gland was subdivided into 3 equal parts. No differences in puff size and the rates of occurrence were found in these puffs (Table 1).

Variability observed in all the samples was mainly due to the differences between the cells within a gland. However, these differences do not depend upon cell localization (Table 1).

The authors are very indebted to Dr. L.S. Kaidanov (Leningrad) for sending NA and VA flies and stimulating discussions.

Reference cited: Lychev, V.A. 1965, Tsitologia (USSR) 7:325-333.

Hoenigsberg, H.F., L.E. Castro and H.R. von Prahl. Universidad de los Andes, Bogotá, Colombia. The Drosophila willistoni group from Colombia.

The geographic distribution of the willistoni group in tropical America raises several questions concerning sympatric speciation of certain members of some of the species of the paulistorum complex (Spassky, et al., 1971). Furthermore, some of the courtship and sexual isolation within the paulistorum complex which break up when Andean and Interior "species" from the Yaguaracacan (Amazon River branch) come together, permit hybridization which at least temporarily swarm the recently erected sexual barriers (Hoenigsberg, et al., 1973).

Moreover, the more general problem of the sexual isolation found within several "subspecies" of the paulistorum complex may gain somewhat if complete collecting records are shown. The following data may serve those interested in the speciation of this group. These figures constitute the complete records from 1968 - 1971.

	Collected Species										
	willistoni		equinoxialis		tropicalis		insularis		paulistorum		
Localities	#	%	#	%	#	%	#	%	#	%	Total
AMAZONIA											
Yaguaracaca A											
Leticia (Bajo)	64	24.52	172	65.90	4	1.53	-	-	21	8.05	261
Yaguaracaca B											
Leticia (Alto)	66	13.75	40	8.33	8	1.67	-	-	366	76.25	480
Marco - Brazil	201	21.16	308	32.42	315	33.16	-	-	126	13.26	950
CAQUETA											
Valparaiso - 1	204	51.78	50	12.69	62	15.74	-	-	78	19.80	394
Valparaiso - 2	348	40.80	92	10.79	95	11.14	-	-	318	37.28	853
VAUPES											
Mitú-1 (Rio arriba)	182	25.96	166	23.68	1	0.14	-	-	352	50.21	701
Mitú-2 (Misión)	438	43.93	141	14.14	17	1.71	-	-	401	40.22	997
MACARENA	31	18.67	80	48.19	30	18.07	-	-	25	15.06	166

Acknowledgements: We are grateful to Colciencias for financial support and to Drs. Th. Dobzhansky and R. Richmond's company in our 1968 collection.

References: Hoenigsberg, H.F. and L.E. Castro 1973, Genetica (in press); Spassky, B., R.C. Richmond, S. Pérez-Salas, O. Pavlovsky, C.A. Mourao, A.S. Hunter, H.F. Hoenigsberg, Th. Dobzhansky and F.J. Ayala 1971, Evolution 25:129-143.

Vaidya, V.G. and N.N. Godbole. University of Poona, India. First report of genus Chymomyza (Drosophilidae) from India: Chymomyza pararufithorax sp. nov.

The genera of Drosophilidae other than Drosophila so far reported from India are Gitonides, Leucophenga, Scaptomyza, Zaprionus and Cacoxenus. To this list is here added the genus Chymomyza represented by C. pararufithorax sp. nov.

DESCRIPTION OF THE MALE IMAGO: General features and head: Body about 2.2 mm in length. Eyes dark red with pile. Antenna yellowish brown, third segment pubescent. Arista with about 4 branches above and 3 below including terminal fork. Palpus with a few prominent setae. First oral prominent, about twice the length of the second. Carina broad below and narrow above. Second orbital about 1/2 the third and about 2/5 the first. Postverticles small.

Thorax: Thorax dark brown, shining. Humerals 2, upper longer. Acrostichal hairs in 8 somewhat irregular rows. Dorsocentrals 2 long. Cross distance between dorsocentrals about two times the length distance. Prescutellars absent. Anterior scutellars slightly divergent. Posterior scutellars crossing each other. Sterno-index about 1.1.

Legs: Yellowish. Forefemur, tibia and proximal tarus dark. Preapicals on all three tibiae and apicals on first and second. Forefemur swollen, with a row of few prominent bristles.

Wings: Wing with a white patch apically. Costa and cell R_1 black. Costal index about 0.9. 4th vein index about 1.8. 4C-index about 1.5. 5 X-index about 1.7. C 1 bristles 2. C 3 bristles on basal 3/4.

Abdomen: 1 T yellowish. Remaining tergites totally black and shining.

Periphallic organs: Dark brown.

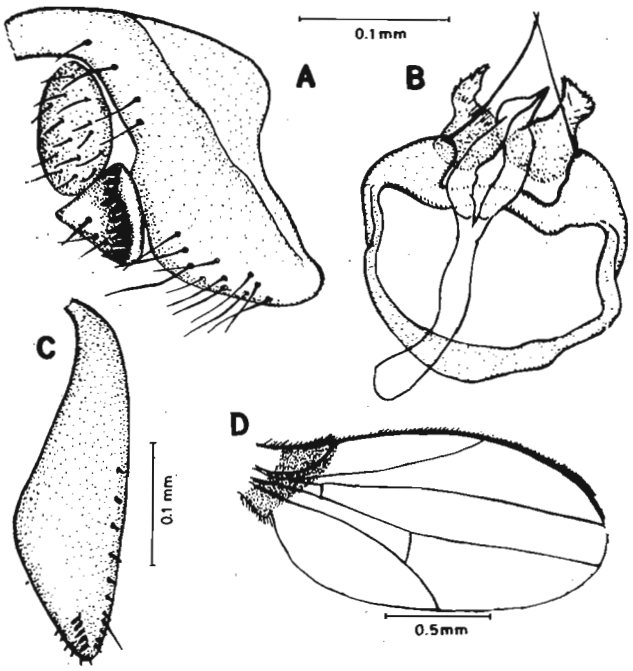


Figure: Chymomyza pararufithorax sp. nov. A. Periphallic organs. B. Phallic organs. C. Egg guide. D. Wing.

Genital arch broader below with about 4 setae on its dorsal half and about 11 setae on its ventral half. Heel absent. Toe rounded. Primary clasper roughly triangular with a sinuous row of 11 teeth and with about 3 marginal bristles. Anal plate roughly oval, separate from the genital arch and with about 15 long setae.

Phallic organs: Aedeagus simple, long and curved. Apodeme of aedeagus short. Novasternum with two long submedian lobes. Anterior paramere elongate, apically hairy. Posterior paramere absent. PI about 0.8.

Rectal index about 2.2.

DESCRIPTION OF THE FEMALE IMAGO: Similar to male. Egg guide: Lobes pale yellow, swollen in the middle, broadly rounded at the tip, with about 15 marginal teeth. Five discal teeth arranged in a row. One subterminal hair. Basal isthmus short and narrow.

MATERIAL: Holotype: Male: Poona (India) July 1972 (Vaidya & Godbole). Deposited with the Department of Zoology, University of Poona, Poona-7, India.

Paratypes: 7 males, 2 females. 1 male deposited with Prof. T. Okada, Department of Biology, Tokyo Metropolitan University, Tokyo, Japan.

HABITAT: The flies were collected in vegetable garden plots by sweeping with net. They appear only in the wet season. It was not possible to rear them in the laboratory.

RELATIONSHIP: This new species seems to be allied to Chymomyza rufithorax (de Meijere) from Indonesia. It resembles C. rufithorax in having yellowish orange mesonotum, white apex of wing and foreleg with black femur and tibia. It however differs from the later species in

having black fore metatarsus.

Acknowledgement: The authors are grateful to Prof. Toyohi Okada of the Tokyo Metropolitan University for checking the description and for confirming the identification of the species.

Gethmann, R.C. University of Maryland Baltimore County, Catonsville, Maryland.

A case of sex chromosome meiotic drive that is age dependent.

An age dependent case of meiotic drive involving the sex chromosomes has been found. Attached-X males of the constitution $Y^S X.Y^L$, $In(1)24^L$ + $X.Y^S$ $A2^R$, y $\overline{v}$/Dp(1;f)60g, y^{31d} produce equal numbers of $\overline{XY}$ and Dp60 bearing sperm for the first six days after emergence, but for the next six days (days 7-12), they produce, on the average, nearly twice as many duplication bearing sperm as $\overline{XY}$ bearing sperm. (See Table 1). The attached-XY is composed of the left end of In(1)24, which carries Y^S distally and is essentially a reinversion of In(1)EN, and

Table 1. Number of progeny by age of parental male.

Paternal age (days)	Regular Progeny y ♀	Regular Progeny y^{31d} w^{sp} ♂	Exceptional Progeny ♀ nondisj.	Exceptional Progeny ♂ nondisj.	Total progeny	average no. prog/male	♂/♀ ratio
1-2	652	684	1	0	1337	191.0	1.05
3-4	1491	1511	4*	4	3010	430.0	1.01
5-6	835	877	0	0	1712	285.3	1.05
7-8	262	477	0	1	740	148.0	1.82
9-10	236	491	0	0	727	145.4	2.08
11-12	370	600	0	4	974	243.5	1.62

* Recovered as a cluster from one mating.

the right end of a detached attached-X, A2, which carries $Y^L(X.Y^L)$. Thus, the euchromatin is in normal sequence, except for a small distal duplication (Novitski, DIS 25:122). Dp(1;f)60g is an X-chromosome duplication carrying the tip of $In(1)sc^8$ and at least one complete dose of the X heterochromatin. It is marked by y^{31d} from sc^8. It occurred spontaneously in a triploid female and was recovered along with its reciprocal exchange product, C(1)RA60g (Mohler, DIS 34:52).

The experimental procedure was as follows: Single males less than 36 hours old were mated to 3 y w^{sp} virgin females. Every two days, the males were transferred to new virgin females without etherization. The females were subcultured every three days for a total of 12 days. There was no change in the sex ratio in the subcultures. A total of 9 males were tested. The number of fertile males for each successive brood was 7, 7, 6, 5, 5, and 4.

For the first six days, males produced $\overline{XY}$ and Dp60 bearing sperm in equal numbers. The slight excess of males over females for the first six days is not significant ($X^2 = 1.46$). There is an obvious excess of male progeny starting with the seventh day. Nondisjunction in the males was low throughout the experiment. The nine exceptions included 7 nullo-$\overline{XY}$, Dp sperm.

There was also a drastic drop in the total number of progeny after day 6, although part of this was due to fewer fertile males in the later broods. On a per male basis, there is still a large drop in the number of progeny after day 6. Although controls were not run on these experiments, this type of a drop in total progeny was not expected, based on an examination of similar brooding experiments taken from the literature (Hiraizumi and Watanabe, Genetics 63:121; Yanders, Genetics 51:481). Considering all progeny, 63% were recovered over the first 6 days, whereas for comparable experiments, the average is around 53%. For just Dp60 progeny, 57% were recovered over the first 6 days. All of this suggests that part of the drop in total progeny is probably due to the missing $\overline{XY}$ sperm, but that all of the drop cannot be accounted for by dysfunction of the $\overline{XY}$ bearing sperm.

Supported by a grant from NSF (GB-38446).

Sidhu, N.S. Indian Veterinary Research Institute, Izatnagar, India. Electron microscopic study of the wall of the vesicula seminalis of D. melanogaster.

Electron microscopy reveals the real form and thus even function of an organ. Certain state of a cell reveals real functional aspects which can be understood through the experience of examining large numbers of electron micrographs taken. Reproductive cells of insects have been studied through use of electron microscopy by workers like Afzelius (1959), André (1961, 1962) Kiefer (1966, 1968, 1970), Sjöstrand and Afzelius (1956), Sidhu (1963, 1970) etc. However, no report has come to the present author's notice, in spite of his best efforts, describing the wall of the vesicula seminalis in Drosophila in detail. Hence the study was carried out with an interest in knowing whether trophocytes are produced by the epithelium of the testicular wall.

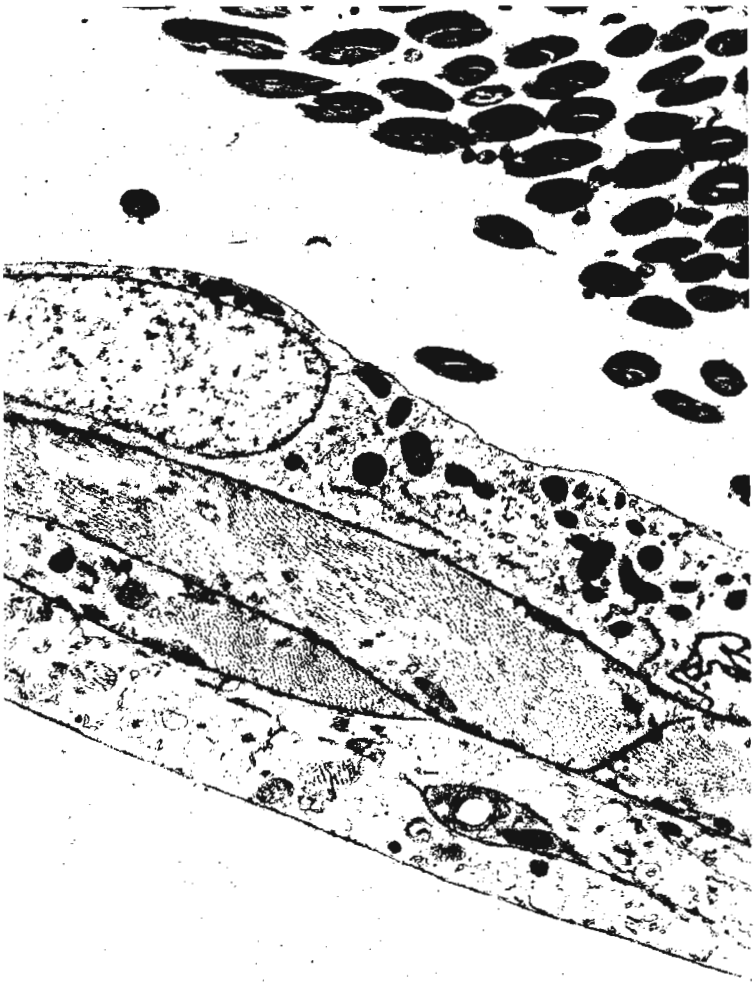


Figure 1. An electron micrograph of a section of the vesicula seminalis of D. melanogaster.

Or-K strain of Drosophila melanogaster was utilized to prepare the material for this study. Seminal vesicles were fixed in Osmic acid vapors and araldite blocks were prepared and sectioned with 500Å thickness using a Porter Blum ultratome. Staining was done using Uranyl acetate and a Philips E.M.75 electron microscope was used for taking the pictures at the Institute of Animal Genetics, Edinburgh, during 1962.

The wall of the vesicula seminalis (Figure 1) has three layers as described briefly for insects by Imms (1960) with light microscopy. The outermost is the peritoneal coat, followed by a middle muscle fibre supporting the innermost coat of epithelial cells. The peritoneal coat is about 1μ in thickness and a few μ in length. The layer has single cell thickness except for overlapping of cells at their ends. A number of mitochondria, endoplasmic reticulum membranes, and some ribosomes are seen in the peritoneal cells, indicating lack of any secretory activity of the layer. The muscle fibres are usually single cell in thickness but at some places even double cells are seen. There are a few mitochondrial bodies in them. Chromatin mass is also observed which appears to be lacking nuclear membranes. The thickness of muscle cell is about 1μ. The innermost epithelial layer is a single cell in thickness, 1μ except being wider at the region of large nucleus. The nucleoplasm seems to possess chromatin fibres in interphase state. Deoxyribonucleic acid (DNA) as well as some Ribonucleic acid (RNA) appear to be present in the nucleoplasm.

The epithelial cell has ribosome-like structures on the endoplasmic reticulum in addition to the mitochondria, large numbers of which are seen. The nuclear membrane is double-layered. The inner membrane of the epithelium is very thin and the secretory function of the epithelial cells is evident from their structure. The trophocyte cells are perhaps also produced by

these epithelial cells (Sidhu, 1973, unpublished). No details regarding formation of the trophocyte cells could be obtained by the study of the testicular wall. The total thickness of the wall of the vesicula seminalis is 3 μ approximately.

References: Afzelius, B. 1959, J. Biophys. Biochem. Cytol. 5:269-278; Andre, J. 1961, J. Ultrastruct. Res. 5:86-108; Imms, A.D. 1951, A General Text Book of Entomology (Methuen & Co., London); Kiefer, B.I. 1966, Genetics 54:1441-1452; _____ 1968, Genetics 60:192; _____ 1970, J. Cell Sci. VI (1):177-194; Sidhu, N.S. 1963, Ph.D. Thesis, Univ. of Edinburgh; _____ 1970, Ind. J. Hered. 2:15-38; Sjostrand, F.S. and B.A. Afzelius 1956, Exp. Cell. Res. 14:268-285.

Miglani, G.S. and R. Parkash. Punjab Agricultural University, Ludhiana, India.

Effect of certain chemicals on salivary chromosomes of Drosophila.

The effect of five chemicals, namely, ethylmethane sulphonate (EMS), methylmethane sulphonate (MMS), diphenethyl alcohol (DPA), hydroxylammonium sulphate (HAS) and maleic hydrazide (MH), was studied on salivary chromosomes of D. melanogaster (Oregon-K stock), through induction of chromosomal aberrations. The chemicals were mixed with food and fed to the larvae. Chromosomes were examined for aberrations in fully grown third instar larvae. Inversions were almost the only type of chromosomal rearrangements induced and hence their frequency was considered as a measure of chromosomal damage.

Table 1 shows the number of inversions observed. Out of 35 inversions observed in larvae fed with EMS, eleven were in X, four in 2L, six in 2R, nine in 3L and five in 3R chromosome.

Table 1. Distribution of inversions induced by EMS, MMS, DPA, HAS and MH.

Chemical	No. of larvae	Total no. inversions induced	No. of observed and expected* inversions induced										Aberrations/ 100 larvae
			X		2L		2R		3L		3R		
			Obs.	Exp.	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.	
Control	90	Nil	0	0.00	0	0.00	0	0.00	0	0.00	0	0.00	0.00
EMS	113	35+1**	11	6.57	4	6.23	6	7.01	9	6.41	5	8.30	31.86
MMS	66	16	4	3.04	0	2.88	3	3.24	1	2.96	8	3.84	24.24
DPA	115	48	1	9.15	4	8.66	10	9.74	16	8.91	17	11.54	41.74
HAS	84	35+1***	0	6.84	0	6.49	13	7.21	11	6.88	11	8.54	42.86
MH	75	10	1	1.87	0	1.78	2	2.00	2	1.83	5	2.37	13.33

* Calculations explained in text
** Translocation
*** Deletion

MMS induced sixteen inversions: four in X, three in 2R, one in 3L and eight in 3R chromosome. DPA induced 48 inversions: one in X, four in 2L, ten in 2R, 16 in 3L and 17 in 3R chromosome. HAS induced 35 inversions: 13 in 2R, 11 in 3L and 11 in 3R chromosome. MH induced ten inversions: one in X, two in 2R, two in 3L and five in 3R chromosome. Surprisingly, MMS, HAS and MH did not affect 2L chromosome and HAS did not affect the X chromosome, suggesting thereby specificity of different mutagens. One translocation and one deletion were also induced by EMS and HAS, respectively. HAS induced the highest frequency of inversions (42.86%) and it was followed by DPA (41.74%), whereas, EMS, MMS and MH induced 31.86%, 24.24% and 13.33% of inversions respectively. In addition to the number of the observed inversions induced by each chemical in a particular chromosome, Table 1 also depicts their expected numbers. The expected numbers were calculated by applying the formula a/b x c where 'a' is length of a particular chromosomal arm, 'b' is total chromosomal length of the complement and 'c' is the total number of inversions induced by a particular chemical. Comparison of observed and expected numbers reveals that in no case these numbers coincide, indicating that induction of abberrations is not a random event.

A comparative analysis of percentages and distribution of the inversions induced by these chemicals in different chromosomal arms depicts that each chemical affects different parts of the genome differently and that a specific chromosome or its specific segments are affected differently. Mutagen selectivity refers to not only specific arms but also to specific parts in each chromosome.

Sulerud, R.L. Augsburg College, Minneapolis, Minnesota. Two types of sensitivity to carbon dioxide in D. affinis.

Two CO_2-sensitive strains of D. affinis were established from isolated wild-caught affinis subgroup females (Minneapolis, Minnesota) in 1968. These strains were designated S35 and S52. Resistant strains designated R48 and R50 were also established at that time. "Sensitivity" was determined by a failure to recover from a 15 minute exposure to pure CO_2 at 14°C (standard test).

Reciprocal crosses have been made of S35 and S52 flies to R48 flies. The recovery of progeny from a standard CO_2 test is shown in Table 1.

Table 1.

	R48♀ x S35♂ (N=410)		S35♀ x R48♂ (N=191)		R48♀ x S52♂ (N=760)		S52♀ x R48♂ (N=360)	
Rec. time	N rec	% rec	N rec	% rec	N rec	% rec	N rec	% rec
15 min.	232	57	0	0	341	45	0	0
30 min.	254	62	0	0	534	70	0	0
1 hr.	283	69	0	0	625	82	0	0
2 hrs.	286	70	0	0	642	84	0	0
4 hrs.	285	70	0	0	645	85	0	0

Carbon dioxide sensitivity was efficiently transmitted maternally in both strains, but paternal transmission was only partial. None of the progeny produced by sensitive females recovered, whereas after four hours the recovery percentages for progeny of S35 and S52 males crossed to resistant females were 70 percent and 85 percent respectively. Recovery often required longer than 15 minutes (maximum recovery time for resistant flies), especially for progeny of S52 males. Therefore, if the percentage of sensitive progeny is determined by failure to recover within 15 minutes after testing, S52 males would be considered to have transmitted sensitivity more efficiently than S35 males (55 percent and 43 percent non-recovered progeny respectively).

Another difference between the S35 and the S52 strains was revealed by testing the two strains at various temperatures. Flies of resistant strains R48 and R50 were tested for comparison, as shown in Table 2.

Table 2.

Temp. °C	R48 and R50		S35		S52	
	rec/N	% rec	rec/N	% rec	rec/N	% rec
4	112/112	100	0/108	0	25/122	20
9	40/40	100	0/37	0	7/26	27
14	70/70	100	2/102	2	39/220	18
19	69/70	99	9/16	56	5/33	15
24	108/110	98	23/23	100	7/34	21
28	70/70	100	34/34	100	2/36	6
30	64/71	90	65/72	90	0/64	0

Few if any S35 flies recovered when tested at temperatures of 14°C or lower, but when tested at 24° or 28°C they all recovered and all but seven flies recovered following a 30°C test. In contrast, all but two S52 flies failed to recover when tested at 28°C, and no flies of this strain recovered from the 30°C test. Between 15 percent and 27 percent recovery took place when S52 flies were tested at temperatures below 28°C. Although not shown in the table, this recovery usually took longer than 15 minutes.

The inability of S52 flies to recover at higher testing temperatures and the delay in recovery beyond 15 minutes from testing at lower temperatures are features which are shared by "delayed-recovery" melanogaster as described by McCrady and Sulerud, 1964. However, delayed-recovery in melanogaster is due to a third chromosome gene, while in affinis a cytoplasmic agent is implied by the maternal inheritance of the condition. Characteristics of S35 sensitivity are more similar to the well-known CO_2 sensitivity in melanogaster caused by virus sigma. Injection experiments have not been conducted to determine whether an infectious agent

is involved in either S35 or S52, but such an agent has been demonstrated in other affinis strains by Williamson, 1961, and others.

Unfortunately both S35 and S52 have been lost in a fire, but new CO_2 sensitive affinis strains are now being established with the aim of studying the variation in sensitivity which seems to exist in this species.

References: McCrady, W.B. and R.L. Sulerud, 1964 Genetics 50:509; Willismson, D.L. 1961, Genetics 46:1053.

Søndergaard, L. University of Copenhagen, Denmark. Studies on the behavour of the paralytic mutant Out-cold^{ts}.

When females heterozygotic for Ocd^{ts} are transferred from 25°C to 19°C (or below) they show a constant sequence of behavoural patterns: 1) uncoordinated movements of the legs so that they fall on their backs, 2) flexion of the first and strong deflexion of the second and third pairs of legs, 3) flutter of the wings so that the flies flop around in the vial (20% of the flies do not show wing flutter). After this sequence the legs are relaxed and the flies are immobilized. When shifted back to 25°C immobilized flies recover mobility within 1-5 min dependent upon how long they have been kept at a low temperature. The duration of the behavoural patterns 1 and 2 does not vary between specimens, but varies with the magnitude of temperature shift down from 25°C (Fig. 1).

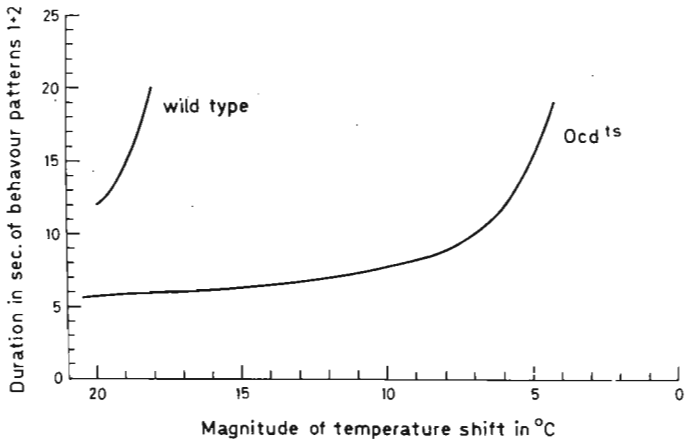


fig.1

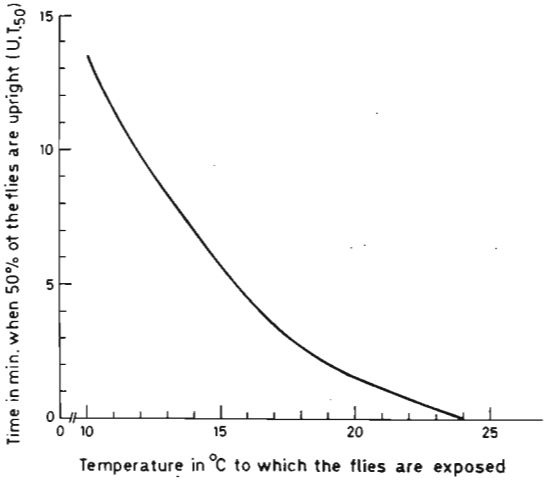


fig. 2

Even when flies are shifted from 25°C to between 20°C and 23°C they are affected, but an increasing number show only uncoordinated leg movements (39% at 20°C and 80% at 23°C).

Paralyzed flies kept at low temperatures regain normal behavour after some time (Fig.2). Wild type flies when shifted to a temperature of 7°C show a similar behavour, but take a longer period of time to become paralyzed.

Ocd^{ts} males at 25°C walk in a reeling manner and fall over frequently; only after a long period of time of kicking do they rise again. Usually only about 50% of the males in a population are upright at a given time. About 40% hold their wings in a drooped position. Ocd^{ts} males are smaller than normal males, and tend to stick in the media immediately after eclosion. They are weak and even when prevented from drowning they will not survive for 48 hours. The males as well as the females are affected by low temperatures. Although incapable of flying, they flutter their wings after cold shocks, as do the females.

Ocd^{ts} flies show leg shaking when etherized, although not as much as the mutant HK_2. Etherized flies and flies injected with tubocurare will show no paralytic behavour in connection with cold shocks. Wild type flies fed a sublethal dose of DDT behave as a phenocopy of Ocd^{ts} males. These observations suggest that Ocd^{ts} mutants are in some way affected in the nervous system.

Orevi, N. The Hebrew University, Jerusalem, Israel. Time of pn-Kpn interaction in males of D. melanogaster.

It was claimed that pn/Y; Kpn/+ males die in early second larval instar (Glassman 1962, Lifschytz and Falk 1969). Hackstein (1971) suggested that some of the larvae of the genotype pn/Y; Kpn/+ die at approximately 36 hours after hatching, while others survive as long as 60-96 hours after hatching.

We studied the time of death of pn-Kpn larvae by taking advantage of the larval marker cho (chocolate-red Malpighian tubules, 1-5.4). Larvae from the mating y pn cho/y pn cho; +/+ x +/Y; Kpn/Kpn were studied by two methods.

a) Counted samples of more or less synchronous larvae were collected at given ages, the proportion of larvae that survived to imagoes was determined (Lifschytz and Falk 1969).

b) The proportion of yellow-chocolate larvae on petri-dishes in which eggs were layed for 2-3 hours was determined at various time intervals.

Three different pn alleles were studied. Although there were differences between alleles in details, the general pattern was uniform enough to be summarized as follows:

pn-Kpn larvae are smaller and their metamorphosis is much slower than that of the normal larvae (see also Hackstein 1971). The difference in size between pn-Kpn and normal larvae is conspicuous already at 28 hours after hatching.

As late as 90 hours after hatching up to 40% of the larvae were yellow-chocolate. Many pn-Kpn larvae survived long after the normal ones pupated; they became extremely large and accumulated melanotic tumors in the hemolymph. Some died as 3rd instar larval 180 hours after hatching.

References: Glassman, E. 1962, DIS 36:66; Hackstein, J.H.P. 1971, Molec. Gen. Genetics 111:373-376; Lyfschytz, E. and R. Falk 1969, Genet. Res. 14:53-61.

Tracey, M.L., O. Pavlovsky and M.M. Green, University of California, Davis, California. Hybridization of D. melanogaster and D. simulans. A frequency estimate.

The process of speciation may be thought of as the formation of reproductive isolating barriers between previously compatible populations. In the genus Drosophila almost all interspecific crosses produce progenies which are inviable or sterile. Under natural conditions sexual isolation precludes, in general, the production of such hybrids; nevertheless, hybrid flies have been collected (Ehrman, 1962).

Interspecific crosses between D. melanogaster females and D. simulans males yield sterile females; the reciprocal cross yields sterile males. We have used this fact to estimate the frequency of hybridization between these sibling species in nature. The occurrence of unisexual progenies produced by single females may be due to i) sex ratio condition, ii) presence of tightly linked, complementary lethals on both maternal X chromosomes, or iii) hybridization. The first and second explanations are readily distinguished from the third because only the hybrid progeny are sterile.

At the McDonald Ranch collecting site in Napa County, California, populations of melanogaster and simulans are very large in September and October when the two species are roughly equal in frequency. To detect possible hybrid matings we distributed 1114 females, collected October 3, 1972, to individual vials on October 4, 1972. Between collection and distribution the females were held together with males and other species in half-pint bottles in a 16°C incubator.

Four of the 1114 females distributed yielded more than 25 females and no males. All other females (1110) produced bisexual progenies. At least 20 females, from each of the four unisexual progenies, were individually tested with simulans v and melanogaster Cy males. All 80 females were sterile.

Although the degree of sexual isolation between these species is high, it is known to be affected by sex ratio, male and female age, density, and genotype (Parsons, 1972; Manning, 1959). It is probable that some, if not all four, of the hybrid matings we have found took place in the half-pint holding bottles prior to distribution. Nevertheless, a hybridization frequency of 3.6×10^{-3} between wild melanogaster and simulans is offered as an approximate upper limit.

References: Ehrman, L. 1962 Quart. Rev. Biol. 37:279; Manning, A. 1959 Animal Behaviour 7:60; Parsons, P.A. 1972 Can. J. Genet. Cytol. 14:77.

Michinomae**M. and S. Kaji. Kyoto Prefectural University of Medicine, Kyoto, and Konan University, Kobe, Japan. The various lysosomal frequencies during the development of the Bar eye disc.

Fristrom (1969, 1972) has demonstrated that degenerating cells appear in the presumptive eye cells during the initial stage of development of the Bar eye disc. Previously, we reported that this cell death is associated with the presence of lysosomes (Kaji and Michinomae 1973; Michinomae and Kaji 1973). During the development of the Bar eye disc, different types of specific structures were observed by electron microscopy. These are simple vesicular figures, myelin figures, fragments of cell organelles each bound with a single membrane and lipid droplets. The presence of these specific structures are observed by acid phosphatase reactions by heavy deposition of osmium black. These acid phosphatase-positive and membrane-bound structures may be defined as lysosomes.

In this paper will be examined various lysosomal frequencies during the development of the Bar eye disc, asetamide-treated Bar eye disc and wild type disc. The lysosomal frequencies and their types are differenciated during the development of the eye disc. The developmental process of the eye disc was correlated with the changes in specific fine structures. In the Bar eye disc, the first visible signs of the specific structures appeared already in the 48 hour larvae after hatching as simple vesicular figures. In the 60 hour disc, there were found simple vesicular figures and myelin figures. Later in this stage, the simple vesicular figures were gradually decreased, accompanied by progress of the eye development. In the 70 hour disc, myelin figures and cell fragments were mainly detected. In the 85 hour disc lipid droplets were observed in addition to myelin figures and cell fragments. After puparium formation, most of the degenerating bodies were changed to lipid droplets. Acid phosphatase activity could be detected in the specific structures of the eye discs except in the case of simple vesicular figures. From this sequence of changes it is interpreted that these fine structures are lysosomes which are undergoing change from the primary (simple vesicular figure) to secondary (myelin figure and cell fragment) and derivative types (lipid droplet). The results of these observations are summarized in Table 1.

Table 1. Relative value of the various lysosomal frequencies of Bar, acetamide-treated Bar and wild type during the development of the eye discs.

Time after hatching	Strain	Primary lysosomes	Secondary lysosomes	Derivative lysosomes
	B	+	-	-
48 hours	BA*	+	-	-
	W	+	-	-
	B	+ +	+ +	-
60 hours	BA	+ +	+ +	-
	W	+ +	+ +	-
	B	+	+ + +	±
70 hours	BA	±	+	-
	W	-	+	-
	B	±	+ + + +	+
85 hours	BA	-	+	-
	W	-	+	-
	B	-	+ + + +	+ + + +
95 hours	BA	-	+	±
	W	-	+	±
	B	±	+	+ + + +
100 hours	BA	+	±	+
(pre-pupae)	W	+	+ +	+

B: Bar eye disc. BA: acetamide-treated Bar eye disc. W: wild type (Oregon-R) eye disc.

*The Bar larvae for the acetamide treatment, were reared in the normal medium from hatching to 42 hours and then transferred to 1.5% acetamide mixed medium for growth until they reached the specific stage.

As apparent in the Table, lysosomal precursor has appeared already in the 48 hour discs. However, in the case of the acetamide-treated Bar eye discs, relative values of various lysosomal frequencies were highly decreased during the development of 70 hour to pre-pupal stage. Moreover, in the case of the wild type discs, these frequencies were also less than that of the untreated Bar eye discs.

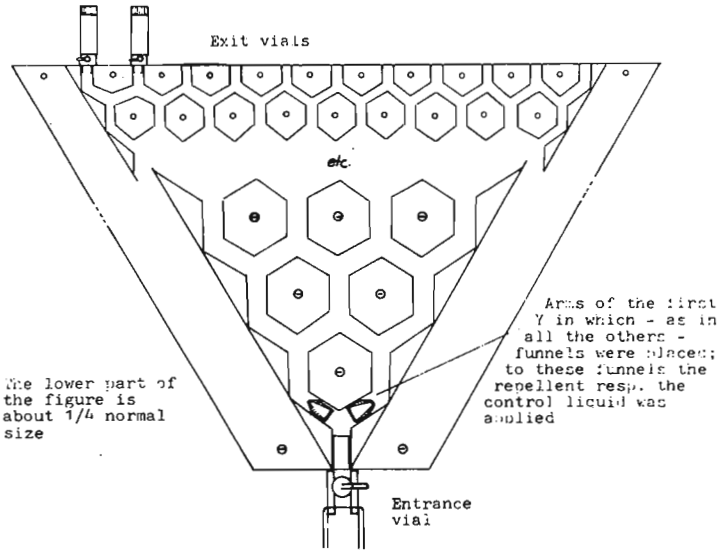
These results suggested that acetamide acts to inhibit the appearance of lysosomes on the metabolic process of the mutant Bar eye disc during development.

References: Fristrom, D. 1969, Molec. Gen. Genet. 103:488-491; _____ 1972, Molec. Gen. Genet. 115:10-18; Kaji, S. and M. Michinomae 1973, Genetics 74:Suppl. 22,2:130; Michinomae, M. and S. Kaji 1973, Japan. J. Genetics 48:297-300.

** will be on leave from Kyoto Prof. Univ. of Medicine to Konan Univ., April 1974.

Gerresheim, F. Universität München, Germany. An attempt to induce and select mutants with abnormal chemotactic behavior.

A search was made for mutants which do not show the avoidance reaction of normal flies toward an insect repellent, i.e. for mutants which behave indifferently or at least show a significantly lower sensitivity. Wild-type Berlin males were fed with EMS and crossed to attached-X females. F_1 males with abnormal behavior were selected and then, in order to determine whether an X-chromosomal mutation had actually been induced, their male progeny in the F_2 were tested.



In order to distinguish the abnormal flies from the normals, a Y-maze, described by Becker (M.G.G. 107:194-200, 1970), was used (Figure). In each of ten consecutive Y's the flies choose between, on one side, the repellent (primarily N,N-Diethyl-meta-toluamide) and, on the other side, a neutral control liquid. After ten choices, the exit vial a fly arrives at tells how often the repellent containing arms of the Y's have been avoided. And the mean of the distribution of a group of animals in the exit vials reflects the average orientation behavior of the group.

Large groups of F_1 males showed in general a strong asymmetric distribution. In 35 test series, out of 13500 F_1 males 60 deviating ones were singled out, i.e. ones that ended up in one of the central exit vials. From each of them an F_2 was raised. None of these showed a significant deviation from the controls. In 3 doubtful cases the test was repeated in the F_3. They, also, did not differ from the controls.

Of all the possible explanations for the lack of success two seem to be most prominent. (1) The success depends on the degree to which the distribution is asymmetric. Among my test series there were too many in which normal flies arrived at one of the central exit vials. Under such conditions the selection for possible mutants was not sufficiently effective. More important is probably (2) that the chemical sense in insects is generally based on a number of different sensory receptors, some being excitable by only specific substances, others being less specific. Correspondingly, some chemical compounds excite only a single receptor type, others excite several types or all of them. The former, specific type of compound is probably suitable for a study of the kind described; the repellent, however, probably belongs to the latter, non-specific type of compound. Although mutants insensitive to such non-specific compounds are conceivable, mutants insensitive to specific compounds seem more probable. - Investigations which take these considerations into account are under way.

Orevi, N. The Hebrew University, Jerusalem, Israel. Prune temperature-sensitive mutations in D. melanogaster.

A search for prune temperature sensitive (pn^{ts}) mutations was attempted in order to gain more information on the interaction between prune (pn) and killer of prune (Kpn).

Wild-type (Q.A.) males were treated with EMS and mated en-masse to sc^8 pn^1/FM6 females. pn^1 is a deletion in the prune region. Cultures were maintained at 29°C. F_1 females which were suspected to carry a pn-mutation were mated individually to FM6 males. Two cultures were established from each to these females, one was kept at 22°C, the other at 29°C. Sons were checked for the pn phenotype.

50 mutations that proved to be allelic to classic pn mutations were recovered. Of these, 10 were temperature-sensitive, namely at 29°C the eye color was brownish while at 22°C it resembled the wild-type. The pteridine pattern of the ts mutants could be distinguished also chromatographically from that of the wild-type. Nine of the ts mutations are insensitive to Kpn at both temperatures. Females heterozygous for the pn^{ts} alleles and classic pn alleles were uneffected by Kpn at both 22°C and 29°C, while their eye color was intermediate at 22°C and prune at 29°C.

One ts allele displayed temperature-sensitivity also in relation to the pn-Kpn interaction: at 29°C pn; Kpn flies died, while at 18°C at least some survived.

Woodruff, R.C. and J. Bortolozzi. University of Texas, Austin. Induction of mutations in D. melanogaster by 2-nitrosofluorene (a frameshift mutagen in prokaryotes).

Ames et al. (1972) have observed that 2-nitrosofluorene (2NF) is a potent frameshift mutagen in Salmonella typhimunium. A preliminary experiment (Woodruff and Johnson, 1973) indicated that by larvae feeding 2NF was not mutagenic in Drosophila melanogaster. Yet, it was conjectured that this lack of detected mutagenic activity may have been due to the method of administration. This report shows that this conjecture was correct, 2NF when administered to adults by injection increases the frequency of recessive sex-linked lethal mutations.

Oregon-R-C males 2-3 days old were injected with 2NF that was dissolved in dimethylsulfoxide (DMSO) and added to 0.7% saline so that the final concentration of 2NF was 0.06% and DMSO was 5%. The control solution contained 5% DMSO in 0.7% saline. A standard recessive sex-linked lethal mutation experiment was performed using FM7, y^{31d} w^a 1z v B/sc^{10-1} females. The P1 flies were pair mated and all crosses were coded to facilitate the detection of lethal clusters. To determine if visible light has an influence on the mutagenicity of 2NF, treated and control flies were divided into two groups. One group (light) received light for about nine hours per twenty-four, and the other group (dark) was kept in the dark for the entire experiment. The results of this experiment are shown in the accompanying table.

Frequency of recessive sex-linked lethal mutations

2-nitrosofluorene treatment		control	
Light	Dark	Light	Dark
6/911 (0.66%)	11/795 (1.38%)	1/956 (0.10%)	5/925 (0.54%)

The frequency of lethal mutations in the treated group (17/1,1706 = 1.0%) is significantly higher at the 1% level (Stevens, 1942) than the control group (6/1,881 = 0.32%). Among the 23 total lethal mutations recovered in this experiment one 2NF induced lethal mutation which was recovered in the light was temperature sensitive, i.e., it was lethal at 24°C but partially viable at 29°C.

There was an unexpected result in this experiment which should be mentioned. The frequency of lethal mutations produced in the dark (16/1,720) is significantly higher at the 1% level than the frequency of lethal mutations produced in the light (7/1,867). The implications of this preliminary observation are unknown. One possibility is that the absence of light increases the frequency of mutations. We have further tested this possibility and the results are reported elsewhere in this volume (Bortolozzi, Woodruff and Johnson).

References: Ames, B.N., E.G. Gurney, J.A. Miller and H. Bartsch 1972, PNAS 69:3128; Woodruff, R.C. and T.K. Johnson 1973, Genetics 74:s299; Stevens, W.L. 1942, J. Genetics 43: 301.

The junior author was supported by Fundação Amparo À Pesquisa do Estado São Paulo, Brazil.

Köhler, W. Institut für Genetik der Freien Universität Berlin, Germany.

Interaction of selection and recombination.

The character of DDT-resistance of Drosophila melanogaster was used to investigate the interactions of selection and recombination. Our experiments started with two lines, Iso Null and Plus Null, out of our stock Berlin wild + K. Iso Null was isogenic and Plus Null heterogenic in the second chromosome. Each line was subjected to a high (5% survival rate; Iso S, Plus S) and a low (41.7% survival rate; Iso L, Plus L) selection pressure. In the 11th generation of selection, we started two new lines, Iso LS and Plus LS, out of the low selected ones by decreasing their survival rate to 5%. The influences of the 1st and 3rd chromosomes should be pointed out in the Iso-lines, and if recombination would have an important effect on selection response it should be seen in the Plus-lines. To compensate the environmental variation within and between generations of selection we corrected the average survival rates (LD_{50}) of each line by their corresponding controls (adding 10 for arithmetical convenience). Therefore the selection curves in Figure 1 represent the gain of selection in each line in relation to their respective controls. Furthermore we smoothed data five times by the three point formula and these data are shown in Figure 2.

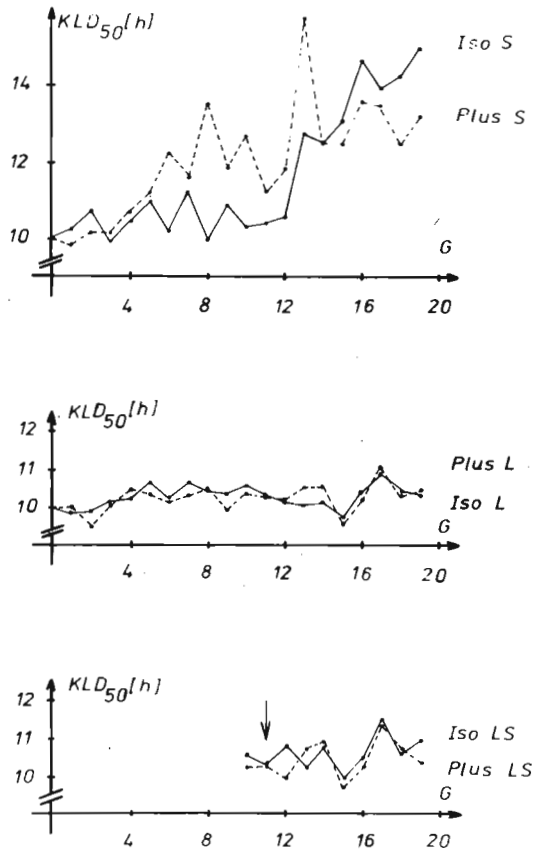


Fig. 1. Corrected average survival rates $KLD_{50}(h)$ of the selected lines plotted against generations (G). The arrow indicates increasing of selection pressure in the low lines.

In order to analyse our experimental data we started computer simulation. First, we used the deterministic model of Lush assuming two unlinked autosomal loci responsible for DDT-resistance with an intermediary and a recessive mode of inheritance. In this case, we got the best fit with the assumption of 5% alleles at each locus responsible for resistance in the initial populations and a coefficient of selection of about 0.70. Secondly, we started another model with 15 loci and three linkage groups using the Monte-Carlo-technique. The values of resistance were simulated by 0 (normal) and 1 (resistant), and between the five loci in each of the linkage groups we fixed the frequencies of recombination at 5, 10, 15 and 20 per cent. The mode of inheritance of the alleles for resistance was defined as recessive in the 1st and 3rd linkage groups and intermediary in the 2nd one without deviation from additivity. In the initial population we started with 5% alleles for resistance and 95% normal alleles at each locus, which were distributed at random. In this model the influence of recombination was strong, especially with respect to the level of resistance and the number of alleles which were fixed, but the effects were suspended in case of low survival rates (10% and 20%, resp.) and by recessive inheritance of the alleles for resistance. It could be pointed out that in our simulated populations the two plateaus observed during selection were not due to recombination, but to the effect of increasing percentages of the recessive alleles for resistance. On the other hand, recombination smoothed the selection curves just to a sigmoid.

Our experimental data and their analysis by computer simulation lead to the conclusion that recombination did not determine the twofold increase of the selection curve of Plus S. This must be much more due to the selection of each an intermediary and a recessive system of alleles responsible for DDT-resistance which may be unlinked. On the basis of our results we

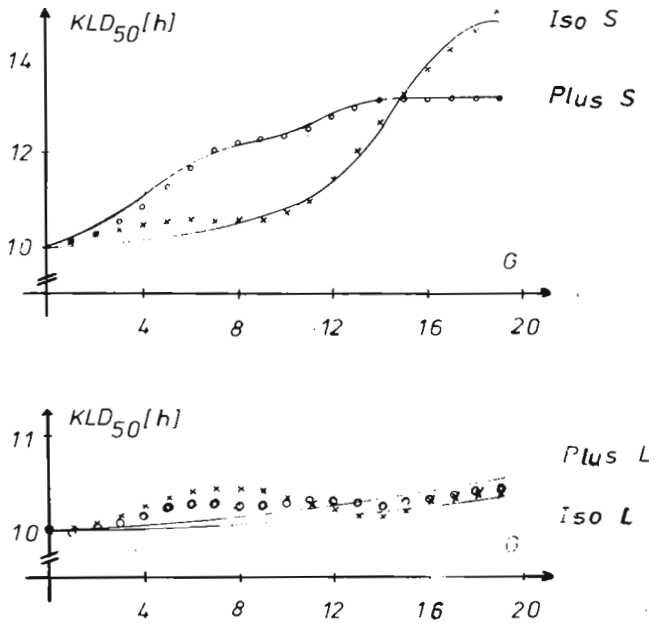


Fig. 2. Circles (o) and crosses (x) indicate the corrected average survival rates of high (uppermost) and low (bottom) selected tines after smoothing. According to the model of Lush the selection curves are the best fit to the experimental data.

can support Robertson's assumption about the importance of recombination or linkage, "that the effects of linkage are much less than we (I) had expected and less than might be assumed for discussions of linkage in literature".

References: Crow, J.F. and M. Kimura 1970, Harper and Row, New Nork; Köhler, W. 1973, Dissertation der Freien Universität Berlin; Robertson, A. 1970, Biomathematics 1:246-288.

Lamb, M.J. and L.J. Lilly. Birkbeck College, University of London, and Middlesex Hospital Medical School, London, England. No detectable increase in sex-linked recessive lethal frequency after feeding male D. melanogaster with the fungicide Benlate.

Benomyl (trade name "Benlate") is a widely used systemic fungicide. Hastie (1970) has reported benomyl-induced instability in Aspergillus nidulans diploids and Boyle (1973) has found cytogenetic effects in Allium cepa and Secale cereale; Dassenoy and Meyer (1973) showed that benomyl induced forward mutations in Fusarium oxysporum. In view of this evidence that benomyl may cause genetic damage, we decided to investigate possible mutagenic effects in D. melanogaster and report here the results of an experiment in which the M-5 technique was used to test for the induction of sex-linked recessive lethals.

Number of chromosomes tested and lethals found after feeding Benlate.

Brood	Fed Benlate		Fed DMSO	
	tests	lethals	tests	lethals
I	340	0	340	0
II	340	1	339	0
III	335	0	339	0
IV	310	0	338	0
V	300	0	320	0
VI	270	0	317	0
Total	1895	1	1993	0

A freshly prepared solution of 0.1% Benlate in 0.5% DMSO was fed to starved 3-day-old Or-R males; each male took approximately 0.14 mg of solution. Control males were fed with 0.5% DMSO. Each treated male was mated with 2 M-5 females in each of six 3-day broods. Approximately 10 chromosomes from each male in each brood were tested for the presence of sex-linked recessive lethals. The results obtained are given in the table. The data provide no evidence of a mutagenic effect of Benlate in Drosophila. It should be stressed, however, that the data are small, only one concentration has been used, only one type of mutation has been investigated, and tests of the mutagenicity of BCM, the breakdown product of benomyl which is formed in aqueous solution, have not yet been made. Further experiments to investigate possible mutagenic effects of benomyl and BCM are in progress.

References: Boyle, W.S. 1973, J. Heredity 64:49-50; Dassenoy, B. and J.A. Meyer 1973, Mutation Res. 21:119-120; Hastie, A.C. 1970, Nature 226:771.

Elens, A. Facultés Universitaires N.D. de la Paix, Namur, Belgium. Initiation of an ethological isolation between ebony and wild Drosophila melanogaster?

Three strains of D. melanogaster have been used in the present experiments: a wild one (Canton special), the mutant strain ebony e^{11}, and a new ebony strain "B" selected after 40 generations of crossing between Canton special and ebony e^{11}.

The first object of this work was to investigate the frequency of heterogamic matings between the wild strain and the ebony ones. It is known that in some cases the sexual isolation can be greater between sympatric than between allopatric races of the same species, as e.g. in the Drosophila paulistorum species complex (Ehrman, 1965). Recently, Nikheev and his collaborators have succeeded in selecting for sexual isolation between the black and vestigial mutants of D. melanogaster. The population was started with 25 pairs of each of both mutant strains. Four days after the beginning of the eclosion period, ten males and ten females of each mutant phenotype were taken as founders of the following generation. It is quite probable that some of the females had been inseminated by heterozygote males; however, the selection was very effective: after eleven generations "a reduction of the proportion of heterozygotes down to below 10% has been attained" (Mikheev et al., 1973). Consequently, it was interesting to consider the hypothesis that the ethological isolation could be greater between wild and the new ebony "B" strain than between wild and the original ebony e^{11} strain.

A second object of the present investigation was to draw a comparison between the sexual activity of the males and the females of a strain in competition with flies of another strain and their sexual activity in presence of flies of their own strain only. It is known that in the presence of wild males, the sexual activity of the ebony males is low but remains constant for a longer time than the sexual activity of wild males (1973). It has been established that on some occasions the experimental inhabition of mating of males can induce later matings of such males and more constancy of mating success, as e.g. for the dragonflies studied by Jacobs (1955). Consequently, it was interesting to examine whether the activity of the ebony males was significantly lowered in the presence of wild males.

In each experiment, the mating successes of three groups of 30 virgin pairs of flies, 4 to 5 days old, were recorded in three observation chambers, according to the multiple choice direct observation method which has been previously described elsewhere (1958). The first chamber contained 30 pairs of wild flies, the second one 15 pairs ebony and 15 pairs wild, the third one 30 pairs ebony. The temperature was 25°C, the relative humidity 40-60%, and the light intensity 500 Lux. For each of the competing pairs of strains (wild with ebony e^{11} and wild with ebony "B") the experiments were repeated 10 times. The results are shown in Table 1.

Although the frequency of heterogamic matings does not differ between the experiments with ebony e^{11} and with ebony "B", the mating between wild males and ebony females are much more frequent for the strain e^{11}. Such results are in good agreement with the conclusions of Mikheev et al. (1973). An ethological isolation could be established between ebony and its wild allele.

Compared to previous observations, the sexual activity of the wild flies was rather low when they were not in the presence of ebony flies. On the contrary, the sexual activity of both ebony strains was particularly high when they were not in the presence of wild flies. It is all more surprising that, in the presence of ebony flies, the wild males become very active. On the contrary, the ebony males were inhibited by the presence of wild flies. As previously

Table 1. Matings recorded in four hours observation.

	Wild alone	Both strains in competition				Ebony alone
	♂+ ♀+	♂+ ♀+	♂+ ♀e	♂e ♀+	♂e ♀e	♂e ♀e
1.	131	51	80	10	32	163
Total flies	300 pairs wild	150 pairs wild			150 pairs ebony e^{11}	300 pairs ebony e^{11}
2.	127	66	55	17	32	159
Total flies	300 pairs wild	150 pairs wild			150 pairs ebony "B"	300 pairs ebony "B"

mentioned, the ebony females of the strain e^{11} seemed to be preferred by the wild males; consequently, their level of sexual activity is significantly higher when they are in the presence of wild flies. However, the level of sexual activity of the females of the strain "B" is unchanged.

Further analysis will have to show if this initial frustrating inhibition of the ebony males could be compensated by a more constant success in the ulterior phases of the competition, as it could be conjectured from our previous observations (1973) and by analogy with the observations of Jacobs on the dragonflies (1955).

References: Ehrman, L. 1965, Evolution 19:459; Elens, A. and J.M. Wattiaux 1964, DIS 39:118; Elens, A., J. Van den Haute, J. Delcour 1973, Evolution (in press); Jacobs, M.E. 1955, Ecology 36:566; Mikheev, A.V., A.G. Kreslavsky and V.M. Solomatin 1973, Genetika (Russ.) 9:169.

Rapport, E. Simon Fraser University, Burnaby, Canada. On the action of the vital stain 2,2'-Dipyridyl.

A vital stain which colours imaginal discs, brain and ring gland of Drosophila, has been described (Rapport and Menon, 1973). The stain, 2,2-dipyridal (Dip) is known to chelate ferrous ions (Fe^{++}) and form a red product. Indeed this reaction is used quantitatively in Fe^{++} determinations. We wished to determine if the in vivo staining reaction could be used as a measure of high Fe^{++} concentration.

Larvae were grown on either cream of wheat media seeded with yeast or the same media made with water containing 1 mg $FeCl_2$ per cc. Larvae were removed from the media by flotation with NaCl solution at either 65 or 72 hours and placed in petri dishes with paper pulp and either water or a Dip solution. In addition to usual sites of staining the $FeCl_2$ fed larvae treated with Dip at 72 hours had deep red granules in the base of the gastric caecae. By visual inspection we could not detect enhanced colouration of organs which usually stain. These results are at variance with those of Poulson and Bowen (1952) whose histological studies using fixed material revealed a direct relationship between stainability and iron concentration in the media and who never detected ions in the caecae. The pulp in dishes with $FeCl_2$ treated larvae was stained pink apparently due to excretion of Fe^{++} ions by the larvae.

These results suggest that at least some of the stain in vivo could be the result of Fe^{++} localisation.

To further investigate the action of Dip in the living organism the effect of somewhat toxic levels of Dip on pupation ability and ability to evert imaginal structures was studied in the different feeding regimes. Table 1 gives the results of this study. Larval development was impeded by Dip treatment and this effect could be reversed to some extent by prior

Age removed from feeding	Treatment	Number of organisms	% failing to pupate	95% confidence interval	% pupae failing to evert heads	95% confidence interval
65	H_2O	153	2.0	0 - 4.2	1.3	0 - 3.2
65	Dip	225	89.3	85.5 - 93.1	18.5	3.8 - 33.2
65	Dip + $FeCl_2$	184	67.9	61.2 - 74.7	23.7	12.9 - 34.6
72	H_2O	214	1.9	0.1 - 3.7	0.5	0 - 1.4
72	Dip	218	55.0	48.4 - 61.7	33.7	24.3 - 43.0
72	Dip + $FeCl_2$	208	26.0	20.0 - 32.0	31.2	23.8 - 38.5

feeding with $FeCl_2$. Among the pupae, head eversion was not appreciably influenced by $FeCl_2$ feeding. These results weakly suggest that preventing Dip from chelating with normal cell constituents (by addition of exogenous ions) reduces its toxicity.

Clearly more work needs to be done to determine the substance(s) to which Dip binds in vivo. While detection of stain in gastric caecae only after $FeCl_2$ feeding suggests that Dip will bind to intracellular iron, the failure of our staining results to confirm those of Poulson and Bowen indicates that more work must be done to determine if this stain faithfully reflects ferrous ion concentration in Drosophila organs.

References: Poulson, D.F. and V.T. Bowen 1952, Exp. Cell Res. (Suppl.) 2:161-179; Rapport, E. and M. Menon 1973, Experientia 29:734-735.

McCrady, W.B. University of Texas at Arlington. Search for variation in response to CO_2 in wild-caught Drosophila melanogaster.

Collections of D. melanogaster were completed during the latter part of June and the first part of July, 1973, at three sites in Dallas and one in Arlington, Texas. A total of 564 strains were developed from isolated wild-caught females. Progenies were reared at 19-20°C to prevent temperature curing of CO_2 sensitivity symptoms and were first screened for any abnormal response to CO_2 by exposure to pure carbon dioxide at 19-20°C. A total of eighty (14.2%) of the strains tested showed departures from control (Oregon) resistant flies in proportion and/or time required for recovery.

For further screening, 72 of the above strains were treated with CO_2 for fifteen minutes at 9°C and their recovery behavior was recorded. Twenty of these strains showed departures in response to CO_2 from that observed in resistant flies. In each of the seven strains shown on the left in Table 1 over 30% of the flies were killed by CO_2 treatment; no recovery occurred later than fifteen minutes following exposure as is typical for classical sensitivity caused by virus sigma. Two strains (80 and 210) seem to exhibit stabilized CO_2 sensitivity and will require no selection. Selection will be carried out in the other five strains in an attempt to establish new stabilized lines.

Table 1. % recovery following CO_2 treatment at 9°C for 15 minutes.

Strain	n	15 min	30 min	1 hr	2 hrs
7	150	50%	50%	-	-
80	52	0%	0%	0%	0%
101	39	18%	18%	18%	18%
210	16	0%	0%	0%	0%
375	42	14%	14%	14%	14%
379	18	56%	56%	-	-
381	18	67%	67%	-	-

Strain	n	15 min	30 min
18	100	70%	85%
19	100	80%	90%
20	150	73%	83%
37	150	83%	87%
41	125	84%	88%
71	70	93%	86%
72	90	83%	94%
74	140	86%	93%
84	70	94%	94%
106	90	94%	96%
257	33	88%	94%
469	21	76%	90%
481	20	80%	85%
Oregon	75	100%	100%

If it is found that these seven strains are the only ones with classical CO_2 sensitivity, the proportion (1.24%) is slightly lower than that determined by Williamson (1961) for wild-caught D. melanogaster in Nebraska. He found 1.6% sensitives in 6,300 flies tested. However, interesting departures from resistant type behavior were observed in thirteen other strains as shown on the right side of the table. In two of these (strains 71 and 84) less than 100% recovery occurred during the first 15 minutes following CO_2 exposure and no recovery occurred later; in fact, fewer flies were able to stand after an additional 15 minutes in strain 71. These may be non-stabilized strains. Recovery behavior in the other strains suggests delayed-recovery as described by McCrady and Sulerud (1964). In all cases recovery was slower than for control flies and less than 100% had recovered within 15 minutes. Furthermore, additional recovery was observed during the next 15 minutes. Some of these flies may be homozygous or heterozygous for gene Dly, the determiner of delayed-recovery. Testing of this hypothesis is now being attempted.

At the present time only two delayed-recovery stocks exist, TDR (Texas Delayed Recovery) and TDR-B. The latter stock has only recently been developed by selective breeding and testing for Dly, although the strain was started with a female collected at the same time and site (Pittsburg, Texas in 1959) as the originator of TDR. Delayed-recovery in TDR-B has been shown to be determined by a gene apparently identical in function and location to that responsible for the delayed-recovery phenomenon in TDR. It appears evident that the two females were a part of the same gene pool. Therefore, the establishment of delayed-recovery strains from other areas seems desirable and will be attempted by selection within the aberrant stocks now available. Investigation of the causative mechanism of delayed-recovery in different strains could be very informative in illumination of the broad question of the relationship

between gene Dly and virus sigma.

References: McCrady, W.B. and R.L. Sulerud 1964, Genetics 50:509-526; Williamson, D.L. 1961, Genetics 46:1053-1060.

Gold, J.R. and M.M. Green. University of California, Davis, California. mu - a mutator gene in Drosophila melanogaster.

In two previous reports, an apparently new mutator gene, mu, in D. melanogaster was identified and genetically characterized (Green, 1970; Green and Lefevre, 1972). In these reports, it was shown that mu significantly increases the reversions of the sex-linked mutants y^2 and f^{3N} to their respective wildtype alleles, and the frequency of sex-linked lethal mutations in homozygous mu females.

In addition to the frequent reversions of y^2 and f^{3N}, several other visible mutations have been recovered from experiments using single P_1 homozygous mu females. Some of these "forward" mutations are listed in Table 1, and are presented to demonstrate the influence on spontaneous mutability of the mutator gene. Most of the newly recovered mutations were progeny tested to determine the origin, i.e. somatic or germinal. Multiple events or clusters

Table 1

Forward visible mutations recovered from experiments using homozygous mu females.

No	Phenotype of the mutation	Number of occurences	Somatic or germinal**
1	achaete	2	?
2	bithorax-like	1	S
3	Beadex	1	S
4	Blistery wing	1	S
5	bulgeing eye (extreme)	1	G
6	cut wing	2*	G
7	Delta wing	4*	G
8	Dicheate-like	1	S
9	Hairless	5*	?
10	hairy eye (extreme)	10	S
11	held-out wing	1	G
12	Lobe or reduced eye	many	S
13	lozenge spectacle	1	sterile
14	Minute	12*	S,G
15	Notch	16*	G
16	roughened eye	6*	G
17	scute	2	S
18	zeste eye color	1	sterile
19	Ultrabithorax-like	2	S
20	several bristle irregularities	many	-
21	several eye shape mutations	many	-
22	several synanders (mitotic loss)	many	-

* Recovered as clustered events

** Somatic - S (not recovered in F_1 progeny tests)
Germinal - G (recovered in F_1 progeny tests)

from single P_1 females were found in several instances and are noted in the Table. Three conclusions can be drawn from the results: 1) mu induced mutability is not gene or allele specific; 2) mu induced mutations occur in both somatic and germinal cells; 3) at least some of the mu induced mutations occur premeiotically as evidenced by the clustered mutations. All three observations were made previously and are extended by the observations reported here.

References: Green, M.M. 1970, Mutation Res. 10:353-363; Green, M.M. and G. Lefevre, Jr. 1972, Mutation Res. 16:59-64.

Marques, E.J. and L.E. de Magalhães.
University of Mato Grosso and University of São Paulo, Brazil. The Frequency of SR-female of Drosophila nebulosa in a natural population.

Four samples of D. nebulosa collected in nature near Campo Grande, State of Mato Grosso, Brasil were analysed to detect the occurrence of SR-females. The results observed were the following:

Date	No. of ♀♀ collected	No. ♀♀ "SR"	%
1/18/73	206	13	6.31
4/12/73	259	9	3.47
5/24/73	65	0	-
6/ 7/73	99	6	6.06

It was found that the SR-condition was due to the presence of Treponema as described by Poulson and Sakaguchi, 1961.

Reference: Poulson, D.F. and B. Sakaguchi 1961, Science 133:1489-90.

Supported by grants from FAPESP.

Kirschbaum, W.F. and R.L. Cabrini.
Comisión Naçional de Energía Atómica, Buenos Aires, Argentina. Lineal microphotometric scanning of Drosophila melanogaster salivary gland chromosomes.

Considering that the microphotometry could be a contribution to the cytogenetic analysis of polytenic chromosomes, several tests have been made under different measurement conditions.

Two systems have been used, direct microphotometry and microdensitometry of the photographic image. In both cases equipment consisting of a Zeiss Photomicroscope I, a Zeiss Photometerhead, a Zeiss case with an R.C.A. Photomultiplier model 1P28 and a Zeiss monochromator model M4GII, using monochromated light of 560 m μ, was used. A lineal scanning of the chromosomes was performed following the longitudinal axis of the chromosomes and of the photography of the chromosome according to the technique of Cabrini, R.L. et al. (Acta histochem. 36, 399:403, 1970).

In direct microphotometry a Feulgen stain of the chromosomes was used, because this is a permanent stain and allows a stequiometric determination of the chromosomes DNA. The chromosome staining technique was used with the modification of the hydrolysis at room temperature with 5N HCl, because better results were obtained with it (DIS 50, 1973). For the photographic method the chromosomes stained with Feulgen have been photographed with positive Ferrania film of 36 mm, using therefore the same optics of the same microscope used for the direct microphotometry.

For comparative purposes always the same piece of the X-chromosome was taken for the direct microphotometry and this same piece photographed for the determination of the photographic densities.

Testing with direct microphotometry we have seen that the best resolution was obtained with a great optic magnification associated with the least possible measurement diaphram, using a field diaphragm that does not surpass the surface illumination of the measurement diaphragm.

With direct microphotometry, the maximum magnification of the optic microscope was used. One could not diminish the diaphragms of measurements and of field to a considered optimum, because of the limiting factor of quantity of light.

The second method, obtention of the optical density of photographies was measured using the same equipment as for the direct microphotometry.

Comparing both methods, the analysis of the photographs gives a superior resolution of the number of bands than direct microphotometry. On the other hand, the direct microphotometry is the best method of giving a quantification of the DNA of the chromosomes.

In pilot tests both methods demonstrated sufficient reproducibility and there one could think of using them in routine analysis of polytene chromosomes giving objective data in a way that eliminates the personal factor of observation.

The possibility of obtaining absolute or relative numeric data of the distribution of DNA in these chromosomes, shows that computation methods may be used for this kind of analysis.

Moree, R., K.R. Baumann and M.B. Durtschi
Washington State University, Pullman.

Rapid population experiments with D. melanogaster.

Experimental populations which run their course fairly quickly, provide a comparison of the relative fitnesses of different wild type strains, and offer interesting possibilities for the study of stable polymorphisms, can be initiated by invading balanced lethal populations with various wild types. In the present study two types of populations, each run in duplicate, were as follows: 80% SM1/Tg invaded by 20% +Wawawai and 80% SM1/Tg invaded by 20% +Canton-S. Adults were placed in half pint culture bottles and removed after one week. At the end of week-2 their progeny were removed, counted and discarded. At the end of week-3 all emerged adults were transferred to a fresh culture bottle and at the end of week-4 they in turn were removed, counted and discarded and the cycle repeated (see Figures, for which time is given in weeks). Thus whole population counts were made on an approximate per generation basis, i.e. every two weeks. This technique was first suggested to me a number of years ago by David Suzuki.

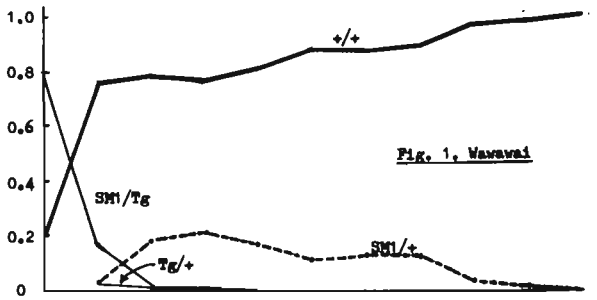


Fig. 1. Wawawai

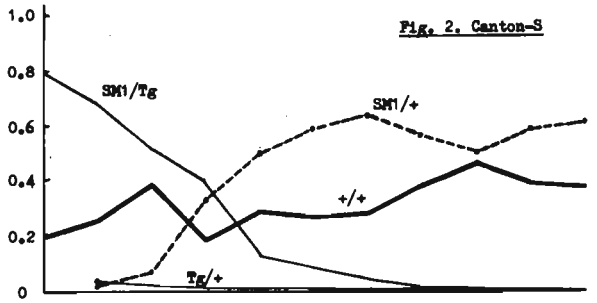


Fig. 2. Canton-S

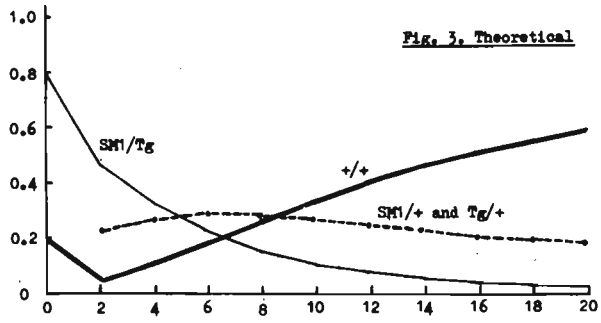


Fig. 3. Theoretical

The results, based on pooled duplicates, are shown for the Wawawai series (Figure 1) and for the Canton-S series (Figure 2), and are obviously quite different. The Wawawai strain, which took over very rapidly, was collected in 1964; the Canton-S strain is of course much older. Both may be compared with corresponding theoretical curves (Figure 3) calculated on the assumption of panmixis, discrete generations, and unity fitness for all nonlethal types. Early changes in the experimental populations were strongly influenced by recombination, though clearly not wholly so. Tg/+, after its first appearance, never reached anything like the frequency of SM1/+. SM1/+ only attained stability in competition with Canton-S wild type (+/+), but did so at a frequency actually higher than this particular wild type (Canton-S). Differences between theory and experiment yet to be clarified are that at week-2 no dip in the experimental frequency of wild type occurred and that initial frequencies of SM1/+ and Tg/+ were lower than the theoretically calculated. Average adult population size per culture for the Canton-S series was 547 flies and for the Wawawai series 360. The difference appears to be chiefly due to the superior fitness of SM1/+ in the Canton-S series.

Nirmala Sajjan, S. and N.B. Krishnamurthy
University of Mysore, Manasagangotri, India.

Erratum in previous note.

DIS 49:60 (1972), D. albomicans - a race of D. nasuta. Erratum: the first part of paragraph three is incomplete. It should read: D. albomicans (Duda 1923) is morphologically similar to D. nasuta, but differs cytologically from it (Sajjan and Krishnamurthy 1971). Both of them are highly polymorphic and even share some inversions (personal communication by Wilson 1971). D. albomicans was recognized as a biologically valid species by Wilson et al. (1969) only because of the difference encountered in the karyotype.

Kambysellis, M.P. New York University, New York. Ultrastructure of the chorion in Drosophila species.

During the last stages of egg maturation the follicle cells synthesize and lay around the oocyte a protective shell, the chorion. This complex structure consists of lipids, carbohydrates and several proteins (King and Koch, 1963; Paul et al., 1972) and exhibits an intricate morphology when viewed under a scanning electron microscope (Figure 1).

The general chorionic pattern in more than 20 species analyzed was found to be the same, a network of hexagons. Significant differences in the size and shape of the hexagons were found among the species (Figures 1.2, .5, .7, .8) and also between different positions in the same egg (Figures 1.7, .11). Each hexagon represents the imprint of a follicle cell. The rims, which correspond to the borders of the follicle cells, are in relief, augmented by continued secretion, while the center of the cell remains concave with numerous pores. The thickness and shape of the hexagonal rims and the number and size of the pores differ among species, and at different locations on the same egg. We suspect, on the basis of preliminary data from a correlated ecological and morphological study, that the diversity of the pore size is correlated with the respiratory needs of the developing embryo (Hinton, 1960). The process of chorion formation during oocyte maturation is now under investigation. (Supported by NSF Research Grants GB-29288 and GB-34168 and Arts and Sciences Institutional Grant.)

References: King, R.C. and E.A. Koch 1963, Quart. J. Micr. Sci. 104:297; Paul, M., M.R. Goldsmith, J.R. Hunsley and F.C. Kafatos 1972, J. Cell Biol. 55:653; Hinton, H.E. 1960, J. Insect Physiol. 4:176.

Figure 1 (next page) Representative scanning electron micrographs of mature Drosophila oocytes:
.1-.3 D. mimica (140, 3,000 and 10,000 x)
.4-.6 D. gymnobasis (300, 3,000 and 10,000 x)
.7 D. virilis dorsal view (3,000 x)
.8-.9 D. melanogaster (3,000 and 10,000 x)
.10-.12 D. virilis microphyle triangular (300, 3,000 and 10,000 x)
All photographs were taken with a Jelco model JSM-U3 scanning electron microscope. The specimens were fixed in 3% glutaraldehyde and coated with 60% gold - 40% paladium in a model Jee-4C vacuum evaporator.

Breugel, F.M.A. van. University of Leiden, The Netherlands. Some new phenes of lethal tumorous larvae and adult characteristics of some "Durchbrenner".

An array of larval characteristics of the late larval lethal mutant ltl (lethal tumorous larvae) have been described by Kobel and van Breugel (1967) and recently by Zhimulev and Lytchev (1972). At the moment sampling of lethal larvae in our ltl/TM_3 strain is facilitated because ltl larvae show rather early in development a heavily bloated caudal end, more pronounced than it seemed six years ago. The stock is still perfectly balanced and in normal cultures only Serrate (ltl/TM_3) flies emerge. However, from 25°C subcultures some "Durchbrenner" may arise if larvae with bloated caudal ends are isolated and placed on fresh standard food. The (pseudo)pupae that eventually arise may show some degree of imaginal differentiation or even a complete imago that emerges. The flies thus obtained can be easily divided in a male and a female class because of their size, pigmentation and sexcomb, but usually they lack outward genitalia. Some of the flies showed extra scutellar bristles, but this happens to occur also in sibs. In contrast to their sibs, the exceptional flies have normal wings, so are most likely indeed ltl homozygotes. Out of 181 assumed ltl larvae emerged 24 flies all with wildtype wings. Of them, 8 males and 8 females completely lacked outward genital structures, 3 more of both sexes had abnormal genitalia and 1 male and 1 female looked normal but proved to be sterile. Some of the genitalless flies showed rudiments of internal gonads after dissection. In one male the outward genitalia hang on the inside of the abdomen. It seems reasonable to accept, that there is a correlation between the bloated caudal structure of the larval body and the lack or abnormal outgrowth of the outward genitalia of the adult.

References: Kobel, H.R. and F.M.A. van Breugel 1967, Genetica 38:305-327; Zhimulev, I.F. and V.A. Lytchev 1972, DIS 48:49.

Kambysellis, M.P., Ultrastructure of the chorion in Drosophila species: legend preceding page.

Narise, S. Josai University, Sakado, Saitama, Japan. Substrate specificity of α- and β-esterase isozymes of D. virilis.

In gel electrophoretic techniques, non-biological substances, such as naphthyl derivatives, have been usually used as substrates of esterases. Purified α-esterases of D. virilis hydrolyze α- and β-naphthyl acetates at a similar rate, while β-esterases are active only on β-form. Neither esterases act on naphthylesters of long carbon chain fatty acids (Narise, S. 1973).

Activity of α- and β-esterase with aliphatic esters and glycerides

	α^3	β^A
Methyl acetate	0.0	35.1
Methyl propionate	12.6	193.7
Ethyl butyrate	50.2	71.2
Ethyl valerate	119.8	62.9
Ethyl caproate	55.5	14.8
Ethyl caprylate	28.4	11.9
Ethyl laurate	0.0	0.0
Ethyl stearate	0.0	--
Monoacetin	8.6	65.2
Monopropionin	47.2	146.7
Monobutyrin	--	--
Monolaurin	17.7	24.3
Monostearin	0.0	0.0
Triacetin	71.4	76.0
Tripropionin	139.4	214.1
Tributyrin	128.5	78.5
Trilaurin	0.6	0.3
Tristearin	0.0	0.3
α-naphthyl acetate	14.3	12.4
β-naphthyl acetate	17.4	27.1

The present work aims to get information on substrate or substrates in vivo of the esterase controlled by α- and β-esterase loci. Partially purified isozymes from homozygous stocks of each allele were used. Although the specific activity of these enzymes was about ten fold that of the crude extract, these samples were almost free from other esterases except that controlled by these loci. Esterase activities were assayed by the manometric technique at pH 7.4 with a 5% CO_2-95% N_2 mixture in the gas space. Substrates were added at a final concentration of 2.5×10^{-2}M. Specific activity was given as microliter of CO_2 produced per 30 min per mg of protein.

The activities of α^3 and β^A are shown in the Table. As presented in the Table, both enzymes attacked more actively methyl or ethyl esters of fatty acids having C^3 to C^8 than the naphthyl acetates. With the valeric acid ester as a substrate, the activity of α-esterase was highest, while that of β-esterase was best with the propionic acid ester. These two esterases also hydrolyzed more actively the lower mono- and tri-glycerides than the naphthyl acetates. However, they did not act on lauryl and stearyl esters except monolaurin. No significant difference was found in the enzyme activities among isoallelic isozymes.

Boström, G. and L.R. Nilson. University of Uppsala, Sweden. On tetrazoliumoxidases in Drosophila melanogaster.

Tetrazoliumoxidase isoenzymes in Drosophila melanogaster were first described and mapped by Jelnes, 1971. Starch gel electrophoresis of 19 wildtype stocks from our laboratory revealed that all strains except one were homozygous for the fast mobility allele To^F. The only strain homozygous for the slow allele To^S was Gruta. The strains homozygous for the To^F allele were Algeria, Amherst-3, Boa Esperanca, Canton-S, Crimea, Curitiba, Formosa, Hikone-R, Karsnäs, Kochi-R, Oregon-R, Salvador, San Miguel, Stäket, Tunnelgatan, Ultuna, Örebro and Knivsta.

Homogenates from Gruta (To^S/To^S) and Hikon-R (To^F/To^F) were subjected to iso-electric focusing (LKB 8100 Ampholine Electrofocusing Equipment) in a 100 ml column. The density gradient was formed by sucrose and the carrier ampholyte used was LKB Ampholine Ampholyte with pH-range 3-10. After focusing the column was bled in fractions of 1 ml with pH determination of each fraction. The carrier ampholyte was separated from the proteins by gel-filtration (Sephadex G-25) and starch gel electrophoresis was performed after concentration of the protein fractions. The isoenzyme pattern of To^S/To^S showed activity in the fraction of pH 4.5 and the To^F/To^F band was found at pH 4.8.

Tetrazoliumoxidases from mammals have been reported to catalyze the NADI-reaction and the oxidation of dichloroindophenol. The tetrazoliumoxidases from the strains studied by us did not catalyze the oxidation of dichloroindophenol. The NADI-reaction was more obscure (one, not reproducible, positive staining).

The isoenzymes did not loose their tetrazoliumoxidase-activity after incubation at 50°C for 45 min.

Berg, R.L., U.S.S.R. 190121, Leningrad F-121, pr. Maklina 1, kv.6. A further study of the rate of "abnormal abdomen" (aa) in geographically isolated D. melanogaster populations.

A simultaneous rise in the rate of abdominal segmentation abnormalities designated as abnormal abdomen (aa) was observed in geographically isolated populations of D. melanogaster in 1968 and especially in 1969 (Berg 1972a). Segregations in the progeny of hundreds of aa males and females individually crossed with non-aa flies proved the predominant role of heredity in the etiology of this fly disease (Berg 1972b). The hypothesis was put forward that the selective values of mutant genes responsible for the abnormality is changing on a global scale. Investigation of 12 populations during 1970 and 1971 substantiated this view, since enormously high rates of aa were observed in each of the twelve populations. In 1972 the study of the phenotypical polymorphism in some of the previously studied populations was repeated and several "new" populations investigated (Table 1). The hypothesis of the global increase of aa rates is substantiated by the high frequency of the aa phenotype observed in 1972 in 13 populations. During the last five years 20 populations were studied and the aa phenotype was abundant in all of them.

References: Berg, R.L. 1972a DIS 48:67-69; Berg, R.L. 1972b DIS 48:67.

Table 1

Concentration of abnormal abdomen phenotype in geographically isolated populations of Drosophila melanogaster in 1972

Population		Collection date	Males			Females		
			n	number	%	n	number	%
Moscow District	Kashire	11-13.IX	1297	70	5.4	549	56	10.2
Transcarpathians	Goriani*	18.IX	1115	80	7.9	402	139	34.6
	Khust*	22.IX	790	73	9.2	879	341	38.8
Crimea	Nikita Botanical Gardens (Magarach)	28.IX	749	64	8.6	541	233	43.1
Kuban	Gelendzhik*	6.X	665	96	14.4	878	442	50.3
Transcaucasus	Kutaissi	14.X	333	36	10.8	683	268	39.2
	Dilizhan	18.X	274	90	32.5	288	130	45.1
	Erevan	19.X	504	73	14.5	500	275	55.0
	Burakhan	21.X	382	83	21.7	302	144	47.7
Middle Asia	Alma-Ata conserve factory	9.XI	378	47	12.4	543	280	51.5
	Alma-Ata winery	10.XI	517	62	12.0	458	266	58.1
	Frunze	30.X	628	83	13.2	492	193	39.2
	Przhevalsk*	4.XI	445	37	8.3	462	130	28.1
	TOTALS	11.IX-10.XI	8077	894	11.1	6977	2897	41.6

Populations designated * were studied for the first time.

Garcia-Bellido, A and P. Ripoll. Centro de Investigaciones Biológicas, C.S.I.C., Madrid, Spain. A mwh^+ duplication on the tip of the first chromosome.

Three independent Dp(1:Y:3)M1-3 have been recovered by X-radiation (3000r) of premeiotic stages of the spermatogenesis in T(Y:3)P6, mwh^+ ve^+ (62 A-B, Lewis 1972, DIS 48:188)/sc^8 Y^S X, y^+ KS y cv v f;mwh ve males. Genetically, the duplication mwh^+ on the tip of Y^S X chromosome locates distal to KS factors. Dp(1:Y:3)M males are viable and fertile over a Y^L chromosome. Homozygous females though, appear to be lethal. The allele mwh^+ of the Dp does not variegate in nullo-Y males.

Basden, E.B. Institute of Animal Genetics, Edinburgh, Scotland.

Some early Morgan specimens.

In the Royal Scottish Museum, Edinburgh, are 50 specimens, entered under No. 55, 1 to 12, for July 22, 1922 as follows:

"Series of specimens of Drosophila melanogaster Mg., of the original varieties bred (bred from original sets used erased) by Professor T.H. Morgan in his experiments on heredity. Presented by J.H. Ashworth, Zoological Department, University of Edinburgh. Spineless bithorax 16 specimens (9♂7♀); Plexus brown speck 6(3♂3♀); Lm.5. (15 on label) Haplo IV. 4(2♂2♀); Spineless glass. III. 2(2♀); Ebony 4. III. 3(1♂2♀); Dichaete hairless. 3(3♀); Black vestigial. 2(1♂1♀); Eosin miniature I 1(♂); Forked Bar I 4(1♂3♀); Yellow White I 3(1♂2♀); Vermilion I. 3(3♀); L.7.5/20 3(1♂2♀)".

The specimens are on micro-pins on papered cork mounts and are labelled in the same hand that made the above entry in the Museum's accession book. Empty pin-holes on all mounts suggest that more specimens were originally present. The number of specimens still there tallies with the numbers recorded above, to which I have added the sexes in brackets.

They agree with the usual descriptions of the respective mutants except:

plexus brown speck - speck is not apparant.

lm haplo-4 - no obvious abnormalities, no trident.

spineless glass - postscutellars not erect.

Dichaete Hairless - heads not deformed, veins not shortened.

vermilion I - eyes wild-type in colour.

Of L.7.5/20, which I cannot properly locate, 1♂1♀ are very pale, including eyes, veins and bristles, but 1♀ is darker.

It is surprising that so few "type" specimens of mutants are preserved for posterity. If others are known it is hoped they will be recorded.

Thanks are due to E.C. Pelham-Clinton for access to the specimens.

Mason, J.M. University of Washington, Seattle, Washington.

A relationship between daughterless and the Y-chromosome.

daughterless (da) is a recessive on chromosome 2 in D. melanogaster which, when homozygous in female parents, results in the production of 100% male progeny regardless of the genotype of the male parent. The abnormal sex ratio is due to differential zygote mortality (Bell, 1954). Sandler (1972) has shown that heterozygous da females produce homozygous da progeny whose viability depends upon the amount of sex chromosome heterochromatin either in the mother or in her offspring. In order to ask whether this relationship also applies to the viability of sons of homozygous da mothers, the following experiment was performed. Females with normal X-chromosomes and either da/da or +/+ were mated to males that were either X/Y; +/+ or $\overline{XY}$/O; +/+. The results were:

mating	number of eggs	progeny		male survival
		female	male	
X/X;da/da x X/Y	2613	0	954	0.90
X/X;+/+ x X/Y	1034	465	415	
X/X;da/da x $\overline{XY}$/O	2299	0	685	0.66
X/X;+/+ x $\overline{XY}$/O	2029	898	922	

In the mating to X/Y males, the male survival is 0.90 where survival is defined as the ratio of adult male progeny to eggs in the da cross divided by that ratio in the control cross. In the mating to $\overline{XY}$/O, the male survival is 0.66. These data show that X/O males produced by da/da mothers do indeed survive less well than X/Y males produced by such mothers.

Two additional points are worth noting. First, in this series of tests da females produced 5050 eggs while + females produced 4812 eggs under the same conditions. Second, if the male survival of 0.90 is different from 1.00 and due to da, then while only male progeny from homozygous da females survive, only a fraction of them do.

References: Bell, A.E. 1954, Genetics 39:958-959; Sandler, L. 1972, Genetics 70:261-274.

Beck, H. University of Geneva, Switzerland. Some observations on Y-chromosomal bobbed mutants in D. hydei.

In D. hydei bb mutants are abundant in natural populations (Spencer, 1944) and many laboratory strains can be shown to carry bb alleles of varying strength on their X-chromosomes when crossed to a suitable bb-tester stock. Although the Y-chromosome in this species carries a number of rRNA genes similar to that on the X-chromosome (Hennig, 1968) no bb mutants of the Y-chromosome have been recorded. To advance studies of bb in D. hydei, it seemed desirable to look for such mutants. For this purpose wild type ♂♂ (stock Madeira) irradiated with 5000 or 6000 rad were crossed to ♀♀ homozygous for a strong bb allele and a number of recessive markers on the X-chromosome. 320 ♂♂ (2.13%) out of a total of 15002 ♂♂ from the two experiments showed either a reduction of bristle size or abnormal abdomen (aa). Out of 273 bb phenotypes tested, only 17 (6.22%) were fertile and two among them were bb mutants of the Y-chromosome. The remainder of the fertile bb phenotypes are dominant mutations resembling the Minutes in D. melanogaster.

The two mutant Y-chromosomes, named Y^{bb^d} and Y^{bb^k}, each are maintained in two stocks of the following composition: w, lt•=/Y^{bb};bb^+/Y^{bb}, and bb^+/bb^+;bb^+/Y^{bb}, as a safeguard against eventual magnification phenomena (Ritossa and Scala, 1969). To study the effects of these Y-chromosomes, ♂♂ from bb stocks of different origin were crossed to $\overline{XX}$ ♀♀ carrying Y^{bb^d} or Y^{bb^k}. Bristle indices of the resulting F_1 ♂♂ and of the different X^{bb}/X^{bb} ♀♀ shown in Figure 1 were measured as described earlier (Beck, 1972). With the exception of X^{bb^R}/Y^{bb} and, less pronounced, of $X^{bb^{19}}/Y^{bb}$ the effects of the two Y chromosomes on bristle length are similar or identical and apparently are not additive; bb^1/Y^{bb^k} for example has the same index as bb^8/Y^{bb^k}, although bb^8 in homozygous females produces only a weak bb phenotype. In ♀♀, on the other hand, perfect additivity of bristle length has been found in a number of crosses between strains bb^4, bb^9, bb^{11} and bb^{19}. Bristle length measurements alone may not be sufficient in our ♂♂ combinations to predict the rDNA content of a given genotype from the known values of some genotypes at the extremes of the scale of phenotypes as proposed by Weinmann (1972). If other components of the pleiotropic pattern of bb, such as fertility and aa effects, are considered, the differences between the two Y chromosomes become more apparent (Table 1). In

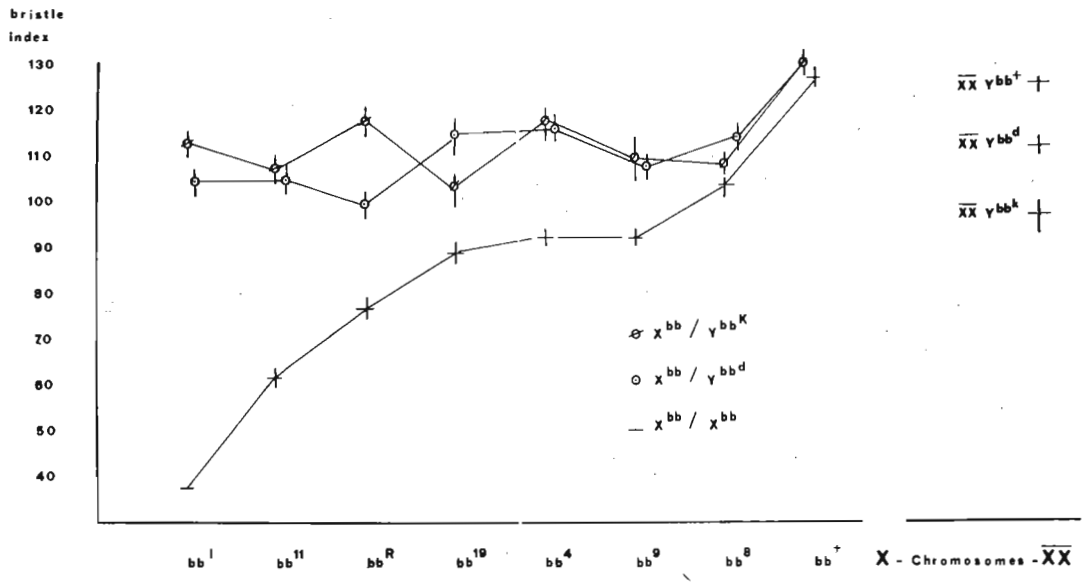


Figure 1. Relative bristle length of different bb genotypes based on measurement of 25 ♂♂ or 50 ♀♀. Confidence intervals are indicated by vertical bars. bb^1 is a lethal bb, the value given is calculated from a viable combination bb^1/bb^{A1}.

all cases where the homozygous ♀♀ and bb^1/bb^1 show aa effects, the corresponding ♂♂ carrying Y^{bb^k} are sterile or nearly so at 25° while all combinations with Y^{bb^d} are fertile and do not show the aa effects associated with Y^{bb^1}/Y^{bb^k} and $X^{bb^{11}}/Y^{bb^k}$. That bb^k is the stronger bb

allele can also be shown by comparing $\overline{XX}$ ♀♀ carrying bb^k or bb^d (Figure 1). $\overline{XX}$ Y^{bbk} produces a distinct reduction of bristle length and a strong aa effect while in $\overline{XX}$ Y^{bbd} only bristle length is slightly affected.

The $\overline{XX}$ strain used, carries no X-chromosomal rDNA (Hennig, 1968), and it is therefore expected that $\overline{XX}$ Y^{bb} should be phenotypically bb. This observation has been extended to a number of $\overline{XX}$ strains of independent origin. In all 9 cases examined, the $\overline{XX}$ Y^{bbk} combinations show similar bristle length and strong aa effects. It seems therefore that the formation of $\overline{XX}$ in D. hydei is generally accompanied by a complete loss of rRNA genes. This supports cyto-

bb allele	1 aa effect in X/X ♀♀	2 aa effect in ♂♂ bb^k	2 aa effect in ♂♂ bb^d	3 Fertility of ♂♂ bb^k	3 Fertility of ♂♂ bb^d
bb^1	*++++	++	-	-	+++
bb^{11}	+++	+	-	-	+++
bb^R	+++	-	-	+	+++
bb^{19}	-	-	-	+++	+++
bb^4	-	-	-	+++	+++
bb^9	++	-	-	-	+++
bb^8	-	-	-	+++	+++

Table 1. Intensity of abnormal abdomen (aa) effect in ♀♀ of different bb strains (1) and aa effect and fertility of different X/Y^{bb} genotypes (2 and 3). Abnormal abdomen effect is a rough estimate based on several inspections. Sterility of ♂♂ was observed in crosses XX Y^{bb} x X^{bb}/Y^{bb}.

* Estimate from ♀♀ bb^1/bb^{11}.

logical observations (Van Breugel, 1970) locating the X-chromosomal nucleolar organizer-region in the distal part of the heterochromatic arm.

For a better understanding of the effects of the Y^{bb} mutants described, it will be necessary to know their rDNA content and rate of rRNA synthesis. Such studies are currently in progress.

References cited: Beck, H. 1972, DIS 49:76; Breugel, F.M.A. van 1970, Genetica 41: 589-625; Hennig, W. 1968, J. Mol. Biol. 38:227-239; Ritossa, F.M. and G. Scala 1969, Genetics Suppl. 61:305-317; Spencer, W.P. 1944, Genetics 29:520-536; Weinmann, R. 1972, Genetics 72:267-276.

Supported by Fonds National Suisse de la recherche scientifique, Grant 3.224.69.

Imberski, R.B. and M. Olds. University of Maryland, College Park. Electrophoretic analysis of alcohol dehydrogenase and octanol dehydrogenase in Drosophila hydei and related species.

Using the agar gel and formazan staining system of Ursprung and Leone (1965) we have, to date, examined single fly homogenates of thirty strains of D. hydei and one strain each of D. neohydei, D. eohydei, and D. mercatorum for possible electrophoretic variants of alcohol dehydrogenase (ADH) and octanol dehydrogenase (ODH).

For all strains and species we observe two (possibly three) clustered bands of ADH activity and one band of ODH activity. This pattern is similar to "ADHII, ODHII" in D. melanogaster (Ursprung and Leone, 1965) except for the degree of mobility in the cathodal direction. All thirty hydei strains show identical patterns with the ADH cluster and ODH single band migrating to slightly more cathodal positions than in melanogaster. In neohydei and eohydei the ADH has the same mobility as in hydei, but the ODH is shifted even further toward the cathode. The mobility of ODH in mercatorum is identical to neohydei and eohydei, but ADH is at the most cathodal position of any of the species examined.

Reference: Ursprung, H. and J. Leone 1965, J. Exp. Zool. 160:147-154.

Supported by a grant from the General Research Board of the University of Maryland.

Zhimulev, I.F. Institute of Cytology and Genetics, Novosibirsk, U.S.S.R.

Comparison of puffing patterns in proximal and distal parts of the salivary gland in D. melanogaster.

The salivary gland of Drosophila consists of two parts: the distal part where the mucoprotein secretion is synthesized and the proximal part where this secretion is absent (Berendes, 1965; von Gaudecker, 1972).

To study the puffs of the proximal part of the salivary glands, larvae of the Batumi-L stock were used at an age of 110, 115 and 118 hours after oviposition. The methods used were described earlier (Zhimulev, in press). Figure 1 is a diagrammatic representation of the differences in puffing patterns observed between the proximal and distal parts. Series of photographs also demonstrate these differences (Figures 2 and 3). A single specific puff ($61F_3$) was found in the proximal part (Figure 2) and only one very

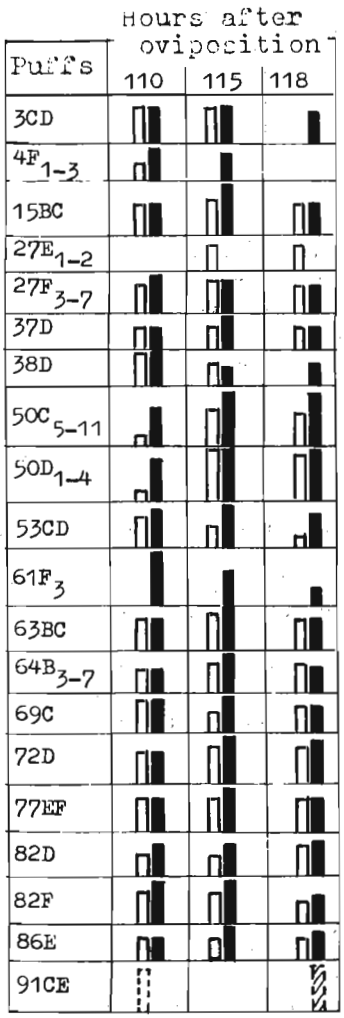


Figure 1. Histogram showing puffing differences between proximal and distal parts of the salivary gland. Height of bars gives the average puff size; black bars represent proximal part, white bars distal part, dotted bars irregularly occurring puffs.

small puff ($27E_{1-2}$) was observed in the distal part. Furthermore 18 puffs differ in size and in the time of appearance Figures 1-3). In the other 294 regions puffing pattern of the proximal part was similar to the one observed in the distal part of larvae at 110-118 hours (Zhimulev, in press).

Thus, during the last hours of larval development, when mucoprotein secretion appears in the cells, no specialized puffing was found in the distal part as compared with the proximal one.

References: Berendes, H.D. 1965, Chromosoma 17:35-77; von Gaudecker, B. 1972, Z. Zellforsch. 127:50-86.

See Figure 3 on next page.

Figure 2. Puffing differences between two parts of gland. a) proximal part, 110 hrs. b) proximal part, 115 hrs. c) distal part, 110 hrs.

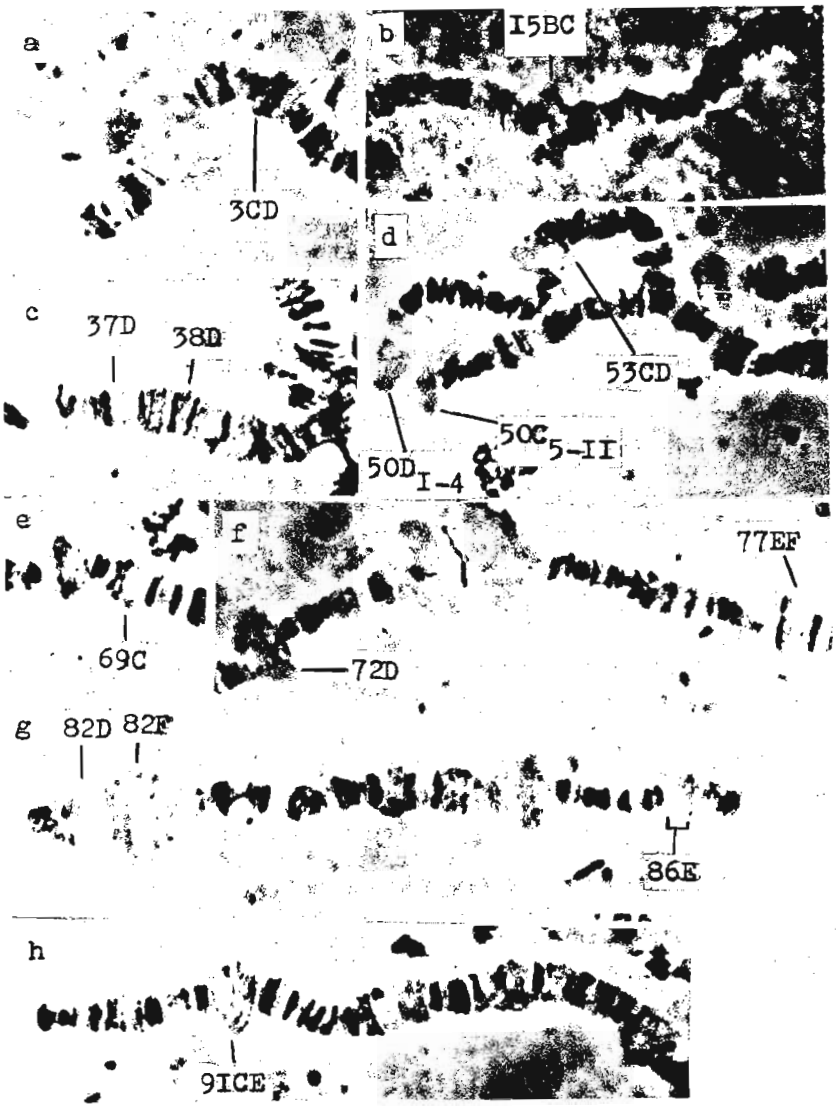


Figure 3. Some puffs of the proximal part differing in behaviour from the puffs of the distal part.
a, h - 118 hrs.
b, c, d, e, f, g - 115 hrs.

McDonald, J. Monash University, Clayton, Victoria, Australia. Selection for high and low percentage vibration in the courtship of D. melanogaster.

Earlier experiments by Crossley (1964) showed that there were individual differences in the male wing vibration component of courtship in D. melanogaster. The possible genetic basis of these differences is being investigated by selection for high and low percentage vibration in Oregon-R males, (based on the selection scheme of Manning (1963)). After four generations of selection, the high and low percentage vibration lines differ considerably from the control, in the expected directions, suggesting a genetic component in the control of this behaviour. The effects and significance of altered percentage vibration on mating success, courtship, and general activity are being investigated in both males and females of the lines as selection proceeds.

References: Crossley, S. 1964, D.Phil. thesis, University of Oxford; Manning, A. 1963, Anim. Behav. 11:116-20.

Spofford, J.B. University of Chicago, Chicago, Illinois. Variegation for dm in $Dp(1;3)N^{264-58}$.

This duplication, inserting 3B4-3D5 into region 80, is cited in Lindsley and Grell, p. 373, as seeming "to carry a mutant allele of dm." According to Lefevre, the rightmost band identified in the duplication is 3D5 and the locus of dm is in 3D4 or 3D5.

It seemed likely that the uniform dm phenotype heretofore recorded for the complete translocation - duplication-bearing 3 plus deficiency-bearing X - and for dm/Y;Dp/+ hyperploid males was the result of extreme variegation rather than mutation accompanying the adjacent break. To check this, dm/Y;Dp males were constructed whose own and whose mother's Su(var) genotypes would improve the chance that bristles of normal thickness would develop from the resulting strong suppression of variegation.

Males taken from a dm/C(1)DX, y f stock were crossed to y w;Su(var) ♀♀. Daughters (heterozygous for Su(var)) were crossed to y w/Y;Su(var) Dp/Su(var) ♂♂. Sons that were phenotypically y^+ w^+ were found to fall into three classes for bristle thickness: Approximately half had typical dm bristles while the remainder were either indistinguishable from dm^+ or intermediate. Males of these three types were individually mated to C(1)RM, y w/Y;$Su(var)^+$/$Su(var)^+$ ♀♀ and presence of pigment in the eyes of daughters (verifying presence of Dp in the tested male) and/or presence of dm^+ in sons (indicating that the tested male had resulted from crossing over in his F_1 mother) were noted. Four kinds of arrays of progeny-types were found:

Phenotype of Tested Male:	Number Tested	Progeny Classes ♂ all dm ♀ all w	♂ dm or semi-dm ♀ w^m or w	♂ all dm ♀ w^m or w	♂ all dm^+ ♀ all w
"non-dm"	6	-	2	3	1
"semi-dm"	7	-	-	7	-
"dm"	8	8	-	-	-

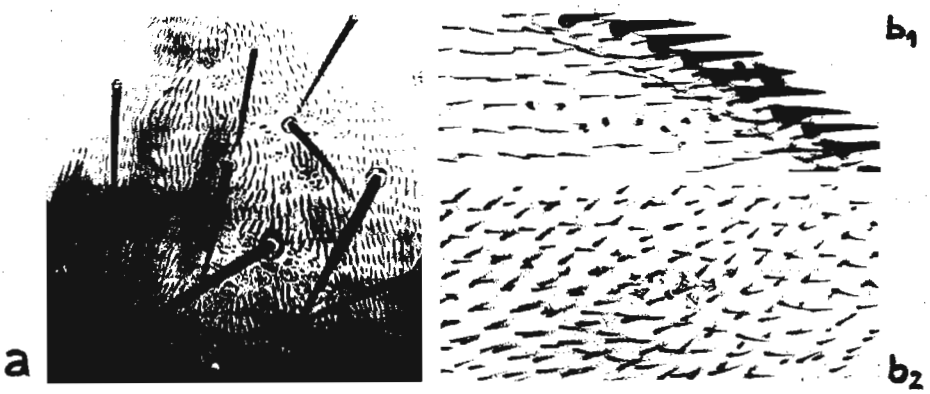
Among the "non-dm" males, one was evidently a crossover, not carrying the Su(var) Dp chromosome. The remainder whose bristles approached wild-type to a noticeable degree all carried the Su(var) Dp chromosome. Presumably, homozygosity for Su(var) completely suppressed dm-variegation when other factors (Su(var) in the maternal genotype, paternal source of the Dp) were favorable, while heterozygosity for Su(var) and the third chromosome from the dm stock only partially suppressed this variegation. Among the progeny of these suppressed-dm^m males, heterozygosity for Su(var) was insufficient to permit expression of the dm^v gene in any but a few sons when the mother lacked the Su(var) allele, even though the mothers carried a Y.

Sharma, A.K., K.S. Gill and G.S. Miglani. Punjab Agricultural University, Ludhiana, India. Studies on chromosomal polymorphism in natural populations of D. busckii.

Seasonal distribution, habitat, and chromosomal polymorphism were studied over a period of two years (1970-71 and 1971-72) in and around the campus of the Punjab Agricultural University, Ludhiana. The fly is found only from November to June; the peak period being from January to March, the mean temperature in February 1971 and 1972 was 14.6°C and 11.7°C respectively. Flies were easily available on the decaying vegetables and fruits, particularly on vegetables. Chromosomal polymorphism was studied in the progenies of 172 female flies; 31 (18%) of these progeny showed inversion loops in the salivary chromosomes of the third instar larvae. The number of inversion loops varied from one to four per progeny; 15, 7, 7 and 2 progenies had 1, 2, 3 and 4 inversions respectively. These different inversions are classified into 21 different types on the basis of their break points. All inversions are paracentric; one inversion is 6 units long, the others vary from 0.5 to 3.5 units. Frequencies of different types of inversions ranged from 1.11 to 4.88 per cent. Nine different types of inversions were present in both years. Inversions are distributed non-randomly in different chromosomes and in right and left arms of the larger chromosomes; 2 inversions are present in the X, 6 in 2L, 8 in 2R, 4 in 3L, 1 in 3R and none in fourth chromosome. The presence of 14 out of 21 inversions in the second chromosome is perhaps related to the occurrence of many desirable gene blocks in this chromosome.

Garcia-Bellido, A. Centro de Investigaciones Biológicas, C.S.I.C., Madrid, Spain. The corrected number of adult epidermic cells of the tergites.

In a previous paper on the clonal parameters of the tergite development (Dev. Biol. 26:264, 1971), we already cast some doubts on the assumption that each trichome in the tergites corresponds to a single cell. The argumentation was that the minimal mwh mitotic recombination spots on the tergites had more processes than those found in the wing. A new cell marker mutant (flare, flr 3: 38.8) shows, in mitotic recombination clones, plates of abnormal cuticle (Figure 1a) corresponding to 4-5 wild-type cell processes in the IV tergite and of the same size on all the tergites. However, the same spots in the wing correspond to a single trichome (Figure 1b).



Direct counts on orcein stained epidermis of IV tergite preparations of 72 hrs. old pupae show (phase contrast) a density of 4.5 ± trichomes per nucleus. Consequently, the number of adult cells per hemitergite must be 3.800 instead of 17.000 - as previously assumed - and 9 the number of cell divisions needed by the 8 larval histoblasts to reach that final number. Since the last mitotic recombination spots appear 24 hrs. after puparium formation the cell division cycle of the histoblasts during this period should be of 2.7 hrs.

Broadwater, C., L.V. Owens, R. Parks, C. Winfrey and F.R. Waddle. Fayetteville State University, Fayetteville, North Carolina. Male recombination from natural populations of Drosophila melanogaster from North Carolina.

In the past two years the phenomenon of male recombination has been discovered among the descendants of wild-caught Drosophila melanogaster from Harlingen, Texas (Hiraizumi) and northwestern Ohio (Waddle). The phenomenon is due to an easily transposable, possibly in some instances extra-chromosomal, factor (Waddle).

The genetics class of Fayetteville State University (C.B., L.V.O., R.P. and C.W.) collected eighteen wild Drosophila melanogaster males from the Fayetteville, North Carolina area and mated them to cn bw;ve e females. Two sons of each male were testcrossed to cn bw;ve e females. For the testcross, each son was mated to 2 females for 7 days. Progeny were counted on the 14th day after mating. The identification of all exceptional progeny was verified by the instructor (F.W.).

Both sons of each of 16 wild-caught males produced progeny. One son of each of 2 males produced progeny. Of 2412 total progeny, 14 were recombinants including 1 (cn;ve) that was recombinant for both chromosomes. At least 1 son of each of 9 wild-caught males produced at least 1 recombinant, indicating that in excess of 50% of the wild flies in this area carry the factor. Considering the limited nature of the test, it may be that the actual incidence of the factor is considerably higher.

The known range of the male recombinant factor has thus been extended to the east coast. It is hoped that the factor will be searched for in the western states and abroad and that the results of such searches will be published in DIS.

References: Hiraizumi, Y. 1971 P.N.A.S. 68:268-270; Waddle, F.R. 1972 Ph.D. Dissertation, Bowling Green State University, Bowling Green, Ohio.

Steiner, W.W.M., W.E. Johnson and H.L. Carson. University of Hawaii, Honolulu and Western Michigan University, Kalamazoo, Michigan. Molecular differentiation in D. grimshawi.

Drosophila grimshawi is currently known from all major islands of the Hawaiian chain with the exception of the island of Hawaii. Here, it's homosequential relative D. pullipes replaces it. In the Maui complex of islands (West and East Maui connected by a narrow, low-elevation land bridge; Molokai and Lanai) D. grimshawi occurs generally above 300 meters altitude.

Carson and Sato (1969) found populations of Drosophila grimshawi differentiated on the Maui complex with respect to the 4th chromosome. Although Molokai and Lanai populations were similar, subpopulations of D. grimshawi on East and West Maui appeared genetically distinct. As a result of these findings a preliminary electrophoretic survey was undertaken to determine the extent of genic differentiation (techniques to be published elsewhere). The results are presented in Table 1, which also includes data for the Hawaii representative D. pullipes. The abbreviation N designates the number of flies examined for each locus.

The expectation of gene pool differentiation with regard to different gene frequencies is met at the Esterase-2 (ES-2), Leucine Aminopeptidase-2 (LAP-2), Octanol dehydrogenase (ODH),

Table 1. Allelic frequencies in Drosophila grimshawi and D. pullipes.

Species:		D. grimshawi								D. pullipes	
Collection No.:		R11,S15,Q80		R8		S16		Q20			
Locus	Allele	W. Maui	N	E. Maui	N	Molokai	N	Lanai	N	Hawaii	N
ES-2	.97		19	0.050	20		72		6		3
	.98			0.050							
	.99	0.210		0.025		0.028					
	1.00	0.158		0.775		0.375		0.750		1.000	
	1.01	0.026		0.050		0.069					
	1.02	0.553				0.361					
	1.03	0.053		0.050		0.132		0.250			
	1.04					0.028					
	1.05					0.007					
LAP-2	.96	0.021	24	0.025	20	0.019	77		6	0.166	3
	.98					0.006					
	1.00	0.458		0.950		0.442		1.000		0.333	
	1.02	0.521				0.526					
	1.04			0.025		0.006				0.500	
ODH	.90	0.368	19				65		6		3
	1.00	0.632				0.885		1.000		1.000	
	1.10					0.115					
MDH-1	.94		23	0.350	20	0.006	83		6		3
	1.00	0.328		0.625		0.982		1.000		1.000	
	1.06	0.652		0.025		0.012					
PGM	1.00	0.800	20			0.972	72	0.500	6		
	1.04	0.200				0.028		0.500			
GOT-1	96		16		13	0.012	83				3
	1.00	0.969		1.000		0.982				1.000	
	1.04	0.031				0.006					
GOT-2	.96	0.028	18		13		81				3
	1.00	0.916		1.000		0.988				1.000	
	1.04	0.056				0.012					

(Table continues)

Locus	Allele	W. Maui	N	E. Maui	N	Molokai	N	Lanai	N	Hawaii	N
IDH	.96		15		20	0.012	81		3		3
	1.00	0.967		1.000		0.988		1.000		1.000	
	1.04	0.033									
HK-1	1.00	1.000	8	1.000	2	0.971	52			1.000	3
	1.02					0.029					
ME	.98		21		20	0.008	59		6		3
	.99					0.008					
	1.00	0.952		1.000		0.949		1.000		0.833	
	1.01	0.048				0.034				0.166	
ADH	1.00	0.969	16	0.975	20	0.973	75			1.000	3
	1.04	0.031		0.025		0.027					
α-GPDH	.90		24	0.050	20		81		6		3
	1.00	0.979		0.950		1.000		1.000		1.000	
	1.10	0.021									

and Malate dehydrogenase-1 (MDH-1) loci. These loci plus Glutamate Oxaloacetate Transaminase-1 (GOT-1), Glutamate Oxaloacetate Transaminase-2 (GOT-2), Isocitrate dehydrogenase (IDH), Hexokinase-1 (HK-1), Malic Enzyme (ME), and alpha-Glycerophosphate dehydrogenase (α-GPDH) display different low frequency alleles between the islands.

Fractionation of the gene pool of D. grimshawi as found by Carson and Sato (1969) is bourne out by these molecular data. In both ES-2 and MDH-1, populations from Molokai, West Maui and East Maui are different from one another. ODH and LAP-2 provide further examples of differences between Molokai and West Maui and East Maui respectfully. It is interesting to note that the Molokai sample displays more alleles per locus than any other population and the extent this reflects sample size should be investigated.

Despite the small sample sizes, the genic similarity between D. pullipes and D. grimshawi is remarkable. The occurrence of the 1.04 allele at the LAP-2 locus suggests a relationship To east Maui or Molokai populations. Deeper invesitigation of the extent of inter- and intra-island differentiation appears warranted and should yield an exemplar of Hawaiian Drosophilid evolution.

Acknowledgements: We wish to thank K.Y. Kaneshiro for identifying and supplying many of the flies used in this study, and Gwen Arakaki for laboratory assistance. Dr. Jayne Ahearn supplied technical assistance in the writing of this paper.

Research supported by NSF grants GB-23230, GB-27586 and GB-29288.

References: Carson, H.L. and J.E. Sato 1969, Evolution 23:493-501.

Fountatou-Vergini, J. Agricultural College of Athens, Greece. Is Drosophila subobscura monogamic?

It is generally considered that adult females of Drosophila subobscura (Col.) mate only once during their lifetime. We have recently collected data indicating that this is not true. As a by-product of another research work we have electrophoretically determined Est-5 genotype of six F_1 progeny separately from each female captured in the wild. These females originated from two different natural populations, from Mt. Parnes in Attica, and from Alikianon village in Crete. Est-5 located near the centromere end of chromosome O (an autosome), is polymorphic (at least six electrophoretically detectable alleles). At least fourteen out of 474 females (= 0.03 ± 0.008) studied from Mt. Parnes were found on qualitative grounds to be digamic (or polygamic), one on the grounds of the number of alleles found in her progeny (exceeding 4) and 13 on their genotypes. In the Cretan population 8 out of 199 females (= 0.04 ± 0.014) studied were found to be digamic (or polygamic), all on the qualitative grounds of the genotypes of their progeny. Since we have studied only one gene and only six individuals from each female progeny the estimate of the frequency of digamic females is the most conservative one. Furthermore, allele frequencies in the populations, sperm stratification, or a nearly complete sperm utilization before the second mating would tend also to underestimate greatly this frequency.

Ouweneel, W.J. Hubrecht Laboratory, Utrecht, Netherlands. Replacement patterns of homoeotic wing structures in halteres.

It is known that the mutation bithorax alters the anterior parts of the metathorax (i.e., notum III and haltere sclerite)[1] and of the haltere into the anterior parts of the mesothorax and of the wing, whereas postbithorax (pbx) effectuates an analogous alteration in the posterior parts. A number of homoeotic halteres were closely analysed in order to establish which cells of the haltere discs are determined to follow a homoeotic pathway, and which structures in the wing and haltere are homologous (cf. ref. 1). As a bithorax allele halteroptera (hl) was used, while for the posterior alteration pbx +/+ Ubx (Ultrabithorax) flies gave excellent results. The pbx halteres exhibited a very consistent pattern. The two sensilla groups in both the pedicel and the scabellum are unaffected; only the posterior parts of these segments are replaced: the scabellum carries an alula, and the pedicel an alar lobe. Usually parts of the cubitus and the 1st vannal vein are seen. The distal segment is much enlarged. Anteriorly it carries a patch of capitellar tissue, but the main part is wing spread. Posteriorly always the "posterior row" of wing border hairs was observed. Usually a pattern of mediae and in one case the medial cross-vein was encountered. The metathoracic bristles, stigma, and papillae[1] are normal; the only homoeotic mesothoracic structure is the postnotum.

The hl halteres are more varying in the expression of the homoeotic effect. Here, the anterior, yellow-haired parts on the scabellum and pedicel usually are partly or entirely replaced by proximal and medial costa, respectively, while the distal segment anteriorly carries the distal costa, and the "triple row" and "double row" of wing border bristles and hairs. The bulk of this segment consists of wing spread. At high degrees of expressivity a clear veinous pattern, comprising the radius and its ramifications, can be seen; in these cases the remaining capitellar tissue is always posteriorly split off from the segment as a small spheroid appendage. The dorsal and ventral sensilla groups on scabellum and pedicel appear to correspond to similar sensilla groups on the proximal and medial wing radius. Further study of the varying replacement patterns suggests that the two "metathoracic papillae" correspond to a similar group of three papillae on the mesothorax (scutum), the metathoracic stigma to the mesopleura with its stigma, the area of the "metathoracic bristle group" probably to the pteropleura, and the scutum to the dorsal notum III.

These alteration patterns and the known organ map of the haltere disc[1] enable, by way of inference, the constitution of an organ map for the wing disc which, however tentative, is more detailed than any wing map so far. It appears that pbx affects a medioposterior segment of the haltere disc, and hl a more irregular, latero-anterior part of the disc. Interestingly, because of the large size difference between the two discs, the homoeotic wing structures in the halteres are produced on a much smaller scale than in situ. It is obvious to assume that in the wing and haltere disc an identical set of positional information is specified (only on different scales, or, if we think of linear morphogenetic gradients, with different slopes). The interpretation of this positional information would then depend on the genome: homoeotic mutations are able to alter this interpretation locally and to produce allotypic, but site-specific, structures. A similar explanation has been given for the leg and antennal disc[2].

References: 1. Ouweneel, W.J. and J.M. van der Meer 1973, Wilhelm Roux' Arch. 172:149-161; 2. Postlethwait, J.H. and H.A. Schneiderman 1971, Devel. Biol. 25:606-640.

Williamson, J.H. University of Calgary, Calgary, Alberta, Canada. Y66d: A Y-chromosome with two nucleolar regions.

Y66d (Y^S y^+ $Y^L.Y^S$) arose spontaneously in an attached-XY/0 male (129-16/0; Parker, DIS 1968). Attached-XY 129-16 (X y^+ $Y^L.Y^S$) was recovered as a detachment of C(1)RM involving the sc^8 Y. Genetic analysis of a series of detachments verified that, genetically, Y66d is KS y^+ KL·KS. Cytological observations of larval neuroblasts have revealed that Y66d has two NO regions. Usually both Y^S elements of Y66d are found to be associated with one nucleolus. In particularly good preparations two nucleoli were clearly visible and separate from each other in each of two cells. Each nucleolus was closely associated with heterochromatic elements presumed to be a short arm of the Y. In both cases heterochromatic elements presumed to be Y^L connected the two nucleoli. Y66d must be KS NO y^+ KL·NO KS.

Hall, L.* University of British Columbia Vancouver, Canada. Conditional dominance of temperature-sensitive locomotor mutants.

The use of an attached X mating scheme has been very successful for the isolation of temperature-sensitive (ts) mutations affecting adult locomotor ability (Grigliatti et al., 1973). When 22°C was used as the permissive temperature and 29°C as the nonpermissive temperature, all ten of the temperature-sensitive locomotor mutants recovered from this screen of 1.1 million progeny were recessives on the X chromosome. This suggests that autosomal dominant mutations of this type either do not occur or are too rare to merit consideration. It would be very interesting to begin to isolate and characterize locomotor mutations on the autosomes but such studies have been hindered to date by the laborious procedure necessary to make an autosome homozygous in order to detect the relatively rare recessive temperature-sensitive mutants. However, recent studies on the ts X-linked locomotor mutants have suggested a procedure which would greatly simplify the task of screening for similar mutants on the autosomes.

The X-linked mutants that show temperature-dependent effects on adult locomotor ability fall into three complementation groups: shibire^{ts} (shi^{ts}), paralytic^{ts} (para^{ts}), and stoned^{ts} (stn^{ts}). All of these mutants when homozygous or hemizygous show wild type behavior at 22°C and are severely but reversibly crippled at 29°C. As heterozygotes these mutants all show wild type locomotor ability at 29°C acting as recessive mutations at this temperature. However, at 40°C as shown in Table 1 two out of the three complementation groups are reversibly

Table 1

Stock	29°C	40°C
Oregon R	+ (wild type)	+ for first ten minutes after which flies show signs of debilitation
Oregon R/FM6	+	Same as Oregon R
shi^{ts1}/FM6	+	Debilitated within 1 minute
shi^{ts2}/FM6	+	Debilitated within 1 minute
shi^{ts3}/FM6	+	Debilitated within 1 minute
shi^{ts4}/FM6	+	Debilitated within 1 minute
shi^{ts5}/FM6	+	Debilitated within 2 minutes
shi^{ts6}/FM6	+	Debilitated within 1 minute
para^{ts1}/FM6	+	Debilitated within 1 minute
para^{ts2}/FM6	+	Debilitated within 1 minute
para^{ts3}/FM6	+	Debilitated within 1 minute
stn^{ts1}/FM6	+	Behavior same as Oregon R
stn^{ts2}/FM6	+	Behavior same as Oregon R

paralyzed under conditions in which the locomotor ability of wild type flies (Oregon R) is unaffected. All of the alleles of both the shibire^{ts} and paralytic^{ts} complementation groups can be distinguished as heterozygotes from wild type flies after a short incubation at 40°C. This suggests that temperature-sensitive autosomal locomotor mutants could be isolated as heterozygotes from the progeny of mutagenized parents using the standard screening device (Williamson, 1971) but modifying the screening conditions to use short incubation times (less than 10 minutes at 40°C. Experiments to test the fertility of flies exposed to 40°C showed that such short exposures have no effect on the fertility or viability of either the wild type or the heterozygous mutant stocks tested.

Table 2

Temperature	Time required for crippling of shi^{ts1}/Oregon R hybrids
36°C	25-35 minutes
37°C	10 minutes
38°C	6 minutes
40°C	1 minute

The conditionally expressed dominant effects of the shibire^{ts} and paralytic^{ts} alleles should be very useful in future genetic manipulations of these stocks since it allows the experimenter to fractionate a population of +/+, +/mutant, and mutant/mutant individuals very rapidly simply by varying the temperature.

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Milkman, R., R. Zeitler and L. Layfer. University of Iowa, Iowa City. Multiple paternity in cage populations.

Individual D.m. females were removed from a polymorphic cage population and immediately allowed to lay eggs in vials, then genotyped with respect to α-glycerophosphate dehydrogenase, malate dehydrogenase, and alcohol dehydrogenase. For each double or triple homozygote, 20 F_1 progeny flies were genotyped. All 3 loci are on one chromosome, so the occurrence of more than two genotypes in a progeny sample indicates multiple paternity. Frequencies of the fast allele at each of the loci are 0.30, 0.38, and 0.22, respectively.

Nine of forty-five double homozygotes produced progeny with more than two genotypes; five of thirteen triple homozygotes did also. Additional double inseminations can be inferred: the observed certain cases are those where at least one male parent was heterozygous and the other male parent had a genotype differing from that of the first. The observed frequency, then, is equal to the true frequency of multiple mating multiplied by the frequency

Table 1. Number of progenies in each class.

Number of genotypes	Number of loci at which mother is homozygous: 2	3
1	7	1
2s*	27	6
2A*	2	1
3	7	3
4	2	2
Total	45	13

*2s - more common genotype ≤14; 2A - more common genotype >14 of the sample of 20. The 2A progenies may have resulted from sampling, differential viability of chromosomes, meiotic drive, or the participation of two different homozygous male parents.

of fulfillment of the above conditions. For doubly homozygous females, the value of 0.20 (9/45) is corrected to 0.38, and for triple homozygotes, 0.38 (5/13) is corrected to 0.44, the correction being less when more loci are involved. Smaller previous experiments using two loci and flies from different cages suggested that multiple paternity was rare in the populations studied. For this reason, and because culture conditions are quite diverse, the present estimated frequency, about 0.4, is not intended as a generalization. Also, when one of two male parents makes the larger contribution by far in an egg sample, the multiplicity may go undetected.

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The effect of varying the temperature on flies heterozygous for the shibire^{ts-1} allele are summarized in Table 2. This is one of the most extreme of the shibire^{ts} alleles. It may be seen that the dominant effect is evident at lower temperatures but the time required for crippling is increased at lower temperatures.

References: Grigliatti, T.A., L. Hall R. Rosenbluth and D.T. Suzuki 1973, Molec. Gen. Genet. 120:107; Williamson, R. 1971, DIS 46:148.

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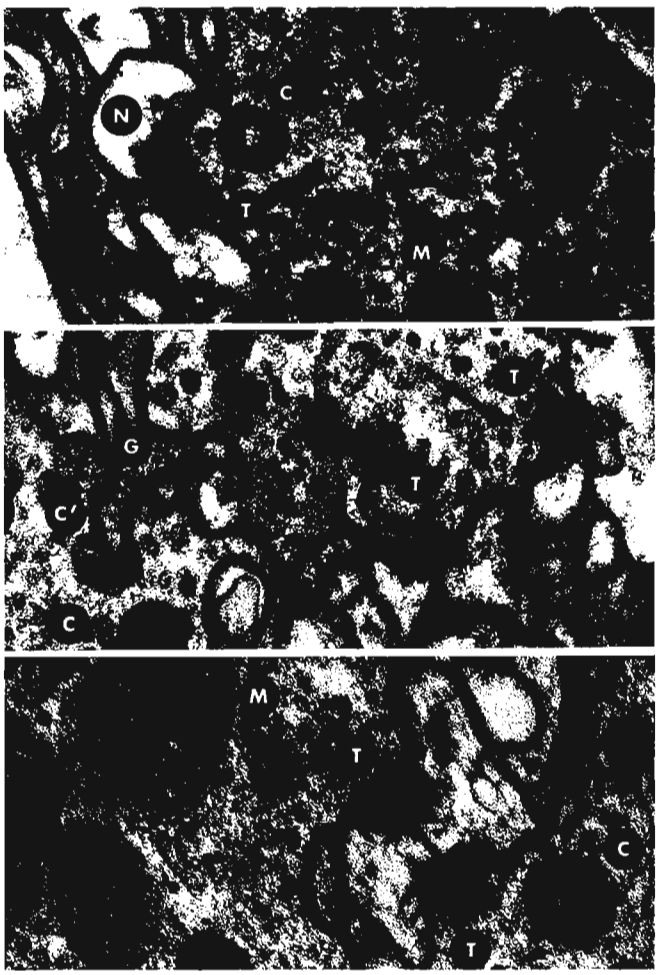
Stark, W.S.* and A.W. Clark. University of Wisconsin, Madison. Visual synaptic structure in normal and blind Drosophila.

The discovery that the nonphototactic Drosophila mutants tan and ebony had ERG defects interpreted as impairment of first order synaptic transmission (Hotta and Benzer, 1969) has led to a search for the physical basis of the functional lesion. While there has been some recent progress in pinpointing the biochemical lesions in tan (Konopka, 1972) and ebony (Hodgetts and Konopka, 1973), Pak, Grossfield, and White (1969) reported a lack of clear structural differences between mutant and wild type.

The accompanying electron micrographs (magnified x 61,500) demonstrate some similarities of membrane -associated synaptic structures in white-eyed wildtype (w), tan (w t), and ebony (w;e). Dissected heads, prefixed in 1% each gluteraldehyde, formaldehyde, and acrolein in 2% sucrose phosphate buffered to pH 7.4 and postfixed in 1% OsO4, were embedded in Spur, sectioned, stained with uranyl acetate and lead citrate, and photographed in a Phillips electron microscope. The top micrograph shows a typical synapse in w with a membrane-bound T-shaped synaptic ribbon (T) in the photoreceptor axon. The synaptic morphology is similar to that discovered in the lamina ganglionaris of Sarcophaga by Trujillo-Cenóz (1965). The round bodies found in the photoreceptor axon (C) are double membrane surrounded projections from glia called capitate projections by Trujillo-Cenóz. Part of a mitochondrion (M) in the presynaptic cytoplasm is also shown. It is noteworthy that there does not appear to be a dense clustering of synaptic vescicles near the ribbon as in typical synapses. Numerous small membrane enclosed bodies present in the presynaptic cytoplasm may be flat viscicles but they appear more like smooth endoplasmic reticulum. There are two collaterals of monopolar neurons (N) on the postsynaptic side with a subsynaptic cisterne of smooth endoplasmic reticulum shown in one where the plane of section was favorable. This one-presynaptic to two-postsynaptic arrangement is typical in Diptera. The middle micrograph shows a photoreceptor axon synapsing onto two separate pairs of collaterals in w;e (but also commonly observed in w and w t). This is commonly found in Dipteran optic cartridge, a unit in the lamina ganglionaris in which six photoreceptor axons synapse onto a meshwork of collaterals from two second order cells (Trujillo-Cenóz, 1965; Boschek, 1971). The pre- and post-synaptic morphology appears similar for w and w;e. Note the capitate projection (C') (in an adjacent photoreceptor axon) which is continuous with a glial cell (G). The bottom micrograph shows T-synapses from two separate photoreceptor axons onto a pair of postsynaptic fibers in a w t preparation (but also seen in w and w;e preparations with a good plane of section). All aspects of the synaptic morphology are the same as in w and w;e with the possible exception that the presynaptic endoplasmic reticulum (or flat vescicles) may show less definition.



Given that the first order synaptic structure is qualitatively the same for normal and blind Drosophila, what differences might exist? It is possible that the numbers of synapses

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Ranganath, H.A. and N.B. Krishnamurthy.
University of Mysore, Manasagangothri, India.

Chromosomal morphism in D. nasuta Lamb. I. Altitudinal variability.

Cryptomorphism is one of the genetic mechanisms evolved by many species of Drosophila to meet the adaptive needs in a dynamic environment. The high order of structural variability in the natural populations of D. nasuta has been recorded by Nirmala and Krishnamurthy (1972). The present report deals with the adaptive polymorphism of D. nasuta at different altitudes of Biligirirangana Hills and Sampaje Ghats (Mysore, India). Table 1 illustrates the trends in the response of chromosomal variability to different elevations.

The observations point to certain conclusions. The data evinces the genetic plasticity of the genotype of D. nasuta to the variation in the altitudes. The incidence of heterokaryotypes ascends with the increasing altitude; the mean number of heterozygous inversions per third chromosome and also per larva is more at higher elevations; particularly the frequency of the overlapping inversions H+K increases at higher altitudes; irrespective of the altitude the fact that the heterokaryotypes are always more than 50% demonstrates the prevalence of

Table 1. Trends in the altitudinal variability of the chromosomal structure of D. nasuta in Biligirirangana Hills and Sampaje Ghats.

	Altitude of Mts.	Total larvae scored	Heterokaryotypes (%)	Heterozygous H+K inversion (%)	Mean values of inversions: Chromosome II	Chromosome III	Larva
Biligiriran-gana Hills	820	106	64.2	16.0	0.25	1.16	1.41
	1040	138	81.9	24.6	0.29	1.68	1.97
	1300	95	94.8	42.1	0.28	2.07	2.35
Sampaje Ghats	500	68	67.6	8.8	0.24	1.36	1.50
	800	65	81.5	21.5	0.24	2.12	2.36
	1100	89	87.3	32.5	0.42	2.12	2.54

heteroselection at all altitudes.

Thus, the existence of these altitudinal variabilities in the chromosomal morphism represents an expression of the flexible nature of the polymorphism present in D. nasuta.

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Reference: Nirmala, S.S. and N.B. Krishnamurthy 1972, DIS 49:72.

(Continued from preceding page)

are different for blind mutant and wild-type. Or perhaps there are differences in synaptic transmission resulting from transmitter differences rather than structural differences. It is also possible that synapses other than receptor to monopolar neuron synapses, some of which may involve the centrifugal fibers described by Trujillo-Cenóz (1965) and Boschek (1971) may be altered in blind mutants. Further work may establish morphological differences between the synapses of wild-type and the nonphototactic mutants.

References: Boschek, C.B. 1971, Z. Zellforsch. 111:369-409; Hodgets, R.B. and R.J. Konopka 1973, J. Insect Physiol. 19:1211-1220; Hotta, Y. and S. Benzer 1969, Nature 222: 354-356; Konopka, R.J. 1972, Nature 239:281-282; Pak, W.L., J. Grossfield and N.V. White 1969, Nature 222:351-354; Trujillo-Cenóz, O. 1965, J. Ultrastructure Res. 13:1-33.

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Sakaguchi, B. and S. Tsuchiyama. Kyushu University, Fukuoka, Japan. Self-induced interaction of D. equinoxialis sex-ratio spirochete.

Interactions among three "Sex-Ratio" (SR) spirochetes from D. equinoxialis, D. nebulosa and D. willistoni in the host of D. pseudoobscura have been demonstrated by Sakaguchi et al. (1968). The interaction phenomena occur between two different SR spirochetes among any of the three spirochete species when mixed infections in the same host were made. During the course of these experiments, we found interaction phenomena between the same SR spirochetes from D. equinoxialis.

To test the effects of superinfections of the D. equinoxialis SR spirochete, hemolymph of the SR strain of D. equinoxialis (ES) was introduced into young adult females. The females were members of the seventh generation of the artificially established SR strain of D. pseudoobscura (PES).

Results of the experiments, Table 1, showed that superinfections of ES hemolymph result in the production of some male progeny in the second brood and in successive broods. Males appeared in the offspring from 16 mothers out of 17 tested. On the other hand, the controls, which were not superinjected, produced very few male progeny. Furthermore, ES or PES females were homogenized in Drosophila saline and the homogenate then centrifuged at 5,000 g for 20 min. The supernatant was treated by heating for 10 min at 60°C. After the treatment it was quickly chilled and centrifuged again at 10,000 g for 20 min. The supernatant, which was almost free of spirochetes, was injected into the PES. The proportion of female offspring in successive broods in the experiment was gradually reduced in subsequent broods from 99% in the first brood and finally reached about 50% in the eighth brood (21 to 24 days after injection). In this experiment, ten females were injected and male offspring were produced from every mother.

As a control, 0.7% NaCl solution was injected into eight PES females. A few males appeared in every brood. The proportions of male offspring was higher in the early broods than in the later broods and all injected females produced some male progeny.

The progenies, from second to fifth generations, from females injected with ES hemolymph, the heat-treated supernatant, and NaCl solution were tested. The SR condition, production of only female offspring, was cured in the series with the heat-treated supernatant and showed normal sex-ratio. The other series did not show such marked effects.

The hemolymph of injected females and their progeny was examined by phase-contrast microscopy. Many morphologically abnormal, as well as a few clumps of the spirochetes, were observed in the females on the second day after injection with the heat-treated supernatant. However, these phenomena gradually disappeared and the spirochetes increased after successive days. The number of these spirochetes was reduced markedly after about 12 days and many abnormal spirochetes were observed. The spirochetes were observed in the hemolymph of male and female adults in the early broods (1 to 9 days) of the second generation. The number of spirochetes in their hemolymph showed difference between individuals, but the spirochetes almost disappeared in the later broods (15 to 21 days). Little effect of injection of the ES

Table 1. Progenies of females of SR pseudoobscura with equinoxialis SR spirochete injected with hemolymph and other materials.

Injected materials	BROODS (DAYS) 0-3	-6	-9	-12	-15	-18	-21	-24	-27	-30	-33	-36
-	0	99	81	63	72	76	77	98	120	93	53	79
% of ♀		97.9	100	100	98.6	98.7	100	100	100	100	100	100
ES hemolymph*	310	555	682	635	472	528	477	505	429	247	120	64
% of ♀	100	96.5	96.1	83.0	78.1	81.4	80.2	78.3	74.8	85.3	82.5	87.5
ES supernatant**	230	214	338	440	414	366	229	245	161	128		
% of ♀	99.1	75.2	56.2	58.9	57.2	57.1	55.0	48.2	50.9	46.1		
0.7% NaCl	227	113	108	65	123	74	14					
% of ♀	84.3	87.7	97.2	96.9	98.4	98.7	92.3					

*, ** see text

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Puro, J., T. Nygrén and M. Nuutila. University of Turku, Finland. Cytogenetic localization of Pc in Drosophila melanogaster.

Attempts at exact genetic localization of the "extra-sexcomb" genes, Pc and Scx, in the third chromosome of D. melanogaster have yielded unsatisfactory results (Hannah-Alava 1969, DIS 44: 75-76). It was only determined by Hannah-Alava that the order was Pc-Scx (not Scx-Pc as listed in Lindsley and Grell, Genetic Variations of D.m.) but until quite recently there has been a complete lack of cytological information on the position of these genes. Pc and Scx were earlier considered as pseudoalleles (Hannah and Strömnaes 1955, DIS 29:121-123) but if so, the distance between the subloci (about 0.3 of a unit, Hannah-Alava 1969) is greater than found for other complex loci. Because of the antennapedia-like phenotype of Pc, as well as semilethality of $Pc^2/Antp^{49}$ and lethality of Scx/Antp compounds, Hannah-Alava (1969) further suggested that both Pc and Scx could be functionally related, if not allelic, to Antennapedia mutants and, thus, possibly in the right arm between 83E and 84D. From the lethal interaction of $Antp^B$ and four X-ray induced revertants of Ns (Nasobemia) sharing a recessive lethal effect and having cytologically detectable change at 84B1-2 Denell (1972, DIS 48:45) concluded that Antp and Ns are allelic. Subsequently he has shown that these Ns revertants also fail to complement the recessive lethality of Scx. This he considers as evidence for Scx, too, being allelic, or possibly pseudoallelic, to the Antp mutants (Denell, Genetics in press). This implies that Antp as well as Scx and Ns are located at 84B1-2 in the salivary chromosome map. On the other hand, since Pc shows no lethal interaction with Scx and the revertants of Ns, Denell concluded that Pc is neither an allele of Antp nor of Scx.

This last conclusion is supported by evidence from our recent experiments (to be published in detail elsewhere) which demonstrate that Pc is in the left arm of the third chromosome. The results of three independent analyses are briefly reviewed here.

(1) It was shown by analyzing 79 recombinant chromosomes derived from crossing-over in the in-p interval from h th st cp in ri Pc^2 sr^{61j2}/eg rn^3 pP bx sr e^s ca compounds that Pc^2 is to the right of ri but to the left of both eg and rn^3. This together with the result of Holm et al. (1969, DIS 44:112) that eg invariably is associated with C(3L) chromosomes induced in homozygous eg/eg females suggests that Pc^2 is in 3L.

(2) In an attempt to induce compound-3 autosomes in th st cp Pc^2/+ females, eight new C(3L) chromosomes with, presumably, heterozygous Pc^2 and one with homozygous Pc^+ were recovered. At the same time, none of eight newly induced C(3R) chromosomes had the Pc^2 gene.

(3) The recessive lethality associated with Pc^2 was found to be suppressed in flies homozygous for Pc^2 but covered by Dp(3;2)FM27, a segregational derivative of an insertional translocation, T(2;3)FM27, comprising a piece of 3L - from 75A-B through 80B(C?) - inserted in 2L at 21F-22A (Puro, unpublished). Such flies exhibited an exaggerated Pc phenotype with extreme pleiotropic effects of wing position and texture in all flies and an increased penetration of extra sex combs in males.

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hemolymph and NaCl solution on the spirochetes was observed.

Oishi (1970, 1971) has shown that SR spirochetes have a virus in each spirochete species of D. nebulosa and D. willistoni, and possibly D. equinoxialis (Poulson, 1973). The viruses act as a virulent to the different SR spirochetes. We examined homologous superinjection in each case of SR pseudoobscura with nebulosa and willistoni spirochete. Little effect of the superinjection was shown in the SR pseudoobscura with willistoni spirochetes, but not nebulosa spirochete.

It is interesting that the results may correspond to behavior of temperate phages. In order to clarify the mechanisms of the self-induced interaction, additional experiments are now underway.

References: Oishi, K. and D.F. Poulson 1970, Proc. Nat. Acad. Sci. USA 67:1565-1572; Oishi, K. 1971, Genet. Res. Camb. 18:45-56; Poulson, D.F. and K. Oishi 1973, Genetics 74: s216; Sakaguchi, B., H. Chikushi, D.F. Poulson and K. Oishi 1968, Proc. Int. Cong. Genet. 12th 2:88.

LeFever, H.M. Kansas State Teachers College, Emporia. Analysis of three White mutants resulting in two new recombination sites at the white locus in Drosophila melanogaster.

Three viable white alleles have been analyzed in respect to their location in the locus and their relationship to the mutant zeste. One of the mutants was white-carrot (w^{crr}) which arose spontaneously in a wild type X-chromosome (Judd 1964). The two remaining mutants were recovered by this author and designated as w^{65a25} and sp-w^4. When tested with sp-w^1, the mutants w^{crr} and w^{65a25} gave the uniform brown eye color characteristic of a white allele rather than a white deficiency. The spotted white phenotype is relatively rare among white mutants and three previous mutants of this type were localized at what is now site 7 (Figure 1). Females with the genotype sp-w^1/sp-w^4 had a phenotype indistinguishable from females with the genotype sp-w^4/sp-w^4 or sp-w^1/sp-w^1 indicating that sp-w^4 was possibly a true allele of site 7.

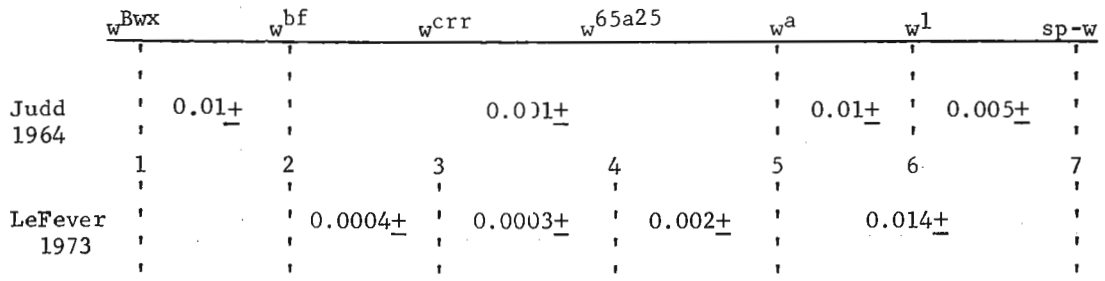

Figure 1. Map illustrating the seven recombination sites of the white locus. For descriptions of the mutants and symbols used, see Lindsley and Grell, 1968. The map distances are corrected for the presence of the autosomal inversions which increase the rate of crossing over by a factor of approximately 3X.

Recombination analysis was used to determine the spatial relationship of the three mutants with reference to the white locus. All constructed parental females carried the autosomal inversions SM1/+ and Ubx^{130}/+ which were employed to increase crossing over in the distal portion of the X-chromosome. Judd (1964) reported that w^{crr} recombined with w^a and was located to the left of it (Table 1). Females were constructed with the genotype w^{crr}/ y z w^{Bwx} and mated to y w spl sn^3 males. Ten exceptional progeny were recovered which place w^{crr} to the right of w^{Bwx} (Table 1). Females were constructed with the genotype w^{crr}/y z w^{bf} and mated to y^2 w^a spl sn^3. Two exceptional progeny were recovered which place w^{crr} to the right of w^{bf} (Table 1). Females were constructed with the genotype z w^{65a25} spl sn^3/y^2 w^a spl ec and mated to y w spl sn^3 males. One exceptional male progeny was recovered which places w^{65a25} to the left of w^a (Table 1). Females were constructed with the genotype z w^{65a25} spl sn^3/w^{crr} and mated to y w spl sn^3 males. Two exceptional progeny were recovered which place w^{65a25} to the right of w^{crr} (Table 1). The mutant sp-w^4 was tested in the following manner: females were constructed with the genotype y^2 sp-w^4 spl sn^3/y^2 w^a spl ec and mated to y w spl sn^3 males. Nine exceptional progeny were recovered which place the mutant sp-w^4 to the right of w^a (Table 1).

When the mutants w^{crr} and w^{65a25} were placed in the heterozygous state with two doses of zeste (z w^+ dup ec/w^{crr} and z/z w^{65a25} spl sn^3) the results were a female with a zeste phenotype. This indicated that w^{crr} and w^{65a25} do not affect the expression of zeste. The mutant sp-w^4, according to its placement by recombination, should be a zeste suppressor (Green 1959c). This was the case as females with the genotype y^2 sp-w^4 spl sn^3/z w^{+dup} ec had a nearly wild type phenotype characteristic of a zeste suppressor effect.

Analysis of the mutant w^{crr} and w^{65a25} which places them in separate locations between former recombinational site 2 (w^{bf}) and site 3 (w^a) indicated that the white locus has at least 7 recombination sites of which sites 1 through 5 are non-suppressors of the mutant zeste (Figure 1).

Acknowledgments: I wish to thank Dr. Burke H. Judd for his assistance during the early part of this study, undertaken while I was a graduate student at the University of Texas, and

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Van Valen, L. University of Chicago, Illinois. A method that might estimate age in Drosophila.

In a previous note (DIS 46:125) I reported failure in an attempt to estimate age in Drosophila by use of daily growth layers in the cuticle. Recently Schlein and Gratz (1972) have had success with this method for mosquitoes and three families of muscoid flies. Their methods differ from ours in perhaps relevant ways. Success with Drosophila could make its ecology anemable to standard ecological procedures. I now lack the relevant equipment but suggest that my previous failure not be taken as definitive.

Reference: Schlein, J. and N.G. Gratz 1972, Bull. World Health Org. 47:71-75.

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Table 1. Exceptional recombinant types recovered from heterozygous females.

Heterozygous female	Exceptional recombinant types recovered	Number	Total offspring
$\frac{y^2 + \quad w^a \text{ spl ec}}{+ \; w^{crr} + \quad + \quad +}$ **	$+ \; w^{crr} \; w^a$ spl ec	4	52713
	$y^2 + + +$	1	
$\frac{+ + + \quad w^{crr}}{y \; z \; w^{Bwx} +}$	$y \; z \; w^{Bwx} \; w^{crr}$	3	103586
	$z \; w^{Bwx} \; w^{crr}$	1	
	$+ + + +$	6	
$\frac{+ + + \quad +^{crr}}{y \; z \; w^{bf} +}$	$+ + + +$	2	170725
$\frac{+ \; Z \; w^{65a25} + \text{spl} + sn^3}{y \quad + + \quad w^a \text{ spl ec} +}$	$y^2 + \text{spl} \; sn^3$	1	14506
$\frac{z + \quad w^{65a25} \text{ spl } sn^3}{+ \; w^{crr} + \quad + +}$	$z + + + +$	2	255341
$\frac{y^2 + \quad sp\text{-}w^4 \text{ spl} + sn^3}{y^2 \; w^a \quad + \quad \text{spl ec} +}$	$y^2 + + \text{spl ec}$	5	21550
	$y^2 \; w^a \; sp\text{-}w^4 \text{ spl } sn^3$	4	

** Burke H. Judd personal communication

for his continued support and encouragement on this project.

References: Green, M.M. 1959c, Hered. 13:302-315; Judd, B.H. 1964, Genetics 49:253-265.

Varma, M.B. and M. Sanjeeva Rao. Osmania University, Hyderabad, India. Studies on the genetic effects induced in Drosophila melanogaster cultured on irradiated glucose medium.

The treatment of food with ionizing radiation is known to be an effective means of prolonging the storage life of food. This fact led to the extreme view of the possibility that irradiated food may be mutagenic and thus hazardous to animals including human beings. Stone, Wyss and Hass (1947) studied the effect of UV rays on irradiated medium in Staphylococcus aureus and reported an increase in mutation frequency. Henderson et al. (1957) cultured Drosophila melanogaster on food made from a yeast-water suspension irradiated with 10^6 gamma radiation and found no significant differences. Swaminathan et al. (1963) found an increase in dominant lethality as well as an increase in frequency of visible mutations in Drosophila melanogaster cultured on food given 150 kr of X rays. Prakash (1965, 1967) reported a high incidence of sex-linked recessive lethals in Drosophila following the addition of DNA irradiated with 100 kr of X rays to media. Rinehart and Ratty (1965, 1967) also reported an increase in sex-linked recessive lethal frequency in D. melanogaster cultured on food given 150 kr, 500 kr, and 3,000 kr of X rays or gamma rays. Reddy et al. (1965) cultured D. melanogaster on a food medium irradiated with 150 kr and 300 kr from a gamma source and observed no induction of mutation. Chopra (1965) tested Drosophila for the production of mutation by feeding the larvae on irradiated medium or irradiated DNA. The experiments did not provide any evidence for a mutagenic effect.

Since the results reported above on irradiation of media are conflicting, experiments were undertaken to assess the effect of irradiated glucose. The present investigation reports the work done on feeding Drosophila on medium containing irradiated glucose and also to assess the damage, if any, of feeding on the same medium for 4 generations.

Glucose was irradiated with a dose of 200 kr of X rays delivered at an exposure of approximately 4000 r per minute. The Oregon strain was allowed to feed on a normal medium containing 50 gms of irradiated glucose dissolved in 100cc of medium. Half of the males developed on this medium were crossed to y sc^{S1} In49 sc^{8};bw st virgin females to screen the incidence of sex-linked recessive lethals and translocations. A brood pattern of three day intervals was used and 6 broods were studied. Each male was allowed to mate with three virgin

Table 1. Comparison of frequencies of sex-linked recessive lethals in a control (1) and induced in four generations (2, 3, 4 and 5) of Drosophila cultured on medium containing 50% irradiated glucose.

	BROOD PATTERN																	
SL	A			B			C			D			E			F			TOTAL		
No	T	l	%	T	l	%	T	l	%	T	l	%	T	l	%	T	l	%	T	l	%
1	861	2	0.23	827	4	0.48	874	2	0.23	89[illegible]	4	0.45	764	0	-	783	2	0.25	5005	14	0.27
2	777	6	0.77	758	5	0.66	620	3	0.48	478	4	0.83	595	3	0.50	435	7	1.60	3663	28	0.77
3	810	7	0.87	452	5	1.10	811	6	0.98	572	5	0.87	438	3	0.68	187	2	1.07	3070	28	0.90
4	902	9	0.99	547	5	0.91	428	4	0.93	390	4	1.02	311	3	0.80	285	3	1.05	2923	28	0.96
5	673	7	1.04	555	5	0.90	439	4	0.91	180	2	1.1	-	-	-	-	-	-	1847	18	0.97

T = Total number of X-chromosomes scored l = lethals recorded

Table 2. Comparison of frequencies of translocations in a control (1) and induced in four generations (2, 3, 4 and 5) of Drosophila cultured on a medium containing 50% irradiated glucose.

	BROOD PATTERN																	
SL	A			B			C			D			E			F			TOTAL		
No	T	t	%	T	t	%	T	t	%	T	t	%	T	t	%	T	t	%	T	t	%
1	849	0	-	846	-	-	832	0	-	862	0	-	737	0	-	731	0	-	4902	0	-
2	546	1	0.18	575	0	-	613	1	0.16	425	0	-	290	1	0.34	200	0	-	2469	3	0.12
3	762	1	0.13	217	2	0.92	383	1	0.26	418	1	0.23	259	1	0.38	172	1	0.58	2211	7	0.31
4	694	2	0.28	310	1	0.32	320	1	0.31	380	2	0.53	282	1	0.35	270	2	0.73	2256	9	0.39
5	333	2	0.62	235	1	0.42	371	3	0.80	200	0	-	-	-	-	-	-	-	1139	6	0.53

T = Total number of F_1 sons tested t = Translocations recorded

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Gupta, J.P. Banaras Hindu University, Varanasi, India. A preliminary report on Drosophilids of Manipur, India.

During recent years a considerable amount of attention has been paid in this country to the field observation of Drosophila, especially in connection with the taxonomy, ecology and geographical distribution. As a result of this several new and newly recorded species have been added to the list of Indian Drosophilids. Still now a vast area of great ecological interest remains virgin or very poorly exploited.

Recently collections were made twice, once in the month of October and once in February 1972 at Khongnangpheidekpi (Manipur) and its adjoining vicinity by using various fermenting fruits as bait. A total of 527 specimens were collected; they comprised ten species belonging to the three subgenera of the genus Drosophila, viz., Sophophora, Drosophila and Dorsilopha.

Drosophila species collected at Khongnangpheidekpi (Manipur, India)

Species	Subgenus	Number
1. D. immigrans	Drosophila	207
2. D. nasuta	"	165
3. D. takahashii	Sophophora	15
4. D. eugracilis	"	42
5. D. malerkotliana	"	27
6. D. bipectinata	"	32
7. D. melanogaster	"	5
8. D. kikkawai	"	7
9 D. nepalensis	"	8
10. D. busckii	Dorsilopha	19
TOTAL		527

Among the species lured to fermenting fruits, D. immigrans and D. nasuta were seen in large numbers. Another interesting feature noticed during these collections was that D. nasuta and D. eugracilis were only seen in October whereas D. immigrans and D. nepalensis in February only. This very clearly indicates that the natural populations of these four species observe seasonal activity.

Acknowledgements: Author is very thankful to the U.G.C. for extending financial assistance.

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Table 3. X^2 values for the differences of sex-linked recessive lethals in controls compared with different broods of 4 generations.

Sl No	Generation	BROODS A	B	C	D	E	F	TOTAL
1	control vs F_1	2.15	0.68	0.14	0.27	1.72	5.26	11.96
2	control vs F_2	2.04	2.58	2.53	0.45	1.66	0.81	16.00
3	control vs F_3	3.02	1.75	1.70	0.67	1.64	1.36	17.46
4	control vs F_4	2.95	1.70	1.68	0.19	-	-	10.72

females. F_1 females were mated individually with y sc^{S1} In49 sc^8 males while the F_1 males were mated individually with bw;st females to score for sex-linked recessive lethals and translocations, respectively.

The other half of the males were allowed to feed again on a medium containing 50 gms of irradiated glucose in 100 cc of medium along with fresh females of ORK. The experiment was repeated in the above manner for four generations. The results are presented in Table 1 and 2.

A X^2 test has been done to compare the following groups: 1) Control vs F_1, 2) Control vs F_2, 3) Control vs F_3, and 4) Control vs F_4. The results of the statistical analyses are presented in Table 3.

The present investigation revealed that irradiated glucose when mixed in medium could induce mutations in Drosophila melanogaster.

Vidyavathi, T. and M. Sanjeeva Rao.
Osmania University, Hyderabad, India.
Induction of mutations in D. melanogaster with Deuterium and gamma rays.

Deuterium is a non-radioactive isotope of Hydrogen resulting from a neutron, emerging from an atomic reactor, which is captured by the nucleus of a hydrogen atom. The study of effects of Deuterium interested many biologists for the reason that Hydrogen, being the most common element in biological systems, its substitution may cause drastic effects. Zamenhoff and Demerec (1943) cultured Drosophila melanogaster on food medium containing Deuterium and reported the failure of this isotope to induce lethal mutations. Hughes et al. (1963) reared flies on medium containing various concentrations of Deuterium and observed the ineffectiveness of this isotope to increase the mutation frequency. Konard (1960) failed to induce any forward mutations in Bacteriophage T_4 with 50% Deuterium. However, he did observe a few wild type revertants from r-II mutants which was attributed primarily to base pair substitution. Rosalic de Giovanni (1960) reported that the growth of several strains of E. coli and B. subtilis was inhibited by the presence of Deuterium and the degree of inhibition in each strain was specific. He thought that the mutagenic effect of Deuterium might be due to the replacement of hydrogen by Deuterium in the Deoxyribonucleic acid molecule during its synthesis which might upset the bonding and geometry of the Deoxyribonucleic acid molecule resultimg in replication error or possibly due to the effect of Deuterium on the enzymes involved in Deoxyribonucleic acid synthesis. Usha Purnima (1972) demonstrated the failure of this isotope to induce sex-linked recessive lethal mutations in D. melanogaster by feeding on a medium containing 30% Deuterium. Manohar Rao (1971) reported an increase in mutation frequency with Deuterium in Oryza sativa.

Pollard (1961) raised cultures of E. coli in Hydrogen oxide and Deuterium oxide and irradiated the two cultures simultaneously with gamma rays where no visible alterations were found in bacteria grown in Deuterium. Manohar Rao (1971) observed an increase in the frequency of chlorophyll mutations in Oryza sativa when Deuterium is used in combination with Ethylmethanesulphonate (EMS). The present investigation reports the work carried out to assess whether Deuterium would interact with gamma rays to alter the radiation damage in D. melanogaster.

Oregon-K strain flies were allowed to feed on a normal medium containing 30% Deuterium. The males developed on this medium were exposed to 3000r of gamma rays. A series of experiments as detailed below were conducted and the criteria for assessing the mutations induced was sex-linked recessive lethals.

1) Control;
2) Irradiating the flies with 3000r of gamma rays;
3) Feeding the flies on a medium containing 30% Deuterium;
4) Rearing the flies on a medium containing 30% Deuterium and irradiating the F_1 males with 3000r gamma rays.

Oregon-K males and females were allowed to feed on a medium containing 30% Deuterium. After 72 hours the flies were removed and the eggs laid were allowed to hatch. The emerging F_1 males were divided into two groups. The first group was used for studying the effect of Deuterium. The second group was exposed to a dose of 3000r gamma rays so as to study the interaction if any. The genetic damage induced was studied by screening for sex-linked recessive lethal mutations. To study the differential response of various stages of spermatogenesis, a brood pattern of three days each of six broods was employed. Each treated male was mated with three females of y sc^{S1} In49 sc^{8};bv;st stock. After three days of mating, the females were transferred to fresh vials while the male was transferred to a fresh vial along with three more females. In F_2 the absence of a wild type body colour male is an indication that a sex-linked recessive lethal has been induced.

The Chi-square test has been done to compare the following groups:

1) Control versus flies reared on Deuteriated food; 2) control versus flies exposed to 3000r gamma rays; 3) flies exposed to 3000r gamma rays versus flies reared on a medium containing 30% Deuterium and then exposed to 3000r gamma rays. If the calculated values exceed the chi-square value at 5% level for one degree of freedom the groups compared are taken to be significantly different from each other. The results are presented in Table 1. The results of the statistical analysis are presented in Table 2.

This analysis clearly indicated that Deuterium failed to interact with gamma rays broodwise, whereas there was a statistically significant increase when the total frequencies of all the six broods were considered where flies were exposed to 3000r gamma rays after culturing on Deuterium medium. The chemical basis of interaction between Deuterium and gamma rays is not clear.

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Paik, Y.K. University of Hawaii, Honululu.

Frequency of heterozygous inversions from a Korean population of D. immigrans.

In October, 1972, a collection of Drosophila was made at Yonsei University Forest, Seoul, at 300 feet. Daily mean temperature in the area was 15°C. In the sample of 369 flies taken, D. immigrans comprised the greatest fraction (62%) and D. bizonata occurred in the next largest numbers (14%). There were eight other species of the same genus present.

The 135 larvae (one/female) of the wild D. immigrans females were examined cytologically for the types and frequencies of inversions and the data are summarized in the table. The notations A, B and C are the same as in Brncic's (1955) data. The result is very similar to that in Japanese populations (Hirumi 1961; Toyofuku 1961), but strikingly different from those reported from other widely separated geographic regions.

Larvae tested		No. of heterozygous inversions observed			No. of inversions per larva (S.E.)
		A	B	C	
135		9	14	1	0.18 ± 0.03
	%	6.7	10.4	0.7	

Acknowlegement: The author expresses his sincere appreciation to Yonsei University for providing the facilities which made this work possible.

References: Brncic, D. 1955, J. Hered. 46:59-63; Hirumi, H. 1961, Jap. J. Genetics 36: 297-305; Toyofuku, Y. 1961, Jap. J. Genetics 36:32-37.

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Table 1. The incidence of sex linked recessive lethals induced in various experiments conducted.

Treatment	Brood A			Brood B			Brood C					
	N	L	%	N	L	%	N	L	%	N	L	%
Control	861	2	0.23	827	4	0.48	874	2	0.23			
30% Deuterium	214	4	1.86	440	1	0.22	500	1	0.20			
3000r γ rays	763	23	3.01	240	14	5.83	348	7	2.01			
30% Deuterium & 3000r γ rays	741	31	4.01	485	28	5.77	323	7	2.1			

	Brood D			Brood E			Brood F			Total		
	891	4	0.45	764	-	-	783	2	0.25	5005	14	0.27
	212	1	0.47	99	-	-	15	-	-	1480	7	0.47
	955	11	1.15	883	5	0.57	582	1	0.27	3771	61	1.61
	519	15	2.8	557	1	0.18	338	2	0.59	2963	84	2.83

N = Total number of X-chromosomes scored
L = Total number of lethals recorded

Table 2. χ^2 values for the differences in sex linked recessive lethal frequency for the groups compared.

Group	BROODS						
	A	B	C	D	E	F	Total
Control vs 30% Deuterium	5.58	1.35	0.40	0.28	-	-	1.32
Control vs 3000r γrays	20.01	17.4	8.51	1.93	2.67	0.83	45.43
3000r γrays vs 30% Deuterium & 3000r γrays	1.48	0.0	0.21	5.28	2.33	0.23	13.40

References: Hughes, A.N., E.H. Phillip and G.C. Becker 1963, Genetics 49:715; Konard, M 1960, Ann. N.Y. Acad. Sci. 84:678; Manohar Rao, D. 1971, M.S. Disser., Osmania University; Pollard, E. 1961, Nature 192:177; Rosalic de Giovanni 1960, Ann. N.Y. Acad. Sci. 84:644; Usha Purnima 1972, M.S. Disser., Osmania University; Zamenhoff, F.C. and M. Demerec 1943, Amer. Nat. 77:380.

Prabhakar Rao, K. and M. Sanjeeva Rao.
Osmania University, Hyderabad, India.

Screening of Ovral and Lyndiol for genetic effects in D. melanogaster.

Sanjeeva Rao et al. (1971) reported a significant increase of mutations in D. melanogaster by feeding Sodium Saccharin. Tranquilizers containing Thioridazine Hydrochloride and Chlordiazepoxide Hydrochloride have also increased the mutation frequency in D. melanogaster by feeding technique (Sanjeeva Rao et al. 1973; Pratap 1970). In view of the positive results obtained in Drosophila with tranquilizers and artificial sweeteners, experiments were undertaken to assess the genetic damage induced if any by oral contraceptives commonly used. The present study reports the work carried out with two oral contraceptives namely Ovral and Lyndiol.

Oregon-K strain flies were allowed to feed on a normal medium containing two tablets of Ovral dissolved in 100 cc of food medium. In another experiment, flies were allowed to feed on a medium containing two and three tablets of Lyndiol for every 100 cc of the medium. After 72 hours the flies were removed and the eggs laid were allowed to hatch. The emerging males were tested for genetic effects if any. The genetic damage induced was studied by scoring for sex-linked recessive lethals and translocations. A brood pattern of three days each of six broods was employed to screen the differential response of various stages of spermatogenesis. Each treated male was mated to three females of y sc^{S1} In49 sc^{8};bw;st stock. After three days of mating, the females were transferred to fresh vials while the male was transferred to a fresh vial along with three more females. The F_1 females were mated individually to y sc^{S1} In49 sc^{8} males, while the F_1 males were mated individually with bw;st stock to score for sex-linked recessive lethals and translocations respectively in the F_2 generation. The results are presented in Tables 1 and 2.

Table 1. Showing the percentage frequencies of sex-linked recessive lethals induced by Ovral and Lyndiol in Drosophila melanogaster.

Treatment	Brood A			Brood B			Brood C					
	N	L	%	N	L	%	N	L	%	N	L	%
Control	1336	3	0.22	1716	8	0.46	1894	3	0.24			
Ovral	975	1	0.102	1035	7	0.676	824	2	0.243			
Lyndiol (2 tab.)	597	-	-	479	-	-	693	-	-			
Lyndiol (3 tab.)	526	1	0.190	707	-	-	658	-	-			
	Brood D			Brood E			Brood F			Total		
	1599	7	0.43	1015	4	0.39	1073	3	0.27	8633	28	0.324
	620	5	0.806	539	1	0.186	293	-	-	3578	16	0.447
	920	4	0.435	456	-	-	153	-	-	3298	4	0.121
	452	-	-	300	-	-	182	-	-	2825	1	0.035

N = Total number of X chromosomes scored L = Number of sex-linked recessive lethals induced

Table 2. Showing the percentage frequencies of translocations induced by Ovral and Lyndiol in D. melanogaster

Treatment	Brood A			Brood B			Brood C					
	N	T	%	N	T	%	N	T	%	N	T	%
Control	758	nil	nil	748	nil	nil	834	nil	nil			
Ovral	946	nil	nil	895	nil	nil	606	nil	nil			
Lyndoil (2 tab.)	616	2	0.325	420	nil	nil	646	nil	nil			
Lyndiol (3 tab.)	721	nil	nil	176	nil	nil	525	nil	nil			
	Brood D			Brood E			Brood F			Total		
	769	nil	nil	660	nil	nil	683	nil	nil	4452	nil	nil
	447	nil	nil	383	nil	nil	143	nil	nil	3420	nil	nil
	280	nil	nil	795	nil	nil	266	nil	nil	3023	2	.066
	269	nil	nil	698	nil	nil	427	nil	nil	2816	nil	nil

N = Total number of sons scored T = Number of translocations induced

(Continued at bottom of next page)

Singh, B.N. Banaras Hindu University, Varanasi, India. Heterosis owing to basal inversion in D. ananassae.

Several inversions in the natural populations of Drosophila ananassae, a cosmopolitan and domestic species, have been reported from various parts of the world. Most of the inversions have a very restricted distribution while the three paracentric inversions originally described by Kaufmann (1936) from Alabama, U.S.A. and termed by him as subterminal (IIL), terminal (IIIL) and basal (IIIR) seem to be coextensive with the species (see the geographic distribution of inversions arranged by Shirai and Moriwaki, 1952; Singh, 1970). These three widely distributed and frequently reported inversions have been called 'cosmopolitan' by Futch (1966).

It has been found in many species of Drosophila that heterotic buffering is associated with chromosomal polymorphism. In D. ananassae heterosis has been found to be associated with subterminal and terminal inversions when heterozygous (Moriwaki et al, 1956; Moriwaki and Tobari, 1963; Tobari, 1964; Singh, 1972, 1973; Singh and Ray-Chaudhuri, 1972). The literature, however, lacks information regarding basal inversion. In the present investigation, a wild laboratory stock of D. ananassae containing this inversion in IIIR and the standard gene sequence in the other chromosomes, has been utilized. This stock was raised from a female captured in Lowari, Chakia forest area, Varanasi, in November 1968. In order to determine the frequencies of different genotypes (karyotypes), the larvae were squashed with the usual acetocarmine method.

All the three karyotypes for the basal inversion were distinguished. Their frequencies are shown below:

	Standard Homozygote	Heterozygote	Inversion Homozygote
Observed	54	119	38
Expected	61.53	104.82	44.65

$\chi^2 = 3.82$
$P > 0.05$

In a random sample of 211 larvae, 119 (56.4%) are heterozygous for the inversion. Thus the frequency of heterozygotes is more than 50 per cent. This suggests that the inversion heterozygote is adaptively superior to the corresponding homozygotes. The expected values of the three genotypes have been calculated on the basis of Hardy-Weinberg frequencies. The χ^2 test shows that the differences are statistically insignificant ($P > 0.05$).

Thus it can be suggested that heterotic buffering is associated with basal inversion as is the case with subterminal and terminal inversions. Now it is known that all the three inversions in D. ananassae which are coextensive with the species, exhibit heterosis in heterozygous condition. So it is proposed that this may be one of the factors which enabled these inversions to spread almost throughout the distribution range of the species.

References: Futch, D.G. 1966, U.T.P. 6615:79-120; Kaufmann, B.P. 1936, PNAS 22:591-594; Moriwaki, D., M. Ohnishi, and Y. Nakajima 1956, Proc. Int. Genet. Symp. pp.370-379; Moriwaki, D. and Y.N. Tobari 1963, Genetics 48:171-176; Shirai, M. and D. Moriwaki 1952, DIS 26:120-121; Singh, B.N. 1970, Ind. Biol. 2:78-81; Singh, B.N. 1972, Genetica 43:582-588; Singh, B.N. 1973 (submitted); Singh, B.N. and S.P. Ray-Chaudhuri 1972, Ind. J. Exp. Biol. 10:301-303; Tobari, Y.N. 1964, Evolution 18:343-348.

(Continued from preceding page)

The results showed the inability of the two oral contraceptives to induce any mutations in Drosophila melanogaster.

References: Pratap, C. 1970, M.Sc. Disser. Osmania University; Sanjeeva Rao, M., B.C. Samuel and A.B. Qureshi 1971, Ind. J. Hered. (in press); Sanjeeva Rao, M., P. Nair and C. Pratap 1973, Ind. J. Exp. Biol. (in press).

Kuhn, D.T. Florida Technological University, Orlando. Effect of maternal age upon an aberrant sex ratio condition in a tumorous-head strain of D. melanogaster.

Most tumorous-head strains of Drosophila melanogaster are characterized by sex ratios in favor of males. However, extensive variability in sex ratio exists among samples within any of the tumorous-head strains (Kuhn 1971). Maternal age was investigated as a possible cause for this variation.

The strain used was one in which Cy/Pm balancer comprised the second chromosome pair, while the X and 3rd chromosomes were retained from tumorous-head. Individual heterokaryotypic females (1/1; Cy/Pm; h 3A/3B), aged for 1, 5, 10, 15, 20, 25 and 30 days were crossed with 3 homokaryotypic males (1/Y; Cy/Pm; h 3A/h 3A), ranging in age from 1 to 3 days. The flies were mated for 5 days in 25 x 95 mm shell vials with standard media at 25 ± 1°C. All F_1 adult flies from each cross were sexed and their karyotypes determined.

Results of this investigation are presented in Table 1. Female sterility was rather high 33.7%, and was generally associated with females from 1 to 10 days of age. Apparently many of the sterile females died by 15 days of age. Productivity was extremely low, 22.0 offspring per female. However, 50.0% of the potential offspring do not survive because Cy/Cy and Pm/Pm are lethal combinations. The average proportion of homokaryotypes, 45.7% is typical for this cross since a differential selection against homokaryotypes exists within the tumorous-head strain. Although some variations in sex ratio exist between females of different ages, no recognizable trend was established. The average sex ratio in favor of males was 55.1%, ranging from 52.6% to 56.9%. A small reduction that may have resulted from a sampling error occurred in 10 and 15 day old females. Except for those two samples, the sex ratios were extremely consistent, with homokaryotypes generally showing slightly more aberrant sex ratios. It is concluded that age of the mother does not significantly contribute to the variability noted in the sex ratio in favor of males.

Thirteen separate samples are taken during the course of this study. Percentage sex ratios for these samples were 52.4, 52.7, 52.8, 53.5, 54.1, 54.3, 54.4, 54.6, 54.9, 56.9, 59.1, 59.6 and 59.8. Since the temperature, the age of the media, the age of males, and the maternal age were well controlled, a yet to be determined variable is still operative.

Reference: Kuhn, D.T. 1971, Genetics 69:467-478.

(Table 1 on next page)

Pinsker, W. and E. Doschek. Institut für allgemeine Biologie, Vienna, Austria. Light dependence of D. subobscura - courtship and ethological isolation.

In the courtship of D. subobscura, visual signals are of vital importance. Therefore this species needs light for successful mating.

By selection, however, a strain has been obtained (see the contribution of R. Springer) which is able to reproduce in absolute darkness. In the courtship of that strain the visual signals have lost their importance in favor of tactile stimuli.

As the courtship behavior is one of the most important isolating mechanisms, experiments were made to investigate to what degree the mutant behavior of this light-independent strain would affect the isolation barriers, when its females were paired with males of the mutant "yellow" and the related species D. pseudoobscura.

	"yellow" - ♂♂		D. pseud. - ♂♂	
	n	% insem.	n	% insem.
light-independent ♀♀	200	29.5%	200	13.0%
wild-type ♀♀	300	9.3%	300	1.0%
	$\chi^2 = 32.57$		$\chi^2 = 29.47$	
	$p < 0.0005$		$p < 0.0005$	

For this purpose groups of 10 virgin females and 10 males were set up in 15 ccm plastic vials under constant illumination during 72 hours. After that time the females were dissected and examined for sperm. Equal tests were made with non-selected wild-type females for comparison.

The results are shown in the table. The percentage of the inseminated females shows that the females of the light-independent strain copulate significantly more frequently than the wild-type in both cases. That means that the isolation barriers were distinctly reduced.

From this fact may be concluded that in the non-selected wild-type strains of D. subobscura visual signals will decisively impede interspecific copulation.

(Continued from previous page: Kuhn, D.T., Effect of maternal age upon an aberrant sex ratio condition in a tumorous-head strain of D. melanogaster.)

Table 1. Measurements of sex ratios, sterility, productivity, and proportion of homokaryotypes of 1/1; Cy/Pm; h 3A/3B females of various ages when individually crossed with 1/Y; Cy/Pm; h 3A/h 3A males.

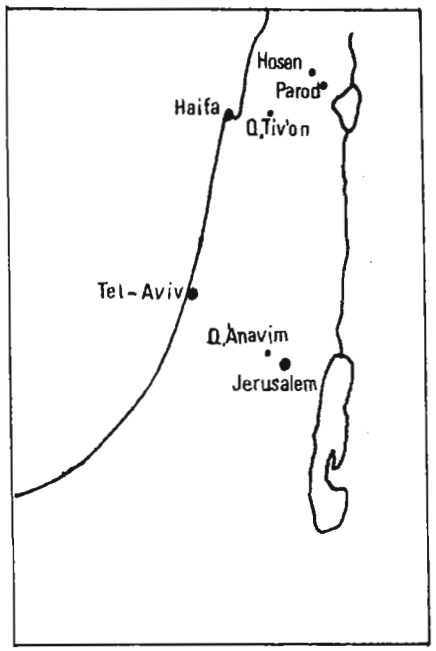
φ^1 1/1; Cy/Pm; h 3A/3B x σ^3 1/Y; Cy/Pm; h 3A/h 3A

♀ Age in days	♂ Age in days	% Sterility	♀ Productivity	% h 3A/3A	No. ♀♀ Productive	h 3A/h 3A ♂	♀	T	% ♂♂	h 3A/3B ♂	♀	T	% ♂♂	Totals ♂	♀	T	% ♂♂
1	1-3	48.6 51/105	20.6 1114/54	45.0 501/1114	54	281-	220=	501	56.1	340-	273=	613	55.5	621-	493=	1114	55.7
5	1-3	50.0 23/46	20.8 479/23	42.4 203/479	23	115-	88=	203	56.7	150-	126=	276	54.3	265-	214=	479	55.3
10	1-3	58.1 18/31	4.7 61/13	55.7 34/61	13	20-	14=	34	58.8	13-	14=	27	48.1	33-	28=	61	54.1
15	1-3	15.4 8/52	20.9 918/44	44.7 410/918	44	224-	186=	410	54.6	259-	249=	508	51.0	483-	435=	918	52.6
20	1-3	27.6 8/29	15.2 320/21	51.3 164/320	21	92-	72=	164	56.1	90-	66=	156	57.7	182-	138=	320	56.9
25	1-3	12.0 6/50	27.9 1226/44	47.1 577/1226	44	325-	252=	577	56.3	360-	289=	649	55.5	685-	541=	1226	55.9
30	1-3	20.9 14/67	26.8 1423/53	46.2 658/1423	53	366-	292=	658	55.6	416-	349=	765	54.4	782-	641=	1423	55.0
Totals		128/380	5541/252	2547/5541	252	1423-	1124=	2547	55.9	1628-	1366=	2994	54.4	3051-	2490=	5541	55.1

Grossman, A.I. The Hebrew University of Jerusalem, Israel. Study of alcohol dehydrogenase and α-glycerophosphate dehydrogenase in the Israel natural populations of Drosophila melanogaster.

Several independent studies have shown, in a variety of natural populations of Drosophila, variation in the loci Adh (Alcohol dehydrogenase) and α-Gpdh (α-glycerophosphate dehydrogenase). The aim of the present work is to extend the survey of these loci to additional populations of D. melanogaster.

Flies were collected from four natural populations in Israel, in Qiryat Anavim, Qiryat Tiv'on, Hosen and Parod (Figure 1). Examination was done by standard electrophoretic procedures, on flies collected in nature and on two male progeny of each pregnant female collected in nature. The results are given in Figure 2. No difference was found between the four populations in the frequencies of alleles in locus αGpdh. The Parod and Hosen populations showed a higher frequency of alleles A_S (Adh) compared to the Qiryat Tiv'on and Qiryat Anavim populations. In all populations, in both loci, there is a shortage of heterozygotes (Table 1).



Comparison of males (145 individuals) and females (134 individuals), in the pooled data of Q. Tiv'on and Q. Anavim, reveals differences between the sexes in the frequencies of both alleles and genotypes. The frequency of allele A_S (Adh) is 68.6% in males and 72.0% in females, while the frequency of α_S (αGpdh) is 37.6% in males and 48.1% in females. The distribution of genotypes in the two sexes is given in Table 2. No difference exists with respect to locus Adh while in locus αGpdh there is a shortage of heterozygotes in the males and an excess of heterozygotes in the females.

Table 1. Distribution of genotypes at loci Adh and α-Gpdh in males of four wild populations of D. melanogaster in Israel

	Q. Anavim		Q. Tiv'on		Parod		Hosen	
	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.
A_SA_S	33	30.7	42	37.6	45	43.8	83	81.2
A_SA_f	24	28.6	25	33.8	10	12.3	33	36.6
A_fA_f	9	6.7	12	7.6	2	0.9	6	4.1
$\alpha_S\alpha_S$	12	8	14	12.6	13	6.7	22	20.1
$\alpha_S\alpha_f$	22	30	35	37.9	13	25.6	55	58.8
$\alpha_S\alpha_f$	32	28	30	28.5	31	24.7	45	43.1

Genotype frequencies, combined for both loci, are given in Table 3. The double homozygotes, $A_SA_S\alpha_S\alpha_S$ and $A_fA_f\alpha_f\alpha_f$ are in excess, compared to the frequencies expected according to random assortment. It seems that selection is operating mainly against the homozygotes $A_SA_S\alpha_f\alpha_f$ and $A_fA_f\alpha_S\alpha_S$. Similar deviations from expected frequencies, of smaller magnitude,

Table 2. Distribution of genotypes at loci Adh and α-Gpdh in males and females.

	♂		♀	
	Obs.	Exp.	Obs.	Exp.
A_SA_S	75	68.3	74	69.6
A_SA_f	49	62.4	45	53.9
A_fA_f	21	14.3	15	10.5
$\alpha_S\alpha_S$	26	20.5	28	31.1
$\alpha_S\alpha_f$	57	68.0	73	66.9
$\alpha_f\alpha_f$	62	56.5	33	36.0

Table 3. Genotype frequencies for both loci.

		A_SA_S		A_SA_f		A_fA_f	
		Obs.	Exp.	Obs.	Exp.	Obs.	Exp.
	$\alpha_S\alpha_S$	22	13.3	4	8.7	-	3.8
♂	$\alpha_S\alpha_f$	32	29.4	21	19.3	1	8.3
	$\alpha_f\alpha_f$	21	31.9	24	20.9	17	8.9
	$\alpha_S\alpha_S$	19	15.4	7	9.4	2	3.1
♀	$\alpha_S\alpha_f$	40	40.2	27	24.5	6	8.2
	$\alpha_f\alpha_f$	15	18.2	11	11	7	3.6

were found in females. The same results were also obtained in a previous study, on flies collected from wild populations in Central Asia (Grossman and Koreneva 1970).

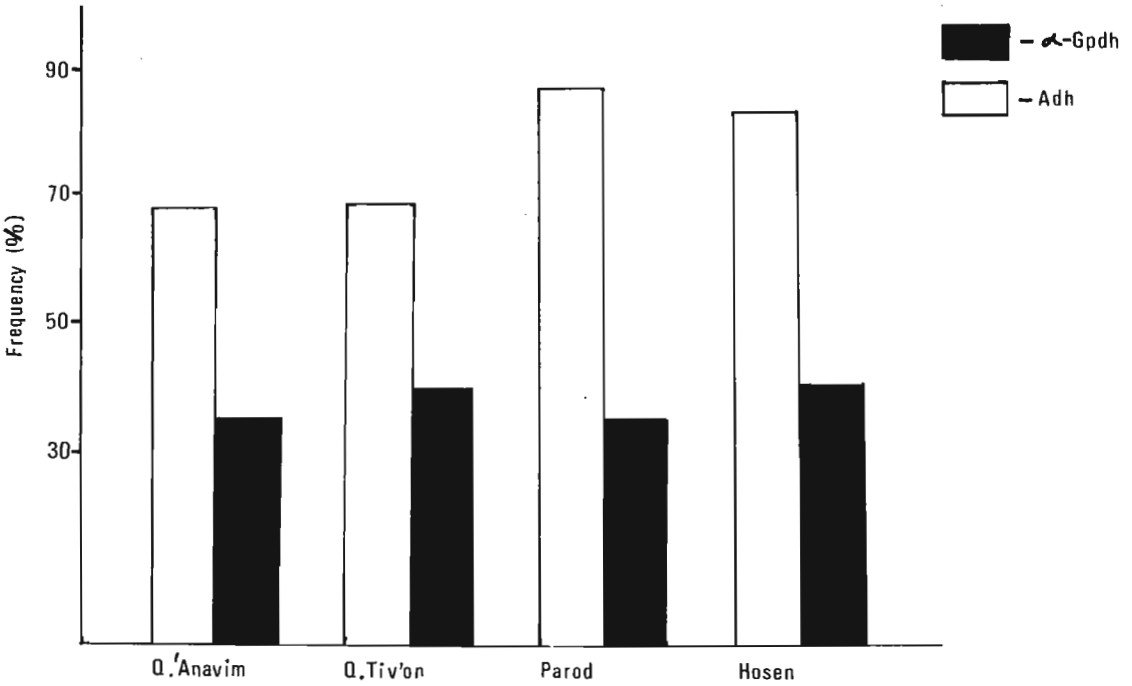


Figure 2. Frequency of allele A_S (Adh) and α_S (α-Gpdh) in males from four wild populations of D. melanogaster.

It is suggested that the intra- and inter-population distribution of the Adh and αGpdh isozymes is specific.

References: Grossman, A.I. and I.G. Koreneva 1970, Genetica (Russ.) 6(8):95-101.

Kenney, J. and A. Hunter. University of the Pacific, Stockton, California. The effect of chlorinated water on D. immigrans.

A. Espinos in DIS 48 reported that when chlorinated tap water (0.4-0.5 ppm) was used to moisten the medium, productivity of D. melanogaster decreased. Since effects on one species may differ from those on others even within the same genus, we have carried out experiments with chlorine in the medium on D. immigrans.

Clorox (NaClO) was diluted with distilled-deionized water to 200-300 ppm as tested with sodium thiosulfate. Instant Drosophila medium was moistened with this solution in the experimental vials and with distilled-deionized water in the controls in a 1:1 volume ratio. Three pairs of virgin flies were placed in 150 ml plastic vials moistened with the experimental or control solution and kept at 25°C. with a 12 hour light-day. The flies were transferred to fresh vials prepared with the corresponding solution every 24 hours.

The average life span of the controls was 30 days as compared to 22 days for the experimentals. Control females produced an average of 13 offspring as compared to 4 for those in chlorinated food. It was noted that the experimental flies were less active than the controls. Handling the flies during transfers it was found that the wings of those on chlorinated medium were more fragile. Experiments on the second generation of offspring gave equivocal results and the experiments have been discontinued due to the difficulty of standardizing and maintaining the concentration of chlorine. It can be concluded however that chlorine does affect the life span and productivity of D. immigrans.

Gvozdev, V.A., V.T. Kakpakov and L.M. Mukhovatova. Kurchatov's Institute of Atomic Energy, Moscow, USSR. Effect of β-ecdysone on cell growth and synthesis of macromolecules in the established embryonic cell lines of D. melanogaster.

The effect of β-ecdysone was studied at the cellular level in the established cell lines of Drosophila melanogaster. The C15 medium enriched with 15% bovine fetal serum was used (Gvozdev and Kakpakov, 1968; Kakpakov et al., 1969; Kakpakov et al., 1971).

0.05-0.1 μg per ml of β-ecdysone causes the efficient inhibition of cell proliferation in the diploid cell line 67j25 D (over 250 passages). 2-deoxy-α-ecdysone is as active as β-ecdysone. On the other hand, α-ecdysone has no effect at a concentration of 1 μg per ml but the concentration of 5 μg per ml inhibits cell multiplication to 60% of control.

The inhibition of cell growth by β-ecdysone may be reversed after 24 hours of treatment but is irreversible after 48 hours. The pre-treatment of cells with α-ecdysone has no effect on the subsequent inhibitory influence of β-ecdysone.

RNA synthesis measured by H^3 uridine incorporation decreases 2-3 fold 24-48 hours after the addition of β-ecdysone. The similar inhibition of DNA synthesis estimated by H^3-thymidine incorporation was observed after 48 hours. The pronounced inhibition of RNA synthesis with no influence on DNA replication was observed in some experiments 24 hours after the addition of β-ecdysone. Protein synthesis measured by C^{14}-lysine incorporation does not decrease after 48 hours of hormone action.

Several other sublines (diploid, triploid and tetraploid) were also sensitive to 0.1 μg per ml of β- ecdysone. The subline 67j25 Da carrying spontaneous X;3 translocation and characterized by elevated rate of cell proliferation as compared to sister cells without translocation was relatively resistent to 0.1 μg per ml of ecdysone.

References: Gvozdev, V.A. and V.T. Kakpakov 1968 Genetika (Russ.) 2:129; Kakpakov, V.T., V.A. Gvozdev, T.P. Platova, L.G. Polukarova 1969 Genetika (Russ.) 5:67; Kakpakov, V.T. L.G. Polukarova, V.A. Gvozdev 1971 Ontogenesis (Russ.) 2:295.

Hennig, W. Institut für Molekularbiologie II der Universität Zürich, Switzerland. A remarkable status of Drosophila pseudoneohydei?

Wheeler (1972) states an unintentional naming of this Drosophila species by us, which was repeatedly supplied to the Max-Planck-Institut für Biologie, Tübingen, by the Austin, Texas, stock center as D. neohydei. It was initially suspected that a "conversion" of the lampbrush loop morphology in the primary spermatocytes from the D. neohydei type (Hess and Meyer, 1963) into another morphology takes place in the new environment. But our studies (Hennig, Hennig and Stein, 1970) revealed that, in fact, another Drosophila species was supplied. This could more recently be substantiated by studies on the metaphase chromosomes of D. hydei, D. neohydei and D. "pseudoneohydei" (Hennig, Leoncini and Hennig, 1973). In addition, the genome sizes of all three species were measured by microspectrophotometry. The genome of D. "pseudoneohydei" is 103% of the D. hydei genome, while the D. neohydei genome is only 84% (Hennig, Leoncini, Hennig and Zacharias, in preparation). A comparison of the lampbrush loops in primary spermatocytes suggested that D. "pseudoneohydei" actually represents D. eohydei. This has been substantiated by our investigation of several strains of D. eohydei, kindly provided by Professor M.R. Wheeler.

In a similar way we identified a stock of D. nigrohydei obtained from Austin, Texas, as D. eohydei. Differences in the sperm length observed in two stocks of D. nigrohydei from Tübingen and Edinburgh could also be explained by the same error (Dr. G.F. Meyer, personal communication). It seems, therefore, that confusions of this type could be easily avoided if characters more easily accessable for recognition than the conventional morphological criteria in the Drosophila genus, would be provided to non-taxonomists. The karyotype, the morphology of the Y chromosome lampbrush loops in primary spermatocytes and DNA density patterns, which are known to be species specific, are obviously excellent criteria for the differentiation of taxa.

References: Hennig. W., I. Hennig and H. Stein 1970, Chromosoma (Berl.) 32:31-63; Hennig. W., O. Leoncini and I. Hennig 1973, In: Heterochromatin in Man (R.A. Pfeiffer, ed.) F.K. Schattauer-Verlag, Stuttgart - New York. p. 87-99; Hess, O. and G.F. Meyer 1963, Portg. Acta Biol. A, VII 29-46; Wheeler, M.R. 1972, DIS 48:154.

Horton, T.D. and C.P. Wright. Western Carolina University, Cullowhee, N.C.

Development of Tyrproless-2, 1(1)EN15, a lethal mutant of Drosophila melanogaster.

Tyrproless-2, 1(1)EN15, is a sex-linked, lethal mutant of Drosophila melanogaster which was X-ray induced by Novitski (1963). In this mutant death usually occurs in the pupal stage. The present investigation involved a study of developmental characteristics of this mutant. All individuals were timed (± 1 hour) and kept under the same controlled conditions. As a control y, w, spl, sn individuals, which came from the stock in which Novitski (1963) induced the lethal mutant, were used.

All control larvae formed puparia at about 110 ± 17 hours after oviposition. About 92 percent of the 1(1)EN15 larvae formed puparia at about 131 ± 23 hours after oviposition. The rest of the larvae remained in the larval stage until death. So, the mutants formed puparia about a day later than controls.

All controls emerged from the pupae as adults at about 90 hours after puparium formation. None of the 1(1)EN15 pupae ever emerged as adults. Instead, they remained in the pupal stage for a long period after the normal time of emergence. The peak of development in 1(1)EN15 pupae was at approximately 130 hours after puparium formation. After this point developmental changes were slight and confined to only a few pupae. At 130 hours some of the pupae showed adult structures such as head, legs, wings, and body hairs, but none of the pupae were completely developed. This peak of development might also be considered the beginning of a complete developmental breakdown, because after this point the pupae showed increasing amounts of desiccation and darkening. Some of the pupae showed little or no development for the entire observation period.

The 1(1)EN15 pupae were observed until about 400 hours after puparium formation. At this point none had emerged as adults. Most of the pupae had become darkened in color and almost completely dried out. This seems to indicate that all had died as pupae.

Reference: Novitski, E. 1963, DIS 37:51-53.

Schwinck, I. University of Connecticut, Storrs.

Autonomy of the aurodrosopterin fingerprint pattern in imaginal eye transplants between the mutants prune and garnet.

In earlier studies, the two-dimensional thin-layer chromatography revealed at least 5 separable drosopterins (Schwinck, Genetics 68:s59, 1971). A survey of various eye color mutants showed quantitative differences of these patterns for 17 mutants and 5 alleles (Schwinck and Mancini, Archiv für Genetik 46:41-52, 1973). In particular, the fingerprint fraction (d) - recently designated aurodrosopterin - varied in the absolute amount as well as in the relative amount in reference to the sum of all drosopterins. Recently, the response to phenylalanine crystal implantation was studied in a number of mutants with large amounts of aurodrosopterin and in a number of mutants with very small amounts of aurodrosopterin. Only the mutants which have a relatively large amount of aurodrosopterin, i.e., garnet, pink-peach, orange, etc., responded with increased post-eclosion synthesis of all drosopterins, whereas the other group of mutants, i.e., rasberry, prune, purple, etc., did not show any response to the phenylalanine implant (Schwinck, Genetics 74:s245, 1973 and unpublished). Two mutant stocks with the extreme drosopterin fingerprint pattern, garnet^{50e} and prune^{2}, were chosen for surface transplantation of imaginal eyes, the technique of which has been described in DIS 49:96, 1972. An imaginal eye from a newly eclosed fly was transplanted onto an incision of the host abdomen, taking care that the "open" back of the transplant eye was exposed to the host hemolymph. Reciprocal transplants and controls, g^{50e} cn eyes onto pn^{2} cn hosts, pn^{2} cn eyes onto g^{50e} cn hosts, and the controls pn^{2} cn eyes onto pn^{2} cn, and g^{50e} cn onto g^{50e} cn, were carried out on a large scale, resulting in 40-65 survivors in each group. The eye color changes during aging appeared normal according to the genotype of transplant eyes and host eyes. After 10-12 days the transplant and host eyes were extracted and the drosopterin fingerprint pattern was developed on thin-layer cellulose plates. For the transplants as well as the hosts, the fingerprint pattern was found to be autonomous according to the respective cell genotype.

(Supported by the University of Connecticut Research Foundation Grant.)

Gress, R.E.* and H. Nickla. Creighton University, Omaha, Nebraska. Choice of oviposition sites by laboratory strains of Drosophila melanogaster.

Previous observations in our laboratory indicated oviposition behavior varied consistently among laboratory strains of D. melanogaster. Four stock populations (maintained by standard mass-culture methods) were used to determine the reliability of these observations: w (1-1.5) lt (2-55.0), cl (2-16.5), and In(2L+2R)Cy. For each replication, ten virgin females and one male were selected from each of the above strains. Virginity was the only criterion for selection of females. After 48 hours of "preincubation" with the respective males, the males were removed, and the females from the various strains were mixed. After 36 hours the 40 females were allowed to lay eggs on Stendor dish lids which had been previously filled with standard medium and covered with a yeast-soaked, circular piece of paper toweling. At 24-hour intervals the eggs were removed from the toweling, separated by pattern, and placed in medium-containing shell vials. Placement patterns of eggs were characterized as to solitary, double (two eggs in contact), or clumped (three or more eggs in contact). Resulting offspring were scored by strain. Table 1 presents the mean percentages of single and clumped egg placement for the various strains. Arcsin transformations were used on all percentage data prior to statistical analyses.

Table 1. Percentage of single and clumped egg placement among four laboratory strains of D. melanogaster.

		SINGLE		CLUMPED	
Strain	Replications*	Mean	95% Confidence interval	Mean	95% Confidence interval
cl	14	12.5**	± 6.5	80.0**	± 8.0
In(2L+2R)Cy	13	31.0	± 10.0	51.5	± 11.5
w	10	30.0	± 11.0	54.5	± 12.5
lt	14	24.5	± 8.5	60.5	± 10.5

* Variation in the number of replications resulted from variability in production of offspring.
** Significant at the 0.05 probability level.

Different preferences for egg placement exist among the four strains studied. Although all strains favored clumped egg placement, clot was sufficiently prejudiced against solitary egg placement to differentiate it statistically from the other three strains. The genetic basis for the selection of oviposition sites is presently under investigation. (Supported by a grant from the National Science Foundation to Creighton University.)

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McCrady, E. III. University of North Carolina, Greensboro. Thoracic macrochaet variation in D. virilis.

Recent transplants of wing discs from a stock of D. virilis (McCrady, 1973) have revealed a regular reduction in the number of macrochaetae produced. Such a reduction was also reported earlier (McCrady, 1971) following in situ operations on the right wing disc. A pilot study of normal variation of each of the 11 macrochaetae on the right side of adults of both sexes has been completed. Of 2483 animals examined, 2391 (96.4%) had the normal complement of bristles. 74 animals (2.98%) lacked both the anterior notopleural bristle and its socket. 12 animals exhibited split notopleurals or extra macrochaetae located between the normal sites of the anterior and posterior notopleurals. No such variation was seen at any of the other nine macrochaet locations, with the exception of one case of a missing dorsal supra-alar bristle on a male. It is thus possible that the area of maximum variation in macrochaet location and development is centered in the distal portion of the thoracic blastema (see fate map of Murphy, 1972), close to the point at which the tracheal supply enters the disc. Further studies are in progress to determine the genetic source of this variation, and to measure its extent in D. melanogaster.

References: McCrady, E. 1973, J. Insect Physiol. (in press); _____ 1971, DIS 47:76; Murphy, C. 1972, J. Exp. Zool. 179:51-62.

Golubovsky, M.D. and K.B. Sokolova. Institute of Cytology and Genetics, Novosibirsk, U.S.S.R. The expression and interaction of different alleles at the 1(2)gl locus.

The mutations of 1(2)gl locus typically cause the death of homozygotes in the larval or prepupal stage. We found these lethal mutations to occur very often and persistently in Geographically isolated populations of D. melanogaster of the U.S.S.R. Is the ubiquitous occurrence of 1(2)gl mutations connected with diffusion of the only mutant allele or with the independent appearance of several? The main task was to make this question clear. If allelic lethals are non-identical in the sense of their genetic topography, it is possible to try to detect some phenotypical or genetical differences between them. Upon detailed and careful analysis of stages of the death of homozygotes we succeeded in dividing the sample of 12 alleles into four groups. Group I - larval, more than 96% of homozygotes die at the second larval instar. Group II - mainly larval, 85-90% of homozygotes die at 2nd larval stage. Group III - larval-prepupal, about 50% of homozygotes die at larval and about 50% at prepupal stage. Group IV - prepupal, more than 80% of homozygotes die at the prepupal stage. By special crosses we showed that the manifestation character of alleles is not connected with influence of genetic background. The expression and interaction of lethals from groups I, II, III and IV was studied in 10 compounds. The following preliminary conclusions may be made: 1) Compounds formed by different alleles of the same group have the same effective lethal stage. 2) Different alleles of the same group had similar type of interaction in compounds with lethals of other groups. Hence it follows that attribution of lethals to group I, II, III or IV reflects some essential genetic characters of alleles. 3) All possible types of interaction are discovered in different heteroallelic combinations: independent action of each allele, dominance of mutant with developmentally earlier expression, and on the contrary, dominance of phenotypically later allele and after all, partial interallelic complementation, leading to viability of 30-40% of heterozygotes. It is interesting that complementation was found only in combinations of lethals of group I with different lethals of any other groups: I/II, I/III, I/IV. In overpopulated cultures the complementing compounds almost all died or were detected only on the tenth day of mass flight. But we obtained the four stocks of viable flies having in each second chromosome a lethal allele of the 1(2)gl locus. So, the high frequency of these lethals in natural populations is a result of the mutation occurrence and not of diffusion. We found also that under normal conditions the lethal alleles of 1(2)gl decrease the viability of heterozygotes but under the conditions of lowered temperature (12°C) the viability of heterozygotes increases by 20%. So lethal heterozygotes on 1(2)gl mutations are strongly favored by natural selection at over-wintering period. It is possible to predict also the existence of viable compounds on different lethal alleles in natural populations.

Bennett, J. and A.M. Hathaway. Northern Illinois University, DeKalb, Illinois. Aging and behavioral correlates of the w, w^+ gene substitution.

Previous studies of the behavioral correlates of the w, w^+ substitution have shown that differences do exist (DIS 48:94). Twenty five flies of each sex from both the Oregon-R-Inbred (ORI) and Oregon-R-Inbred-white (ORI-w) strains (coisogenic except for the substitution) were observed at two and eight days of age. Flies were transferred to new culture vials on the day of eclosion and examined during their second day. They were then maintained singly in numbered vials until observed again at eight days of age. All observations were made between 9 February 1972 and 10 May 1972 from 3:00 to 5:30 p.m. Individual flies were viewed for ten minute periods in 16mm Blister™ slides (DIS 47:75). Observations were recorded on a thirteen trait checklist. Only one incidence of a behavior pattern was recorded per period no matter how frequently it recurred. In the ORI stock only total activity (sum of all recorded activity) was significantly greater at day 2 than day 8. All behaviors but one showed the same pattern but without significant differences. The ORI-w stock showed significantly higher frequencies for two behaviors, (different in each sex) at day 2, but with no consistent pattern. Total activity was not noticeably different at the two ages. Other comparisons only supported the notion that the small sample size precluded definition of the single trait differences. The observations do suggest that age interacts with the w, w^+ substitution. Perhaps the white-eyed flies mature more slowly than the wild type. That is ORI shows peak activity early (108 on day 2, 53 on day 8) and tapers off, while ORI-w is either stable or builds up more slowly (67 on day 2, 71 on day 8). Further observations are in progress.

Korochkin, L.I. and E.S. Belyaeva. Institute of Cytology and Genetics, Novosibirsk, U.S.S.R. Esterase isozymes of Drosophila imeretensis.

Starch and polyacrylamide gel electrophoresis were used to examine the esterase patterns in D. imeretensis stock 27 (from the collection of Prof. N. Sokoloff, Institute of Developmental Biology, Moscow) and also in ♀ D. virilis (stock 9, Batumi) x D. imeretensis ♂ hydrids in the process of ontogenesis. Figure 1 presents an esterase pattern of D. imeretensis. Noteworthy is that D. imeretensis esterases, on the whole, surpass in electrophoretic mobility the esterases of all the other Drosophila of the virilis group (D. texana, D. virilis, D. littoralis) we have studied. 6 fractions are distinguished in D. imeretensis; just as in D. virilis, some of these fractions are represented by 2-3 subbands. Two fractions stain usually more intensely; one of them is β-esterase (esterase-3 on Figure 1). A number of fractions exhibit some individual variability such as absence of some α-esterases, differences in electrophoretic mobility and activity.

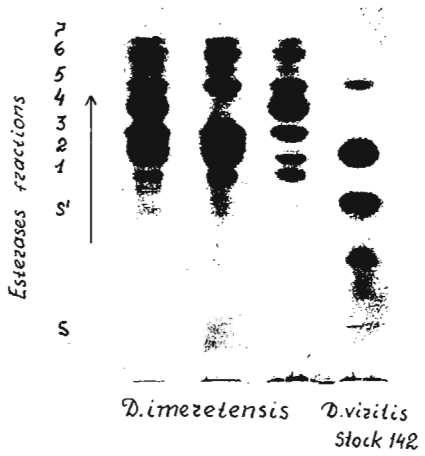

Fig. 1. Electrophorogram

We have produced a number of inbred substocks. One of these stocks, 10F, is characterized by the presence of an additional slow S-esterase belonging to β-esterases. In D. imeretensis β-esterase is well expressed at third instar and stains very heavily, which is quite in contrast to D. virilis, D. texama and D. littoralis. In ♀ D. virilis stock 9 x D. imeretensis stock 27 ♂, a differential expression of parental esterases is observed in that esterase-4 of D. virilis stock 9 is weakly expressed and the activity of the homologous esterase of D. imeretensis predominates; an inverse pattern is observed with respect to the fastest esterase.

Angus, D.S. Salisbury College of Advanced Education, Adelaide, S.A., Australia. Polymorphism in D. immigrans.

The ubiquitous species, Drosophila immigrans, forms a high proportion (up to 80%) of the Drosophila fauna of Adelaide.

Collections were made during September 1973 by sweeping over fermenting banana baits at Toorak Gardens, Adelaide, South Australia.

Examination of individuals revealed that Drosophila immigrans exhibits phenotypic polymorphism for markings on the thorax. The thoracic markings, when present, consist of two or four dark brown longitudinal stripes, as shown in Figure 1. The medial pair lie between the dorsocentral bristles and extend on to the scutellum; the lateral pair lie laterad to the dorsocentral bristles and do not extend on to the scutellum. The proportion of individuals which exhibited these markings are shown in the table. A genetical analysis of the material will be made in order to determine the mode of inheritance of the striped thorax condition. The results of this investigation will be reported elsewhere.

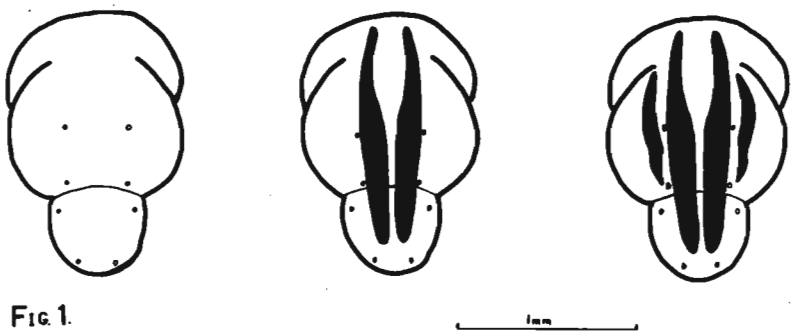

FIG. 1.

	THORAX MARKINGS			
	4 stripes	2 stripes	unstriped	total
♂	123	194	108	425
♀	9	34	9	52
TOTAL	132	228	117	477
%	27.7	47.8	24.5	100

LeFever, H.M. and R.C. Moore. Kansas State Teachers College, Emporia.
Characterization of a micro-organism in Drosophila medium.

In many laboratories the control of bacterial and fungal contamination in Drosophila food medium has been of concern for many years (Felix 1969). Some stock cultures in various laboratories throughout the country have been infected with bacterial growth which gradually spreads to vials and in some instances to all viable cultures.

The objectives of the present study were to bring about isolation, culture, identification and ultrastructural examination of the organism in question.

Isolation and culture: A sample of the organism in question was obtained from contaminated Drosophila media and a streak for isolation was carried out on nutrient agar plates. Following inoculation of the plates there was a 24 hr period of incubation at 37°C. After this period, observations were recorded and inoculation of metabolic media was carried out.

Antibiotic sensitivity: Sensitivity tests were carried out using petri plates containing a blood agar base of 5.0% human blood. Antibiotics used were the following: chloromycetin, neomycin, erythromycin, kanamycin, novobiocin, penicillin, streptomycin and tetramycin. The diameter of the zone of inhibition around each sensitivity disc was measured and recorded.

Growth: Observation of growth was carried out on triple sugar iron agar and nutrient ag agar slants; recordings were taken at 4.0 hrs, 6.0 hrs, 8.0 hrs, 10.0 hrs, 24.0 hrs and 48.0 hrs.

Staining: A gram, spore, and capsule stain were carried out using standard laboratory methods. A gram stain was procured at each recording taken during the period of growth observation.

Electron microscopy: Fixation and embedding were carried out using a standard procedure developed by Kellenberger, Ryter and Sechaud (1958). Photomicrographs were taken on a Hitachi model HS8 electron microscope.

Metabolic tests employed in this study are listed in Table 1 with their results.

Table 1. Results of metabolic tests read at 48 hrs.

Triple sugar iron agar	no gas, no H_2S, alkaline slant, acid butt	Triple sugar iron agar	no gas, no H_2S, alkaline slant, acid butt
Nutrient broth	positive	Lactose	negative
Nutrient agar	positive	Mannitol	slight positive
Blood agar	positive	Urea	negative
Citrate	negative	Gelatin	negative
Motility	positive	Nitrates	negative
Indol	negative	Lysine decarboxylase	slight positive
Salicin	negative	Methyl red	negative
Sucrose	slight positive	Vogues-proskauer	negative
Dextrose	positive, no gas	Catalase	positive

When inoculated upon solid media this organism has a mucoid growth with separate colonies being slightly raised with smooth margins after four hours incubation.

After six hours, growth is palisade and motility is apparent at this stage.

From eight to ten hours, the organism appears pink to red in color with the palisade arrangement not as pronounced; the pigmentation taking on a red-brown appearance after twelve hours. A crust-like covering may be present at this time, thus making the colonies appear dry and folded.

Gram stains taken at 1.0 hr, 2.0 hrs, 6.0 hrs, 10.0 hrs and 24.0 hrs indicated a paucity of gram positive rods with the presence of coccoid forms. Upon using the same technique of examination at 48.0 hrs the organism appeared gram negative. To rule out the possibility of contamination further isolation procedures were carried out, but the results remained the same.

Table 2
Antibiotic sensitivity determined with respect to diameter of zones of inhibition

	Concentrations		
	High	Medium	Low
Chloromycetin	2.3 cm	2.0 cm	1.8 cm
Neomycin	1.8	1.5	1.5
Erythromycin	2.5	2.3	2.0
Kanamycin	2.0	1.7	1.5
Novobiocin	2.0	1.7	1.5
Penicillin	2.3	2.6	2.0
Streptomycin	1.0	1.0	0.0
Tetramycin	1.9	1.5	1.3

The capsule and spore stains from a 24.0 hr culture were negative.

Electron microscopy revealed a thick cell wall encasing the organism in all preparations examined; no specialized intracellular structures could be distinguished in any of the preparations.

The microorganism isolated and examined in this study suddenly appeared in culture media of Drosophila melanogaster; its presence seemed to overwhelm the Dipteran population thereby bringing about total loss of the cultures.

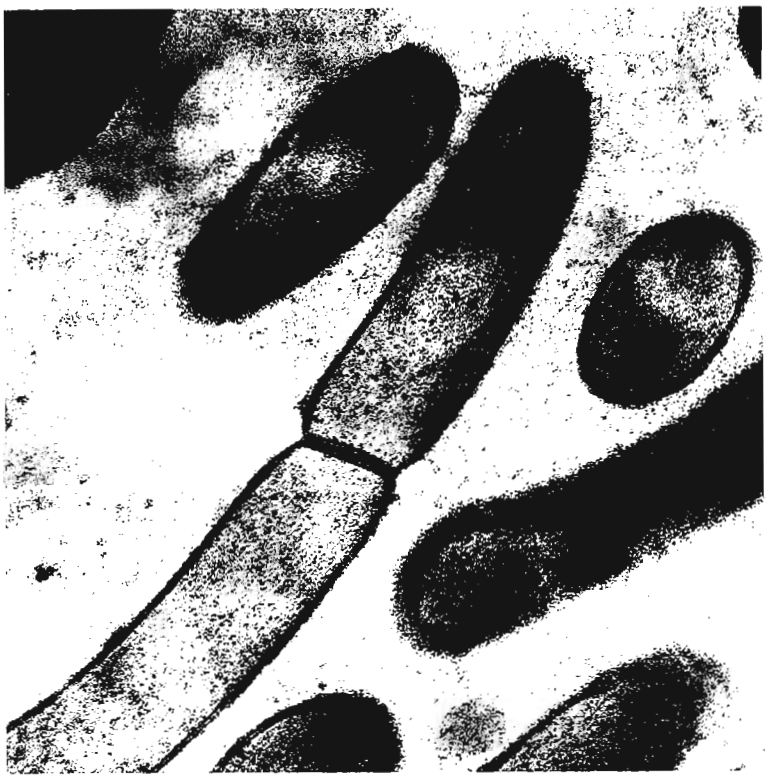

Figure 1. Electron micrograph of bacteria contaminating Drosophila culture media. 48,140 X.

Contamination involving this particular organism is difficult to control in spite of standard sterilization procedures. The organism's resistance to control methods is probably due to the thick cell wall as seen by electron microscopy (Figure 1).

To bring about control of this organism a 50.0% solution of a tincture of Zephiran (Winthrop Laboratories) was used in treating all contaminated media and containers, after which the organism was no longer detectable. Most soaps and alcohol solutions below 80.0% were not effective in controlling the organism; in fact it seemed to thrive in some cases. Antibiotic sensitivity tests indicate a susceptibility to chloromycetin, erythromycin, and penicillin (Table 2).

References: Felix, R. 1969, DIS 44:131; Kellenberger, E., A. Ryter and J. Sechaud 1958 J. Biochem. and Biochem. Cytol.

Gassparian, S. University of Isfahan, Isfahan, Iran. Reproduction of D. melanogaster for different cross breedings.

For the production of virgin females of D. melanogaster in some laboratories, it is customary to separate and transfer the female from the culture medium when the pupa is nine days old, a few hours before the opening of the wings. As this method is time consuming, a new and more satisfactory method has been developed in the Genetics Laboratory at the University of Isfahan. On the seventh day when the pigments of the eye are formed, the pupae are transferred to several new, small, sterile glass containers which have sufficient nutrients; those pupae which were on the glass walls of the original container are moved by a special spatula, and whose which were on cotton wool being transferred by forcepts with the cotton wool, in order to avoid direct contact. Two days after this procedure the virgin females and males are recognizable and thus separated. By this method the time involved is less than the traditional one and the casualty rate is about 10%.

Begon, M. University of Leeds, England.

Preliminary observations on the yeasts associated with Drosophila obscura and D. subobscura.

The Drosophila fauna of Adel Dam Nature Reserve, a mixed woodland near Leeds, is dominated by D. phalerata (a fungal feeder), D. obscura and D. subobscura. The yeasts occurring in the crops of adults of the latter two species have been examined and compared to those occurring in known and prospective feeding sites, as part of an attempt to gain some insight into the field ecology of these species. The results outlined here cover the period late March to late July 1973, during which period the two species occurred in comparable numbers (Shorrocks pers. comm.), and are as follows: (1) During late March and April sycamore (Acer pseudoplatanus) and birch (Betula pubescens) trees bled, and occasionally formed slime fluxes on which males and females of both species fed.

A slime flux of a birch and the crops of flies removed from it showed excellent correspondence with respect to four dominant yeast species, including Rhodotorula rubra and a yeast very similar to Kluyveromyces dobzhanskii (obtained from D. pseudoobscura (Shehata et al. 1955) and D. persimilis (Phaff et al. 1956)). These two species of yeast were also common in crops of flies removed from dripping sycamore sap as was the yeast Aureobasidium pullulans. However only A. pullulans was found in the sap.

No differences emerged between males and females or between D. obscura and D. subobscura in this period.

(2) In late April sap ceased to flow, attempts to cause artificial flows through scoring of bark throughout May, June and July being similarly unsuccessful; during May many bluebells (Endymion non-scriptum) flowered and subsequently rotted. Their yeast flora was as follows: Rh. aurantiaca, Rh. lactosa, Cryptococcus laurentii var laurentii, A. pullulans, plus one other, as yet unidentified.

The crops of D. subobscura caught during this period predominantly contained these same four identified yeasts, plus one other, again unidentified, probably Saccharomyces sp., suggesting the rotting bluebells as at least one of D. subobscura's sources of food; whereas from five D. obscura caught only one Rh. rubra colony was obtained.

However Rh. aurantiaca, A. pullulans and the presumptive Sacch. sp. were all obtained from D. obscura during other periods, and Rh. lactosa was the only one of these species to occur later in D. subobscura, but not in D. obscura. It therefore appears unlikely that D. obscura is incapable of using these yeasts at this time; and, unless explicable by small sample size, the results perhaps suggest an association of D. obscura with its breeding-site as either newly-emerged or actively-breeding adults, since Carson and Stalker (1951) noted a temporal separation between active feeding and breeding, and Carson, Knapp and Phaff (1956) observed that the microflora of breeding sites was strikingly different from that found in the crops of Drosophila adults.

(3) Throughout June and July a total of thirty-two flies were examined. Neither in the yeast species present, nor in the diversity of yeasts per individual fly was any significant between-sex or between-species difference revealed. The yeasts obtained were in the main different species to those obtained during April and May and included A. pullulans, Rh. aurantiaca, Rh. lactosa, Rh. glutinis var glutinis, Torulopsis apicola, Cryptococcus melibiosum and Cr. albidus var diffluens.

Leaf surfaces, rotting flowers, rotting fungi and the rotting vegetation collected in the forks of trees were all examined as potential food-sources, but no pattern of correspondence emerged between these and the crop contents. A. pullulans did however, occur on the leaf surfaces of all eight tree species sampled, and Rh. lactosa on six of them, so that the possibility exists that these constitute part of the food-source.

Thus in June and July, as in April, there appears to be no differentiation of the niches of D. obscura and D. subobscura with respect to adult food.

Had all flies been collected from one site, it might have been argued that the food sources vary spatially, and niche-differentiation varies with food-source. Especially so since Shorrocks (pers. comm.) has found that bait at a height of approximately 6 m. attracts more flies and a higher proportion of D. obscura than bait placed on the ground below it.

Flies caught in this situation, however, revealed no differences in crop contents as a function of bait-height.

Furthermore the food source, whatever it is, appears to be the same throughout the Reserve, since flies caught simultaneously at two sites, 400 m. apart, showed no significant differences in their crop contents.

All of which points towards niche-differentiation, if any, being at the larval or ovi-

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Singh, B.N. Banaras Hindu University, Varanasi, India. Studies on the fecundity of Drosophila ananassae.

Experiments were conducted in order to study the egg-laying capacity of Drosophila ananassae females. Several wild laboratory stocks of D. ananassae raised from flies collected in different localities of India, were utilised during the present investigation. Males and females from each stock were collected within the six hours after eclosion and were grown for 48 hours. The pair mating between females and males from the same stock was done in culture vials. In order to facilitate the counting of eggs a green edible dye was added to the food medium. Each female remained in a culture vial for 24 hours and then transferred to fresh culture vial without etherisation. The eggs laid were counted from the old vial which represented the number of eggs laid by the female within a period of 24 hours. In the similar manner the egg counts were made for all the females for continuously twenty days. If any female died before the twentieth day, the egg-laying data of that female were rejected. The number of females tested varied in different stocks.

The results are summarised in Table 1. The number of eggs laid per female per day varies between 8.67 to 45.64. The χ^2 value has been calculated under the assumption that all the

Table 1. Fecundity of Drosophila ananassae females in different strains.

Strains	Days eggs counted	Number of females tested	Total number of eggs counted	Eggs/♀ for the counted period	Eggs/day/♀
Mughalsarai	20	44	13620	309.54	15.48
Nagpur	20	20	5002	250.10	12.50
Port Blair	20	22	3814	173.36	8.67
Jamsoti	20	47	42902	912.8	45.64
Lowari	20	40	26858	671.45	33.57
ST - Tejpur	20	48	31236	650.75	32.54
a_{25}	20	40	23491	587.27	29.36
Gorakhpur	20	35	24192	691.20	34.56
AL - Tejpur	20	43	22474	522.65	26.13

stocks must possess nearly the same egg-laying capacity. The total χ^2 value is 43.94 which shows highly significant variation. This suggests that the stocks differ in their egg-laying pattern.

The fecundity of D. pseudoobscura is a species characteristic since the several stocks and their crosses yield about the same number of eggs[1]. Thus D. ananassae clearly differs from D. pseudoobscura. Stone et al.[2] investigated the genetic composition of the D. ananassae populations found at the Marshall Islands. The fecundity of D. ananassae was measured. The average eggs per day for the stocks and crosses varied but no consistent relation to genotype was detected. The data reported in the present paper show wide differences as compared to those of Stone et al.[2]. The variability in eggs per day per female recorded in Table 1 reflects genetic differences between strains.

Acknowledgement: The author is indebted to Prof. S.P. Ray-Chaudhuri for guidance.

References: 1. Stone, W.S., F.D. Wilson and V.L. Gerstenberg 1963, Genetics 48:1089; 2. Stone, W.S., M.R. Wheeler, W.P. Spencer, F.D. Wilson, J.T. Neuenschwander, T.G. Gregg, R.L. Seecof and C.L. Ward 1957, Univ. Texas Pub. 5721:260.

(Continued from preceding page)

positional level; a suggestion consistent with the apparent abundance of potential adult food-sources, and the apparent impossibility of finding breeding sites during this period.

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Korochkin, L.I., N.M. Matveeva and A.Yu. Kerkis. Institute of Cytology and Genetics, Novosibirsk, U.S.S.R. Subcellular localization of esterases in D. virilis and D. texana.

Different subcellular fractions of Drosophila were obtained by means of a modified method of differential centrifugation of Schwark and Ecobichon (1968). Fractions A, B, C and D were identified. Each fraction was studied with an electron microscope. Fraction A contained mainly nuclei and cell debris and also large mitochondria. Fraction B contained cell sap. Fraction C contained predominantly mitochrondria as well as lysosomes and a small amount of cell debris. Fraction D was composed mainly of microsomes and of an admixture of small mitochondria. The distribution of esterase activities among the fractions (%) determined by the method of Bamford and Harris (1964) is presented in the Table.

Fraction	D. virilis (stock 140)	n	D. texana (stock 419)	n
A	21.8 ± 0.48	4	13.5 ± 4.5	4
B	24.0 ± 4.0	4	17.3 ± 3.5	4
C	32.0 ± 3.0	4	35.2 ± 4.0	4
D	22.2 ± 3.0	4	34.0 ± 2.0	4

It cannot be ruled out that differences in the activity of the water-soluble esterase between D. virilis and D. texana (this esterase being much fainter in D. virilis) are due to the different distributions of esterase fractions in the cells of these two species of Drosophila.

The zymograms of the subcellular fractions of D. virilis and D. texana yielded by starch get electrophoresis are shown in Figure 1. It should be noted that esterase-2 is mainly expressed in cell sap. In the cell sap of D. virilis esterase-4 is more active than in that of D. texana. There is a certain specificity in the distribution of the fast (F) and show (S)

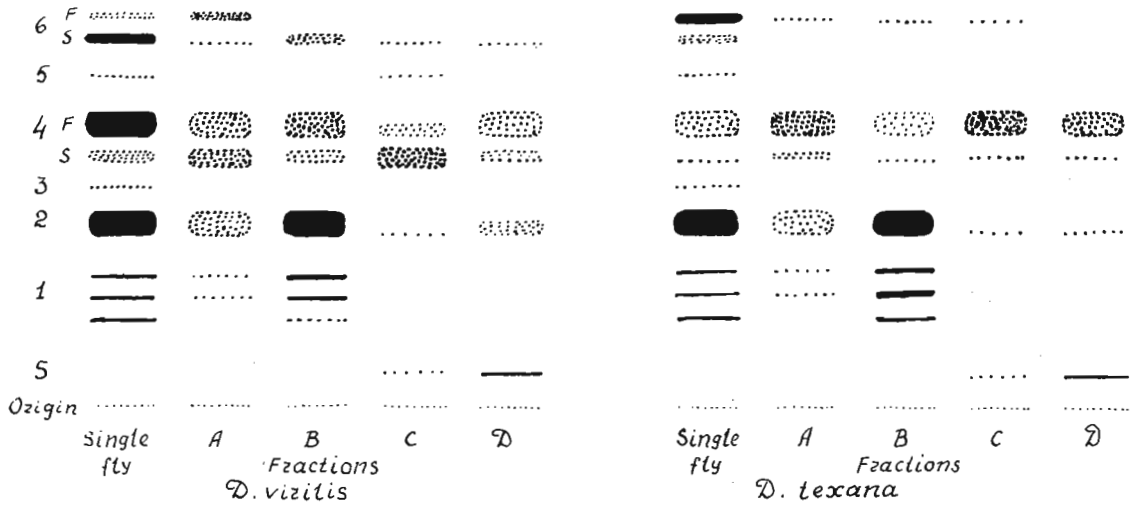

Fig. 1. Electrophorograms

subfractions of esterase-4. Thus, in D. virilis stock 140 esterase-4 predominates in fractions B and D, while esterase-4S predominates in fractions C. In adult flies the activity of the F-subfraction of this esterase is higher. In fractions A both fractions stain equally well, but in contrast to the other fractions, only the fast (F) subband is distinct in esterase-6 (Figure 1). After treatment of the lyophilized fractions with 0.1% Triton X-100 it was possible to extract the slow fraction, which is usually undetectable in D. virilis and D. texana, but seen under certain experimental conditions (insertion of fragments of D. texana 5th chromosome into the 5th chromosome of D. virilis). Furthermore, we have isolated a mitochondrial fraction from D. virilis (stock 140) by the method of Polan et al. (1973). The electropherograms are similar to the ones described above. The data obtained should be taken into consideration when analyzing changes in the relations between different esterase fractions in the course of Drosophila ontogenesis and also when estimating organ specificity in esterase

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Milkman, R. and M. Kratoska. University of Iowa, Iowa City. Cage Drosophila go away to die.

Eleven standard 20-vial Lucite cages were established from one inseminated female (derived from a common cage) each. The terminal pair of vials was left empty. Dead and dying flies accumulated there primarily. An array of experiments has been undertaken to investigate the possibility that old and otherwise "inferior" flies flee or are driven away from food vials and the flat cage surfaces. These include the following: 1) Food vials were removed and the medium searched. Very few adult remains were seen (when food pulls away from the side, many flies are often trapped; this is a frequent but unnecessary occurrence). The floors of the eleven cages revealed a total of four dead flies over several days, while scores of flies accumulated in the death vials. 2) Death vials were left in the cage for varying periods. Accumulation of dead flies was roughly linear; number of "dying" flies (from normal-looking to paralyzed) remained constant. 3) Over 400 "dying" flies and over 400 "normal" flies attracted from the main part of the cage with light were placed in food vials, 20 flies per vial. The "dying" flies' median survival time was 26 days; normal flies', 52 days. The "dying" flies were essentially all fertile (both sexes) in additional tests, with the exception of those that didn't survive etherization. 4) Under these conditions (room temperature, about 20 flies/vial), 348 normal cage flies had a median survival time of 23 hours in plugged empty vials (maximum 71.5 hours). 5) Six vials of marked (n b cn bw) flies were substituted at once for a period of three days in each of two cages. Of the 167 eventually appearing in the death vials, 64 appeared in the first 3 days, and the rest appeared sporadically in declining numbers until 40 days later, after which none appeared. 72 were found in the cage food vials that were removed on regular schedule, of which all were alive. 6) Half of the producing food vials were removed from each of two cages; emergent flies were counted daily for three days, leading to an estimate of emergence per day of 86 and 198, respectively. The cages were censused by direct count and mean adult longevity computed to be 1570/86 = 18 days for one cage and 4010/198 = 20 days for the other. 7) The remaining 9 cages, similarly censused, averaged 2468 flies (range: 1850-3355). With a mean adult longevity of 19 days, a mean daily death (and emergence) rate of 130 is indicated. The mean daily death rate observed in the death vials over a three-month period was 60.

The use of marked strains in cages and in two- and three-vial connecting sets has been undertaken to explore the possibility of determining (transient) behavioral dominance hierarchies and using these status values to permit selection for longevity (result of discussion with F.A. Lints) and other difficult traits. We assume that any two laboratory cultures will each contain flies with a wide range of vitality and so overlap to a degree preventing absolute separation of strains by the emigration of one strain from a mixture. Preliminary results are consistent with this view, although differential emigration is evident. Prescreening to narrow the vitality range will be employed in future experiments. These emigration experiments differ from those of previous investigators in that flies migrate to an empty vial, with no food.

When cages contain no empty vials, the newest food vials contain very large numbers of males and females. Many of these are dead and dying, so that in ordinary cages each food vial serves as a graveyard (as well as a nursery simultaneously) in its turn. This phenomenon is likely to have something to do with the fact that cage flies are sizable and vigorous, rather than scrawny and weak, as they would be if an essential nutrient limited population size directly.

(Continued from preceding page)

distribution.

Acknowledgments: We are grateful to Prof. Sokoloff and Dr. M. Evgeniev (Moscow) for supplying us with flies used in this study.

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Bijlsma, R. and W. van Delden. University of Groningen, Haren, The Netherlands.

Polymorphism at the G6PD- and 6PGD-locus in D. melanogaster.

Eleven D. melanogaster populations from different geographic origins and kept in the laboratory for different periods as cage populations, were assayed for variation of the enzymes glucose-6-phosphate dehydrogenase (G6PD) and 6-phosphogluconate dehydrogenase (6PGD) by means of electrophoresis. Electrophoresis was carried out on polyacrylamide gel using a 0.15 M tris-citric acid buffer pH 7.5 and staining was done in 0.1 M tris-HCl pH 7.5, 100 ml supplemented with 15 mg NAPD, 20 mg substrate (G6P or 6PG), 20 mg M.T.T. and 2 mg P.M.S. Both loci are located on chromosome 1, G6PD at 63.0 and 6PGD at 0.9. For both loci a "slow" and a "fast" allele is known. Allelic frequencies were determined in 1971 and, for some populations also in 1972 (Table 1).

Table 1. Allelic frequencies in the population.

population	origin	in the laboratory since	freq. S-allele G6PD 1971	freq. S-allele G6PD 1972	freq. S-allele 6PGD 1971	freq. S-allele 6PGD 1972
1) Bogota	Columbia	1965	.68	.67	.05	.09
2) Curacao	Neth. Antilles	1971	.78	.74	.12	.04
3) Kaduna	Nigeria	1949	.51	-	.0	-
4) Groningen 67	Netherlands	1967	.05	-	.0	-
5) Groningen 69	Netherlands	1969	.12	.06	.19	.20
6) Groningen 71	Netherlands	197.	.19	-	.0	-
7) Haren 71	Netherlands	197.	.10	-	.0	-
8) Loenen 71	Netherlands	197.	.14	.12	.03	.02
9) Jerusalem	Israel	1970	.04	-	.0	-
10) Pacific 1	Pacific coast USA	1956	.28	-	.0	-
11) Evanston	Illinois USA	1969	.31	-	1.0	-

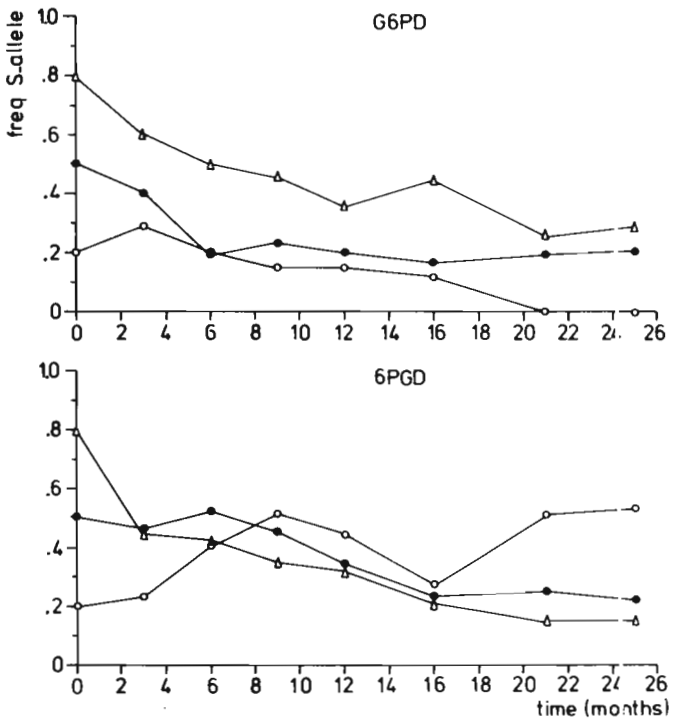

Figure 1. Change in S-frequency in the artificial cage populations (see text).

It can be seen that all populations are polymorphic for G6PD, though allelic frequencies differ considerably. Most populations are monomorphic for 6PGD, with the F-allele fixed (except the Evanston population) in all four polymorphic populations the S-frequency is low. A second determination of the allelic frequencies in the populations 1, 2, 5 and 8, one year later (1972), shows that the frequencies remain fairly constant (Table 1). This suggests that the allelic frequencies are rather stable. It is also interesting to note that populations, in spite of their long laboratory history, can still be polymorphic.

A further investigation was done on artificial cage populations. Three populations were set up with different allelic frequencies for both loci (0.20 S, 0.50 S and 0.80 S) by crossing two reference strains: the Tuscaloosa strain, homozygous slow for G6PD and homozygous fast for 6PGD, and the Oregon-R strain, homozygous fast for G6PD and homozygous slow for 6PGD. The changes in allelic frequencies in these populations were determined at intervals (Figure 1). It must be noticed

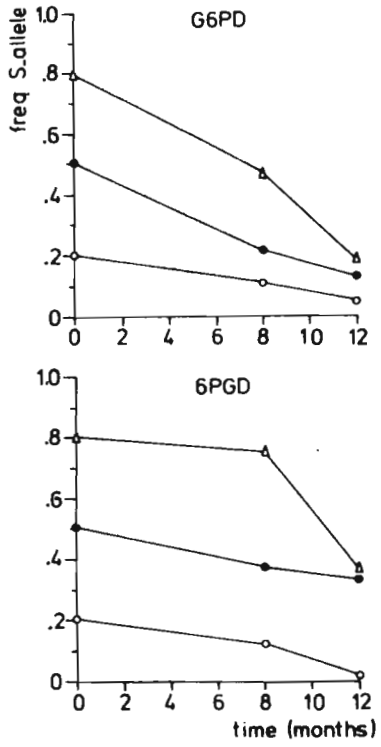

Figure 2. Change in S-frequency in the cages established with isolated lines (see text).

that the strains probably have different genetic backgrounds which probably influence the results. Ten new lines of each homozygous genotype were then isolated from the .50 S cage. This was done 6 months after this cage was established. With these lines new cages were established with initial frequencies 0.20 S, 0.50 S and 0.80 S for both loci (Figure 2).

Figures 1 and 2 show the same trend for both loci in most cages. The initial high frequencies of the S-alleles decrease rapidly. The first and second set of cages do not differ much after twelve months, most allelic frequencies are between 0.10 - 0.20 S. The rapid allelic frequency changes found for both loci strongly suggest the action of selection. Other experiments, now in progress, support this statement. The decrease of the S-frequency of the 6PGD locus is consistent with what is found for the eleven natural populations, in which the S-allele is absent or only present in a low frequency (the Evanston population being the one exception).

Whether the S-frequencies in the artificial populations will stay at the present low level, or the F-alleles will become fixed, as occurred already in one cage, can not be predicted at the moment. Further experiments, including natural populations, are started now to investigate the nature of the selective forces discovered.

Grateful acknowledgement is made to Dr. W.J. Young, University of Vermont, for supplying the Tuscaloosa and Oregon-R reference strains.

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Springer, R. Institut für allgemeine Biologie, Vienna, Austria. Light-independent mating, probably a dominant character of behaviour in D. subobscura.

Selection of a Viennese wild strain (caught in mass in 1966) by means of gradual reduction of invariable day-and-night-illumination to zero within 14 generations gave rise to several strains that have been cultivated in complete darkness for over five years since. Sub-lines brought back into light and cultivated normally for eight generations or more kept their ability to mate in darkness with only a slight decrease of the percentage of successful matings. The F_1 of nearly all crossings of the selected strains with wild type or marked strains of different origin yielded small percentages of individuals able to mate successfully in darkness. The F_1 and F_2 of a crossing between strain Küsnacht and Vienna 30, both structurally homozygous, with the gene-arrangement J_1 as the only deviation from Standard type in the Vienna 30 strain, showed a significantly lower threshold of required brightness for mating, compared to the parental strains. To eliminate the possibility of a non-genetic mechanism of "inheritance" as, for instance, virus infection, Viennese wild strain and "Light-independent" flies respectively were squashed and added to the food medium. Wild strain was tested on "Lin"-infected medium, vice versa, in darkness. Even after three generations of repeated cultivation of the tested strains on infected medium, no influence of the treated food on the mating was observed.

For the present the genetic diagnosis of the "character" Light-independent can be summarized as caused by a dominant major gene heavily influenced in its penetrance by modifiers and environment. Dominant inheritance possibly happens only as an effect of heterosis when outcrossing the heavily inbred Lin strain with other lines. Due to the variable penetrance of the character no attribution to a linkage group has been secured yet.

Sharma, R.P. Indian Agricultural Research Institute, New Delhi, India.

wingless - a new mutant in D. melanogaster.

Among the Ethyl methanesulphonate induced sex linked recessive lethals, maintained for the analysis of temperature sensitive mutants, wingless flies were recovered in one stock. These flies were freed from lethal bearing X-chromosome and were subjected to genetic analysis. The results obtained showed that the wingless condition (see Figure) was governed by a single recessive gene, designated wingless (wgl), and mapped on the left arm of the second chromosome at approximately 30 map units distance. Further, the wingless phenotype was not completely stable. The progeny of wingless parents were comprised of flies with no wings, one wing and two wings in approximately 2:2:1 ratio. The segregation pattern was consistant. Progeny raised by crossing one-winged x one-winged and two-winged x two-winged (isolated from the progeny of wingless flies) also segregated into the three phenotypes. The ratio of the three classes, too, was similar to that obtained in the cross of wingless flies. This segregation pattern suggests that individuals with the three phenotypes are genotypically similar and that changed phenotypic expression is the result of incomplete penetrance.

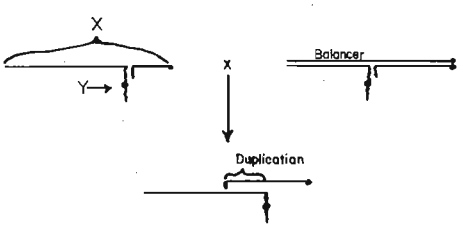
Besides affecting the wing, the wgl mutation has an associated effect on haltere development. In the wingless stock, flies with none, one or both of the halteres occurred. The suppression of wing and haltere development was, however, independent since flies with all combination of wing and haltere number were produced in the progeny of wingless parents. This points to the fact that there is complete autonomy in wing and haltere development but suggests that the critical stages during wing and haltere differentiation are controlled by similar steps.

Williamson, R.L. and W.D. Kaplan. City of Hope Medical Center, Duarte, California.

A duplication that causes leg shaking.

We have found that male and female flies which carry Dp(1;4)f^+ exhibit abnormally rapid and vigorous leg shaking under ether anaesthesia. According to Dr. George Lefevre, Jr. (personal communication) these flies are duplicated for at least 14A1 - 16A1. It was not clear whether the shaking is caused by a duplication of normal material or by a dominant mutation associated with the duplication.

Accordingly we crossed to each other X;Y translocations (obtained from Bowling Green) with different breakpoints to produce duplications in the 14A1 - 16A1 region as shown at left. Using this method, shaking flies were produced from parents which did not shake. Because of a genetic discrepancy in one of the translocation stocks, we decided to examine the progeny of translocations whose cytology had been more recently determined. These stocks were kindly given to us by Drs. Barbara Stewart and John Merriam. A summary of the cytology of the duplications and their associated behaviour is shown at left.



Duplication Cytology	Behaviour
13F - 15DE	Shakes
15B - 17BC	Normal
13EF - 14F	Shakes
13A - 13F	Normal

Thus far the shortest duplication to cause shaking is 13F - 14F. We are attempting to localise the operative duplication and to discover its spacial and functional relation to known behavioural mutations in this region.

Postlethwait, J.H. University of Oregon, Eugene. A quantitative juvenile hormone assay on Drosophila.

The Cecropia juvenile hormone (JH) has been shown to have a number of morphogenetic effects on Drosophila (1-4). These include on the abdominal tergites and sternites a reduction in bristle size, abnormal bristle morphology, a reduction in bristle number, regions without any hairs (trichomes), regions without any pigment, failure of male genitalia to rotate and decrease in eclosion frequency. In order to provide a useful JH assay, these parameters have here been quantified.

Oregon RC animals were cultured as usual and individual animals were collected as white prepupae, either before or after JH treatment, to provide animals of known age at the time of hormone application. JH was dissolved in acetone and delivered topically to animals in 0.3 µl drops. Eclosed or uneclosed animals were fixed in 70% ethanol 7 days after pupariation.

The abdominal cuticle was mounted between two cover glasses and examined under 400X magnification. The 5th tergite of the male and the 6th tergite of the female were scored, as were the three posterior most sternites. A total of 1071 JH treated animals provide the data for Figures 1 and 2. An additional 359 animals provide the data in Table 1.

Table 1. Relative abilities of 24 JH analogues to block metamorphosis.

Analogue	% aberrant bristles per tergite	Cecropia JH equivalent*
Epoxygeranyl sesamole	95	3,400
Iso C_{17}JH	55	120
C_{16}JH	39	56
C_{18}JH	36	34
C_{18}JH	19	19
C_{18}JH + C_{16}JH	19	19
C_{18}JH + sesamex	13	16
C_{17}JH	18	20
Epoxygeranyl-p-ethyl denzene	19 19	19 19
Epoxygeranyl-p-proply benzene	8	10
Geranyl sesamole	5	6
Chloromethyl C_{16}JH	4	4
C_{16}episulfide	4	5
Bishomofarnesoate	1	2
Williams-Law	0.4	2
Ethyl dichloro farnesoate	0.2	1
Farnesenic acid	0.1	1
Farnesol	0	1
C_{18}imino JH	0.1	1
C_{16}imino JH	0	<1
C_{17}aldehyde	0	<1
Geranyl-Methyl-benzoate-dihydro chloride	0	<1
Epoxy geranyl-o-p-bensoic acid methyl ester ether	0	<1
Geranyl-o-p-benzoic acid (methyl ester) ether	0	<1
Sesamex	0	<1
Acetone	0	<1

*Amount of cercropia JH required to cause the same degree of abnormality.

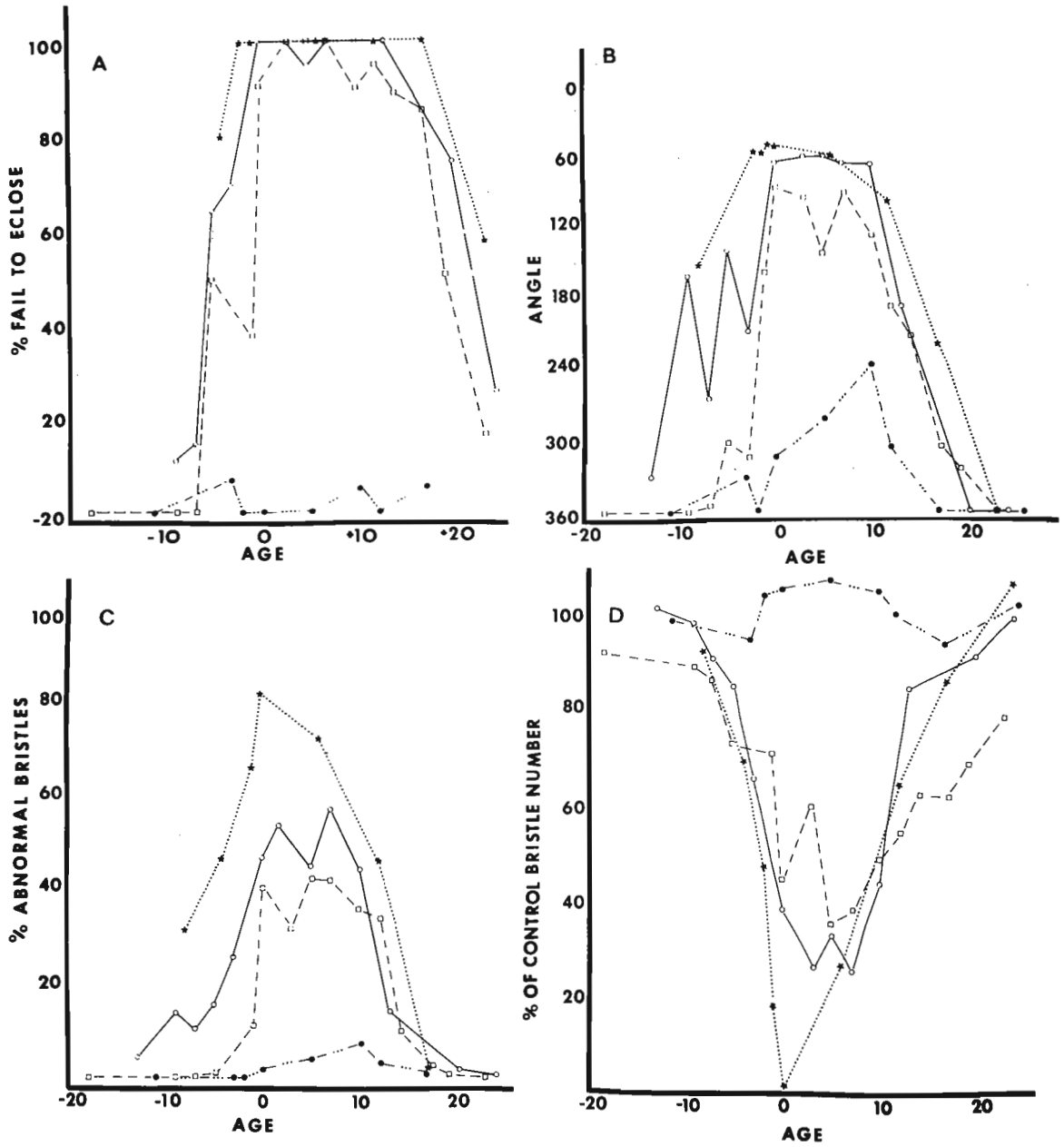


Figure 1. Delineation of the JH sensitive period. A. % of treated animals that fail to eclose by seven days after pupariation. B. The angle of rotation of male genitalia. C. % abnormal bristles on the tergite. D. Bristle number on the tergite as % of control. Stars: 3,400 μg/g; Empty circles: 340 μg/g; Squares: 34 μg/g; Filled circles: 3.4 μg/g.

To insure a sensitive assay the most sensitive developmental stage must be found. Figure 1 shows how four parameters - eclosion frequency, angle of rotation of male genitalia, fre-

quency of abnormal tergite bristles, and bristle number - vary with age. Each point represents 10 - 20 animals. The most sensitive stage includes the first 10 hours after eclosion.

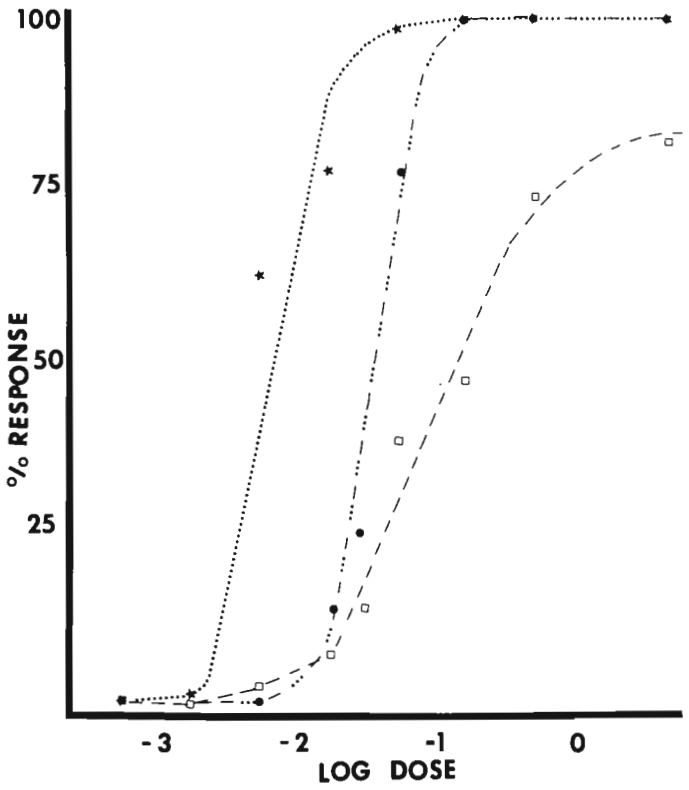


Figure 2 shows the dose-response curve for sternite and tergite bristle number, and eclosion frequency. The threshold response for the posterior sternites is 0.002 µg/individual. Figure 3 shows the sternites from a graded series of JH doses, as well as normal adult cuticle and normal pupal cuticle.

Figure 2. Dose-response curve for JH applied to white prepupae. Filled circles: failure to eclose; Squares: % aberrant bristles on tergite; Stars: % aberrant bristles on sternite.

Several JH analogues were tested using this assay, and the results are given in Table 1. All analogues were delivered at 34 µg/gm animal, and the results are recorded as the dose of cecropia JH required to give quantitatively similar results.

The dose resulting in 50% morphological inhibition of metamorphosis ($I.D._{50}$Morph.,5) is 5 µg/g live weight. The $I.D._{50}$Morph. for topically applied Cecropia JH is about 8 µg/g for Tenebrio and 25 µg/g for Pyrrhocoris (5,6). So Drosophila is about as sensitive as some other insects. The JH sensitive phase is during the period the abdominal histoblasts are dividing most rapidly (7-10).

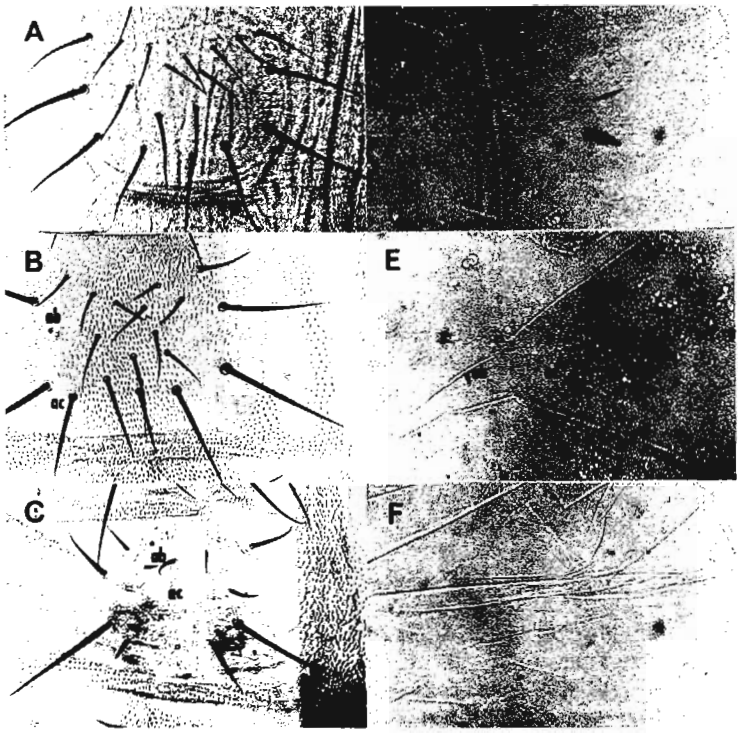


Figure 3. Response of sternites to graded dosages of JH. A. Acetone treated control sternite. B. .002 µg JH/animal. C. .016 µg JH/animal. D. .05 µg JH/animal. E. .16 µg JH/animal. F. Pupal cuticle from an acetone treated control. ab: abnormal bristle; ac: aberrant cuticle.

Supported by a Rockefeller Foundation Grant to Professor C.M. Williams and NIH Grant GM19307 to J.H.P.

References: see next page.

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Vogel, E. Zentrallaboratorium für Mutagenitätsprüfung, Freiburg i. Br., Germany. Strain variations in response to certain indirect mutagens in D. melanogaster.

Strain differences in sensitivity to insecticides such as DDT, parathion and others, as well as cross-resistance are well-known in Drosophila. Among the factors considered to cause such effects is variation in enzyme activity of mixed-function oxidases localized in the microsomes (e.g. R.L. Metcalf, Ann. Rev. Entomol. 12:229, 1967). Since metabolic activation of indirect carcinogens such as aryldialkyltriazenes and azoxyalkanes is also performed by mixed-function oxidases (R. Preussmann et al., Ann. Acad. Sci. 163:697-716, 1960), the question that presented itself was whether similar effects might occur with respect to chemical mutagens.

The strains selected to study this question were our wild strain Berlin K and a resistant one, Hikone R. Dosage-mortality effects and the induction of X-chromosome recessive lethals were analyzed by treating adult males of the two strains. 1-2 day old males were exposed to test solutions of 1.0 mM/l 2,4,6-trichloro-phenyldimethyltriazene or 1.3 mM/l azoxymethane for three days and recessive lethals tested for. To recover stage-dependent sensitivity differences, three broods of three days duration each were set up (Table 1).

Table 1. Frequencies of X-chromosome recessive lethals induced by 2,4,6-trichloro-phenyldimethyltriazene (a) and azoxymethane (b).

Expt.	Strain	Brood I leth./chrom.	%	Brood II l./chr.	%	Brood III l./chr.	%	I-III (II) l./chr.	%
a	Berlin K	131/553	23.7	102/364	28.0	sterile		233/917	25.4 ± 1.4
	Hikone R	29/617	4.7	39/603	6.5	20/753	2.7	88/1973	4.5 ± 0.5
b	Berlin K	6/617	0.97	27/582	4.6	2/202	0.99	35/1401	2.5 ± 0.4
	Hikone R	5/608	0.82	49/603	8.1	sterile		54/1211	4.5 ± 0.6

The experiments revealed pronounced differences in mutation frequencies between both strains. Recessive lethals were induced to a much greater extent in Berlin K males by the triazene, while more lethals were produced by azoxymethane in Hikone R males. Analyses of the data from the different brood pattern experiments (I - III) using the χ^2 test revealed highly significant differences between the samples.

With the compounds so far tested, there was a positive correlation between toxicity and genetic activity for triazenes and azoxyalkanes. Triazenes were more toxic to Berlin K males, while Hikone R males showed higher sensitivity to azoxymethane (as well as the structural isomer of azoxyethane-diethylnitrosamine).

The data are interpreted to be due to genotype-dependent differences in activation of these indirect mutagens resulting in differing concentrations of mutagenic products in various parts of the body including the gonads. This assumption is supported by:

(1) the positive correlation between mutation frequency and the observed sterilizing effects.

(2) the inhibitory action of proper enzyme inhibitors on mutation induction by indirect mutagens (Vogel, unpublished), and

(3) our finding that the mutation frequency in Berlin K - Hikone R hybrids (Berlin K ♀♀ x Hikone R ♂♂) treated with the triazenes is almost exactly half that in the wild strain.

Whatever the correct explanation of the result is, the data show that group-specific cross-resistance to certain chemical mutagens seems to exist in Drosophila.

Nirmala Sajjan, S. and N.B. Krishnamurthy. University of Mysore, Manasagangothri, Mysore, India. Competition studies between D. ananassae and D. melanogaster.

D. ananassae and D. melanogaster, the two cosmopolitan, sympatric and domestic species of the melanogaster species group, were subjected to competition both at the preadult and adult stages of the life cycle. Monomorphic and polymorphic strains of D. ananassae and highly inbred strains of D. melanogaster were employed in these experiments. By adapting the procedure of Ayala (1965) the relative and competitive fitnesses of the above strains have been estimated. Preadult competition was analysed at 4 different densities - 100, 200, 400 and 1,000 eggs per 250 ml milk bottle containing equal amounts of media. The complete dynamics of these species in inter- and intraspecific competition will be discussed elsewhere.

Some of the observations of the above experiment are presented here. The relative fitness, as measured by the productivity and the total population size, is more for D. melanogaster than D. ananassae. The fitnesses of monomorphic strain of D. ananassae and highly inbred strain of D. melanogaster were found to be superior to the corresponding polymorphic and less inbred strains. In interspecific competition, irrespective of the strains in the process, D. ananassae was eliminated by D. melanogaster. The preadult competition has revealed that as the density increases, the mean developmental time of the species also increased while the viability was lowered. D. ananassae manifests a faster rate of development while D. melanogaster exhibits a higher grade of viability. The mean developmental time of the pure cultures of both the species is significantly less than the corresponding mixed cultures. This indicates that some sort of interference occurs when they compete for food resulting in lengthening of the developmental period.

Thus, D. melanogaster is a better competitor because of its higher degree of egg to adult viability and more relative fitness than D. ananassae. In view of this, it is felt that the sympatric coexistence of these species in nature may be mediated by their yet unknown non-overlapping microhabitats.

Acknowledgments: The authors are deeply indebted to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology, for many constructive suggestions and criticisms. This work is financially supported by C.S.I.R.

Reference: Ayala, F.J. 1965, Genetics 51:527-544.

Mulley, J.C. University of Sydney, Australia. Electrophoretic detection of pyranosidase in Drosophila buzzatii.

In D. buzzatii the detection of β-glucosidase and β-galactosidase on duplicate electrophoretograms gave identical banding patterns. This indicated that the one enzyme was involved and a problem of nomenclature therefore exists. The difficulty was overcome by referring to the enzyme as pyranosidase since synthetic pyranosides were employed as substrates.

Electrophoresis was done on density gradient acrylamide gels. Gels were run at 300 V for 2 hours in a tris-borate buffer (0.1 M, pH 8.9). Enzyme detection was extremely simple. The pH of the gel was lowered in phosphate buffer (0.1 M, pH 6.0) for 15-30 minutes. To this was added 10 mg 4-methylumbellicalfenyl-2-acetoamido-2-deoxy-β-D-glucopyranoside or the corresponding galactopyranoside (obtained from Koch Light). After 15-30 minutes the fluorescent bands were scored under ultraviolet light (3,500 Å).

Variation was detectable in Australian populations. Banding patterns were found to be inherited in a simple Mendelian fashion.

$$S \times F \longrightarrow \text{all } S/F$$
$$S/F \times S/F \longrightarrow 11F : 21\ S/F : 8\ S$$

Gene frequency was highly stable (between .83 and .95) in 10 populations sampled from 8 localities where the greatest distance between localities was over 500 miles. This stability in gene frequency may well be a reflection of the uniform habitat of D. buzzatii which colonise only the rotting stems and fruit of certain cacti of the genus Opuntia.

Acknowledgements: This study is part of a program on the relative fitness of Drosophila populations supported by a grant from the Australian Research Grants Committee to Associate Professor J.S.F. Barker. I am grateful to Dr. I.R. Franklin for suggesting the application of this assay to D. buzzatii.

Guzmán, J., De la Rosa, E., Félix, R. and Olvera, O. National Institute of Nuclear Energy, Mexico City, Mexico. Preliminary results on the radioprotective effect of butylated hydroxytoluene on D. melanogaster.

During irradiation with high energy radiation, free radicals are produced which are generally considered responsible for the induction of biological damage. Irradiation of organisms with ionizing radiation causes mutations, some of which can be identified as recessive lethals, as well as chromosome losses due to breakage. There is evidence although indirect, that changes produced by irradiation and those which arise spontaneously in the cell, have free radicals as a common source. These arise respectively, by the dissociation of water and by the interaction of oxidative enzymes with O_2 and H_2O_2 (Harman, 1956). Radiation-induced free radicals are found along pathways randomly distributed throughout the entire cell. Therefore, they are more likely to reach and to react with cellular materials, such as the DNA of the nucleus, than the same number of endogenously originated radicals which act in circumscribed regions (Harman, 1962).

This difference in the distribution of radicals would account for the longer period of time generally required to produce a given effect by radicals of metabolic origin in comparison with the ones formed by irradiation. The damage produced by free radical reactions would be expected to be proportionately greater as the length of the chain reactions increases (Nesrobian and Tobolsky, 1961; Swern, 1961). Lipid peroxidation is cited as a cause of biological damage in various pathological states. In vitro peroxidation of lipid in vitamin E deficiency in numerous animal tissues has been reported; however, there is a paucity of definitive in vivo studies. The reaction of ozone with carbon-carbon double bonds of unsaturated fatty acids has been known for many years (Goldstein and Buckley, 1970).

When one considers that 2-mercaptoethylamine is an efficient free radical scavenger and has therefore a radioprotective action (Harman, 1969), it is reasonable to assume that BHT (2,6-di-tert-butyl-p-cresol), being another active free radical scavenger, might show radioprotection when administered as a food additive to fruit flies. It has been shown previously, that BHT prolonges the life span of mice (Harman, 1968) and Drosophila melanogaster (Félix et al., 1970).

Male flies from a strain "Oster male" containing a marked sc^8 Y chromosome and the closed X^{c2} with the mutations yellow (y) and Bar (B) in the males, were mated after being aged during 3 days to isolated virgin "Oster females", with markers in the X, II and III chromosomes (y sc^{S1} In49 sc^8;bw;st p^P). Each female was aged during 3 days before mated to 2 or 3 males in each vial, in order to identify clusters of XO individuals originated in pre-meiotic events.

The F_1 flies were separated to determine the frequency of exceptional males which appeared yellow in contrast to the normal class of males expected. The latter had non-yellow bodies, since they carried the normal dominant allelomorph (y^+) of yellow in the sc^8 insertion of their chromosome. The yellow males represent cases of loss of the whole or part of the X- or Y-chromosome. The use of the markers Bar and yellow to identify the treated sex chromosome of the males, makes the detection of sex linked recessive lethals fairly easy. F_1 males and females were allowed to mate with each other for at least two days before being separated into vials. The F_1 fertilized females were tested for lethals by examination of the F_2 offspring for the absence of Bar males, which would indicate that a lethal was induced in the paternal chromosome. BHT (Egon Meyer) was added to the culture medium at a concentration of 0.2g/100 ml. As BHT is sparingly soluble in water, it was dissolved in propionic acid, which is a normal component of the medium.

The Oster male flies received the following treatments: while a control group was kept on normal medium, a second group was irradiated when 3 days old with 2,500 r and mated immediately in vials to virgin Oster females. A third group was fed before and after irradiation with 2,500 r, with BHT added. A Co^{60} source Gamma-Cell 200 model was employed for the irradiation treatment.

Table 1. Percentage of X-chromosome loss (Def. 1. Trout, H., 1964)

Control	2,500 r	BHT+ 2,500 r
0.39 (5/1275) ± 0.17	0.88 (7/792) ±0.33	0.55 (3/539) ± 0.32

The chi square and P values for the obtained deviations are as follows:

a. (2,500 r)-(control), $\chi^2 = 4.742$, $P < 0.05$
b. (2,500 r)-(BHT + 2,500 r), $\chi^2 = 0.632$, $P > 0.30$
c. (BHT + 2,500 r)-(control), $\chi^2 = 0.368$, $P > 0.50$

(Continued at bottom of next page)

Godbole, N.N. and V.G. Vaidya. University of Poona, India. Drosophilid survey of Mahabaleshwar.

Mahabaleshwar, a well-known hill station in the State of Maharashtra, India (Lat. 17°51' N, Long. 73°30' E), is situated at an altitude of about 1570 m above mean sea level. The township and the surrounding hilly area of about 130 sq km was chosen for the survey. Most of this area is covered by dense forest and underlying bushy vegetation. The temperature ranges between 18°C and 23°C. The average annual rainfall is about 625 cm. The wet season extends from July to October.

The survey of Drosophilidae was undertaken for a period of four years beginning in August 1969, during which frequent collections were made covering all the seasons of the year. The following localities were visited, which represented many different types of ecological habitats: 1) Bazar area, 2) Chinaman's Waterfall, 3) Bombay Point, 4) Lodwick Point, 5) Dhobi Waterfall Ride, 6) Arthur Seat, 7) Old Mahabaleshwar, 8) Venna Lake, 9) Wilson Point, 10) Babington Point and 11) Tigerpath Ride.

The collections were made mainly by sweeping with net and by placing banana baits. The flies were found on garbage, around decaying leaves, on exuding sap of trees, etc. and were abundant during the months of November to June. During the months of July to October, a period of heavy rainfall, very small numbers of flies could be collected. Most of the species collected except two (shown with asterisks) could be reared in the laboratory on the standard cornmeal-agar medium.

The following twelve species were collected which include two new species and a new report from India. Three genera, Drosophila, Leuphenga and Stegana, are represented.

1 Drosophila (Sophophora) biarmipes
2 Drosophila (Sophophora) melanogaster
3 Drosophila (Sophophora) ananassae
4 Drosophila (Sophophora) malerkotliana
5 Drosophila (Sophophora) jambulina
6 Species of Sophophora (new species)
7 Drosophila (Drosophila) nasuta
8 Drosophila (Drosophila) repleta
9 Drosophila (Scaptodrosophila) latifshahi
10 Leuphenga (Leucophenga) guttiventris
*11 Leuphenga (Leuphenga) subpollinosa (new report from India)
*12 Stegana (a new species of Steganina subgroup allied to S. excavata)

Acknowledgement: The authors are grateful to the Head of the Zoology Department of the Poona University for facilities.

(Continued from preceding page)

As there is not a significant difference in groups b and c, the results for XO chromosome loss are not conclusive.

Table 2. Percentage of X-chromosome recessive lethals

Control	2,500 r	BHT+ 2,500 r
0.37 (1/274) ± 0.37	3.06 (18/589) ± 0.71	0.70 (2/287) ± 0.49

The chi square and P values for the obtained deviations are as follows:
a. (2,500 r)-(control), $X^2 = 14.651$, $P < 0.001$
b. (2,500 r)-(BHT + 2,500 r), $X^2 = 11.223$, $P < 0.001$
c. (BHT + 2,500 r)-(control), $X^2 = 1.006$, $P > 0.020$

These data indicate that BHT added to the food medium of D. melanogaster is an effective radioprotector when the percentage of sex-linked recessive lethals is estimated.

References: Félix, R., J. Ramírez, V.M. Salceda and A. de Garay 1970, DIS 45:121-123; Goldstein, B.D. and R.D. Buckley 1970, Science 169:605-606; Harman, D. 1956, J. Geront. 11: 298-300; Harman, D. 1962, Radiat. Res. 16:753-763; Harman, D. 1968, The Gerontologist 8: 13; Harman, D. 1969, J. Am. Geriat. Soc. 27:721-735; Nesrobian, R.B. and A.B. Tobolsky 1961, Autooxidation of hydrocarbons accelerated by metals, light and other agents. Lundberg Interscience, N.Y., Vol. 1:107-131; Traut, H. 1964, Mutation Res. 1:157-162; Swern, D. 1961, Autooxidation and antioxidants. Lundberg Interscience, N.Y. 1-54.

Sreerama Reddy, G. and N.B. Krishnamurthy.
University of Mysore, Manasagangothri,
Mysore, India. Photomap of the salivary gland chromosomes of D. ananassae.

For the first time, a photomap of the salivary gland chromosomes of Drosophila ananassae of a homozygous strain from Perumalmalai (South India) has been made. The salivary gland chromosome complement consists of six arms radiating from a conspicuous chromocenter. The two short arms represent X-chromosome and the remaining four arms represent the second and third chromosomes. The fourth chromosome being heterochromatic is incorporated into the chromocenter.

The photomap of six arms of the salivary gland chromosomes (Figure 1) is divided into 1/166 sections, approximately of equal lengths. The numbering of sections is continuous, starts from the terminal end of the left arm of the X-chromosome and ends at the tip of the right arm of the third chromosome. The number of sections in each arm indicates the relative length of that arm. For instance, the longest arm 2L has 43 sections while the shortest arm XR has only 11 sections. Of the remaining four arms, XL has 20 sections, 2R-32, 3L-35 and 3R has 23 sections. The diagnostic features of each arm are:

XL is divided into 1/20 sections. It can be easily recognised by a flared tip followed by a puff with two dark bands in the region 1, a large roundish and constant puff in the region 4, which is followed by another small puff in the region 5. Other notable features of this arm are the presence of an oval puff with three dark bands in the region 11, a round puff with a dark band and faintly stained dot band in the region 15, a rectangular puff with four dark bands in the region 19 and a bulb at the basal region followed by a dark band and a thin band in the region 20.

XR is the shortest arm with sections 21 to 31. The guiding features of this arm are the presence of three puffs each in the regions 24, between 27 and 28 and 30. The distal end is fan-shaped, followed by two dark bands. Another diagnostic feature of this arm is the vase-shaped puff in the proximity of the chromocenter.

2L is the longest of all the arms with sections 32 to 75. The characteristic bell-shaped terminal end is one of the constant features of this arm. This is followed by a dark band in the constricted portion of the region 32. Certain remarkably constant puffs are noticed in the regions 37, 39, 42 and 53. The puff in the region 42 has four well-defined dark capsules. Other prominent features of this arm are the presence of two large puffs in the regions 64 and 65 and thick dark bands in the regions 66, 67, 68 and 70. The basal region of this arm shows the presence of two small puffs in the regions 73 and 74 which form important land marks in the identification of this arm.

2R is intermediate in length between 2L and 3L. The sections are from 76 to 108. A small puff-like enlargement between 77 and 78, followed by three distinct dark bands in the region 79, an oval puff in the region 80 and two constant oval puffs each in the regions 93 and 97 are some of the diagnostic features of this arm. In addition, the terminal portion of this arm can be easily recognised by three small puffs in the regions 104, 106 and 107. The arm ends with a slightly flared tip containing small indistinguishable vesicles.

3L is the second longest arm, with sections from 109 - 143. The notable features of this arm are three consecutive puffs in the regions 112, 113 and one between 114 and 115. The puff present in region 113 has two shields. The region 118 has a roundish puff with a central thick dot band and a thin band on either side of it. One of the additional features of this arm is the presence of a series of dark bands and chain bands between the regions 123 and 129, of which the region 127 and 129 are characteristically enlarged to form round puffs. The basal region is characterised by three large puffs of which the first one is present in the region between 135 and 136 and the other two are in the regions 138 and 140. The puff present in the region 140 is highly diagnostic with a thick chain band in the middle and a thin band on either side of it followed by a thin dot band.

3R is shorter than 2R and the sections are from 144 - 166. This arm can be easily identified by the presence of prominent bands throughout its length. The guiding features of this arm are three dark bands at a little distance from the chromocenter in the region 145, a puff in the region 147, followed by conspicuous dark bands in the regions 148 and 149. Other marked features of this arm are the presence of four distinct dark bands in the region 159 followed by a hat-like puff in the region 160. Two moderately large puffs behind the terminal end in the regions 166 and 165 are also of great importance in identifying this arm.

The photomap made and presented here permits more precise characterisation of the natural configurations of the bands and puffs and is helpful in localising with precision the varied gene arrangements found in this species.

(See photomap next page)

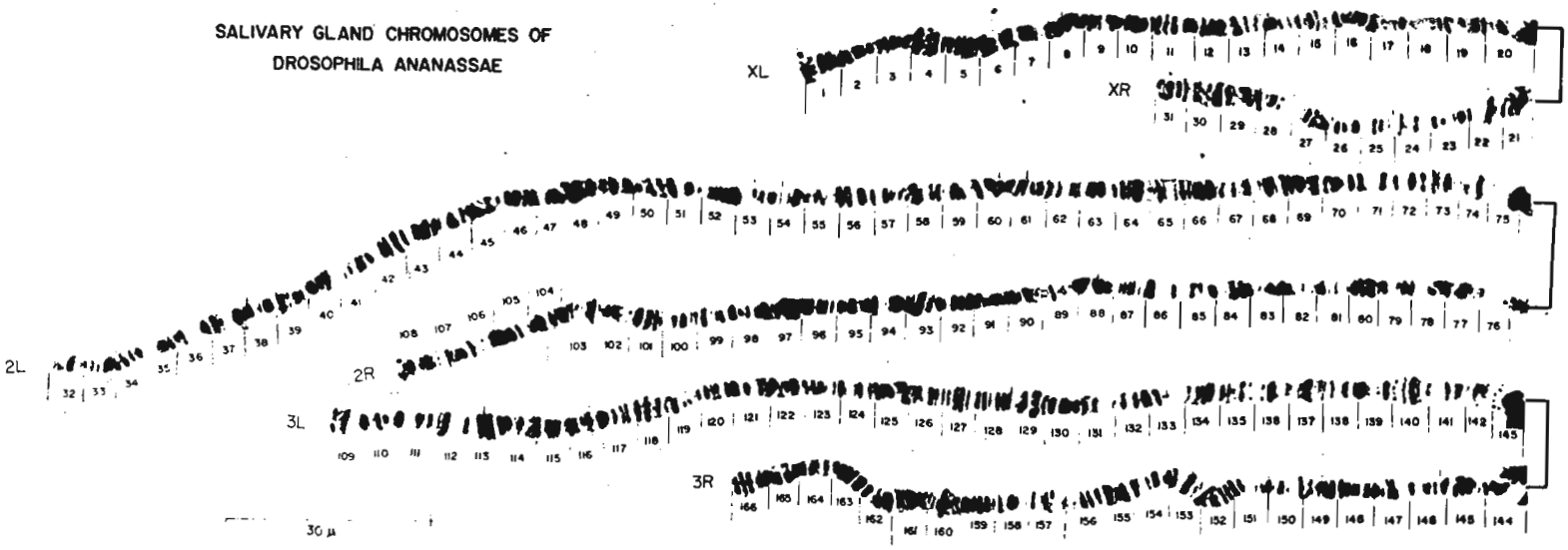


Fig.1

Acknowledgments: The authors are grateful to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology, for his valuable suggestions and encouragements and to Mr. Ramakrishnaraju for preparing the photomicrograph.

Falk, R. The Hebrew University, Jerusalem, Israel. On the structure of the R(1)2, y f chromosome.

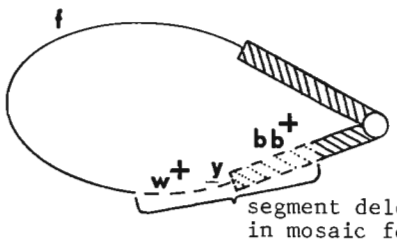
The exact origin of the ring R(1)2 is not known. It is assumed that it originated through a translocation between the two arms of an attached-X chromosome, CR(1)RM (Schultz & Catcheside 1939). Recently Vinikka et al. (1971) reconfirmed Schultz & Catcheside's observations that R(1)2 was duplicated for the bands 20A onward of the polytenic chromosome map. Schalet & Singer (1971) placed lethal l(1)A7 at band 20A1-2. This would mean that all essential loci proximal to l(1)A7 are represented twice in R(1)2.

The frequency of fragments that encompass the proximal loci of the X-chromosome induced in the R(1)2, y f is higher than that induced in a rod X-chromosome (Falk 1973). Dr. A. Schalet suggested to me that the higher frequency of such events induced in the ring than in the rod could have resulted from the fact that the proximal segment was present in duplicate in the ring chromosome.

In a previous study (Falk 1970) we induced recessive lethals in the proximal segment of the R(1)2, y f (obtained from Dr. I.I. Oster in 1965). The proportion of induced lethals that were located in this segment in the ring was not lower than that obtained in the rod X-chromosome. Furthermore, many of these lethals were found, by allelism tests, to be functional deletions of essential genes proximal to the locus of l(1)A7. This would suggest that in the ring studied by us no duplication for the most proximal essential loci existed. In a cytological analysis of the salivary gland chromosomes of R(1)2, y f/+ larvae no duplication for the bands of segment 20 was found (see Figure). Thus, the R(1)2, y f chromosome in our

laboratory is different from that described by Vinikka et al. and, in contrast to the R(1)2 present in the Leiden laboratory (personal communication), the R(1)2, y f in our laboratory is only slightly subvital for homozygous females and XO males. The variation in the structure of the R(1)2 chromosome in various laboratories could be due to mislabelling in the past (mixing R(1)1 with R(1)2 ?), but stems more probably from "evolutionary" diversifying changes that have been taking place in different laboratories which started with similar R(1)2 chromosomes (Schalet, personal communication).

Another clue to the structure of R(1)2, y f was recently obtained by Miss M. Yacoby from a cross of irradiated R(1)2, y f/Y^{bb-} males to $XY^{L}.Y^{S}$ (108-9), y^{2} $su(w^{a})w^{a}$ $Y^{L}.Y^{S}/In(1)sc^{4L}$ sc^{8R} + S, y sc^{4} sc^{8} w^{a} B females. A mosaic female was found, both her eyes were Bar (intermediate), one eye was completely apricot, the other was wild-type, except in a small upper section; the abdomen on one side showed an extreme bobbed effect. There were no indications of the fly being gynandromorphic (although one wing was slightly shorter than the other). The female died without leaving offspring. Upon dissection she was found to have female genitalia and gonads on both sides. We propose that this female, who started as a $In(1)sc^{4L}$ sc^{8R} + S, y



sc^4 sc^8 w^a B/R(1)2, y f zygote was obtained as a result of a chromatid deletion in the ring X-chromosome, including both the w^+ and the adjacent bb^+ loci. In other words, we propose that the single bb^+ segment present in the R(1)2, y f is located in the heterochromatic segment next to the "distal" euchromatic genes, and that the heterochromatic segment on the other side of the centromere, that is connected to the "proximal" genes, is void of bb^+ and probably originated from the long arm of the Y-chromosome. According to this interpretation the R(1)2, y f is an inverted closed X-chromosome (see scheme).

References: Falk, R. 1970, Mutation Res. 10:53-60; _____ 1973, Chromosomes Today 4:(in press); Schalet, A. and K. Singer 1971, DIS 46:131-132; Schultz, J. and D.G. Catcheside 1939, J. Genet. 35:315-320; Vinikka, Y., A. Hannah-Alava and P. Arajärvi 1971, Chromosoma 36:34-45.

Andrews, P.W. and D.B. Roberts. University of Oxford, England. A screen of a number of fluoro-compounds as possible selective agents for biochemical mutants in Drosophila melanogaster.

The current interest in genetic control in eukaryotes has made it desirable to select large number of mutants affecting the synthesis of a single protein. In Drosophila, Grell (1968) has used the sensitivity of Adh^- mutants to alcohol to select for such mutants, Glassman (1965) has used the sensitivity of Xdh^- mutants to purines to select for these mutants and more recently Sofer (1972) has selected for Adh^- flies using the sensitivity of the wild type, possessing the enzyme, to pent-1-ene-3-ol. We have investigated the possibility of using the resistance to a number of fluorocompounds, themselves non-toxic, as indicators of the absence of enzymes involved in their metabolism to toxic fluorocitrate (see Table 1).

Table 1

Fluorocompound	Enzyme mutants examined for resistance
2-Fluoroethanol*	Alcohol dehydrogenase minus - Adh^{nl}
4-Fluroobutanol*	Aldehyde oxidase minus - $Aldox^{nl}$ x
Ethyl α Fluorobutyrate*	Aliesterase minus x - strains carrying bw st sv^n & bw st ss.
5-Fluorotryptophan+	Tryptophan pyrolase minus - V^{36f}
6-Fluorotryptophan+	

* Obtained from K & K Laboratories
\+ Obtained from Sigma
x We are grateful to Professor Ursprung for sending us $Aldox^{nl}$ and to Dr. Ogita for the two Ali^- strains

Hypochlorite sterilised dechorionated eggs of either Oregon-R, Canton-S or mutant stocks (Table 1) were transferred to sterilised yeast/agar medium with, or without, the compound being tested. In some experiments the numbers of individuals hatching, pupating and ecloding were recorded (Table 2) but with two of the mutants, only qualitative observations were made ($Aldox^{nl}$ and Ali^-).

2-fluoroethanol and 4-fluorobutanol proved toxic to both wild type strains and to Adh^{nl}. There were suggestions that the latter were slightly more resistant, especially in the egg. In the cases where development was observed the larvae were generally very sluggish and development time was greatly increased. The mutant $Aldox^{nl}$ was similarly affected by these compounds.

The fluoroderivatives of tryptophan did not affect any stock over the concentration range used, up to 200 µg/ml. Ethyl fluorobutyrate (up to 2mg/ml) was slightly toxic to both Oregon-

R and to the two Ali⁻ strains studied.

Under the conditions used here none of the fluorocompounds look promising selective agents.

Table 2

		% Hatching	Pupation as % Hatching	Eclosion as % Hatching	No. of Eggs Examined
Control - No additions to medium					
	OR-R	81	78	74	150
	Canton-S	88	75	75	50
	Adh^{nl}	63	81	78	150
	v^{36f}	57	53	44	100
With 2-Fluoro-ethanol added					
0.2 µl/ml	OR-R	10	0	0	50
	Canton-S	46	4	0	50
	Adh^{nl}	40	0	0	50
0.5 µl/ml	OR-R	59	0	0	100
	Adh^{nl}	74	0	0	100
1.0 µl/ml	OR-R	8	0	0	50
	Adh^{nl}	50	20	8	50
2.0 µl/m.	OR-R	0	0	0	50
	Adh^{nl}	0	0	0	50
With 4-Fluorobutanol added					
0.1 µl/ml	OR-R	84	79	79	50
	Adh^{nl}	86	91	91	50
0.5 µl/ml	OR-R	23	26	22	100
	Adh^{nl}	24	45	42	100
10 µl/ml	OR-R	4	0	0	50
	Adh^{nl}	0	0	0	50
With 5 Fluorotryptophan added					
2 µgm/ml	OR-R	86	98	98	50
	v^{36f}	64	53	50	50
20 µgm/ml	OR-R	92	94	85	50
	v^{36f}	56	68	63	50
200 µgm/ml	OR-R	98	88	88	50
	v^{36f}	54	70	67	50
With 6 Fluorotryptophan added					
2 µgm/ml	OR-R	82	95	90	50
	v^{36f}	56	68	22	50
20 µgm/ml	OR-R	84	95	93	50
	v^{36f}	46	70	50	50
200 µgm/ml	OR-R	88	93	89	50
	v^{36f}	52	77	62	50

References: Glassman, E. 1965, Fed. Proc. 24:1243; Grell, E., K.B. Jacobson and J.B. Murphy 1968, Ann. N.Y. Acad. Sci. 151:411; Sofer, W.H. and M.A. Hatkoff 1972, Genetics 72:545.

Purnima, U. and M. Sanjeeva Rao. Osmania University, Hyderabad, India. Studies on genetic effects of deuterium in Drosophila melanogaster.

The effect of deuterium on mammals was first observed in 1932 by Lewis who fed heavy water to mice and reported that the treated mice became sterile. Further, in treated males the spermatozoa were abnormal, while in pregnant females fetal abnormalities were reported. Hughes and Calvin (1958) reported the induction of sterility in mice fed deuterium.

Zamenhof and Demerec (1943) cultured flies on a medium containing deuterium and reported the failure of this isotope to increase the lethal frequency. Hughes et al. (1963) subjected flies to a medium containing various concentrations of deuterium and observed its ineffectiveness in increasing the mutation frequency.

Deuterium inhibits the growth of some of the lower organisms such as green algae, E. coli, yeast and the molds Aspergillus and Penicillin (Katz, 1960). Deuterium, having twice the mass of ordinary hydrogen, differs from its common isotope more sharply than any other rare non-radioactive isotope, and, since hydrogen is the most common element in biological systems, experiments were undertaken to assess the genetic effects, if any, produced by deuterium.

The present investigation reports the study of deuterium in producing genetic effects in Drosophila by culturing for four successive generations on a medium containing the same.

Individuals of the Oregon-K strain were allowed to feed on a normal medium containing 30% deuterium. Half of the males developed on this medium were crossed to y sc^{S1} In49 sc^{8};bw;st virgin females to screen the incidence of sex-linked recessive lethals and translocations. A brood pattern of 3 days interval was used and 3 broods were studied. Each male was allowed to mate with 3 females. The F_1 females were mated individually with y sc^{S1} In49 sc^{8} males, while the F_1 males were mated inidvidually with bw st virgins to score for sex-linked recessive lethals and translocations, respectively. The other half of the males were allowed to feed on the medium containing 30% deuterium along with fresh females of Oregon-K. This was repeated for four generations. The results are presented in Tables 1 and 2.

Table 1. Frequency of sex-linked recessive lethals induced in a control (1) and in four generations of Drosophila cultured on medium containing 30% deuterium (2,3,4 and 5)

Sl		BROODS A			B			C			TOTAL		
No	Generation	T	l	%	T	l	%	T	l	%	T	l	%
1	Control	861	2	0.23	827	4	0.48	874	2	0.23	2562	8	-.31
2	F_1 treated	214	4	1.86	440	1	0.22	500	1	0.20	1154	6	0.57
3	F_2 treated	905	4	0.44	196	1	0.56	549	1	0.18	1650	6	0.54
4	F_3 treated	249	2	0.803	182	2	1.22	163	2	1.22	594	6	1.1
5	F_4 treated	293	1	0.34	531	2	0.38	316	4	1.25	1140	7	0.61

T = total number of X chromosomes tested l = lethals recorded

Table 2. Frequency of translocations in a control (1) and induced in four generations of Drosophila cultured on a medium containing 30% deuterium (2,3,4 and 5)

Sl		BROODS A			B			C			TOTAL		
No	Generation	T	t	%	T	t	%	T	t	%	T	t	%
1	Control	849	0	-	846	-	-	832	0	-	2527	0	-
2	F_1 treated	290	1	0.34	293	1	0.34	240	0	-	823	2	0.24
3	F_2 treated	630	1	0.15	108	2	1.8	173	0	-	911	3	0.32
4	F_3 treated	183	1	0.54	99	1	1.01	107	0	-	389	2	0.51
5	F_4 treated	178	1	0.46	126	0	-	253	0	-	557	1	0.17

T = total number of F_1 sons tested t = translocations recorded

A chi-square test has been done to compare four groups, control vs F_1, F_2, F_3 and F_4. The results of this statistical analysis are presented in Table 3. Comparison of the data obtained for each generation gave evidence against a mutagenic effect of deuterium.

(Table 3 next page)

Rajaraman, R. and O.P. Kamra. Dalhousie University, Halifax, N.S., Canada. On Ruby laser mutagenicity to Drosophila melanogaster male germ cells.

In the past decade lasers emitting at different wavelengths have been employed to study the interaction of monochromatic radiations on various biological systems. Biological effects of laser radiations depend on their wavelength, energy density as well as the pigmentation and absorbance of the target (Rajaraman and Kamra, 1970). Ruby laser (RL)-induced (6943Å) changes in somatic cells have been reported by several workers. Contradicting observations like growth enhancement by increase in cell division (Jamieson et al., 1969), blockage of mitosis in prophase and retardation of growth by cell division (Gordon et al., 1968) in mammalian cells in culture due to RL radiation have been reported. Chromosomal clumping and aberrations were observed in RL irradiated rabbit endothelial cells (Okigaki and Rounds, 1972). Exposure of D. melanogaster larvae to unfocused RL (0.1J/mm^2) produced significant variations in the life span of different stages that were transmitted as true breeding mutations (Zuzolo, 1966). In view of these observations, we decided to test the mutagenicity of RL radiation in D. melanogaster by screening for sex-linked recessive lethals.

Male Drosophila larvae of X^{c2} y/sc^8 Y stock were collected from the culture tubes when they were climbing up for pupation and were allowed to pupate on moist filter paper. 24 hrs old male pupae were irradiated with 5 or 10 pulses of RL radiation (217 mJ/pulse) to sample the spermatids which are most sensitive to radiation damage. The pupae were arranged during irradiation so that only the posterior ventral part of the pupae were exposed to radiation, to avoid unnecessary damage to the thoracic and head regions. On emergence, the males were individually mated with six 3-day old y sc^{S1} In-49 sc^8;bw;st p^p virgins for two days. The F_1 offspring were screened for lethal mutations. The treatments and the results are shown in the table.

Treatments	No. chrom. tested	No. lethal	% lethal
Control (a)	383	0	0.1
(b)	473	1	
5 x 217 mJ (a)	431	0	0.0
(b)	394	0	

It is apparent that RL radiation did not induce sex-linked recessive lethal mutation even in the spermatid stage, which is most sensitive to radiation damage. The males that were exposed to 10 pulses of 217 mJ showed temporary sterility (for two days) indicating that either the males were not able to mate due to any possible injury by irradiation or the germ cells in spermatid stage were killed, fertility being regained by repopulation due to continuously maturing germ cells. These results indicate that RL radiation is not mutagenic as regards to sex-linked recessive lethals and it may cause cell death or division delay in the spermatogenous cells probably due to secondary heat damage.

References: Gordon, T.E., C.A. Waldron and L.S. Gordon, Cancer 25:851; Jamieson, C.W., M.S. Litwin, S.E. Longo and E.T. Krementz 1969, Life Sci. 8:101; Okigaki, T. and D.E. Rounds 1972, Radiat. Res. 50:85; Rajaraman, R. and O.P. Kamra 1970, Photochem. Photobiol. 11:121; Zuzulo, R.C. 1966, NASA Rep. 1-66-1:1.

(Continued from preceding page)

Table 3. χ^2 values for the differences of sex-linked recessive lethals for generations compared

S1 No	Group	BROODS A	B	C	TOTAL
1	Control vs F_1 treated	5.58	1.35	0.403	0.9142
2	Control vs F_2 treated	0..21	0.272	0.610	0.07997
3	Control vs F_3 treated	0.523	0.208	1.437	5.317
4	Control vs F_4 treated	0..21	0.546	3.124	1.7804

Hengstenberg, R. Max-Planck-Institut für biologische Kybernetik, Tübingen, Germany.

Responses of cervical connective fibers to visual pattern movement in wild type Drosophila melanogaster.

In female wild type D. melanogaster (stock Berlin) the fused pair of cervical connectives contains 3578 nerve fibers. Their mean diameter is about 5 μm in the two dorsal "giant fibers", 1-3 μm in another set of 70 fibers, and less than 1 μm in the remaining 3506 fibers. The smallest profiles have diameters of less than 0.1 μm. Action potentials of at least some of the larger fibers can be recorded for several hours from the intact connective by means of metal hook electrodes. The records show a continuous spontaneous activity, which fluctuates in time, and which specifically changes in response to visual stimulation. The stimulus consisted of striped patterns, moving at constant speed across translucent screens in front of the two compound eyes. If the patterns on either side move horizontally from front to back, the spike rate in the cervical connective increases to about twice the resting activity. This effect lasts until the movement comes to rest. Pattern movement in the reverse direction elicits very small if any responses. Monocular stimuli give rise to similar but smaller responses than binocular ones. Horizontal rotatory stimuli on both eyes are equivalent to front-to-back stimuli on one eye alone. If the direction of pattern movement is varied with respect to the long axis of the fly, the response is found to be largest for horizontal pattern movement from front to back. Vertical movement is almost ineffective. Varying the speed of pattern movement yields a maximum of the response beyond $w=400^{o}$/sec with patterns of $\lambda=40^{o}$ spatial wavelength.

Studies are under way to analyze, in more detail, the characteristics of these responses, and to establish their relation to visually controlled behavioral traits.

Levison, G.M., J.P. Chinnici and J.N. Gargus. Virginia Commonwealth University Richmond.

Quantitative measurement of red eye pigment in various white mutants of Drosophila melanogaster.

Eyes of wild type Drosophila melanogaster contain two distinct types of pigment, ommochromes (brown) and pteridines (red). The ommochromes are found in most arthropod groups and the biochemcial pathways of these pigments are well known. Pteridines, on the other hand, are unique to Drosophila and their synthesis has not yet been fully elucidated, although some biochemical and physiological information is known.

In reviewing the literature for reports of quantitative measurements of pteridine pigments found in female flies homozygous and heterozygous (with white) for the various alleles at the white locus, we found that such precise information generally either was not available or not comparable. Therefore, we found it necessary and useful to determine these values ourselves for 20 white alleles, wild type (Oregon-R), brown and scarlet mutants. See Table 1 for a listing of these stocks; see Lindsley and Grell (1968) for full descriptions. All stocks were obtained from the Bowling Green Stock Center.

All flies were raised at 25 ± 1°C on a standard dextrose, agar, Brewer's yeast medium containing Tegosept-M as a mold inhibitor. Pigment determinations were obtained on two groups of flies: females homozygous for the genes listed in Table 1, and heterozygous females obtained by crossing females homozygous for the gene in question with white eyed hemizygous males. Flies were aged a minimum of five days post-eclosion before the pigment extraction procedure was begun. The method of Ephrussi and Herold (1944) was followed in extracting the pteridine eye pigment. In each case, 100 females were etherized and then decapitated with a surgical steel blade (size 11). The heads were next split in half vertically and collected with a small camel's hair brush to be put into four ml of Acid Ethyl Alcohol (AEA) solution, where they remained for 20-22 hours at 25 ± 1°C. After the required time, the solution containing the eyes was spun in a Beckman centrifuge for 15 minutes at 15,000 x g to separate the eyes from the pigment which had dissolved in solution. Three ml of the solution was then transferred into a quartz cuvette and placed into a Perkin-Elmer 124-D Double Beam Spectrophotometer in order to measure absorption. The reading at 480 milli-microns was taken as the

Table 1. Pteridine pigment values of female Drosophila melanogaster (25 heads per 1 ml of AEA)

Mutant Tested	Genotype	Absorbance at 480 milli-microns	Mutant Tested	Genotype	Absorbance at 480 milli-microns
1. w^{m4}	w^{m4}/w^{m4}	1.600	13. w^{ec3}	w^{ec3}/w^{ec3}	0.000
	w^{m4}/w	0.565		w^{ec3}/w	0.009
2. w^{co}	w^{co}/w^{co}	0.158	14. w^{bf2}	w^{bf2}/w^{bf2}	0.012
	w^{co}/w	0.060		w^{bf2}/w	0.003
3. w^{e}	w^{e}/w^{e}	0.131	15. w^{bux}	w^{bux}/w^{bux}	0.013
	w^{e}/w	0.057		w^{bux}/w	0.044
4. w^{a3}	w^{a3}/w^{a3}	0.110	16. w^{i}	w^{i}/w^{i}	0.010
	w^{a3}/w	0.054		w^{i}/w	0.002
5. w^{a2}	w^{a2}/w^{a2}	0.086	17. w^{bf}	w^{bf}/w^{bf}	0.013
	w^{a2}/w	0.042		w^{bf}/w	0.012
6. w^{col}	w^{col}/w^{col}	0.084	18. w^{h}	w^{h}/w^{h}	0.017
	w^{col}/w	0.046		w^{h}/w	0.031
7. w^{bl}	w^{bl}/w^{bl}	0.061	19. w^{t}	w^{t}/w^{t}	0.013
	w^{bl}/w	0.031		w^{t}/w	0.005
8. w^{sat}	w^{sat}/w^{sat}	0.074	20. w	w/w	0.005
	w^{sat}/w	0.063			
9. w^{a}	w^{a}/w^{a}	0.037	21. OR-R	$+^{w}/+^{w}$	1.894
	w^{a}/w	0.025		$+^{w}/w$	1.696
10. w^{a4}	w^{a4}/w^{a4}	0.029	22. bw	$+^{w}/+^{w}$, bw bw	0.048
	w^{a4}/w	0.038		$+^{w}/w$, $+^{bw}$ bw	1.612
11. w^{e2}	w^{e2}/w^{e2}	0.037	23. st	$+^{w}/+^{w}$, st st	1.799
	w^{e2}/w	0.034		$+^{w}/w$, $+^{st}$ st	1.917
12. w^{ch}	w^{ch}/w^{ch}	0.035			
	w^{ch}/w	0.025			

peak absorption value, although we wish to note that there was, in some cases, an extremely broad peak from about 480 to 460 milli-microns. The 480 milli-micron value was taken as the peak due to convention. Prior to each reading, a previously collected sample of pigment from scarlet homozygous females was run in order to standardize the spectrophotometer so that data collected at different times would be comparable. Single readings were made for most of the genes tested. However, second readings, from new crosses, were taken for those white alleles which showed a heterozygous value much lower than the homozygous value. In all these cases, the data from the two runs were quite similar, and the values presented in this report are the average of the two runs.

The values obtained are presented in Table 1. Note that of the 20 white alleles tested, 12 of them (numbered 1-12 in the table) gave homozygous values of greater than 0.020 (values lower than 0.020 are too low to be reliably determined on the instrument we used). Of these 12 white alleles giving high homozygous values, seven of them (1-7 in the table) gave heterozygous values of approximately one-half the homozygous value, indicating that for pteridine pigment synthesis these seven white alleles show incomplete dominance in relation to white. The white alleles we tested which gave reasonable amounts of red pigment and which also gave similar values in homozygous and heterozygous conditions were numbers 8 through 12 in the table. Therefore, for pteridine pigment synthesis, these five alleles show more or less complete dominance over white, according to our data.

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Janning, W. University of Muenster, Germany. Distribution of aldehyde oxidase activity in imaginal disks of Drosophila melanogaster.

In Drosophila, little is known about genetic mosaicism in the internal organs of the imago and in larval structures, due to the lack of suitable marker genes. Recently it was found that aldehyde oxidase can be used as a cell marker for Malpighian tubules, the gut and the inner genitalia of the imago (Janning, 1972 and in preparation). By the use of the mutant gene maroonlike on the X-chromosome, mosaic tissues in gynandromorphs can be detected in organs or organ parts which show aldehyde oxidase activity in the wild type. The tissue distribution of aldehyde oxidase activity in larvae and adults has been described by Dickinson (1971). In imaginal disks he found "moderate to heavy activity". For studies concerned with imaginal disks in gynandromorphs a more detailed analysis of the normal distribution pattern was necessary.

Disks of third instar wild type larvae were dissected out and inspected for aldehyde oxidase activity after appropriate staining (for the staining procedure see Courtright, 1967; Dickinson, 1970, 1971). Activity was found in all the prominent disks of both sexes. In the labial, genital and the three leg disks most of the tissue shows heavy activity with patches of low or no activity. Particularly remarkable patterns are found in the haltere, wing and eye-antennal disks. In the wing (Figure 1a) and haltere disks, stripes of activity go through

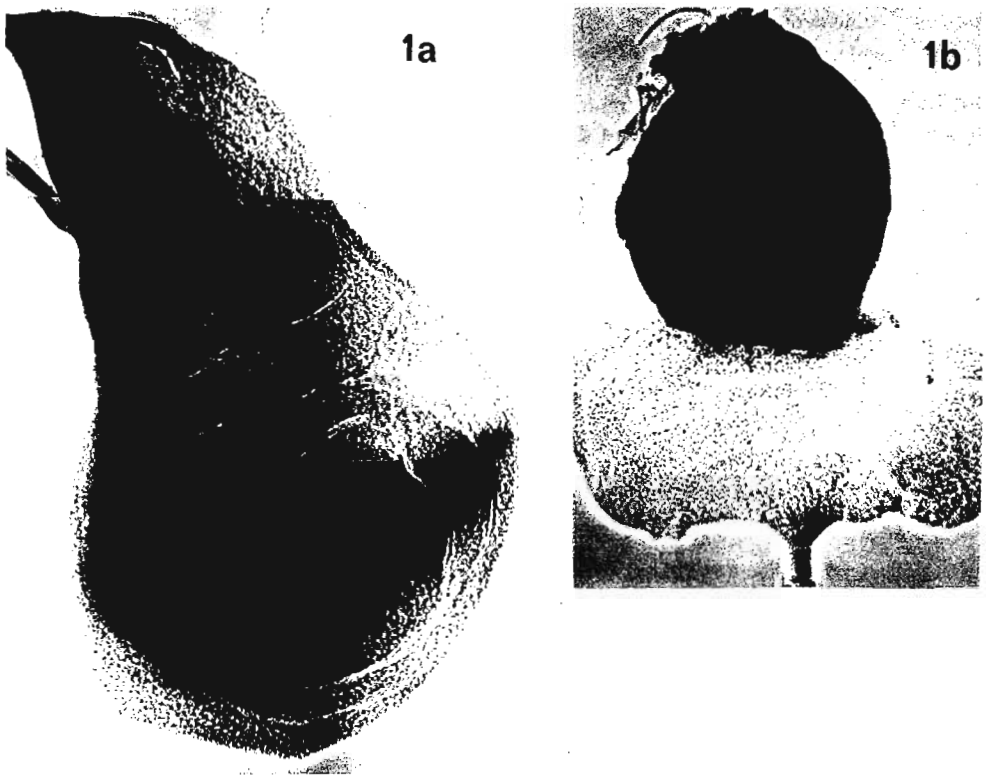

Figure 1. Distribution of aldehyde oxidase activity in imaginal disks of wild type larvae. a) Wing disk with stripes of heavy activity. b) Optic disk with aldehyde oxidase activity in the antennal part, and no activity in the eye part.

otherwise aldehyde oxidase-negative tissue. In the optic disk (Figure 1b), a sharp border separates the areas of activity and no activity. The antennal part of the disk shows heavy activity in all the tissue except for a small spot of low activity that is sometimes present near the center of the disk, whereas in the eye part no activity can be detected. This

borderline of activity in the optic disk seems to cut the disk into the antennal and eye parts. The staining patterns are highly reproducible. To rule out impermeability for certain staining components in the unstained areas, disks were injured or cut into pieces prior to the aldehyde oxidase test. The activity patterns were the same as in intact disks.

Thus at least the antennal disk can be used for studies with genetic mosaics. In gynandromorphs mosaic antennal disks have been found and are now being analysed in detail. This work was supported by the Deutsche Forschungsgemeinschaft.

References: Courtright, J.B. 1967, Genetics 57:25; Dickinson, W.J. 1970, Genetics 66: 487; _____ 1971, Develop. Biol. 26:77; Janning, W. 1972, Naturwissenschaften 59:516.

Chinnici, J.P. Virginia Commonwealth University, Richmond, Virginia. Preliminary data on the effect of mono-sodium glutamate on viability and crossing over in Drosophila melanogaster.

The effect on human health of the food additive mono-sodium glutamate (MSG) has been a topic of concern since 1968 when Schaumberg et al. first described the "Chinese Restaurant Syndrome" in man and associated it with ingestion of the flavor enhancer MSG. Evidence presented by Ghadimi et al. (1971) indicates that the symptoms of this syndrome (headache, sweating, nausea, weakness, thirst, flushing of the face, a sensation of burning or tightness, abdominal pain, and lacrimation) may be the result of "transient acetylcholinosis", since glutamic acid is readily converted to acetylcholine when excess sodium is present, the symptoms being due to the effect of the excess acetylcholine on the parasympathetic nervous system.

More serious concern about the effect of MSG on development has resulted from several reports that MSG causes lesions in the hypothalamus of the brain and/or degeneration of the retina of the eye of mice, rats, and rhesus monkies (see, for example, Olney and Sharpe, 1969; Arees and Mayer, 1970; Olney, 1971; and Burde et al., 1971). However, several other reports have failed to substantiate these findings (see, for example, Adamo and Ratner, 1970; Oser et al., 1971; and Reynolds et al., 1971), so that no clear cut conclusions may be drawn. A possible contributing factor to the effect of MSG on brain development is the finding that MSG briefly but significantly depresses glucose uptake by mice brain cells (Creasey et al., 1971).

Two brief reports on the effects of MSG on development and productivity in D. melanogaster have been published. Turner and Wright (1971) have reported that 1 and 3 percent solutions of MSG do not change the number of adults emerging from cultures. Data from Forman and Majumdar (1971) show that a 10% MSG solution reduces the number of adults emerging from cultures by 57% while the percentage of females emerging from these cultures rises from the control value of 49.95% to 60.1%. Also, flies allowed to drink a 10% MSG solution for 24 hours produced 39% fewer offspring than the controls, with the sex ratio of these offspring not being affected. I am currently studying the effect of MSG on viability, fecundity, and crossing over in D. melanogaster. Some of the preliminary data from this study are presented below.

The effect of a 10% MSG solution (10 grams of MSG added to 100 ml of a standard dextrose-yeast-agar medium) on egg to adult viability in the Oregon-R wild type strain was measured as follows. To each of 30 vials, each containing the 10% MSG supplemented media, 25 eggs were added. A similar number of eggs was added to each of 30 control vials containing media unsupplemented by MSG. The results are presented in Table 1. In the control vials, 74% of the

Table 1

Treatment	N	No. of Adults Produced $\bar{x} \pm s$	Percent male Offspring $\bar{x} \pm SE$
Control 0% MSG	30	18.73 ± 3.45	50.33 ± 2.15
10% MSG*	30	12.83** ± 3.79	51.06 ± 3.20

N = Number of vials set up, each containing 25 eggs.
* = 10 grams per 100 ml of media solution
ANALYSIS OF VARIANCE (TREATMENT VS. CONTROL)
**: $P < .01$
others not significant

eggs developed into adults, while only 51% of the eggs developed into adults in the MSG vials. This is about a one-third decrease in egg-adult viability, a significant reduction, though not as great as reported by Forman and Majundar (1971). However, the sex ratio of the adults is not altered by their development on the MSG media. This finding is at variance with the results of Forman and Majundar.

To test the effect of MSG on altering crossing over, the following experiment was performed. Females from a stock homozygous for the four sex linked mutants scute (sc, 1-0.0), crossveinless (cv, 1-13.7), singed (sn^3, 1-21.0), and miniature (m, 1-36.1) were mass mated with males from a wild type Oregon-R stock in half pint bottles containing media supplemented with 5%, 10%, 15% or no (control) MSG. The offspring (heterozygous females and hemizygous mutant males) were aged two days and then were single pair mated in vials containing control media (no MSG added). After 8 days, the parents were removed from the vials and the offspring were scored to determine the amount of crossing over in the female parents. The results are presented in Table 2. The following facts are evident from this table: (1) Fecundity of the

Table 2

Treatment	N	No. of Offspring $\bar{X} \pm s$	Percent Males $\bar{X} \pm SE$	Mean Percent Recombination and 95% Confidence Limits sc-cv	cv-sn^3	sn^3-m
Control	24	132.20	48.25	14.1	5.6	14.9
0% MSG		± 13.38	± 0.73	13.0-15.2	4.7-6.5	13.7-16.2
5% MSG*	19	142.68	48.31	13.1	4.6	13.5
		± 22.18	± 0.67	11.1-15.2	3.8-5.5	12.0-13.7
10% MSG*	26	124.07	48.73	11.0**	4.7	12.4**
		± 14.76	± 1.3.	9.5-12.6	4.1-5.3	11.2-13.7
15% MSG*	20	141.10	48.30	8.0**	3.7**	10.5**
		± 31.21	± 0.86	6.8-9.2	3.2-4.3	9.1-11.9

N = Number of females tested
* = Grams per 100 ml of media solution
ANALYSIS OF VARIANCE (TREATMENT VS. CONTROL)
**: $P < .01$
others not significant

flies is not reduced by growth on MSG supplemented media; (2) the sex ratio of the offspring is not affected by growth of the parents on MSG media; (3) growth of the parents on media supplemented with 10% or 15% MSG has a significant effect on reducing the amount of crossing over between the four sex linked loci. Additional experiments are currently underway to determine the repeatability of the above results, and to gain more information concerning the heritability and the mechanism of the action of MSG on reducing crossing over.

I would like to acknowledge the assistance rendered by William B. Boiler during the course of the experiments reported above.

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Ranganath, H.A. and N.B. Krishnamurthy.
University of Mysore, Manasagangothri, India. Competitive coexistence is not an invalidation but an extension of the Gause principle.

Competition studies have been made between two sibling and sympatric species, D. nasuta and D. neonasuta, following the procedure of Ayala (1965). Polymorphic and monomorphic strains of both the species were employed in the experiments. In all the cases D. neonasuta was eliminated. Table 1 gives the genotype of the competing species and time of elimination of D. neonasuta. The population dynamics of these two species in pure and mixed cultures will be presented elsewhere.

In the light of the above findings, the authors are tempted to draw certain interesting conclusions. Ayala (1970) has suggested that the niche of a species includes all its relationships to the physical (temperature, light, humidity, food, place to live, etc.) conditions and the biotic (other organisms of the same kind or of different kinds) environment. The functional relationship with these ambient ecological factors determines the functional status of the species in a niche. In a single niche, organisms can have different varieties of functional relationships. For example, the different stages in the life cycle of Drosophila (the eggs, the 1st, 2nd and 3rd instar larvae, the pupae and the imagoes) within a bottle certainly differ in their ecological-functional relationships. The degree of functional relationships can be altered either by changing the genetic constitution of the species or the ecological factors. So functional status of a species is a dynamic character.

Table 1. Results of the competition between D. nasuta and D. neonasuta.

Species	Time of elimination of D. neonasuta in weeks
1 Monomorphic nasuta v/s Monomorphic neonasuta	7 - 8
2 Polymorphic nasuta v/s Monomorphic neonasuta	8 - 10
3 Monomorphic nasuta v/s Polymorphic neonasuta	11 - 15
4 Polymorphic nasuta v/s Polymorphic neonasuta	22 - 27

It is felt that the determination of the ecological niche of a species in general is not important as its functional status in a microcosm. In view of this, Gause principle can be restated as follows: 'Two or more forms with absolutely identical functional status cannot coexist indefinitely in the same environment'.

Let the functional status unit of one species be unity and of the other is 1-s, then no matter how small is s, the less efficient species will be, given enough time, eliminated. By changing the magnitude of the functional status of the competing species it is possible either to shorten or lengthen the duration of their coexistence.

'Time' factor is the central tenet of the Gause Law. It is not possible to predict the time of elimination of any one of the competing species without experimentation. The 1-s should reach a critical and threshold point before one of the species is to be eliminated. It is only a question of time. As the extent of functional status changes due to the alteration either in the genotype or in the ecological factors, the period of elimination also should vary. So it is not possible to forecast the time for elimination of the less efficient species, but only the process can provide this information.

Biological mechanisms attributed by Ayala (1970) for the coexistence of the competing species provide the mechanisms either to alter the functional status completely or their magnitude. If the coexistence is due to the former case, they can do so as there is no clash between them, but in the latter case it is only a question of time. The laboratory experiments of Ayala (1969), which he has used to invalidate the Gause principle fits into our latter explanation. In the competition between D. serrata and D. pseudoobscura, at 25°C D. serrata wins, but at 19°C it is a loser, while at 23.5°C both the species coexist for quite a number of generations. This experiment is a clear indication of the changes in the functional relationship of the species at different temperatures. Further, it suggests, that the observed number of generations at 23.5°C may be insufficient for 1-s to express itself and it requires some more time. The requirement of different lengths of periods to eliminate any one of the competing species is clearly seen in the competition between D. nasuta and D. neonasuta (Table 1). Similarly Park (1954) has demonstrated that the outcome of competition between T. castaneum and T. confusum depends on the amount of food, temperature and relative humidity.

Thus, the competitive coexistence of species for certain number of generations under certain environmental situations cannot invalidate the Gause Law (Neo), as the words 'even-

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Preuss, V. University of Tübingen, Germany. Light-dependent and light-independent mating of D. subobscura.

Successful mating of wild type D. subsbscura depends on light. Springer (1964), however, could obtain a light-independent strain (L1) by selection. In order to investigate the question as to whether or not the elements of courtship are the same in the L1 strain and other strains, flies from a wild type strain, a yellow mutant strain and the L1 strain were observed directly in Elens-Wattiaux observation chambers. The male choice and the female choice method was used. In each individual male choice experiment, 10 males of one strain were brought together with 10 females of the same and 10 females of another strain. For the female choice experiments, 10 females were combined with 10 males of the same strain and 10 males of another. Homogamic and heterogamic matings were registered over an observation time of one hour. During the course of the observations it became clear that most of the matings occured after a significant and pronounced courtship of the males. Such a sort of mating may be called "typical". In some cases, however, matings were performed spontaneously without any proceeding "dance" of the males. These matings may be described as "atypical" matings. The results of the observations are shown in the following table:

MALE CHOICE EXPERIMENT

males	females	matings				total number of matings observed
		homogamic		heterogamic		
		typ.	atyp.	typ.	atyp.	
+	+ and y	139	1	123	1	264
L1	L1 and y	30	33	59	52	174
y	y and +	69	--	23	--	92
y	L1 and +	56	3	4	--	63

FEMALE CHOICE EXPERIMENT

females	males					
+	+ and y	163	5	9	1	178
L1	L1 and y	39	50	8	6	103
y	y and +	30	--	134	6	170
y	L1 and y	31	1	55	54	141

With respect to typical and atypical matings it is rather clear that atypical matings occur almost exclusively in experiments with L1 males. Hence, it may be assumed that this type of courtship behavior of the L1 strain is responsible for its ability to mate in the dark. It can be further seen from the table that yellow males are discriminated by wild type and L1 females. Further, L1 males seem to prefer yellow females, while wild type males do not. The meaning of optical signals for the mating display is obviously different for the wild type and the L1 selection strain. It is generally different also for males and females of all strains.

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tually' or 'given enough time' have broad spectrum. The neo-Gause law also helps to account for the sympatric diversity present in the natural populations occupying the heterogenous environments more aptly than the earlier concept.

Acknowledgments: We are highly indebted to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology, for his constant help and encouragement. This work is financially supported by Mysore University Grants.

References: Ayala, F.J. 1965, Genetics 51:527-544; _____ 1969, Nature 224:1076-1079; _____ 1970, Essays in Evolution and Genetics in honor of Th. Dobzhansky pp 121-158; Park, T. 1954, Physiol. Zool. 37:177-238.

Wright, C.P. Western Carolina University, Cullowhee, North Carolina. Oxygen consumption in tyrproless-1, l(1)EN14, a lethal mutant of Drosophila melanogaster.

Tyrproless-1, l(1)EN14, is a sex-linked lethal mutant of Drosophila melanogaster which was X-ray induced by Novitski (1963). Death in this mutant occurs in the larval stage. Both weight and oxygen consumption measurements were made on individual male larvae from the first instar larval stage until about the time of death. Oxygen consumption measurements were made with small respirometers in a 25°C water bath. A 20% NaOH solution was used to remove CO_2 from the respirometers, which caused movement of the NaOH drop. By measuring the volume of the droplet displacement, the oxygen consumption of a larva was determined. Control larvae were y, w, spl, sn males from the stock of Novitski (1963) in which the lethal mutant was induced. Experimental data were tested statistically against control data by use of the Mann-Whitney U nonparametric test (Tate and Clelland, 1957).

Both fresh and dry weights of l(1)EN14 larvae (Table 1) were significantly less than those in controls (Table 2) at every age measured. Rates of oxygen consumption per larva were

Table 1. Average weights and rates of oxygen consumption for l(1)EN14 larvae.

Age in hr. after oviposition	Fresh weight/ larva in mg.		Dry weight/ larva in mg.		O_2 consumption in cu. mm./ larva/hr.		O_2 consumption in cu. mm./mg. dry wt./hr.	
	n	M. ± S.E.	n	M. ± S.E.	n	M. ±S.E.	n	M. ± S.E.
30 hr.	-	Did not weigh	4	0.0014* ± 0.0001	8	0.071** ± 0.010	8	50.447 ± 7.357
48 hr.	-	Did not weigh	4	0.005* ± 0.001	8	0.131** ± 0.011	8	26.100 ± 2.167
72 hr.	8	0.13** ± 0.01	8	0.04** ± 0.01	8	0.552** ± 0.024	8	14.158** ± 1.424
96 hr.	11	0.22** ± 0.02	11	0.08** ± 0.01	11	0.376** ± 0.104	11	4.735** ± 1.143

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

significantly less in l(1)EN14 (Table 1) than in controls (Table 2) at all ages. When calculated per unit dry weight, rates of oxygen consumption in l(1)EN14 (Table 1) were greater than in controls (Table 2) until 48 hours after oviposition. After this the oxygen consumption per unit dry weight in l(1)EN14 larvae dropped sharply until at 96 hours it was at a very

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

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

low level. Some l(1)EN14 larvae lived a good deal longer than 96 hours, but since very few larvae could be found in mixed cultures after this time and since the rates of oxygen consumption had already begun to decrease, no measurements were made beyond this point.

None of the l(1)EN14 larvae formed pupariа; thus, this is a larval lethal. In order to determine more precisely the time of larval death, 58 l(1)EN14 larvae at 48 hours after oviposition were placed in food containers and observed until death. Results of these observations are found in Table 3. The extent of observable gross morphological development in l(1)EN14 larvae can be correlated with the low oxygen consumption measurements. Soon after hatching, l(1)EN14 larvae looked smaller than normal. After about 96 hours, very few live l(1)EN14 larvae could be found in the cultures, and most of those found appeared quite sick in that they were moving around lethargically, if at all. At 96 hours some l(1)EN14 larvae

still had the second-instar cuticle attached to them. In some cases the cuticle was attached at the larval mouth hooks, so that the larvae were dragging the cuticle around with them. In other cases the cuticle was wrapped around the posterior half of the organism. This seems to indicate that the second molt in 1(1)EN14 larvae occurs at a later than usual time or that the larvae have some difficulty in undergoing the molt.

Table 3. Subsequent development of 58 1(1)EN14 larvae separated from non-lethal sibs and placed in food petri dishes 48 hours after oviposition.

Age in hr. after oviposition	Vigorously alive*	Abnormal**	Dead***	Could not find
72 hr.	42	3	2	11
96 hr.	7	30	5	16
120 hr.	0	5	35	18
144 hr.	0	2	38	18
168 hr.	0	0	39	19

*Crawling around quite actively.
**Crawling around lethargically or moved only when pricked with forceps.
***Darkened in color, looked dried out and did not move when pricked roughly with forceps.

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

Gould-Somero, M., R. Hardy and L. Holland
University of California, San Diego, La Jolla. The Y chromosome and sperm length in D. melanogaster.

It has been reported that the amount of Y chromosome material in male D. melanogaster is directly correlated with the length of the sperm tails. That is, males with two Y chromosomes produce sperm roughly twice as long as males with one Y chromosome (Hess and Meyer, 1963, 1968). This observation interested us because of its obvious implications for the control of protein synthesis and assembly in the developing spermatid. Therefore we tried to repeat the observation but were unable to: motile sperm from XYY males were the same length as those from XY males.

Sperm lengths were measured after teasing motile sperm out of the seminal vesicle in saline (Ephrussi and Beadle, 1936), supplemented with 9% fetal calf serum to reduce stickiness, and spreading them out under a coverslip. The preparations were examined by phase contrast microscopy; motile sperm were selected and photographed when they stopped twitching. The lengths were measured in the photographs. We consciously selected for the longest sperm.

We examined adult males (2 - 9 days after eclosion) of the following sex chromosome constitutions: X/Y (Canton-S males); XY/Y ($Y^{S}X \cdot Y^{L}$, In(1)EN, y B/y^{+}Y); XY/T(Y;3) ($Y^{S}X \cdot Y^{L}$, y/T (Y;3)Df(73AB-D)/In(3LR)TM6, ss^{-} bx^{34e} Ubx^{67b} e); and X/Y/Y (produced by the cross C(1)RM, y pn v/Y; C(4)RM, ci ey ♀♀ x y/y^{+}Y; mei-S332; spa^{pol} ♂♂; the flies for this latter cross were kindly supplied by Dr. Brian Davis).

Genotype	Sperm length (mm)	n
X/Y	1.92 ± 0.014	6
XY/Y	1.86 ± 0.010	5
XY/T(Y;3)	1.85 ± 0.037	5
X/Y/Y	1.69 ± 0.062	7

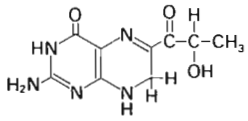
The results of the sperm measurements are summarized in the table. Clearly the presence of an extra Y chromosome per se is insufficient to double the sperm length in D. melanogaster.

References: Ephrussi, B. and G. Beadle 1936, Amer. Nat. 70:218-225; Hess, O. and G. Meyer 1963, J. Cell Biol. 16:527-539; Hess, O. and G. Meyer 1968, Adv. Genet. 14:171-223.

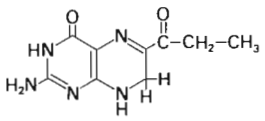
Supported by grant number AEC PA 150-6 from the Atomic Energy Commission, and by grant number HD 03015 from the National Institutes of Health.

Takikawa, S. Kitasato University, Sagamihara, Japan. Eye pigments of D. melanogaster.

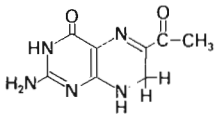
It is well known that wild type flies of D. melanogaster have red and yellow eye pigments, and the structures of the pigments are interesting from the viewpoints of genetics and pteridine metabolism. The mutant sepia of D. melanogaster contains only yellow pigments (Ziegler and Hadorn, 1958). One of them is sepiapterin, whose structure is 2-amino-4-hydroxy-6-lactyl-7,8-dihydropteridine (Nawa, 1960). Another pigment is isosepiapterin (Viscontini and Möhlmann, 1959), whose structure is 2-amino-4-hydroxy-6-propionyl-7,8-dihydropteridine (Forrest et al., 1959). In this communication, the isolation of the third yellow pigment from mutant sepia and the determination of its structure. We call the pigment "neosepiapterin", simply because this particular pigment accumulates in the sepia mutant. The structures of these yellow pteridines are shown in Figure 1.



Sepiapterin



Isosepiapterin



Neosepiapterin

The technique used for column chromatographic separations followed that of Tsusue and Akino (1965) and that of Fukushima and Akino (1968). In order to separate neosepiapterin from isosepiapterin, cellulose powder was used as an adsorbent and it was eluted with the solvent, n-butanol, ethanol, water (2:1:1 v/v). Flies were reared on standard yeast medium at 25°C and were harvested on the 1st, 5th and 9th days after eclosion. The contents of the pigments relative to the ages of the flies is shown in Table 1. Since the eye color of the sepia flies tends to become darker after eclosion, it has been supposed that the content of the pigments increases as the flies grow older. But these data show that the quantity of sepiapterin and neosepiapterin does not increase after 5 days eclosion.

Table 1. Quantity of yellow pteridines related to the ages after eclosion. Starting material was 50 g of sepia mutant of D. melanogaster respectively.

	Quantity (mg)		
Days after eclosion	Sepiapterin	Isosepiapterin	Neosepiapterin
1	19.8	0.36	0.27
5	34.4	1.33	0.36
9	34.4	1.87	0.37

On the other hand, isosepiapterin continues to increase.

The Rf values of the yellow pteridines are shown in Table 2. The molecular formula of neosepiapterin was determined to be $C_8H_9O_2N_5$ by mass spectrometry (molecular ion peak, m/e 207.0774). This pigment is thought to be C-6 substituted pterin because its alkaline potassium permanganate oxidation gives 2-amino-4-hydroxypteridine-6-carboxylic acid. The formation

Table 2. Rf values of yellow pteridines

	Solvents				
Pteridines	1	2	3	4	5
Sepiapterin	0.29	0.34	0.47	0.27	0.36
Isosepiapterin	0.21	0.44	0.52	0.46	0.53
Neosepiapterin	0.21	0.32	0.42	0.30	0.36

Solvents: 1. 3% ammonium chloride
2. isopropanol, 1% ammonia (2:1)
3. isopropanol, 2% ammonium acetate (1:1)
4. n-butanol, acetic acid, water (4:1:1)
5. n-propanol, ethylacetate, water (7:1:?)

of 2,4-dinitrophenyl-hydrazone shows carbonyl group at the sidechain. And the ultraviolet absorption spectrum of neosepiapterin resembles that of isosepiapterin. These data indicate that the structure of neosepiapterin is 2-amino-4-hydroxy-6-acetyl-7,8-dihydropteridine.

The distribution of neosepiapterin in sepia fly remains unknown, but it is supposed to be contained in the eye since sepiapterin and isosepiapterin are the eye pigments. Neosepiapterin was not detected when pterins were extracted from a large amount (1.2 kg) of wild type flies. Many derivatives of pteridine, which have three carbons at the sidechain on C-6 of the pteridine ring, have been isolated from natural sources. But those with two carbons have never been isolated before. Therefore, the occurrence of reduced 6-acetyl pterin suggests an unknown pathway of pteridine metabolism. Studies on the origin of a two carbon sidechain of the pigment is under investigation.

References: Forrest, H.S., C. Van Baalen and J. Myers 1959, Arch. Biochem. Biophys. 83: 508; Fukushima, T. and M. Akino 1968, Arch. Biochem. Biophys. 128:1; Nawa, S. 1960, Bull. Chem. Soc. Japan 33:1555; Tsusue, M. and M. Akino 1965, Zool. Mag. Tokyo 74:91; Viscontini, M. and E. Möhlmann 1959, Helv. Chim. Acta 42:836; Ziegler-Günder, I. and E. Hadorn 1958, Z. Vererbungslehre 89:235.

Bächli, G. University of Zürich, Switzerland. Drosophilidae of Kanha National Park, M.P., India.

Drosophilidae were collected by net sweeping over banana baits during the period of August 26 to September 18, 1972. The 8 collection sites are located in the center of the Kanha National Park, about 200 km northeast of Nagpur. The altitude is around 600 m above sea level. The species collected and the number of specimens are listed in Table 1. Drosophila (Scaptodrosophila) sp. and Hypselothyrea sp. are new species, while Leucophenga flavicosta, Lissocephala sabroskyi and Drosophila minima are reported for the first time from India. Domestic and cosmopolitan species were mostly absent. In the collection period (end of monsoon season), the ecological conditions of the center of the Park are therefore considered non-domestic.

Table 1. List of species collected, in order of frequency.

Species	Number of Specimens	Species	Number of Specimens
D. malerkotliana	10,643	** D. (Scaptodros.) sp.	6
D. jambulina	4,115	Leuc. albicincta	4
D. paratriangulata	1,349	* Leuc. flavicosta	4
D. albomicans	387	** Hypselothyrea sp.	2
D. takahashii	373	* Lissocephala sabroskyi	2
D. latifshahi	62	Leuc. guttiventris	1
D. kikkawai	26	D. silvalineata	1
D. chandraprabhiana	9	D. bipectinata	1
		* D. minima	1

* first record from India
** new species

Hardy, R.W., Le Ngoc Anh and Ng. H. Xuong University of California, San Diego, La Jolla, California. Three dimensional measurements of spermatid nuclei in Drosophila melanogaster from electron micrographs of serial cross sections.

We have developed a method for measuring the volume and morphology of spermatid nuclei. Testes are prepared for electron microscopy by the method of Tokuyasu, Peacock and Hardy (Z. Zellforsch. 124:479-506, 1972). Serial sections of an entire bundle of nuclei of spermatids in the early coiling stage are cut and the thickness of each is estimated from its interference color. Electron micrographs of selected sections are scanned with a digital densitometer connected to an IBM 1800 computer (Xuong, J. Physics E. 2:485, 1969). A program written in

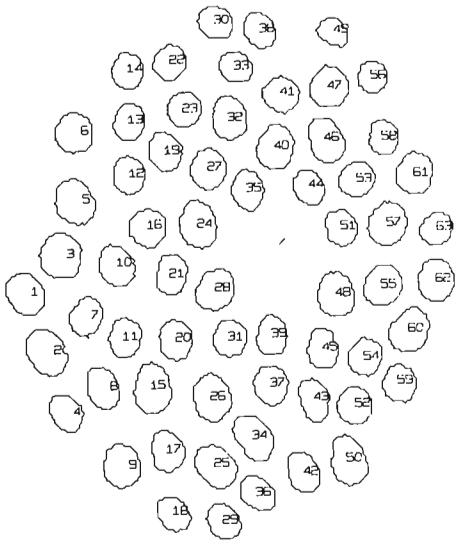
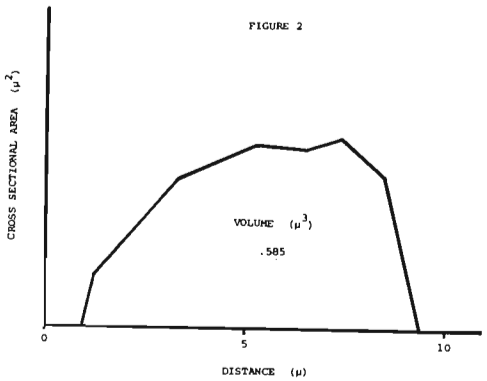
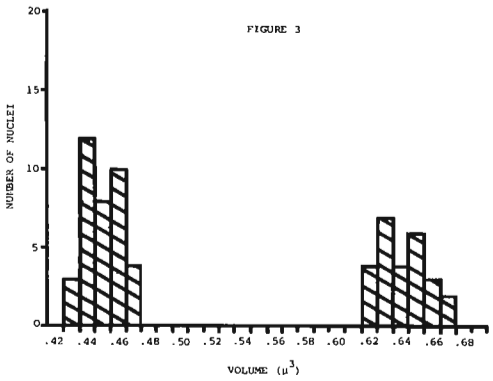
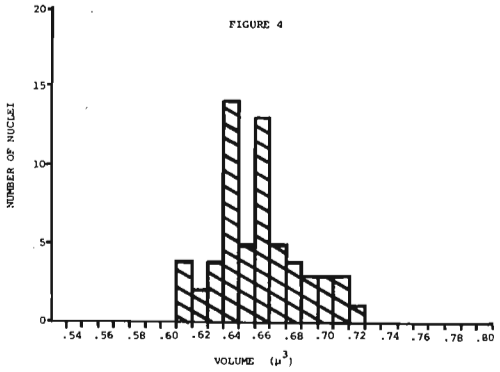


FIGURE 1

Fortran IV examines the digital picture of each electron micrograph and searches for all the nuclear images present. The output is a set of contour points and a cross sectional area for each nucleus. There may be as many as 64 of these contours. Figure 1 is a graphic display on a CalComp plotter from a typical electron







micrograph. When all cross sections are scanned all the various cross sectional areas of a specific nucleus are plotted as a function of the position of the serial section along the length of the nucleus (e.g. Figure 2). The area under the curve gives the volume of the nucleus and the shape of the curve reflects its morphology. This method is being used to investigate the consequences of changing the amount and arrangement of the nuclear chromatin with respect to nuclear morphology.

Wild type males produce nuclei of approximately uniform volume (Figure 4) and shape as might be expected from the similarity in chromosome content of X and Y bearing sperm. Males with an attached XY chromosome produce two types of nuclei; those carrying the XY and a set of autosomes and those with only a set of autosomes. Such males produce a bimodal distribution of nuclear volumes (Figure 3). Nuclear morphologies on the other hand are similar. In principle this method may be applied to a variety of serial section data.

Gelti-Douka, H., T.R. Gingeras and M.P. Kambysellis. Athens University, Greece; New York University, New York. Site of yolk protein synthesis in D. silvestris.

The observation of yolk proteins in the hemolymph of mature Drosophila species suggested that these proteins are synthesized outside the follicle as in the case of other insects (Telfer and Anderson, 1968). We attempted to localize the site of synthesis of the yolk proteins in the large Hawaiian species, D. silvestris. Tissues from single flies were dissected and washed thoroughly, initially in Waddington's Ringers and then in Grace's tissue culture medium; homogenized in borate-saline and either applied on SDS polyacrylamide gels for electrophoresis, or tested against antibodies prepared with D. silvestris crude yolk extracts. Of all the tissues used (muscles, epidermis, middle gut, malpighian tubes, fat body, oocytes and hemolymph with blood cells), only mature oocytes and hemolymph had the characteristic electrophoretic bands of yolk protein and gave immunoprecipitin lines on Ouchterlony double diffusion immuno-plates. Identical results were obtained with D. virilis. In view of these results we cultured muscles, epidermis or fat body of D. silvestris in Grace's medium containing H^3-leucine. After five hours of incubation, the culture medium was analyzed for iso-

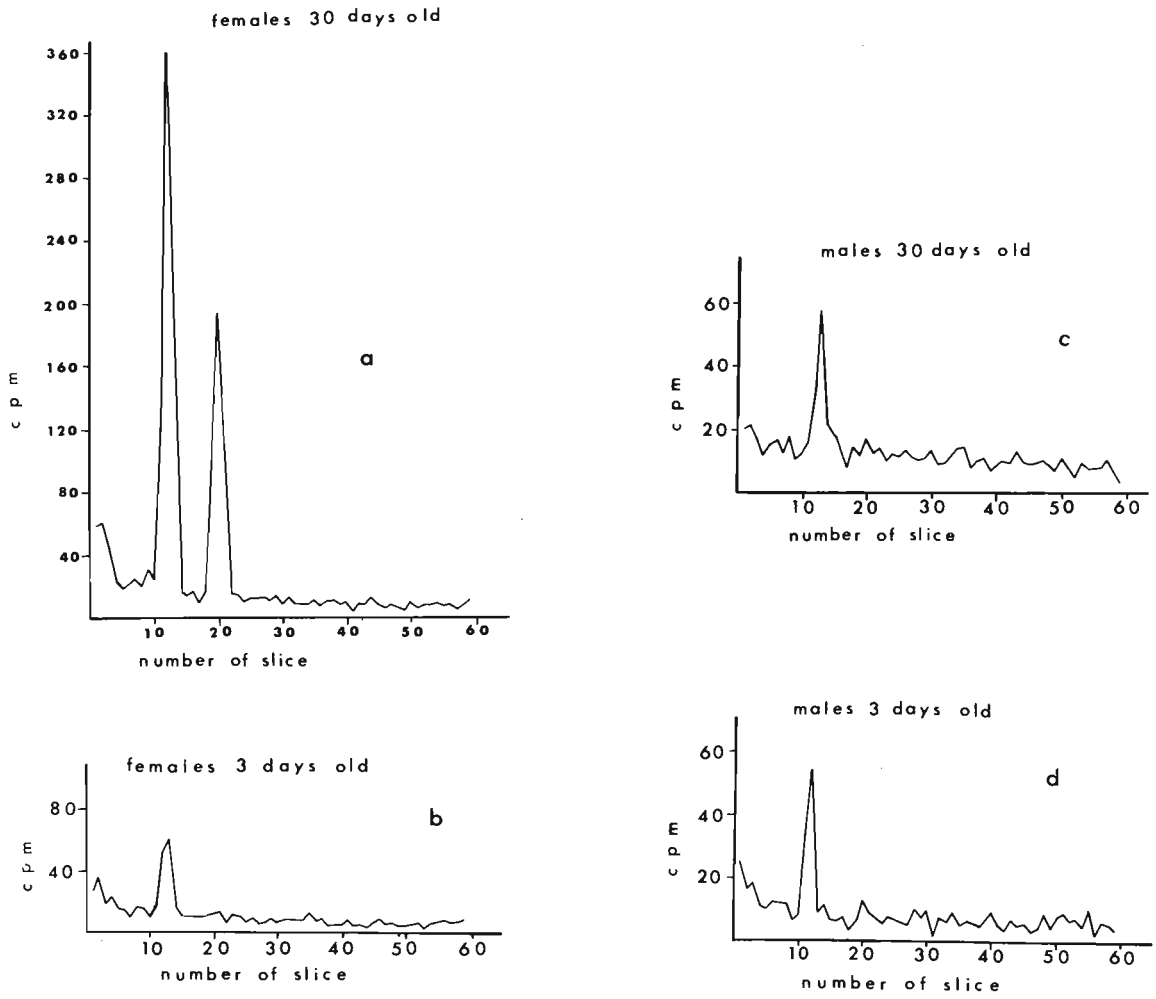


Figure 1. Fat body of D. silvestris cultured in 100 µl of Grace's culture medium for five hours in the presence of H^3-leucine (25 µC/ml). A sample of the culture medium (25 µl) was incubated for one hour with antibody prepared with D. silvestris crude yolk extract. The precipitated material was analyzed by SDS polyacrylamide gel electrophoresis. The gel was sliced into 1 mm slices and the isotope incorporation in each slice was determined by liquid scintillation counting.

tope incorporation into total protein and into antibody-precipitable yolk protein by means of liquid scintillation counting, both directly and after gel electrophoresis. The results demonstrated that the fat body from females was able to synthesize and release into the culture medium several proteins, two of which (one major and one minor) were yolk proteins (Table 1, Figure 1a). Only fat body showed detectable synthesis of yolk proteins. Using fat body from newly emerged female flies (3 day old) we found that only one yolk protein was produced. The other yolk protein appeared during or near adult maturation (Figure 1b). The exact time of its synthesis has not been determined as yet. Fat body from males produces only small quantities of the major yolk protein (Figure 1c, d). From these results it is clear that the yolk proteins in Drosophila species are synthesized in the fat body, released in the hemolymph and taken up by the oocytes. This last step appears to be mediated by juvenile hormone (Kambysellis and Heed, 1974). (Supported by NSF Research Grants GB-29288 and GB-34168.)

Table 1. Fat body of D. silvestris cultured in 100 µl of tissue culture medium for six hours in the presence of H^3-leucine and H^3-phenylalanine.

Fat body	TCA precipitated proteins (cpm)	Antibody precipitated proteins (cpm)
females	7,586	2,026
males	931	878

References: Telfer, W.H. and L.M. Anderson 1968, Devel. Biol. 17:512; Kambysellis, M. P. and W.B. Heed 1974, J. Insect Physiol. (in prep.).

Bortolozzi, J., R.C. Woodruff and T.K. Johnson. University of Texas, Austin.

Frequency of spontaneous lethal mutations in darkness in Drosophila melanogaster.

A preliminary experiment (Woodruff and Bortolozzi, 1973) suggested that the absence of light may increase the frequency of spontaneous sex-linked lethal mutations in D. melanogaster. To test this hypothesis, the following experiment was performed.

Untreated Oregon-RC males 3 days old were pair mated with FM7, y^{31d} w^a 1z v B/sc10-1 virgin females on standard corn meal medium. These matings were divided in two groups. One group (light) was kept under standard laboratory conditions in the light for about nine hours in 24 hours and the other group (dark) was kept in the dark for the entire experiment.

The F_1 FM7/Oregon-RC virgin females were backcrossed with FM7 males and F_2 offspring were scored for the absence of Oregon-RC males. The results of this experiment are summarized in Table 1.

The analysis of Table 1 shows that the frequency of lethal mutations in the dark is significantly higher at the 1% level (Stevens, 1942) than the frequency in the light. The reasons for this increase in spontaneous mutations are unknown. We are currently performing complementation tests with all of the lethal mutations recovered in this experiment. This will enable us to determine if any lethals belong in clusters and to determine if nondisjunction is involved.

Dark	Light
11/1,741 = 0.63%	4/1,862 = 0.22%

Table 1. Frequency of spontaneous recessive sex-linked lethal mutations in the dark and in the light.

It is of interest to speculate that D. melanogaster may have a light dependent repair system. In the dark, this system would be inoperative and would lead to an increase in the frequency of unrepaired mutations. This possibility is being currently investigated.

References: Stevens, W.L. 1942, J. Genetics 43:301; Woodruff, R.C. and J. Bortolozzi 1973, DIS 50.

The senior author was supported by Fundação de Amparo à Pesquisa do Estado de São Paulo, Brazil.

Cantelo, W.W. and A.L. Boswell. Plant Genetics and Germplasm Institute, ARS, U.S. Department of Agriculture, Beltsville Maryland. Mite control with chemicals in a Drosophila culture.

When we were confronted by a large infestation of mites in our Drosophila colony, we treated the colony with benzyl benzoate, which had been found by others to eliminate mites from their colonies (DIS 20:96, DIS 46:156). However, this treatment was ineffective for us, possibly because we had a different mite species. Our colony was infested with Proctolaelaps hypudaei (Oudemans)(det. by R.L. Smiley) of the family Ascidae, a cosmopolitan mite. It feeds on mites and other small arthropods and probably caused depletion of the Drosophila culture by feeding on the Drosophila eggs and affecting the Drosophila behavior.

To determine whether chemicals used commercially to control plant-feeding mites would control P. hypudaei, we treated 220-ml jars containing ca. 70 ml of diet medium, adult Drosophila and many hundreds of mites with miticides. The miticides used were Dowco® 213 (tricyclohexylhydroxytin), oxythioquinox, binapacryl, dicofol, and propargite. Each was applied to a different jar as a 2% dust (0.034 g ± 0.002 s.e. per jar), with 3 replicates. Also included was a set of replicates treated with 10% benzyl benzoate in alcohol, the air-dried weight of which was 0.075 g ± 0.002 s.e.

When the cultures were examined 3 days later, the mites in the jars treated with binapacryl, dicofol, propargite, and methyl benzoate did not appear to be affected by the treatments. In the 3 jars treated with Dowco® 213, 2 living mites were observed, and the jars treated with oxythioquinox had 3 living mites. Dowco® 213 and benzyl benzoate killed all the flies. Therefore, the chemical of choice for controlling P. hypudaei, when adult Drosophila mortality is unwanted, would be oxythioquinox.

Barker, J.S.F. and I.R. Bock. University of Sydney; University of Western Australia, Perth, Australia. The Drosophilidae of Sulawesi, Indonesia.

As Bock and Wheeler (1972) note that there are effectively no records of the Drosophilidae of Sulawesi (Celebes), two collections were made by J.S.F.B. during a short visit in February, 1973. One collection was made over fermenting banana and pineapple in the grounds of Hotel Victoria, Ujung Pandang (Makassar), and one by sweeping over rotting fruits and refuse in a picnic ground surrounded by rain forest at Bantimurung (about 30 km north-east of Ujung Pandang). The species recorded were:

	Ujung Pandang	Bantimurung
Drosophila (Drosophila) spp. (immigrans sp. gp.)	-	45
Drosophila (Sophophora) melanogaster gp. ♂♂:-		
D. ananassae	1	-
D. atripex	14	2
D. bipectinata	7	1
D. eugracilis	-	1
D. malerkotliana	-	1
D. parabipectinata	10	1
D. pseudoananassae	1	-
Drosophila (Sophophora) melanogaster gp. ♀♀:-	5	21
Drosophila (Scaptodrosophila) spp.	-	3
Liodrosophila sp.	-	1

The difference in sex-ratio of the D. melanogaster gp. between the two collections is quite remarkable, and it is hoped to investigate this further in future collections.

Reference: Bock, I.R., and M.R. Wheeler 1972, The Drosophila melanogaster species group. Studies in Genetics VII, Univ. Texas Publ. 7213:1-102.

Tsusue, M. Kitasato University, Sagamihara, Japan. Metabolism of eye pigments in mutant sepia of D. melanogaster.

Sepiapterin, yellow pigment of sepia flies, have been supposed to be a precursor of drosopterin, red eye pigment of wild type flies. Four red pigments, namely, drosopterin, isodrosopterin, neodrosopterin and drosopterin-D have been found in the wild type flies, but biosynthesis and chemical structures of these red pigments have not yet been established.

Sepiapterin was decomposed by silkworm deaminase to yield corresponding lumazine of sepiapterin (xanthopterin-B_2)[1]. The enzyme was distributed in many tissues of silkworm (lemon) and high specific activity was found in malpighian tubes and integuments. The enzyme had high substrate specificity and only sepiapterin and isosepiapterin were deaminated as far as examined. The pH optimum of the enzyme was at 8.0, and activity of the enzyme was inhibited competitively by many pteridines, for instance 2-amino-4-hydroxypteridine, biopterin and xanthopterin[2]. The enzymatic reaction was formulated as follows.

$$\text{sepiapterin} + H_2O \longrightarrow \text{xanthopterin-}B_2 + NH_3$$

On the other hand, sepiapterin was reduced by silkworm (fat body) enzyme in the presence of NADPH[3]. The product of the enzyme was dihydrobiopterin as well as mammalian sepiapterin reductase[4]. Dihydrobiopterin was further reduced by dehydrofolate reductase to yield tetrahydrobiopterin which was a cofactor of aromatic amino acid hydroxylation[4]. Although a part of biochemical significance of these pteridines have been clarified, the reason why the pigments accumulate in the eyes of flies remains still unknown.

References: 1. Tsusue, M. 1967, Experientia 116; 2. Tsusue, M. 1971, J. Biochem. (Tokyo) 781; 3. Matsubara, M., M. Tsusue and M. Akino 1963, Nature 909; 4. Nagai, M. 1968, Arch. Biochem. Biophys. 426.

Richmond, R.C. and A.W. Finkel. Indiana University, Bloomington. Selection for thermal preference in D. melanogaster.

The ability of Drosophila flies to select a preferred temperature range has been little investigated. We have designed a temperature gradient apparatus for use with small insects and are subjecting a wild collected stock of D. melanogaster to selection for hot and cold temperature preferences (Cozad et al., 1973). After nine generations of selection in the Hot line and ten generations of selection in the Cold line, a clear response to selection has been obtained. Figure 1 shows the responses for females for both the Hot and Cold lines. Approximately 200 flies of each sex are tested in each generation and 5-10% of these flies are selected as parents

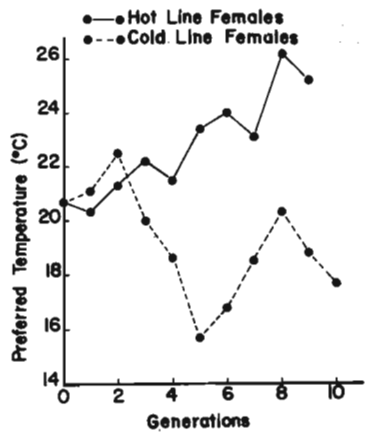


Figure 1. Response to selection for hot and cold temperature preferences in female D. melanogaster.

for the next generation. The regression of mean preferred temperature on generation is highly significant ($p < .001$) for both sexes in the Hot line and borders on statistical significance ($.05 < p < .10$) for both sexes in the Cold line. These behaviors thus appear to have a low but significant heritability and are likely to be of value in studies of the genetics of behavior in Drosophila. Supported by NIH grant ROI-GM 18690.

References: Cozad, S.J., R.C. Richmond and A.W. Finkel 1973, submitted to Ecology.

Frei, H. University of Geneva, Switzerland. New white alleles by recombination in Drosophila hydei.

w^{iv} of D. hydei is a slightly ivory-tinged mutant of the white locus. Only one coloured allele, w^{ak}, is known. Darker alleles (w^{hg}, w^{fr} and w^{gl}) are mutational derivatives of w^{iv}. They arose either spontaneously or in an X-irradiation experiment (w^{fr}) in attached-X chromosomes. After detachment, intragenic exchange properties of these secondary alleles were studied in w^{ak}/w^{hg}, w^{ak}/w^{fr} and w^{ak}/w^{gl} genotypes. Among the offspring of such compound females, wildtype recombinants were found as well as the reciprocal white-eyed exchange types (R). Furthermore, exceptional white-eyed recombinants (E) exhibiting the marker combination of the wildtype recombinants were found among the offspring of w^{ak}/w^{hg} and w^{ak}/w^{fr}. From females w^{+}/w^{hg} and w^{+}/w^{fr}, but not from w^{+}/w^{gl}, white-eyed recombinational types could be obtained (E*), the direction of exchange being the same as in the case of E-types. Recombinant males were individually crossed to attached-X females C(1)RM, w^{iv} y^{lt}, and of four different stocks thus established, male offspring was used for a chromatographic study of allele expression.

By thin layer chromatography of head extracts I examined the pattern and the relative amounts of fluorescent eye pigment components in R of w^{fr}, E and E* of w^{fr}, and E* of w^{hg}. Fluorescence was induced by filtered UV at wavelength 365 m μ, and its intensity measured at different wavelengths of the visible spectrum on a Zeiss "Chromatogramm-Spectro-Photometer." In the examples illustrated, flies reared at 25°C were aged for 1 week and used when 7-10 d. old. Extracts of 20 ♂ heads in 0.1 ml of methanol/NH_3(25%)/H_2O(5:1:4) gave sufficient material for 2 chromatographs. Ascending chromatography on thin layer cellulose foils (Merck) was applied in one-dimensional runs of 8 hours in propanol/NH_3(25%)/H_2O(10:1:4). The chromatograph readings (Figure) were taken at wavelengths in the blue part around 440 m μ, from the starting point to about RF 0.5.

R of w^{fr}, which is a double mutant as shown by its separability by recombination into w^{ak} and w^{fr}, contains a minimum of fluorescent pterins. By contrast, E and E* of w^{fr} contain relatively high amounts of, mainly, biopterin, isoxanthopterin and xanthopterin, thereby

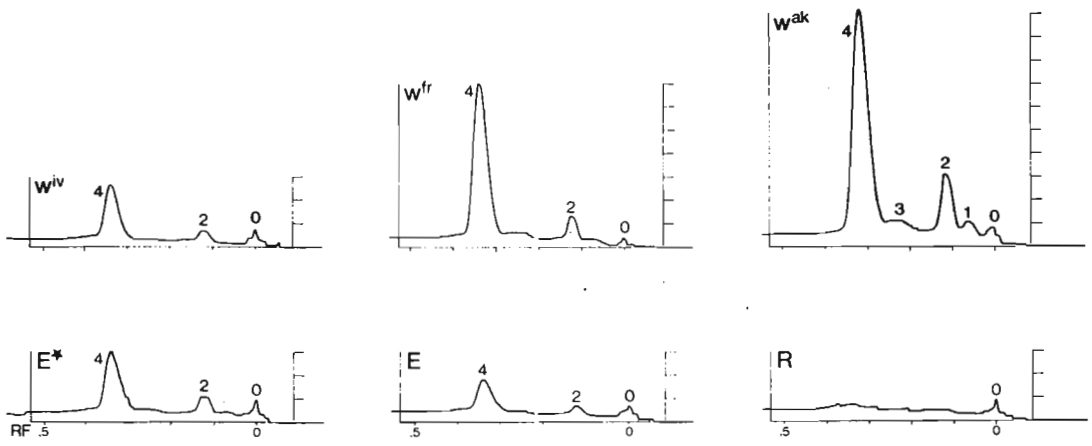


Figure: Pterin fluorescence patterns from two simultaneously prepared series of chromatographs. Top row: original white alleles, w^{iv}, w^{fr} and w^{ak}. Bottom row: White-eyed types from recombination experiments, E* of w^{fr}, E of w^{fr} and R of w^{fr}. 0 = starting point, 1 = pterincarbonic acid, 2 = isoxanthopterin + xanthopterin and allied substances, 3 = yellow pterin and allied substances, 4 = mainly biopterin, admixture of 2-amino-4-hydroxypteridine. The same intensity scale of arbitrary fluorescence units applies to the six examples shown.

resembling rather closely the original w^{iv} allele. However, in 6 independent comparisons, E* showed a higher concentration than E of the substances mentioned, w^{iv} being intermediate in most of the cases. E* of w^{hg} (not shown in the Figure) produced chromatographs that were nearly empty, and similar in pattern to those from R of w^{fr}.(Continued at bottom of next page)

Grossman, A.I. The Hebrew University, Jerusalem, Israel. α-glycerophosphate dehydrogenase in males and females of Drosophila melanogaster.

Three alleles have been recently described in the α-Gpdh locus of Drosophila melanogaster, and the isozymes produced by these alleles have been identified (Koreneva & Grossman 1970). The identification of specific isozymes is achieved by electrophoresis, but in a survey of some wild populations in the Soviet Union we found several flies in which the bands were smeared and did not allow definite identification. The same phenomenon was also found in other studies.

Flies were collected from a wild population in Qiryat Anavim, 10 km west of Jerusalem. Whole fly homogenates were prepared on glass, in a drop of distilled water. These homogenates were run in starch gel electrophoresis, with a discontinuous buffer system, TRIS - citric acid, pH 7.9. Staining was done by the usual procedure.

The front lines of the samples from females showed further movement than those of males, and their bands came out smeared and unclear (Figure 1). It was impossible to determine the

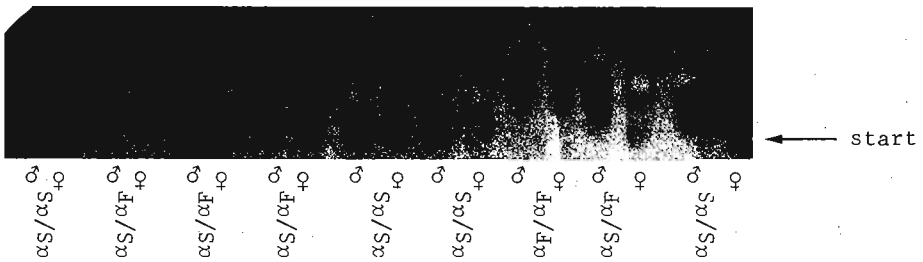


Figure 1

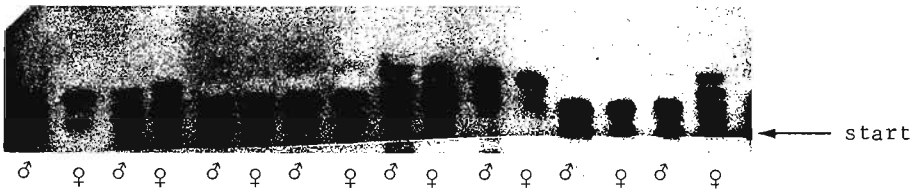


Figure 2

genotypes of the females with respect to the α-Gpdh locus. Clear bands were obtained after electrophoresis was performed with homogenates of females after removal of the abdomen (Figure 2).

It may be that the inability to get clear bands from whole fly homogenates of females is due to the presence, in these homogenates, of proteins associated with developing eggs.

References: Koreneva, L.G. and A.I. Grossman 1970, Genetica (Russ.) 6(6):126-128.

(Continued from preceding page)

Considering that all the exceptions arose through crossing over, but were white-eyed instead of wildtype, it may be postulated that "incorrect" recombination has created new hypomorphic alleles. In line with this, they could be interpreted as more or less important deficiencies of the white gene with meiotic mispairing and unequal exchange at their origin. Alternatively, the w^{iv}-derivatives (w^{fr} and w^{hg}) might already be double mutants of white. In that case, the allelic crosses carried out would represent trifactorial exchange experiments, so that new sublocus combinations would be expected to appear, comprising from zero to three mutated sites.

Supported by Fonds National Suisse de la recherche scientifique, grant 3.224.69.

Stewart, B. and J.R. Merriam. University of California, Los Angeles. Segmental aneuploidy of the X-chromosome.

Aneuploidy, the condition of genetic imbalance, appears to be detrimental in animals, and, when the aneuploid region is sufficiently large, lethal. These deleterious effects of aneuploidy may be thought of as arising from two sources; a relatively small number of genes each, when aneuploid, with a large detrimental effect, or a number of loci, each with a small effect, which cumulatively result in the ill effects of aneuploidy. It should, by examining a series of aneuploids, be possible to distinguish these two possible causes. A gene with a large aneuploid effect should be mappable, where the cumulative effect of many genes will map to a region rather than to a specific locus. It has recently been shown by Lindsley and Sandler et al. (1972) that the latter is the case for the major autosomes of Drosophila melanogaster. The present study was initiated to observe the effects of aneuploidy on the X-chromosome. The X may potentially be different from the autosomes in its tolerance to aneuploidy, as it is itself present in different numbers in the two sexes, and is subject to the regulation imposed by dosage compensation. Furthermore, a series of deficiencies covering the entire X provide a valuable tool for the mapping of mutants and of the structural genes of enzymes.

In order to generate such a series of aneuploids, translocations involving a y-marked X and the $B^{S}Yy^{+}$ were isolated. y females were mated to $y/B^{S}Yy^{+}$ males. Among the progeny of this cross was a $y/y/B^{S}Yy^{+}$ female, which was back crossed to a $y/B^{S}Yy^{+}$ male to establish a stock. From this stock, $y/y/B^{S}Yy^{+}$ females were collected and mated to FM7/Y males. $y/B^{S}Yy^{+}/Y$ males were isolated from this cross, irradiated with X-rays, and mated to either FM7a (a female fertile variant of FM7) females or $\overline{XX}$ females. The $y^{+}B^{S}$ female progeny of the cross of FM7a females to irradiated males were individually mated to FM7a males. From the cross of $\overline{XX}$ females by irradiated males, $y^{+}B^{S}$ males were individually mated to $\overline{XX}$ females. The progeny of both sets of crosses were scored for the presence of translocations involving the X and the $B^{S}Yy^{+}$.

Using these procedures, 75 X;Y translocations were recovered. Information concerning their X and Y breakpoints, male viability and fertility, and aneuploids observed in $\overline{XX}$ stocks is listed in the tables below. Only a minority of the translocations listed could be used in the crosses described below. They were mostly lines with a single break each in the X and Y chromosomes and that could be maintained with viable and fertile males in a $\overline{XX}$ stock.

By crossing two of these translocations with displaced X breakpoints and Y breaks involving different arms, it is possible to recover among the F_1 progeny individuals aneuploid for the region between the two X breakpoints. Duplication-bearing males and females and deficiency-bearing females are generated in each cross, and can be genetically distinguished from each other and from euploid progeny. The results obtained from crossing the X;Y translocations pairwise to generate X chromosome aneuploids are shown in the figure below. Each cross was set up using 15 parents of each sex. Crosses were done reciprocally where possible, and at least in duplicate.

In all cases, hyperploid females were viable and fertile. These duplication-bearing females often had the phenotypic changes associated with hyperploidy, such as smaller size, misshapen wings, and abnormal abdomens.

Males bearing duplications for all the regions of the X-chromosome were recovered with the exception of the two regions, 3A - 3E and 11D - 12E. Males with a duplication of the former region can be generated in other ways; the failure of the duplication-bearing males to survive in this case is thought to be due to lethal genes in the translocation stocks used. The region 11D - 12E most probably does not survive due to the large size of the region.

Hypoploidy of the X-chromosome is less well tolerated than is hyperploidy. Using the translocation stocks, deficiencies uncovering about one third of the X have been generated. In the rest of the chromosome, the X breakpoints of the translocations are farther apart than the four lettered subunits set by Lindsley and Sandler et al. (1972) as the standard for testing a region for deficiencies. One haplo-lethal locus, 7B - 7C (B5 x B17), has been identified.

Fertility of the duplication-bearing males and females carrying heterozygous deficiencies appears to be a function of the size of the aneuploid region. Flies with smaller aneuploid regions are more likely to be fertile than those aneuploid for a larger region. This size relationship is not constant over the entire chromosome, however; some areas of the X seem able to tolerate more aneuploidy than others.

A number of phenotypic effects were associated with the aneuploids. Males hyperploid for the region 8F - 11A (B52 x B53) had curled wings; females hypoploid for this region had out-

stretched wings. Aneuploidy of the region 8C - 11A (J8 x B53) gave the same phenotypes, but duplications or deficiencies of smaller portions of this region did not. The phenotypes were variable; not all of the flies aneuploid for the region displayed abnormal wings. Of the seven Minutes reported on the X, five occur in regions not expected to survive as heterozygous deficiencies because of the large size of the region. No evidence was found for the existence of Minute 1(k); heterozygous deficiencies of the reported location of Minute 1(k) were not associated with a Minute phenotype. A Minute phenotype appeared in deficiencies of the region 14F - 15D/E (B25 x B10, B35 x B10, B35 x B18), which corresponds to the location of Minute 1(o).

From the results of the viability and fertility of aneuploids of the X-chromosome, it would appear that the detrimental effects of aneuploidy result from the cumulative effects of many genes rather than from a few genes with relatively large effects, since aneuploidy is tolerated in almost all regions of the X-chromosome if the region is small enough. The X-chromosome is thus similar to the major autosomes in this respect.

Additional X;Y translocations are currently being recovered and analyzed. With the X-chromosome breakpoints closer together, it should be possible to complete the study of the effects of aneuploidy on the X-chromosome, and to generate a series of overlapping deficiencies uncovering the entire X to be used for mapping new mutants.

Table 1. X;Y translocation stocks

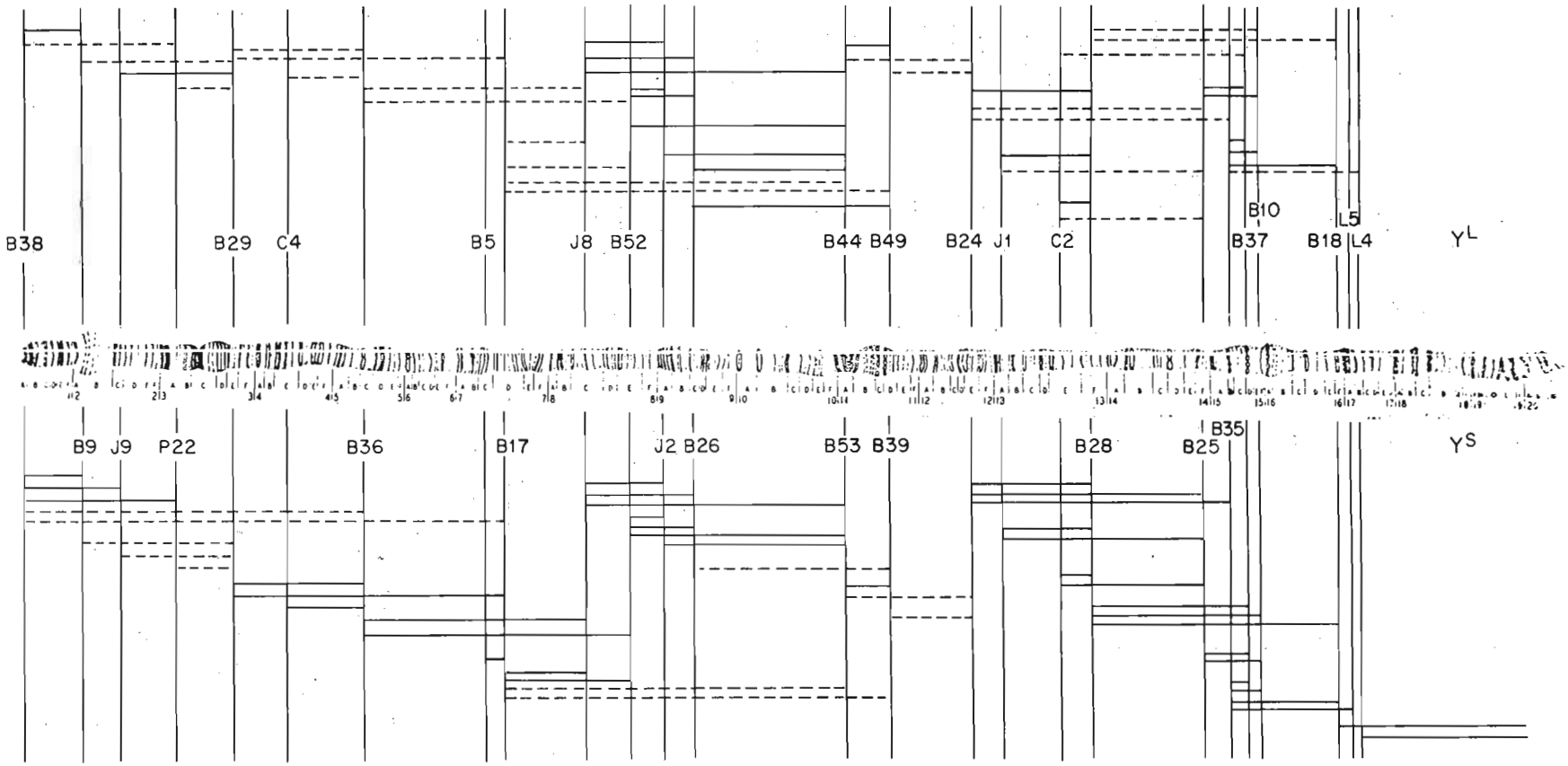
Stock	X Breakpoint	Y Breakpoint	Males	Stocks Kept	Aneuploids in $\overline{XX}$ Stock
B48	tip, 3F	Y^S	viable, fertile	y w f	y w^+ f B^S
B38	tip	Y^L	viable, fertile	y w f/FM7	y^+ w f
B41	tip	Y^S	viable, fertile	y w f	y w f B^S
B11	1B, 4A	insertion	viable, fertile	FM7	-----
J14	1D	?	lethal	FM7	-----
B4	1F, 3C	insertion	viable, fertile	y f	y^+ f B^S
B9	2A	Y^S	lethal	FM7	-----
B27	2B	insertion	viable, fertile	y w f	y^+ w f B^S
J9	2C	Y^S	lethal	FM7	-----
P22	3A	Y^S	lethal	FM7	-----
B33	3A-B, 12E	insertion	viable, fertile	y w f	y^+ w f B^S
P5	3C	?	lethal	FM7	-----
B3	3C2,3	insertion	viable, fertile	y f	y^+ f B^S
B29	3E	Y^L	viable, fertile	y w f/FM7	y^+ w^+ f
C10	3E, 7F	?	viable, fertile	y f/FM7	-----
C4	4C	Y^L	viable (barely)	FM7	-----
B36	5C	Y^S	viable, fertile	y w f/FM7	y w^+ f B^S
B5	7C	Y^L	lethal	FM7	-----
B17	7D	Y^S	viable, fertile	y f/FM7	y f B^S
J8	8C	Y^L	viable, fertile	FM7	-----
B52	8F	Y^L	viable, fertile	y w f/FM7	y w f^+ B^S, y^+ w^+ f
J2	9A	Y^S	viable, fertile	FM7	-----
B26	9C	Y^S	viable, fertile	y w f/FM7	y^+ w f^+, y w^+ f B^S
L3	9C-D	insertion	viable, fertile	y f	y^+ f B^S
P11a	10C	insertion	lethal	lost	-----
P11	10C	insertion	viable, fertile	y f/FM7	-----
B44	11A	Y^L	viable, fertile	y w f/FM7	y w f^+ B^S, y^+ w^+ f
B45	11A	Y^S	viable, fertile	y w f/FM7	y^+ w f^+, y w^+ f B^S
B53	11A	Y^S	viable, fertile	y w f/FM7	y^+ w f^+
J4	T(X;Y;3) 11A, 87	?	lethal	FM7	-----
S15	11B	Y^S	lethal	FM7	-----
B16	11B	Y^S	lethal	FM7	-----
P7	11C-D	?	lethal	FM7	-----
B39	11D	Y^S	viable, fertile	y w f/FM7	y^+ w f^+
B49	11D	Y^L	viable, fertile	y w f/FM7	y^+ w^+ f, y w f^+ B^S
B7	12B-C	?	lethal	lost	-----
L1	12E	Y^L	viable, fertile	FM7	y f^+ B^S

B24	12E	Y^L	viable, fertile	y w f/FM7	y w f^+ B^S
B32	12E	Y^L	viable, fertile	y w f	y w f^+ B^S
B51	12E	Y^L	viable, fertile	y w f	y w f^+ B^S
C8	T(X;Y;2) 12E, 26	?	lethal	FM7	-----
B46	T(X;Y;2;3) 12E, 2R, 3R	Y^S	viable, fertile	y w f	y^+ w f^+
J1	13A	Y^L	viable, fertile	y f/FM7	y f^+ B^S
C2	13E-F	Y^L	lethal	FM7	-----
P12	13E-F	Y^S	viable, fertile	FM7	-----
B28	13F	Y^S	viable, fertile	y w f/FM7	y^+ w f^+
M1	14A	?	lethal	FM7	-----
B6	14E-F	Y^S	viable (barely)	FM7	-----
B25	14F	Y^S	viable, fertile	y w f/FM7	y^+ w f^+
B35	15B	Y^S	viable, fertile	y w f/FM7	y^+ w f^+
B13	15D	Y^L	viable, fertile	lost	y f^+ B^S
B37	15D-E	Y^L	viable, fertile	y w f/FM7	y w f^+ B^S
B10	15E	Y^L	viable, fertile	y f/FM7	y f B^S
P21	15E	Y^L	viable, fertile	FM7	y f^+ B^S
B47	15E	Y^L	viable, fertile	y w f	y w f^+ B^S
B8	16D	Y^L	lethal	FM7	-----
L5	16F	Y^L	viable, fertile	y f/FM7	y f B^S
B18	16F-17A	Y^L	viable, fertile	y f/FM7	y f B^S
C7	17A5,6	?	viable, sterile	FM7	-----
L4	17B-C	Y^L	viable, fertile	y w f/FM7	y w f B^S
J10	17 D-E (complex)	Y^S	viable, fertile	y f/FM7	y^+ f
P8	18A	?	viable, sterile	FM7	-----
B50	18A	Y^L	viable, fertile	y w f	y w f B^S
P6	18B	Y^L	viable (barely)	FM7	-----
S33	20		viable, fertile	y f/FM7	-----
P3(y)	20		viable, fertile	FM7	-----
J11(y)	20		lethal	FM7	-----
B12(y)	20		viable, fertile	y f/FM7	-----
S23	20		viable, fertile	FM7	-----
J6	20		viable, fertile	y f/FM7	-----
B31	20		viable, fertile	y w f	y^+ w f
B34	20		viable, fertile	y w f	y w f B^S
C1	complex		lethal	FM7	-----
C5	complex		lethal	FM7	-----
B43			viable, fertile	y w f	y^+ w f B^S

Table 2. Summary of the analysis of 75 X;Y translocations.

	Procedure A	Procedure B
Male viable and fertile		
Single X break; Y^L	9	11
Single X break; Y^S	3	9
Double X break; insertion into Y	5	3
Complex	2	2
Other	5	3
Male viable and sterile		
Single X break; Y^L	2	-
Single X break; Y^S	1	-
Other	2	-
Male lethal		
Single X break; Y^L	3	-
Single X break; Y^S	5	-
Double X break; insertion into Y	1	-
Complex	2	-
Other	8	-

Heterozygous deficiencies in females



Duplications in males

—— = *viable*

---- = *nonviable*

Kirschbaum, W.F. and H.G. Fromme.
Comisión Nacional de Energía Atómica, Buenos Aires, Argentina. Method of obtaining sections for electron microscopy of squashed salivary gland chromosomes.

A method of obtaining sections for electron microscopy of squashed Drosophila melanogaster salivary gland chromosomes, that we consider to have the following advantages, was developed: 1) Practically with one squash of embedded polytene chromosomes enough material is obtained to study a problem and enough prepared chromosomes left to repeat or pursue further studies; 2) The possibility of selecting, by optical microscopy, the chromosomes or regions of chromosomes already embedded, that are to be studied by electron microscopy; 3) To obtain sections parallel to the chromosome axis of a whole polytene chromosome or a special region that contains several chromosomes.

The procedure is as follows: a) Squash of salivary gland chromosomes with lactic acid orcein using siliconed slide and a cover slip with a carbon film; b) Remove cover slip with liquid N, discard slide; c) 95% ethanol saturated with uranyl acetate for 10 min; d) Wash with 95% ethanol for 10 min; e) 100% ethanol for 10 min; f) 100% ethanol and propylene oxide (1:1) for 10 min; g) Propylene oxide for 10 min; h) Propylene oxide and Epon (1:1) for 10 min; i) 3 passages in liquid Epon for 2 min each; j) 12 hours in liquid Epon in a refrigerator at 4°C; k) Fix cover slip with Epon on a 2 mm thickness plate of Epon; l) Once the Epon is polymerized, remove cover slip with liquid N; m) Select desired chromosome or region of chromosomes with optical microscope and cut the chosen piece with a fine saw; n) Fix plate piece with chromosomes facing up to the block; good care has to be taken that the plane of the chromosomes is perpendicular to the block axis.

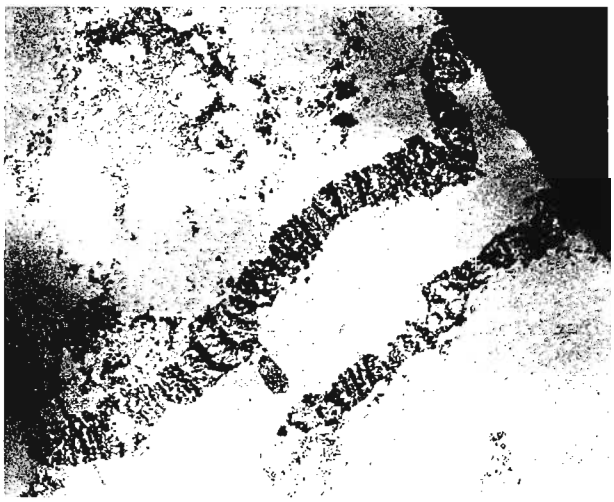


Figure 1. Section of a single whole U-shaped chromosome, which is partially covered by the grid; scanning electronmicrograph x 1540 magnification.

Figure 2. Detail of chromosome x 16000 magnification.

Good squashes and stained chromosomes are obtained using lacto-acetic orcein which makes it easy to choose the right ones already included in the Epon plate, with the optical microscope. It acts also as a good fixative and gives a good resolution to study the banding pattern of the polytene chromosomes. We found that a careful embedding of the chromosomes was very important and came to the conclusion that best sections for this type of study are those of around 90 mμ thickness.

Bischoff, W.L. University of Toledo, Ohio. Fine structure mapping of three lethal mutants within the deep orange gene of Drosophila melanogaster.

Fourteen mutants of independent origin have been assigned to the deep orange locus (1 - 0.3 ±) in Drosophila melanogaster. The absence of complementation between all possible combinations of these mutants and the failure of five of them (dor, dor^{66g}, dor^{61e}, dor^{169f}, and dor^{69L1}) to map in a manner compatible with predictions based on the operon model have permitted the conclusion that this locus best fits the single cistron model of genetic organization (1). The present study was undertaken to determine the topographical relationships between three more of the original fourteen mutants (dor^{169L1}, dor^{169L2}, and dor^{169L3}) and to assign them appropriate positions on the fine structure map of the dor gene. All crosses utilized a crossover selector system which is based on the unique sterility phene of deep orange females and involved mating y dor/Y tester males with females heterozygous for the appropriate pair of dor mutants. Under these conditions such females will produce no progeny unless an event occurs yielding a dor$^+$ gene which will allow the zygote receiving it to survive. Crosses were performed in 1/2 pint bottles on standard cornmeal-molasses-agar medium at 25 ± 1°C. An estimation of the total number of zygotes sampled for each cross was obtained by counting the number of eggs present in 5% of all cultures ten days after set-up. The mutants yellow (y, 1-0.0) and prune (pn, 1-0.8) were used as outside markers lying to the left and right of dor respectively. A summary of all fine structure crosses, dor$^+$ chromosomes recovered, and recombination frequencies is given in Table 1. A revised genetic fine structure map incorporating the

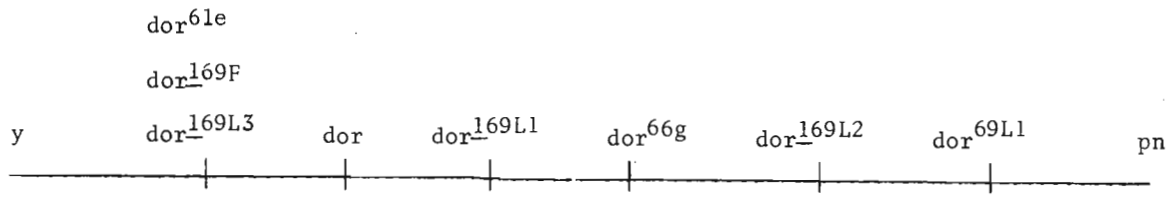
Table 1. Summary of fine structure crosses and surviving progeny.

	Experimental cross	dor$^+$ recombinant chromosomes	dor$^+$ exceptions	Frequency of dor$^+$ alleles
(1)	y dor pn/+ 169L1 + x y dor/Y	2 + + pn	0	2/511,000
(2)	y 66g +/+ 169L1 pn x y dor/Y	1 y + pn	3 + pn	5/890,000
(3)	y dor pn/+ 169L2 + x y dor/Y	2 + + pn	2 + +	4/319,000
(4)	y 66g +/+ 169L2 pn x y dor/Y	3 + + +	0	3/602,000
(5)	y 69L1 +/+ 169L2 pn x y dor/Y	1 y + pn	1 + pn	2/491,200
(6)	y dor pn/+ 169L3 + x y dor/Y	2 y + +	2 + +	4/650,500
	TOTALS	11	9	20/3,463,700

relative positions of the above three mutants is shown in Figure 1. The localization of the lethal mutant dor^{169L1} to the region between the viable mutants dor and dor^{66g} and of the lethal mutant dor^{169L2} to the region between the viable mutants dor^{66g} and dor^{69L1} strengthens earlier conclusions (1) that the various dor alleles do not represent polarity mutants in

Figure 1

Genetic fine structure map of the deep orange locus



several adjacent cistrons of a gene cluster. No attempt was made to determine the relative positions of dor^{61e}, dor^{169L3}, and dor^{169F}, all of which lie to the left of dor since the various heterozygous combinations of these mutants are either lethal or show greatly reduced viability and are therefore not readily adaptable for use in the present selector system. The dor$^+$ exceptions listed in Table 1 represent the products of events which are not associated with outside marker exchange. The possible origins of these exceptions are discussed in detail elsewhere (1).

Reference: (1) Bischoff, W.L. and J.C. Lucchesi 1971, Genetics 69:453-466.

Muhs, H.-J. Forstbotanisches Institut, Freiburg, Germany. Increased frequency of the silent gene (LAP D^0) in laboratory stocks of Drosophila melanogaster during humid-hot summer season.

Homozygous lines were produced for the allele LAP-D^F and LAP-D^S for population genetic experiments. These lines can be distinguished by electrophoresis by the isozyme bands DF and DS of the leucineaminopeptidase (LAP). (See also the technical note on the resolution of a modified micromethod in this issue.) During the humid, hot summertime (from June to the end of August) phenotypes with decreased activity of the enzymes of the D-bands suddenly appeared. The frequency of such phenotypes was able to increase up to 0.3. Therefore it was necessary to stop the already started experiments and to repeat them in the cooler season. Together with the decreased activity of the D-bands one could observe an increased activity, especially of the C1-band and sometimes of the E- and F-bands.

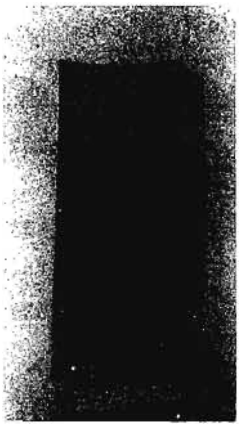


fig.1

fig.2

Figure 1 shows 4 samples of late pupae. The start is at the bottom, the direction of migration leads upwards. The third sample which can be seen on the left side represents a homozygote D^SD^S, the other three samples D^SD^0.

Figure 2 shows as well 4 samples of late pupae. The third sample seen from the left side is a heterozygote D^FD^0, all the others are homozygotes D^FD^F.

The reason for these phenomena is unknown. All during the year the populations were held at a constant temperature of 25°C. It is possible to regulate the temperature exactly but not the humidity. So it happens that warm and humid weather creates condensation water on the inner walls of the incubator. Any mistake in the electrophoretic method can be excluded because parallel samples with full and with decreased activity occur. Phenotypically the samples with the decreased activity of the D-bands look just like the heterozygotes D^FD^0 and D^SD^0 respectively (Muhs, 1973) so that we can take it for granted that they are identical. If all these samples represent such heterozygotes, it remains still inexplicable how the rapid increase of frequency from 0.0 to 0.3 within a few generations is possible. Homozygotes D^0D^0 have a small survival and have only been found twice. As a second interpretation we cannot exclude that in this case a regulator gene is controlling the activity of the D-bands but this had not been proved.

Reference: Muhs, H.-J. (in prep.) Frequency of phenotypes (LAP, Aph and Est) seldom observed in laboratory strains of Drosophila melanogaster.

Postlethwait, J.H. University of Oregon, Eugene. Effects of juvenile endocrine organs and juvenile hormone on the metamorphosis of Drosophila.

Metamorphosis of Drosophila is blocked by substances with juvenile hormone (JH) activity (1-4). The experiments reported here compare the JH syndrome to the effects of juvenile endocrine organs implanted into ready to pupate hosts.

The anterior or posterior thirds of first instar Drosophila larvae were implanted into ready to pupate third instar hosts. In other experiments first instar brain-ring gland complexes or late third instar imaginal discs were implanted into mature larval hosts. After metamorphosis the host was dissected and the position of the implant recorded. The cuticle of the host and the implant were then mounted in Gurr's water mounting medium between two cover glasses. D. virilis and D. melanogaster were used as donors and hosts. The data for both were similar, but were more extensive for D. virilis, and so these appear in Table 1.

Table 1. The effect of implanted parts of first instar upon metamorphosing Drosophila virilis hosts.

Implant	Age of implant	Age of host	Number of recovered hosts	Number of hosts with aberrant cuticle above implant
Anterior third	First instar	Mature third instar	84	17 (20%)
Posterior third	First instar	Mature third instar	87	1 (1%)
Anterior third minus brain-ring gland complex	First instar	Mature third instar	99	5 (5%)
Brain-ring gland complex	First instar	Mature third instar	74	14 (19%)
Imaginal disc	Third instar	Mature third instar	48	0 (0%)

When abdomens of adult hosts were examined abnormalities were evident in 17 (20 percent) of the 84 abdomens that had been implanted with the anterior third of first instar donors. Only 1 abnormality was encountered among 87 abdomens that had received the posterior third of first instar larvae. The abnormalities were localized in the cuticle immediately overlying the implant; they were encountered only when the implant was superficial, never when it was deeply imbedded in the abdomen. As illustrated in Figure 1B, the abnormalities consisted of one or more localized zones closely resembling pupal cuticle (Figure 1D) and differing from adult cuticle (Figure 1A) in terms of the absence of pigment or hairs. Bristles distinctive of adult cuticle were either absent or of aberrant size and shape. Vogt (5) apparently observed similar inhibition of adult differentiation after the implantation of adult corpora allata into mature third instar hosts of D. hydei.

The ring gland of first instar larvae proved to be too small to be dissected and transplanted as such. Therefore, the brain-ring gland complex was implanted into mature larval hosts (Table 1). When the latter emerged as adults, 14 (19 percent) of 74 individuals showed typical integumentary defects. Here again, the local inhibition of metamorphosis was conditional upon the close proximity of implant and overlying cuticle. Control animals received the anterior third from which the brain-ring gland complex had been removed. Only 5 percent of these animals showed the typical defect. This could be due to the retention of the ring gland by some of the anteriors since the ring gland grasps the pharynx rather tenaciously. As a final control imaginal discs from mature larvae were implanted into hosts of the same age, and none of these hosts showed any abnormalities.

These studies, coupled with Vogt's results, show that the ring gland of adult Drosophila and the brain-ring gland complex of first instar Drosophila are capable of inhibiting metamorphosis of the abdomen.

In order to test the effect of exogenously supplied juvenile hormone, 0.005 to 5 µg of C18-Cecropia hormone was applied to white puparia of D. melanogaster or D. virilis in 0.3 µl

acetone. 0.3 µ l acetone served as control solution. Seven days after treatment eclosed and uneclosed animals were dissected and their cuticular parts prepared for microscopic observation.

Individuals receiving in excess of 0.05 µg formed defective pharate adults which failed to emerge. As illustrated in Figure 1C, the abdominal tergites of individuals receiving low doses of JH showed patches of cuticle identical to those produced by the implantation of brain-ring gland complexes. Individuals treated with the highest doses showed underneath the pupal cuticle an entire "adult" abdomen covered by an aberrant cuticle showing an almost complete suppression of pigmentation and of bristles and hairs. The cuticle in many cases was indistinguishable from pupal cuticle. Although the aberrant cuticle could not be distinguished from pupal cuticle it must be noted that the normal pupal cuticle lacks any projections or irregularities which in unstained whole amounts permit its positive and unambiguous identification. But we can conclude that exogenously supplied synthetic JH causes a general syndrome identical to that produced locally by an implanted active young larval brain-ring gland complex, and in both cases the aberrant cuticle is indistinguishable from pupal cuticle.

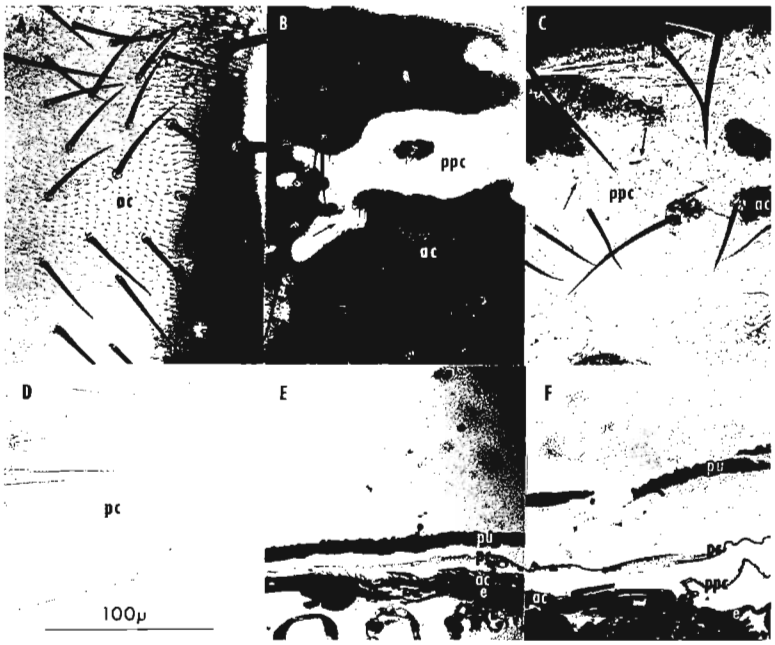


Figure 1. A. Adult cuticle from an acetone treated control.
B. Aberrant adult cuticle lying above the implanted anterior third of a first instar larva.
C. Aberrant adult cuticle from a JH treated pharate adult.
D. Pupal cuticle from an acetone treated control.
E. Histological section of an acetone treated control.
F. Histological section of a JH treated pharate adult.
ac, adult cuticle;
e, epidermis
pc, pupal cuticle
ppc, aberrant adult cuticle
pu, puparium
arrows, abnormal bristles

Experimental animals were also examined histologically by fixation in Bouin's and histological sections were prepared and stained in azocarmine-azan and hematoxylin. All individuals showed three cuticular layers. In the case of the acetone-treated controls, these layers consisted of an external red-staining puparium about 8 µ thick, an intermediate approximately 1 µ thick blue-staining pupal cuticle, and an innermost red-staining adult cuticle about 3 µ thick covered with bristles and hairs (Figure 1E). In the JH-treated individuals (Figure 1F) the two outer cuticles were as in the controls except that the intermediate layer often stained pink rather than blue. Interest of course centered on the innermost cuticle of the experimental flies. This cuticle showed zones which in terms of thickness, staining properties, and surface architecture were indistinguishable from pupal cuticle. Emphasis should be placed upon the color of the aberrant zones, which was blue, a pupal characteristic, rather than red, an adult characteristic.

Although the aberrant cuticle formed under the influence of either JH or juvenile endocrine organs cannot be distinguished from pupal cuticle by the methods here employed, one cannot conclude that the aberrant cuticle is in fact pupal cuticle. Bhaskaran (6) has shown that in Sarcophaga JH causes the production in the abdomen of an abnormal adult cuticle without hairs and bristles in mild cases, and complete suppression of histoblast spreading with no abdominal adult cuticle being formed in severe cases.

References: (1) Dearden, M. 1964, J. Insect Phys. 10:195-210; (2) Bryant, P. and J.

Sang 1968, Nature 220:393-394; (3) Ashburner, M. 1970, Nature 227:187-189; (4) Madhavan, K. 1973, J. Insect Phys. 19:441-453; (5) Vogt, M. 1946, Nature 157:512; (6) Bhaskaran, G. 1972, J. Exp. Zool. 182:127-134.

Supported by a Rockefeller Foundation Grant to Professor C.M. Williams, and grant GM 19307 to J.H.P.

Ganetzky, B. and J. Figenshow. University of Washington, Seattle, Washington. An influence of the compound-generating exchange on ring recoverability in tandem-metacentric compound-X chromosomes.

Lindsley and Sandler (1965) noted that a difference in the pericentric heterochromatic content of two different tandem metacentric chromosomes resulted in striking differences in the transmission of tandem rings derived from these. Since the exchange that leads to the generation of a tandem metacentric can occur in different places in the heterochromatin, it is apparent that the location of this exchange can affect the subsequent behavior of the compounds generated.

We have constructed a number of different tandem metacentrics whose variability in behavior is consistent with this idea. The compounds were recovered from females of the constitution: $X{\cdot}Y^L$, $In(1)sc^4$, EN^R y sc^4 sn g/$Df(1)v^{74}$, $X{\cdot}Y^L$, $In(1)sc^4$, EN^R y sc^4 g/$Df(1)g^1$ y f B, or $X{\cdot}Y^L$, $In(1)sc^4$, EN^R y sc^4 sn g/y cv v f, after irradiation with 2000 R. The compounds recovered showed striking differences, both within and among different constructions, in respect to the ring progeny generated. The results of the constructions are summarized in the following table:

Genetic constitution	No. C(1)TM recovered	No. stable (no ring progeny)	No. unstable (20-25% ring progeny)
$\frac{X{\cdot}Y^L,\ In(1)sc^4,\ EN^R\ y\ sc^4\ sn\ g}{Df(1)v^{74}}$	13	12	1
$\frac{X{\cdot}Y^L,\ In(1)sc^4,\ EN^R\ y\ sc^4\ sn\ g}{Df(1)g^1\ y\ f\ B}$	15	8	7
$\frac{X{\cdot}Y^L,\ In(1)sc^4,\ EN^R\ y\ sc^4\ sn\ g}{y\ cv\ v\ f}$	9	4	5

An example of the behavior in crosses of $C(1)TM/y^+Y$ x $Y^SX{\cdot}Y^L$, y B/O of stable and unstable compounds is:

	Pat♂♂	TM♀♀	Homo.♀♀ (v)	Ring♀♀	Ring♂♂	Exc.♂♂	Exc.♂♂ / Reg.♂♂	TM♀♀ / Reg.♂♂
Unstable	3753	1055	60	1118	1674	200	.05	.28-.30
Stable	1554	613	-	0	0	29	.02	.39

It should be pointed out that in the cases where the tandem metacentric chromosomes are stable they cannot be distinguished genetically or cytologically from reversed metacentrics. However in every case examined where the chromosome was unstable the breakdown product was a ring confirming the compound's identity as a tandem metacentric. Whether stable or unstable, the compounds were heterozygous for the expected markers, consistent with their being tandem metacentrics. Analysis of the data from the stable compounds suggests that rings are formed but they behave as dominant lethals.

Several points of interest emerge from the data presented above. First, since the behavior exhibited by tandem metacentrics can vary quite drastically even when the chromosomes used in their construction are identical, it seems that the initial exchange leading to the

compound does influence the subsequent behavior of ring chromosomes. Second, since the constructions with a vermillion deficiency appear to generate a different collection of compounds, especially with respect to the number of stable compounds, it would appear that the presence of this deficiency influences the behavior of the compound. However, the deficiency could be eliminated by a double exchange and only in one case did loss of the deficiency result in recovery of rings. Thus it seems that it is not the presence of the deficiency in the tandem metacentric itself that affects its behavior, but rather, the deficiency must somehow influence the nature of the compound-generating exchange.

Finally, it should be pointed out that in addition to those tandem metacentrics reported here, Lindsley and Sandler in the course of generating their tandem metacentrics mention the recovery of stable compounds behaving as if they were attached X's and Pasztor (1967, DIS 42: 107) reported the construction of a tandem metacentric which generated rings that had a high frequency of mitotic loss.

Reference: Lindsley, D.L. and L. Sandler 1965, Genetics 51:223-245.

Ripoll, P. and A. Garcia-Bellido. Centro de Investigaciones Biológicas C.S.I.C., Madrid, Spain. A new sc^{VI} translocation to the long arm of the Y.

This translocation arose spontaneously in the stock $Dp(1)sc^{VI}/\mathfrak{C}(1)M3$, $y^2/T(Y:3)P6$ and was detected as a y^+ female. Genetic tests showed that the sc^{VI} element was now translocated to a KL deficient Y chromosome with all the KS factors present. We previously found that the Y, Dp(Y:3)P6 (Lewis, 1972, DIS 48:188) element was a Y chromosome carrying all the fertility factors, besides mwh^+ ve^+. Since the primitive mwh^+ element remained it is reasonable to conclude that the new Y chromosome is sc^{VI}.KS mwh^+ ve^+.

The y^+ of the sc^{VI} element of this Y chromosome variegates very strongly in y males, whereas mwh^+ does not.

Gassparian, S. University of Isfahan, Isfahan, Iran. New mutants in D. melanogaster.

Populations of D. melanogaster from two regions in Isfahan province were studied. A total of 57 mutations were detected from the combined populations of Hossein-Abad and Isfahan region, which was kept for more than 44 generations under artificial selection. The number of mutations detected from the city of Isfahan are 30; the rarest mutation is an allele of miniature called minature of Isfahan, which shows an excellent viability after culturing in Mostashfi medium. One allele, dark red eye color, is a new mutation. This is a single gene mutation on the third chromosome in a distance of 108 ± 1 genetic units. The pure lines of both stocks are available. (Supported by grant #51001 University of Isfahan Research Center.)

QUOTABILITY OF NOTES

Doane, W.W. 47:100
Elens, A. 49:71
Libion-Mannaert, M. & A. Elens 49:77
Mather, W.B. & P. Thongmeearkom 50:60
Nash, W.G., T.B. Friedman & C.R. Merril 50:19?
Portin, P. & M. Ruohonen 49:70
Rizki, T.M. & R.M. Rizki 50:45
Savontaus, M.-L. 45:131
Sharma, R.P. 50:134
Sharma, R.P., K.S. Gill & G.S. Miglani 50:98
Steiner, W.W. 49:125
Williamson, R.L. & W.D. Kaplan 50:134

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

Hanna, P.J. and K.F. Dyer. Monash University, Clayton, Victoria, Australia.
The development of resistance to various organophosphates in populations of D. melanogaster.

Among the main problems associated with the use of insecticides is the development of resistance to particular chemicals and cross resistance to a variety of other chemicals conferred by just one. Over fifty economically and socially important species of insects and mites are now known which show some resistance to one or more organophosphorous compounds. In only a few of these cases has a mechanims of resistance been investigated, but in most of them it is a gene with primarily dominant effects which is responsible.

We have now obtained populations of D. melanogaster which are resistant, either singly or multiply, to six different organophosphates. These range from the simple to the complex and from the relatively nontoxic to the highly toxic. These populations are shown in Tables 1 and 2.

Those populations in Table 1 were started on 18th May, 1972, from a stock of Ore R which had been rendered lethal-free by the Cy BlL technique. The populations in Table 2 were started on 6th February, 1971, as F_1 hybrids between Ore K and Ore R stocks.

The populations are kept in standard metal population cages with 16 food pots which are changed every 2-3 weeks. The phosphates are added to the food medium during the last stages of food preparation. In those populations with two or more phosphates applied, each phosphate is added singly to between 4-8 of the food pots. We have not, as yet, done any experiments in which more than one phosphate is present in all the media. The concentrations are increased when visual inspection shows a sufficient number of larvae emerge from food medium containing the obtained dose to suggest a degree of resistance is present.

Table 1. Resistance of populations of D. melanogaster to organophosphates applied singly.

Compound	Normal toxic level	Now resistant to
TEP	0.018M	0.1M
TEP	0.018M	0.13M
TMP	0.02M	0.04M
TBP	0.02M	0.11M
TIPP	0.004M	0.02M
DEMETON-S-METHYL	10ppm	100ppm
DDVP	0.1ppm	0.8ppm

Table 2. Resistance of populations of D. melanogaster to organophosphates applied multiply.

TEP	0.018M	0.1M
TMP	0.02M	0.06M
TEP	0.018M	0.1M
TBP	0.02M	0.07M
TEP	0.018M	0.1M
TIPP	0.004M	0.03M
TEP	0.018M	0.1M
TMP	0.02M	0.06M
TBP	0.02M	0.05M
TEP	0.018M	0.1M
TIPP	0.004M	0.02M
TMP	0.02M	0.05M
TBP	0.02M	0.05M

TMP - Trimethylphosphate
TEP - Triethylphosphate
TBP - Tributylphosphate
TIPP - Triisopropylphosphite
DDVP - Dimethyl 2, 2 dichlorovinylphosphate

These populations are now available for investigation but one or two points are worth further comment now. Firstly, we have shown in experiments with samples from these populations that there is a measure of cross resistance between a number of these compounds. Resistance to TMP confers resistance to TEP and vice versa, for example, although not very efficiently. More important, perhaps, is the fact that TEP, at least, confers a considerable degree of resistance to DDVP. We have demonstrated this using WHO type aerosol tests on samples from our populations.

In those populations in which TMP only is applied, the maximum dose we have been able to reach is 0.04M. In those in which TEP and TBP are also applied it is 0.06. The difference is due to the male sterilising activity of TMP at doses above 0.002M (Dyer and Hanna, 1972). This sterility is not present in males developing in TEP media which we have mentioned, does confer TMP resistance.

Preliminary tests on the mechanism of TEP resistance by means of diallele crosses among

the various resistant populations suggests that most of the resistance is contributed by the effects of one or a small number of almost completely dominant genes.

Reference: Dyer, K.F. and P.J. Hanna 1972, Mutation Res. 16:327-331.

Garcia-Bellido, A. and J. Dapena. Centro de Investigaciones Biologicas, C.S.I.C., Madrid, Spain. Recovery of cell marker mutants in Drosophila.

This is a preliminary report on some new cell marker mutants detected by mitotic recombination in somatic cells. Wild type males were mutagenized, with EMS (0.3%), and crossed for three subsequent generations with their sisters. The males of the third generation were then crossed to cn bw;e females, their offspring were irradiated (1.000R X-rays) as 84 ± 12 hrs. old larvae to induce mitotic recombination (MR) and the adult males were singly mated to SM5/ $T(2,3)$ e/Ubx^{130};C(4)RM spa^{pol} females. Once their offspring were ensured, the males were sacrificed and one by one mounted for microscopic examination. Their dorsal thoraces and abdomens were then scored for the appearance of clones of abnormal cuticular structures. The treated chromosomes of the presumptive mutant-carrying males were kept balanced over SM5; Ubx^{130}. Identification of the mutant-carrying autosomes and arm localization was again carried out by mitotic recombination. Quantitative analysis of twin spots with known cell marker mutants (Garcia-Bellido, 1972 Mol. Gen. Genetics 115:54 -) gave the approximate mitotic location in the chromosome. In order to locate them meiotically the balanced stocks were crossed to a standard multiply marked (MM) chromosome and the F_1 daughters were backcrossed to the same MM stock. Their offspring were irradiated and the different crossover classes studied for the presence of MR spots of the cell marker mutant. Since this mutant could simultaneously be accompanied by some induced lethals, a viability test of the cell marker mutant was carried out by crossing the complementary left and right recombinant classes carrying the mutant.

As to the usefulness of the new mutants found, a ranking (RK 1-3) has been attempted which states the penetrance of the marker in different organs and cuticular structures. For the sake of comparison mwh is ranked RK 1.

The present method allows the detection of induced - or spontaneous - cell differentiation mutants located anywhere in the Drosophila genome already in heterozygous flies, independently of whether the mutant or the chromosome is a zygotic lethal.

pawn (pwn, 2:58.3 between pr and c); in 2R, 52.2% of the MR between centromere and sdp). Affects both chaetes and trichomes. Chaetes appear truncate due to the depigmentation and subsequent loss of the tip. Trichomes are pin-shaped with a thin transparent process. Homozygous poorly viable in zygote. RK1 in abdomen, RK1 for trichomes and chaetes in the thorax.

sandpaper (sdp, 2:83.1 between pr and px; in 2R, 62.6% of the MR between centromere and y($Dp\ sc^{S2}$). Affects only the pigmentation and the trichome pattern on the tergites (RK1). Cuticle depigmented, trichomes super-numerary, thick, densely packed and uncombed. Lethal - or associated with lethal - in homozygotes. Non-detectable in the thorax.

flare (flr, 3:38.8 between h and th; in 3L, 69% of the MR between centromere and jv). Affects both chaetes and trichomes. The former have a rudimentary socket and their shaft is frequently crooked and branched. Trichomes are transformed into multiple short outgrowths over the entire cell surface. Lethal - or closely associated with a lethal - in homozygotes. An allele of flare, non complementing for lethality, has been independently found. RK1 in both thorax and abdomen. Affects cell viability in thorax.

bald (bld, 3:48.1 between st and cu; in 3R, 87.5% of the MR between centromere and Ki). Affects chaetes, trichomes and cuticle pigmentation. Cuticle completely depigmented, which manifests in transparent chaetes and thin, wooly trichomes. Lethal - or associated with lethal - in homozygotes. RK1 in both thorax and abdomen.

comet (cmt, 3:57.2 between cu and sr; in 3R, bld is 44.5% of the MR between centromere and cmt). Affects chaetes and trichomes. Chaetes small and thin. Trichomes similar to mwh but less regular and with a lower number of processes per cell. Lethal - or associated with lethal - in homozygotes. RK3 in abdomen, for trichomes in thorax RK1.

Ziegler, R. and H. Emmerich*. University of Cologne, Germany. Phosphorylation of chromosomal proteins in Drosophila hydei.

It is generally believed that chromosomal proteins are involved in regulation of the genome in eukaryotes. Histones can be acetylated (Allfrey 1970), phosphorylated (Langan 1969) or varied in their thiol/disulphide ratio (Ord and Stockem 1969). The more tissue specific non-histone chromosomal proteins (NHP) show a correlation between their phosphorylation and the derepression of genes (Allfrey 1970), the electrophoretic pattern of phosphorylated NHP shows specificity for different tissues (Platz et al. 1970, Rickwood et al. 1972). In dipteran giant chromosomes, however, Benjamin and Goodman (1969) could not find a correlation between ^{32}-P-incorporation and puff sites. Their results seemed to us to be worth a re-examination: Those authors used a normal thin layer autoradiography, which has for ^{32}P a resolution of 5 - 10 µm only; they examined only already existing, but not newly induced, puffs and they didn't include biochemical examinations of the chromosomal proteins in the giant chromosomes. We therefore investigated the phosphorylation of chromosomal proteins in D. hydei during puff induction by track autoradiography of ^{32}phosphorous and by electrophoretic separation of the NHP.

Manually isolated salivary glands were incubated in Gehring's balanced saline for 45' with 100 µCi/1.5 ml ^{32}-orthophosphate either in the presence of 10^{-6}M β-ecdysone or, in order to induce temperature puffs, at 37°C. cAMP was used at 10^{-3}M, puromycin at 100 µg/ml. One gland of each isolated pair was used as a control. Mass preparations of salivary glands were made after Boyd et al. (1968). To examine the phosphorylation of NHP in the chromosomes, the glands were squashed in orcein-acetic acid; for the study of the phosphorylation of histones they were fixed in 2.5% glutaraldehyde. After postfixation with 10% formaldehyde in 0.1 M phosphate, the slides were treated with ribonuclease. Track auto radiography was performed with Ilford G5 emulsion after Rogers (1967). NHP from the incubated mass preparations of glands were isolated after Helmsing and Berendes (1971) and separated by iso-electric focussing after Gronow and Griffith (1971). The polyacrylamide gels were sliced and counted in a Packard Liquid Scintillation Counter.

Phosphorylation of NHP: Fixation of the chromosomes with alcohol-acetic acid extracts part of the histones, especially the phosphorylated ones, so in the chromosomes mainly NHP remained. As shown the chromosomes are phosphorylated. We do not think that the label is

Figure 1: Early ecdysone puffs (78B) are induced; temperature puffs (e.g. 48B) already existed. Note low label of the nucleolus.

Figure 2: Early ecdysone puffs are only poorly labeled.

RNA phosphorous as the squashes had been treated with RNase and the nucleolus retained only slight labeling (Figure 1; in looking at the autoradiographs one must keep in mind that this is a track autoradiography and the grains must be followed back to their source). The cytoplasm shows also some label. The label of the chromosomes is randomly distributed and their is no preferential incorporation in pre-existing or newly-formed puffs, either after ecdysone or after temperature treatment (Figures 2 and 3).

Figure 3: Induced temperature puff (48B).

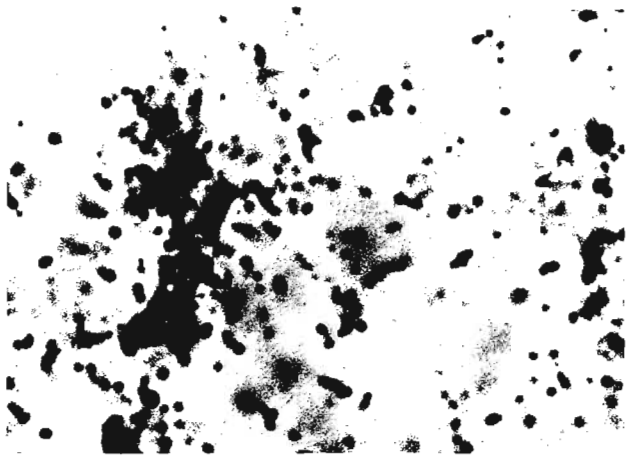


Figure 4: Induced early ecdysone puff (78B), glands fixed with glutaraldehyde.

Phosphorylation of histones and NHP: When the salivary glands were fixed with glutaraldehyde, which does not extract histones, we found a higher incorporation of ^{32}P, but the distribution was essentially like that observed for NHP (Figure 4). There were reports about a histone specific protease which might preferentially attack phosphorylated histones (Marushige and Dixon 1971, Elgin and Bonner 1972). The label could therefore be absent from puffs after prolonged induction, so we incubated the salivary glands for only 10' with ecdysone. The chromosomes just started to develop small ecdysone puffs, but the incorporation of phosphate was very low and not preferential at the sites of puff formation. The suppression of protein synthesis by puromycin did not diminish the ^{32}P-labeling, so it seems to be improbable that newly synthesized proteins would be phosphorylated to a higher extent than pre-existing ones.

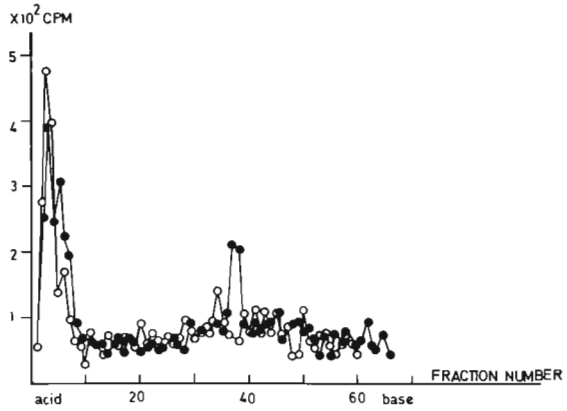


Figure 5: Isoelectric focussing of chromosomal NHP in 10% acrylamide containing 8M urea, 30 min. after temperature shock in the presence of 100 µCi ^{32}P.

Isoelectric focussing of phosphorylated NHP: The NHP of the chromatin from 350 mg salivary glands which were kept at 37°C for 45' in the presence of 200 µCi ^{32}-orthophosphate showed one additional phosphorylated protein band with an isoelectric point of 6.5, whereas the controls kept at 21°C only showed ^{32}P in the very acid part of the gel. The addition of cAMP enhanced the incorporation of ^{32}P into glutaraldehyde fixed chromosomes as compared with cAMP-free controls, but the peak in the isoelectric focussing could not be enlarged by that treatment.

Despite the fact that we could not find a histological correlation between puff induction and chromosomal phosphorylation, we were able to demonstrate a specifically phosphorylated NHP

after temperature shock of mass prepared salivary glands. Since the chromatin was carefully washed and no cholesterol was demonstrable in it by the Liebermann'Burchard reaction, we do not think that this newly phosphorylated protein is a cytoplasmic contamination or deriving from membranes. We have no explanation for the origin or function of that phosphoprotein. It might have a cellular transport or disposal function as Sheperd et al. (1971) have suggested for phosphorylated histones.

References: Allfrey, V.G. 1970, Fed. Proc. 29:1447-1460; Benjamin, W.B. and R.M. Goodman 1969, Science 166:629-631; Boyd, J.B., H.D. Berendes and H. Boyd 1968, J. Cell Biol. 38:367-376; Elgin, S.C.R. and J. Bonner 1972, Proc. Austr. biochem. Soc. 5:18; Gronow, M. and J. Griffith 1971, FEBS Letters 15:340-344; Langan, T.A. 1969, PNAS 64:1276-1283; Marushige, K. and G.H. Dixon 1971, J. Biol. Chem. 246:5799-5805; Ord, M.G. and L.A. Stocken 1969, Biochem. J. 112:81-89; Platz, R.D., V.M. Kish and L. Kleinsmith 1970, FEBS Letters 12:38-40; Rickwood, D., G. Threlfall, A.J. MacGillavary, J. Paul and P. Rickes 1972, Biochem. J. 129:50p; Rogers, A.W. 1967, Techniques in autoradiography, Elseviers Amsterdam; Sheperd, G.R., B.J. Noland and J.M. Hardin 1971, Arch. Biochem. Biophys. 142:299-302.

The work was supported by the Deutsche Forschungsgemeinschaft and by the SFB 74.

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Stark, W.S* and G.S. Wasserman. University of Wisconsin, Madison, Wisconsin.

Erratum in previous note.

DIS 49:63 (1972), Temporal properties of the ERG on-transient recorded in the retina and lamina. Erratum: In the electrical network (figure, part b) certain components were inadvertantly interchanged. The signal source should be located where the monopolar neuron membrane resistance (r) and capacitance (c) were and vice versa. The polarity of the relocated signal source should be positive outside the monopolar neuron.

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Roberts, D.B. and S.M. Moffitt. University of Oxford, England.

Studies on antigens of wild type strains of Drosophila melanogaster.

Extracts of eggs, third instar larvae and flies from 75 different wild type strains either collected locally (4) or at Strömsvreten (10) in Sweden or obtained from different laboratories around the world were analysed on immunoelectrophoresis plates using antisera prepared against extracts of eggs, third instar larvae or flies of the strain Oregon-R. 24 different antigens were studied and in only one case was there an obvious difference between strains. Small changes in electrophoretic mobility which could have been detected by other techniques such as polyacrylamide gels, would not have been detected here. The striking difference was an antigen present in 72 of the 75 strains but not present in Bacup, Bannerdale or Berlin. This antigen (No. 2, Roberts, Nature 1971, 233:394) was present in unfertilized Oregon-R eggs and persisted throughout embryogenesis finally disappearing soon after hatching. In reciprocal crosses between Oregon-R and Bacup the antigen was found in eggs when Oregon-R was the female parent but not when Bacup was the female parent. This together with the disappearance of the antigen soon after hatching suggests that the antigen is synthesised during oogenesis and is diluted out during embryogenesis. The eggs of Oregon-R/Bacup heterozygotes possess the antigen.

To locate the gene responsible for the synthesis of this antigen Berlin flies were crossed with strains carrying marked 1st, 2nd or 3rd chromosomes which also carried inversions. The marked strains all possessed the antigen. Eggs from the F_2 females were tested for the presence of the antigen. Females homozygous for the Berlin 2nd chromosome layed eggs which did not possess the antigen while flies homozygous for Berlin 1st or 3rd chromosome layed eggs which possessed the antigen. This suggests that the gene responsible for the synthesis of this antigen is on chromosome II. No further location studies have been carried out.

Anxolabehere, D., P. Girard, L. Palabost and G. Periquet. University of Paris, France. Genic variation and degree of heterozygosity in natural populations of Drosophila melanogaster.

In an attempt to estimate the genic variations in natural populations, D. melanogaster flies were collected in October 1971 in the south of France and kept in mass culture at 20°C+1. This natural population appears to be highly polymorphic when one looks for visible recessive mutants. Population studies revealed the presence of at least 17 mutants (some of which are described in D.m. New Mutants section).

Allelic variation was then studied on 26 loci. 17 of them were randomly chosen from the visible recessive mutation class and 10 randomly chosen from the allozymic mutation class (rosy locus is in both classes). For the first class, analysis was made on single virgin flies crossed with multi-marked strains; for the second class, electrophoresis on starch gel was performed with the current techniques generally used.

The results are presented in Tables 1 and 2.

Table 1. Frequencies of alleles in the visible recessive mutation class.

Locus	Tested number of flies	Observed number of heterozygotes	% of heterozygotes	% of the mutant alleles
black (b)	184	0	0	0
brown (bw)	100	0	0	0
dumpy (dp)	84	0	0	0
ebony (e)	71	0	0	0
forked (f)	127	0	0	0
frisé (fri)	613	2	0.3	0.2
garnet (g)	103	0	0	0
miniature (m)	127	0	0	0
rosy (ry)	43	0	0	0
ruby (rb)	103	0	0	0
scarlet (st)	43	1	2.3	1.2
sepia (se)	119	1	0.8	0.4
vermilion (v)	127	0	0	0
vestigial (vg)	100	0	0	0
vin (vi)	121	1	0.8	0.4
white (w)	613	7	1.1	0.6
yellow (y)	613	3	0.5	0.2

Table 2. Frequencies of alleles in the allozymic mutation class.

Locus	Tested number of flies	Observed number of genotypes			Allelic frequencies	
		FF	FS	SS	F	S
αGPDH	110	36	50	24	0.56	0.44
Acph.1	120		monomorphic		1.00	0
Aph.2	112	27	(85)*		0.49	0.51
ADH	120	101	19	0	0.92	0.08
Est.6 males	60			60	0	1.00
Est.6 females	60	2	24	34	0.23	0.77
Est.C	120		monomorphic		1.00	0
ME (NADP)	120		monomorphic			
ODH	120		monomorphic		1.00	0
To	240		monomorphic			
XDH (ry)+	120		monomorphic			

* FS and SS indistinguishable, allelic frequencies expected from Hardy-Weinberg distribution.

Interesting comparisons can be made between these two genome samples. In the first class mutations, 35% of the loci tested appeared to be polymorphic without any statistical difference between autosomal and sex linked mutations. The same amount of variation is encountered

in the second class, 40%, but the degree of heterozygosity in the individual's genome is considerably different: 0.3 % in the first case and 11.3% in the second.

For the enzymatic polymorphism the amount of heterozygosity per individual observed in D.m. falls in the range (.08 - .25) already observed on Drosophila species.

However, genetic variants studied in our two classes are probably different. In fact, for the rosy locus the electrophoresis technique allows one to see functional allozymic variants whereas the visible recessive phenotype technique shows the mutation corresponding to a "non functional" allozymic variant.

Thus, systematic comparisons in natural populations of the amount of polymorphism for the two mutation classes will provide interesting information on polymorphism, its nature and its maintenance.

Ce travail a été effectué dans le cadre de l'Equipe de recherche associée n°406 du CNRS: "Analyse et mécanisme du maintien du polymorphisme".

Jayasuriya, V.U. de S. and W.E. Ratnayake
Vidyodaya Campus, University of Sri Lanka, Nugegoda, Sri Lanka. Screening of some pesticides on Drosophila melanogaster for toxic and genetic effects.

The mutagenic effects of chemicals were discovered by Auerbach (1943) using Drosophila melanogaster. Sobels (1973) has very convincingly shown that Drosophila melanogaster is still the organism par-excellence for mutagenic studies. Although D. melanogaster has been used extensively for mutagenic studies, this organism has not been widely used for screening mutagenicity of pesticides. Instead, pesticides have been tested widely for genetic and cytogenetic effects on prokaryotes like E. coli, phage and on eukaryotes like Vicia faba and Allium cepa which are plants (Epstein and Legator 1970).

Benes and Sram (1969) studied the mutagenic effects of 34 chemicals (of which 16 were pesticides), by injecting relatively high concentrations into adult D. melanogaster flies and scoring for sex linked lethal mutations. The pesticides tested were mostly insecticides and they did not show any significant mutagenic activity.

In the present study a larval feeding method was employed for testing the mutagenic effects of pesticides. Initially, various concentrations of different pesticides (six - see Table 1) obtained by the serial dilution method were made up in Drosophila food medium and 50 1st instar larvae were introduced into 3" x 1" vials containing about 8 - 10 ml of treated food medium.

Table I. Pesticides used in the study.

1. Fenbar (fenitrithion)	
2. Endrin	1,2,3,4,10,10Hexachloro 6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo-endo5,8-dimethanonaphthalene
3. Gamalin	Benzene hexa chloride
4. Deenol (D.D.T.)	1,1,1, Trichloro 2,2 bis(p-chlorophenyl) ethane
5. Nicotox	3-(1 Methyl-2-pyrrolidyl) pyridine
6. Stam 34	DDVP

The developmental period and percentage of emergence of adult flies were recorded. The results are summarized in Table II. Data obtained from these dosage mortality studies show that within a certain dose range, which varied from one pesticide to another, percentage mortality increased at different rates. It was also observed that development of D. melanogaster larvae was affected. In general, low concentrations did not affect the developmental period, but at increasing non-lethal concentrations of pesticides development was delayed. This is comparable to observations of Ramel & Magnusson (1969) for organic mercury compounds.

The approximate LD 50 concentrations of pesticides obtained from the above studies were then used to treat 1st instar larvae which were subsequently used in genetic tests. The genetic tests carried out were the Muller-5 technique for sex linked lethal mutations and the induced crossing over test in males. Oregon-K (wild type) larvae were treated for the former Test, while + + + +/dp b cn bw larvae were treated for the latter test. Table III summarizes the data obtained from these tests.

The sex linked lethal frequencies for the pesticides tested are almost at the spontaneous

level (0.52%). Nicotox, however, showed a sex linked lethal frequency between 1-2%. But due to the fact that only three lethals were detected out of 249 chromosomes, it is not possible to infer that this pesticide is definitely mutagenic. With regard to induced crossing over, however, almost every single pesticide has produced crossing over in the 1st three day brood. Approximately 1500 chromosomes were scored in these experiments. This would indicate that the pesticides do have some effect on the genetic material.

The fact that no sex linked lethal mutations were detected for any of the pesticides that were tested, it is almost certain that gross genetic damage is not produced by these pesticides. The induction of crossing over in males does indicate, however, that the pesticides do have some effects on the chromosomes of Drosophila. If the induced cross overs which are produced are due to single strand breaks on the DNA (Ratnayake 1970), it is possible that in the event that mutations are produced by these pesticides, they may occur as mis-sense mutations. Hence the sex linked lethal test is perhaps unable to detect such mutations. In order to detect such mis-sense mutations it would be advisable to use the specific locus method. We propose to carry out such tests in the future.

As cancers may arise due to somatic crossing over, a possible carcinogenic role of pesticides is indicated by the fact that they produce crossing over in the larval testes.

This work was partly supported by a grant NSCA/1/29/2 of the National Science Council of Sri Lanka.

References: Auerbach, C. 1943, DIS 17:48-50; Benes, V. and R. Sram 1969, Industrial Med. Vol. 38 12:50-52; Epstein, S. and M.S. Legator 1971, The Mutagenicity of Pesticides, MIT press; Ramel, C. and J. Magnusson 1969, Hereditas 61:231-254; Ratnayake, W.E. 1970, Mut. Res. 9:71-83; Sobels, F.H. 1973, Asilomar Conference on Environmental Mutagens.

Table II. Dosage/mortality of pesticides tested.

Pesticide	% Concentration	% Mortality	Remarks
Fenbar	0.00004	32.0	-
	0.00006	53.4	-
	0.00008	80.0	-
	0.00009	90.0	development delayed 1-3 days
	0.00010	92.3	" " " "
Gamalin	0.001	20.5	-
	0.002	83.5	development delayed 1-2 days
	0.003	100.0	" " " "
Nicotox	0.002	10.0	-
	0.004	14.7	-
	0.008	18.0	-
	0.010	75.0	development delayed 3 days
	0.012	90.0	" " " "
Endrin	0.0001	10.7	-
	0.0002	45.7	-
	0.0003	65.7	-
	0.0004	75.0	development delayed 3-4 days
	0.0005	89.4	" " " "
Deenol	0.0010	18.0	-
	0.0015	23.0	-
	0.0020	29.0	-
	0.0025	53.0	50% of emergence delayed 1 day
	0.0050	100.0	" " " " " "
Stam 34	0.035	33.7	emergence delayed 2-4 days
	0.0435	86.7	" " " "
	0.0875	100.0	" " " "

Table III. Results of Sex-linked lethal and induced crossing over experiments.

	Sex linked lethal test					Induced crossing over test		
Pesticide	% concentration	% emergence	No X chromosomes tested	No of lethals	% lethals	Cross over types	No of cross overs	Total scored
Fenbar 1	0.00008	69.6	474	1	0.21	1 bw 1 cn bw	2	2047
2	0.00008	58.3	-	-	-	2 dp b cn	2	1409
3	0.00008	63.	-	-	-	1 bw 1 cn bw 1 dp b 1 b cn bw	4	2306
Endrin 1	0.002	59.3	230	1	0.43			
2	0.003	64.0	-	-	-	-	-	-
3	0.004	61.0	-	-	-	1 dp b	1	1285
Gamalin 1	0.001	60.0	405	2	0.50	4 bw 1 dp b cn	5	2246
2	0.001	70.0	-	-	-	2 bw	2	1668
Deenol 1	0.0025	49.0	314	-	-	1 bw 1 dp b bw	2	2061
Nicotox 1	0.008	72.6	249	3	1.20	-	-	2081
Stam 34 1	0.035	52.0	240	2	0.83	-	-	1650
Control		98.0	381	2	0.52	-	-	4963

Postlethwait, J.H. University of Oregon, Eugene. Molting of Drosophila first instar larval cuticle induced by a metamorphosing host.

At metamorphosis third instar larval tissues degenerate. To find whether larval tissues of the first instar larva molt or degenerate during metamorphosis, the anterior thirds of first instar larvae were transplanted into ready to pupate hosts. As figure 1A shows, the implanted first instar larval cells did not degenerate; rather they molted to produce second or third instar mouth hooks (see Table 1). In contrast, anterior thirds of first instar larvae when implanted into adult females only occasionally molted to form second instar mouth hooks and never molted twice to form third instar mouth hooks (Figure 1B and Table 1). Thus mouth hook molting in a metamorphosing host is due at least in part to the host's endocrine organs since more molts occur in a metamorphosing host than in a non-metamorphosing host.

To ascertain whether the implant's ring gland was responsible for sparing the epidermal cells from degeneration or whether this property was inherent in the cells themselves, first instar mouth hooks without brains or ring glands were implanted into metamorphosing or non-metamorphosing (adult) hosts. The results (Table 1) show that mouth hooks without first instar endocrine organs can molt repeatedly in a metamorphosing host. In one case (Figure 1C) we even observed a total of four pairs of mouth hooks which represents one more molt than occurs in situ. This may have been accomplished in a manner similar to the supernumerary molts found by Žďarek and Slama (1), who injected ecdysone into last stage fly larvae shortly after their molt. In the case reported here, the rapid succession of high ecdysone titers during metamorphosis may have elicited three molts from the first instar epidermis. Only fifteen percent of control mouth hooks implanted into adults molted once. These studies show that the ability to molt rather than degenerate is a property of the first instar epidermis rather than the immediate effect of the first instar brain-ring gland complex upon these cells.

The competence of the imaginal discs of the first instar larva to respond to the metamorphosing host by differentiation can also be studied in these experiments. In only seven percent of the transplants of anterior thirds of first instar larvae into mature third instar larval hosts was there any indication of differentiation of adult parts. In one case there

Figure 1. Response of first instar mouth hooks of D. virilis to metamorphosis.
A. Anterior third of a first instar larva transplanted into a ready to pupate host, and recovered from the adult after metamorphosis. Beside the original first instar mouth hooks are the second and third instar mouth hooks, showing that the tissue molted twice.
B. Anterior third of a first instar larva transplanted into an adult host, and recovered seven days later. Only the first instar mouth hooks are present, showing that no molting has occurred.
C. First instar mouth hooks transplanted into a ready to pupate host, and recovered from the adult after metamorphosis. Four pairs of mouth hooks have been formed, indicating three molts. 1,2,3, first, second and third instar mouth hooks; 4, supernummary mouth hooks; S, molted spiracles.

Table 1. Effect of a metamorphosing host on first instar mouth hooks of D. virilis.

Implant	Age of implant	Age of host	Number of recovered hosts	Number of implants in which the mouth hooks molted			Number of implants in which the imaginal discs metamorphosed
				once	twice	thrice	
Anterior third	First instar	Mature third instar	84	60 (72%)	8 (10%)	0	6 (7%)
Anterior third	First instar	Adult	26	5 (19%)	0	0	0 (0%)
Mouth hooks minus ring gland	First instar	Mature third instar	38	35 (92%)	13 (34%)	1 (3%)	-
Mouth hooks minus ring gland	First instar	Adult	20	3 (15%)	0	0	-

was a little eye pigment, in others there were only brown cuticular vesicles with no bristles or hairs. The overwhelming conclusion was that first instar discs lacked the competence to respond appropriately to the hormonal conditions of the metamorphosing host, as shown also recently by others (2-4). This work shows that neither first instar larval cells nor imaginal tissues have developed competence to respond to the hormonal conditions of metamorphosis.

References: (1) Žďárek, J. and K. Slama 1972, Biol. Bull. 142:350-359; (2) Gateff, E. 1972, Thesis, University of California, Irvine; (3) Mindek, G. 1972, Wilhelm Roux Arch. 169: 353-356; (4) Schubiger, G. 1973, in press.

Supported by a Rockefeller Foundation Grant to Professor C.M. Williams and NIH Grant GM 19307 to J.H.P.

Bortolozzi, J.* and L.E. Magalhães.
Faculdade de Ciências Médicas e Biológicas de Botucatu, Botucatu-SP., Brazil. Gene frequency and population size.

Thoday (1963) has suggested that fluctuation in population size may have pronounced genetic consequences in a population of Drosophila melanogaster. He found a significant correlation between the gene frequency of the mutant white (w, 1-1.5) and population size; as the number of flies increased, selection favored the w gene and vice-versa.

The following experiment was conducted to re-examine the results obtained by Thoday. We used three populations maintained in cages (about 2000 cc) similar to those used by Bennett (1956) with some modifications (Magalhães, Bortolozzi and Sene, 1968). Two stocks of D. mel-

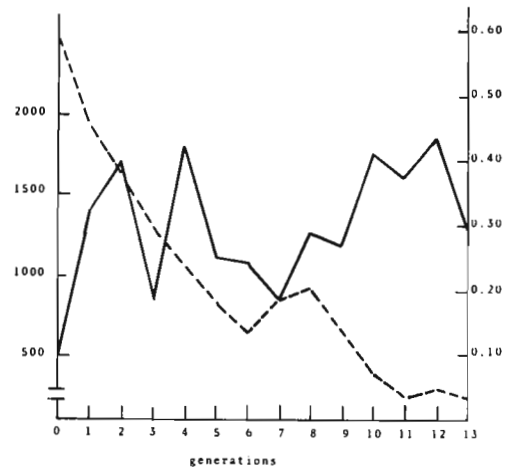


Figure 1A

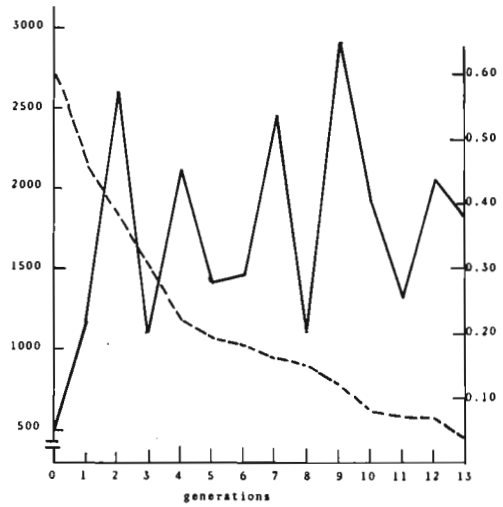


Figure 1B

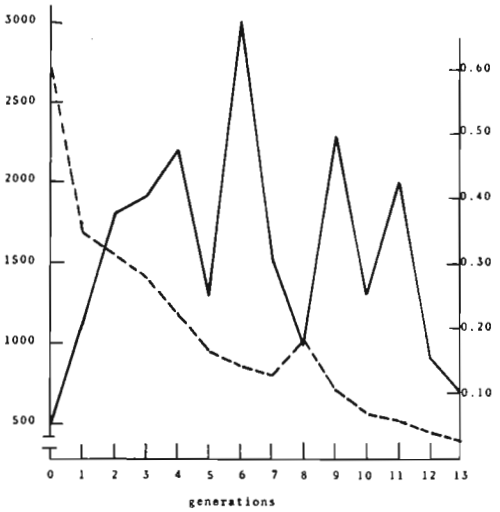


Figure 1C

Figure 1. Population size (————) and mean frequency (-------) of the mutant w in each generation of the cages A (Fig. 1A), B (Fig. 1B) and C (Fig. 1C).

anogaster used in the present experiments had identical genetic backgrounds, except that one stock contained w and the other its wild-type allele. The populations were maintained for 13 generations in such a way that there was no overlapping in generations.

The gene frequency of w (q), in each sex for each generation was estimated by the maximum likelihood method (Cotterman, 1954). The mean gene frequency was calculated using the formula

$$\bar{q} = 2/3\ q(♀) + 1/3\ q(♂)$$

In each population studied, the frequency of the w gene decreased independently of the population size fluctuations. Figure 1, for cages A, B and C, respectively, show clearly that there is no correlation between gene frequency and population size. These results are contradictive to the results of Thoday, but are in agreement with the observations of Thompson (1961). The reason for the contradiction is unknown. One possibility based upon the fact that Thoday used a higher population density than was used in this experiment, is that in a crowded condition the w mutant allele may be favored. We are currently testing this possibility.

References: Bennett, J.H. 1956, DIS 30:159; Cotterman, C.W. 1954, in Statistic and Mathematics in Biology, Ed. by O. Kempthorne et al., pp. 449-465; Magalhães, L.E., J. Bortolozzi and F.M. Sene 1968, Ciência e Cultura 20:197; Thoday, J.M. 1963, Amer. Nat. 97:409-412; Thompson, J.A. 1961, Genetics 46:1435-1442.

Research supported by Fundação de Amparo à Pesquisa do Estado de São Paulo, Brazil.

* Present address: Department of Zoology, University of Texas at Austin, Austin, Texas 78712.

Fattig, W.D. and J.R. Moody. University of Alabama, Birmingham. Recovery of conditional male fertility mutants located on three chromosomes of D. melanogaster.

We have isolated four conditional male fertility mutants located on the Y-chromosome, five located on the 3rd chromosome, and ten putative ones on the X-chromosome. All are characterized by failure to reproduce at 27°C, but are fertile at 22°C. Microscopic analysis of the testes of several of these mutants reveals an absence or reduction of motile sperm at 27°. No conditional morphological variation was observed in electron microscope studies of testes of one of the Y-chromosome mutants. Introduction of males of one of the Y-chromosome strains into population cages containing wild type flies, followed by temperature shift, resulted in a decrease in population fertility. The reduction in fertility was inversely related to the interval of time between introduction of the defective males and temperature shift. Such studies indicate the feasibility of this approach to the biological control of insect pests.

Our data indicate that genes affecting male fertility are widely distributed throughout the genome of Drosophila, and may mutate to conditional states which are useful in studies of the genetic control of fertility.

PERSONAL AND LABORATORY NEWS

Maloglowkin-Cohen, Ch., is now visiting scientist at the Department of Biology, Instituto de Biociências da Universidade de São Paulo, Brasil.

John Sparrow is now at the Department of Biology, University of York, England, after completing a postdoctoral at the University of Virginia, Charlottesville.

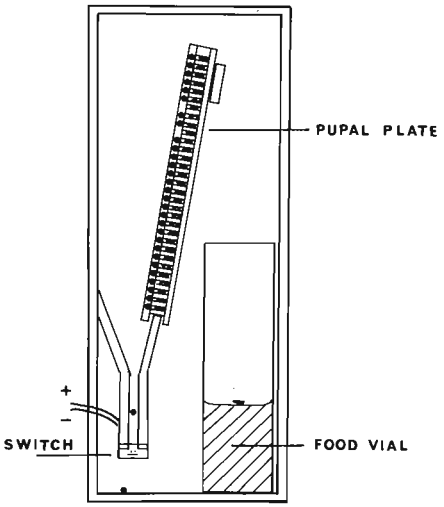
V.G. Vaidya was awarded Fellowship of Alexander von Hunboldt Foundation to work at the Genetics Institute of Freie Universität Berlin for five months (15 Sept. 1973 - 15 Feb. 1974).

R.M. Kothari has moved to the Department of Microbiology, Indiana University, Bloomington and is continuing his work on biochemical genetics.

Part of the population genetics group from the Biology Department of the University of Chicago has moved to the Museum of Comparative Zoology, Harvard University, Cambridge, Mass. 02138. Movers include: A. Gupta, R. Lande, R.C. Lewontin and R. Singh.

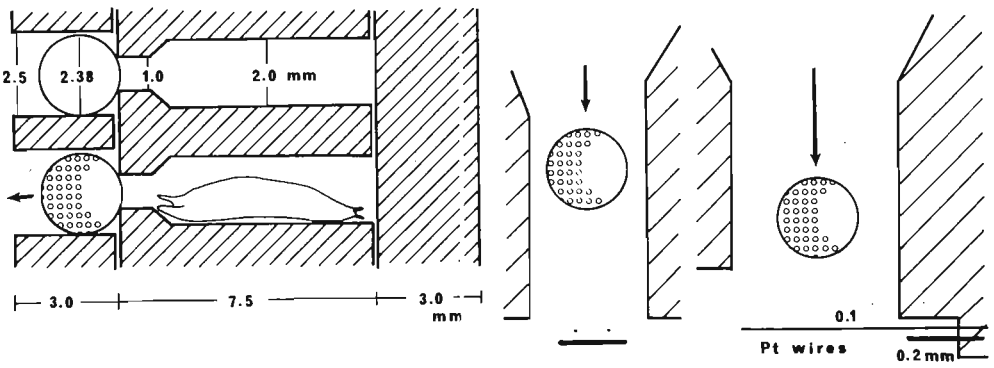
Günther Meyer, of the Max-Planck Institut für Biologie, Abteilung Beerman, is a Horgitt Research Fellow with S.J. Counce and Montrose Moses in the Department of Anatomy at Duke University from July 1, 1974 to January 1, 1975.

Lumme, J. and P. Lankinen. University of Oulu, Finland. An apparatus for recording the eclosion rhythm of Drosophila.



Various devices have been constructed for registering the eclosion rhythm of Drosophila (e.g. Grant et al., 1970; Chandrashekaran et al., 1973). Truman (1972) has devised a recording apparatus for moths, which does not have any mechanically moving parts. Therefore we considered this design most useful for recording the eclosion rhythm of flies because of its simple structure and function. As a further advantage of this design can be mentioned that it creates minimum disturbance to the pupae. We have modified this apparatus for flies of the D. virilis group. The structure and dimensions of the device made of acryl plastic are illustrated in the figures. The pupae are inserted into the holes of the pupal plate, and the openings are closed with balls of stainless steel. When emerging, an adult fly pushes forward the ball weighing 50 mg and drops it down. The falling ball is trapped by a funnel, where it makes a contact in the microswitch of the event recorder. The channel, through which the balls fall, is so wide that adult flies can not block it. The microswitch is insensitive for flies weighing about 4 mg.

The device can easily be constructed for flies which are smaller than those we have studied. Illumination and temperature can be controlled as desired. The same chamber can be used for recording the flight activity of newly emerged adult flies by e.g. the method devised by Nederström and Lumme (1972). For some purpose it is desirable to collect flies emerged within known intervals of time, but this seems



to be difficult with our apparatus.

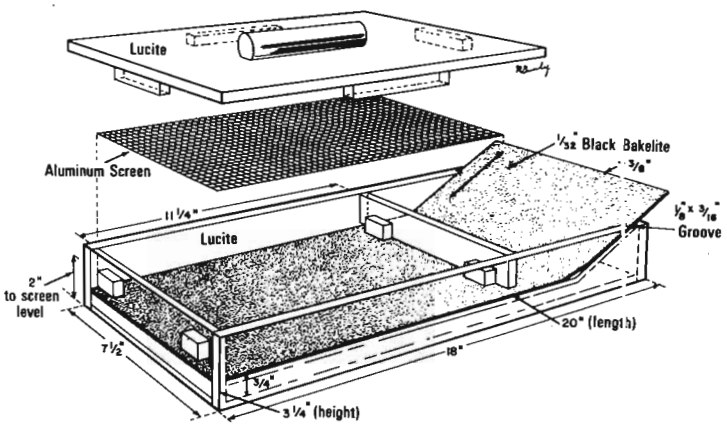
References: Chandrashekaran, M.K., A. Johnsson and W. Engelman 1973, J. comp. Physiol. 82:347-356; Grant, B.S., G. Bean and W.L. Harrison 1970, DIS 45:185-186; Nederström, A and J. Lumme 1972, DIS 48:158; Truman, J.W. 1972, Z. vergl. Physiologie 76:32-40.

Gassparian, S. University of Isfahan, Isfahan, Iran. A simpler method for the separation of larvae of D. melanogaster.

A new simpler technique for collecting larvae of D. melanogaster was developed. After the adult flies are separated from the container, another glass container wetted with distilled water, is superimposed upside down upon the original container which held the remaining larvae and pupae. After 10 hours, about 50 or more larvae go into the new container, the number depending upon the type of medium. Although the time needed for this procedure is short, because of the migration time required, it is better to separate the larvae the evening previous to the next day's experiments.

Nash, W.G., T.B. Friedman and C.R. Merril. National Institute of Mental Health, Bethesda, Maryland. An improved ovitron: A means of collecting large quantities of timed embryos.

Large numbers of synchronous Drosophila eggs or larvae are useful for biochemical and developmental studies. Several devices for collection of the eggs have been reported (1,2,3,4,5). We have extensively modified the design of the ovitron of Yoon and Fox (1) to facilitate the collection of large quantities of embryos. Our device consists of a plexiglass box (see Figure) which is filled with Drosophila Ringer (25°C) to a level approximately 1/2" above the aluminum screen (16 mesh). Females are placed on the surface of the Ringer in the 7-1/2" x 11-1/4" area and the lid is applied. Eggs can be collected at 30 to 60 minute intervals for 12 to 18 hours. Just prior to a collection, the sides of the ovitron are gently tapped to free those eggs caught on the screen, allowing them to fall onto the black Bakelite slide which is then slowly removed. The eggs are collected with a moistened camel's hair brush which is gently stroked across the Bakelite slide. The aluminum screen permits most of the eggs to pass while retaining drowned adults. This procedure allows multiple collection of large quantities of timed embryos. Eggs obtained after the first few hours of operation of the ovitron are permeable to macromolecules (1).



Newly emerged females were fed on a diet enriched with honey and yeast for six to eight days to maximize egg laying (2.5 eggs/female/hour). The non-anaesthetized, well fed females were collected, knocked to the bottom of a half-pint milk jar and sprinkled onto the surface of the Ringer solution. Best results were obtained with a maximum of 30 to 50 females per square inch (i.e., 2300 to 3700 flies per ovitron). Greater than 50 females per square inch resulted in massive drowning. At best, we obtain a yield of approximately 9000 eggs per hour per ovitron.

References: 1) Yoon, S. and A.S. Fox 1965, Nature 206, No. 4987:910; 2) Delcour, J. 1969, DIS 44:133; 3) Maroni, G.P. 1972, DIS 48:158; 4) Majumdar, S.K. and D.S. Novy 1972, DIS 47:150; 5) Würgler, F.E., H. Ulrich and H.W. Spring 1968, Experientia 24:1082.

Breugel, F.M.A. van. University of Leiden, The Netherlands. A simple method of injecting larvae of Drosophila avoiding ether treatment.

In standard injection procedures very often an ether treatment is involved. Some larvae become over-etherized and die and the awakening of other larvae covers a rather long uncontrolled time span that might be of disadvantage in some experiments. It is possible to omit ether anaesthesia by using a cold treatment. Alcohol cleaned and dried larvae can be placed in large groups (e.g. 100 individuals) on a glass slide that is mounted on a block of ice out of a normal refrigerator. The thin layer of melting water keeps the slide at about 0°C and the larvae become quiet after a few seconds. The relaxed larvae can be punctured dry or submerged in a drop of insect salt solution with common Drosophila injection tools. An experienced investigator might handle over 300 larvae within one hour. The awakening is quick and the survival perfect. Injection fluid, needles, etc. logically should be as sterile as possible, but the use of antibiotics is not necessary.

Moree, R., C.Z. Pakonen and N.E. Balkin. Washington State University, Pullman, Washington. Attempts to improve triple balancer strains.

In attempting to improve the chromosomal integrity of triple balancers we have tried the following combinations: 1) Basc/Basc;SM1/Pm;P88/TM1
2) Ext/FM6;SM1/Pm;P88/Sb
3) Ext/FM6;SM1/Pm;P88/TM2
4) FM7a/FM7a;SM1/Tg;TM6/Sb

P88 designates Ins(3LR)P88,In(3R)C,D^3 bx^{34e} e, an intermediate stage in the development of an improved third chromosome balancer by E.B. Lewis. TM6 designates E.B. Lewis's original TM6, to which he has added the excellent marker Ubx^{P15}. In combination 1) Basc, not unexpectedly, broke down too frequently to be useful. Combination 2) had extremely low productivity and some breakdown of P88, and so was discarded. Combination 3) was lost because of extremely low fecundity and viability; many pupae in the final crosses and in the stock failed to emerge. Combination 4), though somewhat better, was lost after several generations because of generally poor productivity. The schedules we used are available for anyone who would like to have them. Special thanks are due to Prof. E.B. Lewis for providing us with the intermediate stage and the improved form of TM6. (Aided by funds from the State of Washington Initiative Measure No. 171 for the Support of Biological and Medical Research.)

Selker, H.P.* and J.H. Postlethwait. University of Oregon, Eugene. Electrophoretic separation and detection of non-enzymatic proteins from single flies.

Although enzymes can be readily separated and detected in homogenates of single flies by standard electrophoretic techniques (e.g. Dickinson, 1971), detection of proteins from single flies using standard disc gel electrophoresis has not been feasible. So to detect non-enzymatic proteins, gels have been made in capillaries (Grossbach, 1965; Ward et al., 1970). These methods have required the use of a pair of expensive micromanipulators to form the stacking gel (Grossbach, 1965), or have involved a specially made apparatus to remove the gel from the tube (Ward et al., 1970). We have developed a simple procedure for capillary disc gel electrophoresis that avoids these problems and provides protein bands from a single ovary from a single fly.

Twenty-microliter Wiretrol^r (Drummond Scientific Co.) capillaries are filled completely with the desired resolving gel (such as used by Jovin et al., 1964) and are allowed to polymerize. Since de-gassing seems to help prevent bubbles from occurring in the gels, the gel solution without catalyst solution is aspirated five to ten minutes in a 25-milliliter filter flask. The catalyst solution is then added and the solution is allowed to aspirate a few more minutes. The gel solution is then introduced into the tube by capillary action.

After the resolving gel has polymerized, the gel is pushed from the bottom of the capillary by the Wiretrol^r plunger until a small length (ca. 3mm) of gel protrudes from the top. The protruding end is then cut off flat with a razor blade, and then pushed back into the tube by air pressure supplied by a 100 cc syringe fitted with an 18 gauge needle and connected to the top of the capillary by a length of polyethylene tubing (i.d. 0.047 in Intramedic^r by Clay Adams). If the gel is pushed back down 5 mm, this allows about three microliters total for the stacking gel and sample.

The stacking gel is applied immediately with a small pipette made by drawing out a 100-microliter Microcap^r (Drummond) until it is about 100 μ O.D.). In our experience, a third to a half of the remaining space should be filled with stacking gel (one to one and one half microliters). After the stacking gel has polymerized, the sample and tracking dye can be applied using a micropipette.

The gels can then be run on a conventional disc electrophoresis apparatus (e.g. Canalco Model 1200), the only adaptation needed being to use the gum rubber plugs from the bulb dispenser that comes in a Microcap^r package as an insert within the usual grommets in order to accomodate the small diameter of the capillaries.

The gels are run at 0.2 ma per gel and are removed from the tube by use of the Wiretrol plunger. They can then be stained with Coomassie Blue stain in 5:5:1 methanol:water:acetic acid, and destained in the same solution without the dye.

This method seems to work well with Acrylamide gels of about 4-5% with SDS, but successful runs have been done with 7.5% gels and without SDS. Higher percentage gels may form bubbles and adhere more strongly to the capillary wall, as do gels that have been allowed to stand for more than 3 hours. Both these problems are lessened by treating the capillaries

with photoflow and chilling them before putting in the resolving gel.

This research was supported by grant GM 19307 from NIH.

References: Dickinson, W.J. 1971, Develop. Biol. 26:77-88; Grossbach, U. 1965, Biochem. Biophys. Acta 107:180-182; Jovin, T., A. Chrambach and M.A. Naughton 1964, Anal. Biochem. 9: 351-369; Ward, S., D.O. Wilson and J.J. Gilliam 1970, Anal. Biochem. 38:90-97.

* Present address: Department of Biology, Reed College, Portland, Oregon 97202.

Guest, W.C. and T.C. Hsu. University of Arkansas, Fayetteville, and University of Texas M.D. Anderson Hospital, Houston.

A new technique for preparing Drosophila neuroblast chromosomes.

The value of D. melanogaster larval neuroblast cells for studying somatic metaphase chromosomes has long been recognized and a variety of squash techniques have been developed. Hsu (1971) used brain squashes to obtain metaphases for demonstrating the similarity of Drosophila heterochromatin to the heterochromatin in mammalian chromosomes. However, it is frequently difficult to obtain numerous division figures with well spread chromosomes using conventional squash techniques.

We have recently applied some of the Giemsa banding procedures widely used in studying mammalian chromosomes to Drosophila using a modification of the technique developed by Stock, Burnham and Hsu (1972) for obtaining well spread metaphase figures from solid tissues. This procedure, in addition to giving large numbers of metaphase and late prophase figures with well spread chromosomes, permits the slides produced to be handled as air dried preparations.

The procedure is as follows:

1. Twenty-five to thirty larval brains are dissected out in physiological saline or insect Ringer's solution. The brains should be washed several times in saline to remove debris. The physiological saline is replaced with a hypotonic solution. Both 1% sodium citrate and physiological saline diluted with equal parts of distilled water were used with ten to fifteen minutes giving good results. The hypotonic saline is removed with 1 ml tuberculin syringe or with a microcapillary pipette.

2. The ganglia are fixed in methanol acetic acid (3:1) with the fixative being changed several times to insure complete removal of water. The fixed material may be used immediately or may be stored in the fixative. Fixed material stored in absolute alcohol is much more difficult to work with than ganglia stored in the fixative.

3. Slides which have been cleaned thoroughly are warmed to 40-45^{o}C on a hot plate or slide warmer.

4. The fixed ganglia are transferred to a well slide. We used a standard depression slide with a cylindrical well 3 mm deep. Remove as much of the fixative as possible with a syringe or microcapillary pipette. It is helpful to perform these transfers under the low magnification of a stereoscopic microscope. Add approximately 0.2 ml of 60% acetic acid to the well. Under the dissecting microscope the tissue will begin to swell and become translucent. Using a Drummond micropipette (see Stock, et al. for description) agitate the material by drawing the ganglia into the pipette several times. As this is done the ganglia will begin to disintegrate. This is a critical step. If the material remains in the strong acetic acid too long the cells will be distorted or destroyed. Best results have been obtained by adding the acetic acid, agitating briefly, then applying the material to warmed slides while some of the brains are still intact but swollen.

5. The cell suspension and intact tissues are drawn into the Drummond pipette and a small drop placed on the heated slide. Immediately the drop is drawn back into the pipette leaving a thin circular film on the slide containing a monolayer of cells. This process is repeated many times over the entire slide very quickly while the slide is warm. An area that can be covered by a 22 x 40 or 22 x 50 mm cover slip can be spotted on each slide. It is important that the slide be kept warm to accelerate evaporation of the acetic acid.

6. The slides are dried thoroughly before using. While we have used slides made in this manner for banding chromosomes or for staining heterochromatin, the slides can be used in a number of techniques that require a monolayer of air dried cells.

References: Hsu, T.C. 1971, J. Heredity 62:285-287; Stock, A.D., D.B. Burnham and T.C. Hsu 1972, Cytogenetics 11:534-539.

Félix, R. National Institute of Nuclear Energy, Mexico City, Mexico. A motorized waterflow devide designed for continuous hard work and rapid washing of countless vials and culture containers.

The washing of numerous culture containers consumes valuable time in Drosophila laboratories, thereby making the installation of automatic washing devices desirable and economical. The motor driven apparatus assembled for our laboratory consists of a rotating brush with water outlets for washing and rinsing the material. During two years of hard work it has expedited the washing of half-pint bottles and vials. The installation and design of the device are illustrated by the photo and diagram. In several hours the automatic washer thoroughly cleans hundreds of bottles and vials, an accomplishment that cannot be equalled by manual labor.

An apparatus based on the same principle is described in DIS 45:180-181 (1970). Its constant hard use has prompted several modifications for improving its durability and resistance. The modifications are described in the following. This system functions effectively for 6 hours a day, requiring only a replacement of the washer at the motor base every 6-8 months. The entire assembly is driven by an a.c. motor (1/10 HP, 110-220V, 0.09 KW, and with 1,340 r.p.m. Mez Mohelnke, Chez). The rotation of the motor shaft (fm) is transmitted to the washing shaft (fd), which turns inside the water chamber (c). This shaft has four perforations which carry pressurized hot water from the water chamber to four openings (s) at the base of the shaft. In this way the water flows directly into the vials or through the hollow cleaning additive (at the right of the figure) into the half-pint milk bottles. The water temperature is regulated by mixing hot and cold water which enters the water chamber through water inlets connected by a rubber tubing.

At the base of the motor a hard rubber washer (r) enveloped by steel is tightly fitted to the motor shaft to prevent water leakage. The washing shaft rotates upon two inoxydizable steel ball bearings (b). The apparatus is screwed to the motor, the lower aluminum plate (pa) being attached to the upper steel plate (pc) with four screws.

An acrylic plastic container (p), available commercially for refrigeration storage, protects

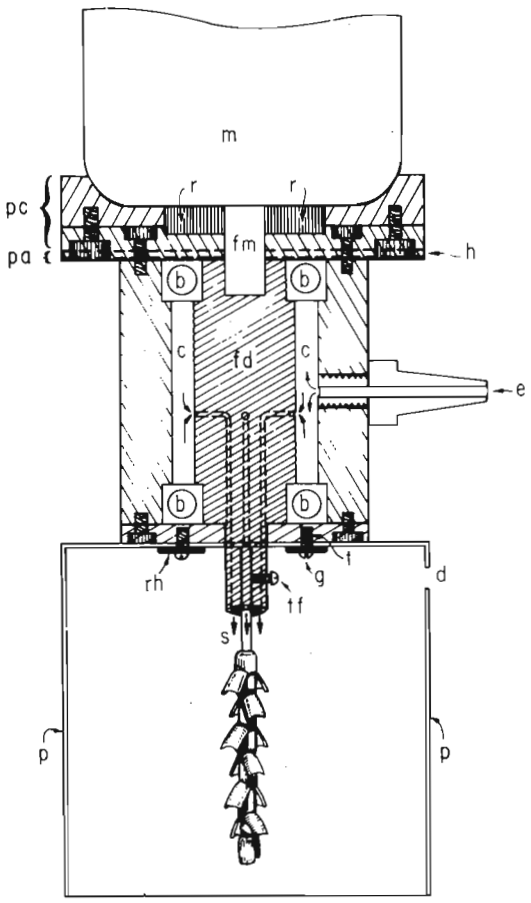
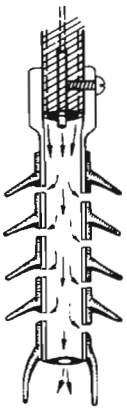
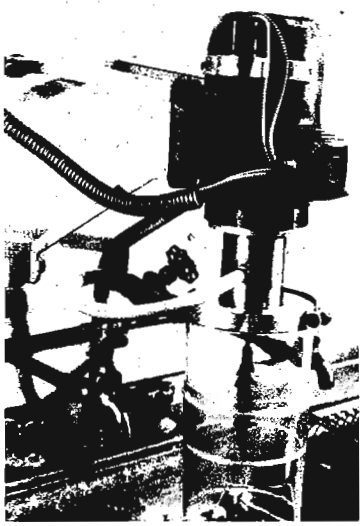


Diagram of the motorized water flow device.





View of the water flow device with the shielding of acrylic plastic.

the operator from being splashed. The container is attached to a circular aluminum plate at the base of the apparatus with screws (g). The washing shaft passes through a circular hole located in the bottom of the plastic container. The cleaning additive for vials, made with strips of latex or rubber tubing, as illustrated, is attached to the distal end of the washing shaft with a screw (tf). Through an orifice (d) a screw-driver is inserted to adjust the cleaning additives. The centrifugal force of the washing shaft lifts up the lateral strips thoroughly cleaning the vials. The interchangeable hollow washing shaft with strips of tubing cut out to the appropriate length is used when half-pint bottles are washed. Water flows from the distal end, as well as through the lateral holes of the hollow shaft, thereby expelling food medium and wastes from the bottles.

The whole waterflow device is mounted on the wall above the sink; the water containing expelled food medium is immediately disposed of in a cesspool. Rubber gloves may be used to protect the operator's hands; however, the few defective vials that break during washing rarely inflict injury because of the softness of the rotating latex strips and the protection afforded by the acrylic plastic container.

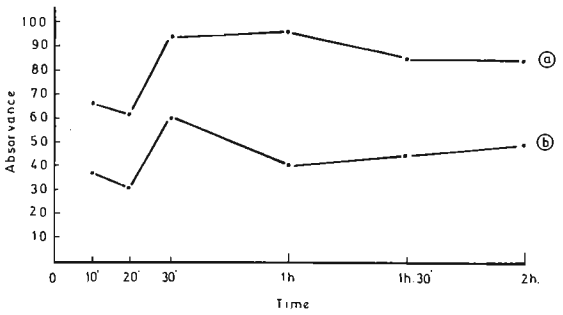
This apparatus was designed and constructed at the Nuclear Center General Workshop of the National Institute of Nuclear Energy, thanks to the assistance of Ing. Luis de la Torre.

Kirschbaum, W.F. Comisión Nacional de Energía Atómica, Buenos Aires, Argentina.

Feulgen stain with hydrolysis at room temperature of Drosophila melanogaster salivary gland chromosomes.

Considering that better results have been obtained with the Feulgen stain using room temperature during hydrolysis with 5N HCl, than with the traditional one of 1N HCl at 60°C (Deitch, A.D. et al., Conditions influencing the intensity of the Feulgen reaction, J. Histochem. Cytochem. 16:371-379, 1968), this new technique has been tried with Drosophila melanogaster salivary gland chromosomes.

With this technique, better images of polytene chromosomes have been obtained specially because no shrinkage of the cells material is produced. This is caused by the high temperature treatment during hydrolysis, with the conventional system.



As the shape of the 5N HCl hydrolysis curve is dependent on the fixative and preparative procedure used and it varies from the material stained, the curve of room temperature hydrolysis with 5N HCl for Feulgen staining has been obtained for Drosophila melanogaster salivary gland chromosomes. The different values were obtained by microphotometric measurements, using the following equipment consisting of a: Zeiss Photomicroscope I, Zeiss Photometerhead, Zeiss case with R.C.A. Photomultiplier model 1P28 and a Zeiss monochromator m4GII.

The data were obtained under the following conditions of the microphotometer:

1. Optical conditions of the microscope - Objective 100x immersion 1.3 ap. Planapo. Field diaphragm. 0.1 mm diameter opening. Optovar 2x magnification. As condenser a 10x magnification objective with 0.22 aperature.

2. Photohead - eyepiece of 10x magnification; measuring diaphragm 2 mm diameter. A total magnification of 2000 was used with a band width of 180 Å.

3. Monochromator - Slit with 1 mm opening; monochromated light of 560 mμ.

For comparative reasons one strong band B_2 was measured, and a soft B 12 one of the X-chromosome. One can see that they have the same optimum time of hydrolysis and follow almost parallel lines. The soft band logically loses staining intensity before the strong one (see Figure).

Method used for staining the chromosomes: 1) Squash salivary gland chromosomes in lacto acetic (1:1) solution; 2) Separate cover slip with liquid N; 3) Alcohol 96%, formol 40% solution (9:1) 10'; 4) Hydrolysis in 5N HCl room temperature; 5) Wash in distilled water; 6) Shiff reactive during 2 hours; 7) Wash in distilled water; 8) 3 passages in sulphurous water 2' each; 9) Wash in distilled water; 10) Alcohol 96%; 11) Alcohol 100%; 12) Mount in Euparal.

Merriam, J.R. University of California, Los Angeles. On setting up a lab and kitchen.

Several people have asked me what equipment and suppliers I use in my lab and kitchen. They particularly want to know about low cost substitutes or new products I have found in setting up. I thought it might be generally useful to write down the sources of equipment and supplies we use (listed in the tables below) and recount some of our experiences with this material. The costs listed for equipment are approximate only and from four years ago, so please be warned. It is to be hoped that labs with other ways of doing the jobs will be encouraged to write DIS and give us all the benefits of their procedures.

---- Lightnin Portable Mixer. Very useful, the shaft needs to be extended 8" (Σ=27") in order to mix small batches or when pumping gets down to the last bit of media.

---- National Filamatic Pipeter. No service by company on west coast, but we have only had to replace the front electrical panel in 4 years. The chief advantage of this machine is that the FKS and FUS cylinders are stainless steel and won't break. The machine will operate both cylinders simultaneously for 2 different quantities if desired.

---- Sherer Reach In Incubator. This box may not be suitable for precisely constant temperatures since it operates by alternating blowing hot and cold air. We use ours for keeping stocks and media at 18° and for this it is the best buy for the money. It comes with 4 shelves but we needed 3 more. We built special long narrow trays to fully use all of the shelf space. It holds 48 cubic ft.

---- Dissecting microscopes and lights. Although we use Wild M5 scopes, I think the new Wolfe or similar scope from Japan ($260 from Carolina Biological Supply with built in light source) would be adequate. The Bausch and Lomb lights listed are fluorescent and are the cheapest acceptable lights we found.

---- Disposable plastic bottles. This is perhaps our major innovation. We originally bought these bottles because they are cheap but they are also much lighter than glass and take up less space. They are autoclavable. We have washed and reused our supply constantly for 4 years without problem. They are semi-transparent in that one can see larval tracks and pupae through the bottle walls but not adult flies. We plug the bottles with dispo foam plugs which we recover after autoclaving and wash in a regular clothes washer. Since we bought our supply of bottles three different companies have sold them. Pacific Plastic Bottle Co. (as of Sept. 17, 1973); Van Waters and Rogers may be interested in supplying them also, which would be easier for small class uses and high schools.

---- Baskets and trays. We made all our baskets and trays in the department shop out of metal sheeting or wire fencing. They are considerably cheaper and far better than any commercial varieties we have yet seen. The wire baskets hold 54 plastic bottles; the trays hold 168 vials.

---- Media. We use the Cal Tech formula for which the ingredients are listed in Supplies. The absorbent cotton balls are used to plug vials. Minor items, such as food coloring and sucrose, we obtain from the food market. Propionic acid by Mallingkrodt. As described in another note in this DIS, we add caffeine to the food for mite control. However, this is not a substitute for throwing out old bottles. Mike Ashburner is reported to have a more powerful technique for getting rid of mites which we also plan to try.

Table 1. Equipment

Quantity	Item Description	Address	Approximate Cost
1	Lightnin Portable Mixer	Mixing Equipment Co., Inc. Rochester, New York 14603	$1,500
1	Groen Steam Kettle Model #FT-20	Dover Corp., Groen Division Elk Grove Village, Illinois	
1	National Filamatic Pipeter Model #DAB-5	National Instrument Co., Ind. 4119-27 Fordleigh Road Baltimore, Maryland 21215	$850
1 each cylinder	#FKS-130-75 for 60 ml bottles #FUS-60 for 7 ml vials		

(Continued next page)

Table 1 Continued

Quantity	Item Description	Address	Approximate Cost
1	Sherer Reach-In Incubator Model R1-48 (2^{o} - 42^{o}C)	Sherer-Gillette Company Environmental Division Marshall, Michigan 49068	$1,500
	Microscope Lamps Cat.#31-35-32	Bausch and Lomb	$45
6800	1/2 pint milk bottles (round)	Pacific Plastic Bottle Co. 305 E. Home Street Rialto, California 92376 (phone: (714) 875-4661)	$18.35/1,000
50+ from 100' sheet	Wire Bottle Baskets Dimensions: 14 3/8" x 17 3/1" x 5 1/8" Wire # 16 Width 24" Mesh 1/2" x 1"	Any hardware supplier	$38.60/100' roll
30	Vial Trays Dimensions: 10 1/2" x 14" x 3 1/8"; 3 perforated carbon steel sheets (36" x 120") with 1/2" holes, 20 gage, .035" thick, staggered pattern.	Ducommun Metals 4890 S. Alameda Street Los Angeles, California (phone: (213) 588-0161)	$25/sheet
		Plated with Cadmium Irridite by Barry Ave. Plating 2210 Barry Avenue West Los Angeles, California (phone: (213) 478-0078	$30/30 trays
200	Shelf Trays Dimensions: 10 1/2" x 14" x 3 1/8" 20 Galvinized steel sheets (36" x 120") @ 26 gage		$5.00/sheet

Table 2. Supplies

Amount Ordered	Item	Supplier	Cost/Unit
50-100 lbs. (100 #/6 mos.)	Agar (fine ground) powder form A	Mcorehead & Co., 14801 Oxnard Van Nuys, Ca. (213) 873-6640	$71.00/25 lb.
400-500 lbs./ 9-10 mos.	Corn Meal (yellow)	The Pennington Company, Los Argeles, Ca. (213) 938-2941	$3.30/25 lb.
200 lbs./year	Anhydrous Dextrose #2401	Corn Products - Chemstat 720 Centinela Ave. Inglewood, Cal. (213)678-3468	$25.00/100 lb.
10-20 boxes (10/4-5 mos.)	Absorbent Cotton Balls #8329 (large) 4,000/box	Johnson & Johnson, 4100 Bayshore Blvd., Menlo Park, Ca. (415) 329-0400	$10.00/box
10-20 pkgs. (6 mos.)	Dispo Plugs T 1385 (28 x 35 mm)	Scientific Products, 1711 Redhill ave., Santa Ana, Ca. 92705 (phone (213) 860-5551)	$5.95/pkg.
100 lbs. (100 lbs./year	Yeast, debittered dry	Philadelphia Dry Yeast Co. Philadelphia, Pennsylvania	$0.28/lb.

Félix, R. National Institute of Nuclear Energy, Mexico City, Mexico. Multi-purpose medium for Drosophila cultures used in teaching or research.

The following procedure for Drosophila cultures has been employed with good results in our research laboratory. Its use is also recommended for small laboratories in schools and universities which use the fruit fly to compliment the theory in genetic and radiobiology courses.

1. Sterilize the polyurethane stoppers of the half-pint milk bottles, used for mass cultures, or those for the vials (see DIS 45:180, 1970) in an autoclave at 120°C and 20 lbs. pressure for 30 minutes.
2. Sterilize the flasks and vials in an oven at 70-80°C for 3 hours.
3. Place the following mixture on a gas stove boiling it for 20 minutes:

Distilled water	4,000 ml
Agar	40 gr
Cornmeal	250 gr
Sucrose	140 gr
Dextrose	100 gr

4. Remove the mixture from the heat and immediately add the mixture of brewer's yeast.
5. Prepare in advance the following mixture of yeast and water, stirring it for 2 minutes with an electric blender or a mechanical agitator (see DIS 45:178, 1970).

Brewer's yeast	120 gr
Distilled water	800 ml

6. After the above mixture is added to the medium, mix it for 3 minutes with the mechanical agitator.
7. Heat it once again; letting it boil for 20 minutes.
8. Remove it from the heat and cool, or chill it in a waterbath placed in the sink, stirring the prepared medium with a large spoon.
9. After the mixture has chilled in the waterbath or by exposure to room temperature, stir it again with the mechanical agitator. When the temperature drops below 60°C, add the two following compounds:

Propionic acid	20 ml
"Tegosept M"	20 ml

The two substances should be added in separate containers. The solution of "Tegosept M" (a trade name for methyl-p-hydroxybenzoate) is prepared by dissolving 12.5 gr in ethanol, equalling 100 ml with 96° ethanol. The propionic acid is added without dilution.

10. Upon reaching the above mentioned step, the milk bottles and vials are removed from the stove, so that they will be hot, at the appropriate temperature for emptying the medium.

This all-purpose food formula is recommended for obtaining a medium that neither will shake out, nor liquify in old overcrowded bottles and vial cultures.

Sometimes the stock cultures in our laboratory become contaminated with a reddish-brown bacterial growth that restrains larval development and kills the adults. Pembritin (ampicillin, Beecham) in a concentration of 0.065 mg/ml of food, effectively controls such contamination (see DIS 44:131, 1969).

However predatory mite infestations have been more difficult to control than the already well-known contaminations of molds or bacteria. The mites are introduced into the laboratory by the collected wild flies, or when Drosophila cultures received from other laboratories are not examined carefully before initiating new cultures. This pest is controlled by a treatment of benzly-benzoate (20%) in ethanol (see DIS 47:127, 1971).

Histiostoma sp. has a hypopus stage which attaches itself to Drosophila, as well as to other insects. The absence of long hairs distinguishes it from other less dangerous mites. In addition the predatory mite is characterized by a squatty body build, in contrast to the longer, thinner bodies of non-parasitic mites. Due to the fact that the female adults produce both male and female progeny, the existence of a single mite in the hypopus stage could infect the entire laboratory.

The small newly eclosed nymphs thrive on the culture medium, and in approximately a week metamorphosize into the migratory (hypopus) stage. These extremely active hypopi develop in large numbers in old, infested cultures. Their ability to penetrate tiny crevices facilitates the escape from the culture medium, thereby endangering other cultures, unless tightly stoppered. The migratory nymphs attach themselves to whatever insect they come in contact with, injecting the mouth parts into the insect. After 10 days they leave the host, growing to the adult stage on the surfact of the medium, where they reproduce.

A heavy infestation extending to the stock cultures occurred in our laboratory in 1970.

The mites entered among samples of wild flies collected at several trappings in Mexico City. In order to eliminate the mites, the infested cultures were treated in the following manner: all contaminated bottles and instruments were heated in a furnace before washing. The instruments used to manipulate flies, as well as the microscope surface, the exterior of bottles, and the table surfaces were repeatedly washed with a solution of benzyl-benzoate (20%) in 90° ethanol. Before starting new cultures all flies were examined under the microscope, and apparently mite-free adults selected for later use. As it is difficult to avoid contaminating the new medium, the adults were allowed to lay eggs on it only during a 24 hour period. When several small hypopus nymphs either from contaminated flies or from other cultures, were found in the new medium, its surface had to be covered with a solution of benzyl-benzoate (20%) in 96° ethanol. This treatment kills the nymphs without any apparent toxic effect on the Drosophila larvae, which then develop into the adult stage unimpeded by predatory mites. The newly emerged flies were transferred to new cultures every day in order to avoid the attachment of any mites which survived treatment.

In the heavily infested cultures all of the flies died; and a crowding of mite nymphs was found among the Drosophila larvae. In this case the application of a second treatment was necessary, thoroughly washing the larvae by immersion in a solution of benzyl-benzoate (20%) in ethanol. Following the 2-4 minute immersion in the benzyl-benzoate solution, the larvae were washed with Ringers solution and then transferred to fresh vials. The pest was controlled effectively after three weeks, adhering to the above steps.

Frankham, R. Macquarie University, Sydney, Australia. Instant mashed potato as a fly food.

The instant Drosophila media available commercially are expensive and must be imported if one lives in the antipodes. They are also unlikely to be available locally to people on field-collecting trips. Instant mashed potato added to water (in 1:2 proportions by volume) with a pinch of granulated live yeast on top provides a satisfactory instant media for culturing Drosophila. It is improved if powdered agar (10g to 160g instant mashed potato) and Nipagin (1.27g to the above) are added. Killed yeast may also be added to this powder. We have found differences among brands of instant mashed potato in their suitability for culturing Drosophila. This media has been used successfully by external students carrying out Drosophila experiments at home.

Merriam, J.R. and B. Howard. University of California, Los Angeles. Mite control with caffeine in Drosophila food.

During a recent set of experiments on behavioral responses to drugs we noticed that the flasks with caffeine were less susceptible to periodic mite infestations. We tested this conclusion more directly by comparing innoculations of flies from an old mite infested culture into vials made up with and without caffeine. The vials were placed in a pan containing mineral oil to prevent the mites from spreading. Visable mite appearance was delayed by about two weeks in the vials containing caffeine although there was no change evident in the times of larval, pupal or adult progeny appearance.

We now routinely add caffeine to about 0.02% of the final concentration of food. This comes to 3.4 g per 300 half pint bottle batch (17 l. H_2O). The caffeine is added after the food has cooked (along with the propionic acid mix) and is thoroughly mixed in. A neutral red food dye is used as marker. Anhydrous caffeine is purchased from the Sigma Co. ($10 for 500 gm).

We feel that this procedure has so far helped keep our lab relatively free of mites. However, caution should be exercised in determining the amount of caffeine added by other labs. Whereas a concentration of 0.02% does not visably affect the flies in our hands, we found that a concentration of 0.05% in vials kills about half the adults within 24 hours. It is interesting that 0.05% is probably about the same concentration as the caffeine in a "strong" cup of coffee. Yanders and Seaton (1962) did not find caffeine in these concentrations to be mutagenic in flies and we have no cause to disagree with their conclusion.

Muhs, H.-J. Forstbotanisches Institut, Freiburg, Germany. Good resolution of a modified micromethod of starch gel electrophoresis on slides used in large series with selection lines of Drosophila melanogaster.

Since 1968 isozyme investigations with D. melanogaster brought us many experiences concerning micromethods. The apparatus used (producer: Fa. Boskamp, 5304 Hersel bei Bonn, W. Germany) was originally constructed for immune electrophoresis. During these experiments it was successfully used as a serial method on starch gel for isozyme separations. If the starch gel (10% w/v) was heated for a short time to about 75°C. With help of the buffer system described by Poulik (1957) separation was accomplished within 40 min using a wattage of 200 v (33 v/cm). Wettex sponges instead of paper were used as electrode bridges in order to avoid capillar buffer-streams (for details see Muhs, 1973).

The characteristics of this micromethod consist of high resolution, good reproducibility and great sensitivity. The last feature is not always desired during a serial method. By extensive standardization very good reproducibility can be reached. The high resolution, however, which is demonstrated by the soluble main protein bands and by the isozymes of the leucineaminopeptidase of late pupae is the most essential feature. Among the three well-known soluble main protein bands (Beckman and Johnson, 1964) it was possible to separate the middle one into two distinct bands (III and II2). The faster band also consists of two bands (I1 and I2) but they cannot be recognized in the scanner because of the great width of the slit. The isozymes of the leucineaminopeptidase were named according to Beckman and Johnson (1964). The AF-band is situated between the protein bands III and II2, the AS-band between the protein bands II2 and III. The AF- and AS-bands can easily be recognized without control because the protein bands are coloured red by the Fast Black K salt whilst the isozyme bands are coloured violet. In the C-region five new bands are distinguished. Some of them can be seen in the figure 3 (bands C1 to C4). Probably the band C4 will be a conformational isozyme of DF. In the case DF is lacking, C4 is lacking too. It is easy to differentiate the homozygotes DFDF and DSDS and the heterozygotes DFDS from one another (see also the research note on the frequency of the silent gene LAP D^{o} in this issue). This is even the case after about 20 min of electrophoresis.

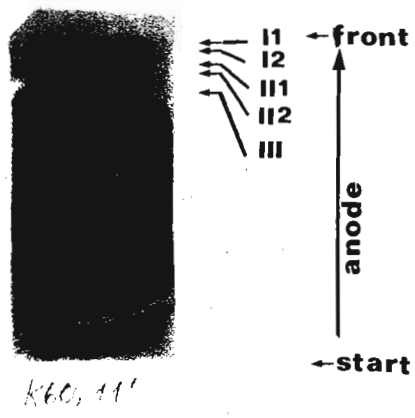


fig.1

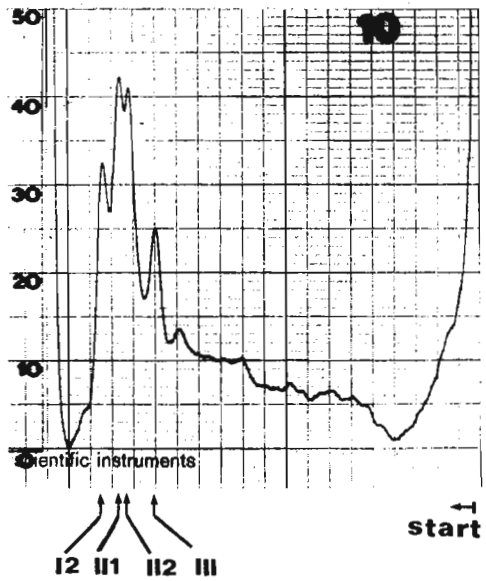


fig.2

Figure 1 shows the soluble main protein bands of three late pupae. According to the decreasing rate of migration the bands are called I1, I2, III, II2 and III.

In Figure 2 one of these samples has been scanned. One can plainly recognize both of the middle bands III and II2.

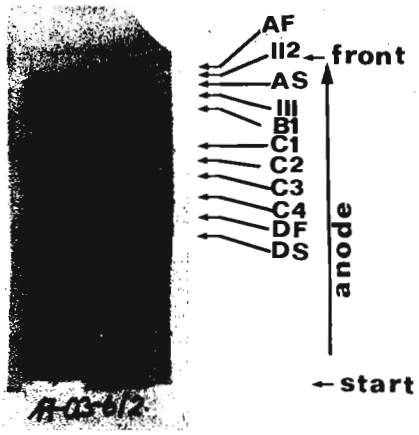


fig.3

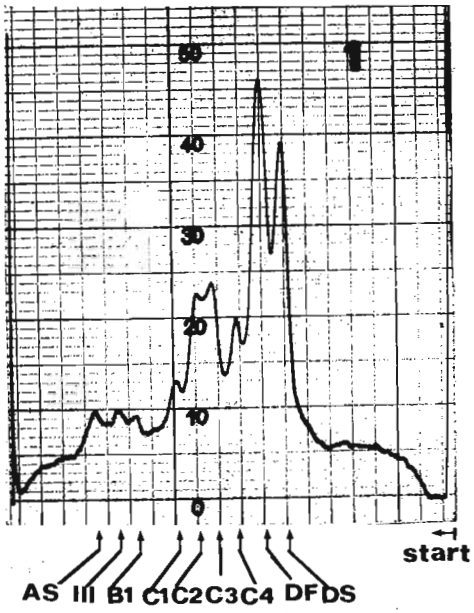


fig.4

Figure 3 shows the typical isozyme patterns of the leucineaminopeptidase (LAP) of 4 late pupae.
In Figure 4 the second sample from the left has been scanned.

References: Beckman, L. and F.M. Johnson 1964, Hereditas 51:212-230; Muhs, H.-J., Frequency of phenotypes (LAP, Aph and Est) seldom observed in laboratory strains of D.m. (in preparation); Poulik, M.D. 1957, Nature 180:1477.

Mulley, J.C. University of Sydney, Australia. Successive enzyme staining on acrylamide gels.

The small body size of Drosophila limits the number of samples with sufficient enzyme concentration that may be applied to gels. This report considers the employment of an agarose overlay for successive staining of a density gradient acrylamide slice. This increases the number of markers detectable in the one individual which is useful in reducing labour and speeding up the analysis of many loci or getting the most information possible from populations of low numbers.

The following method was used for the detection of phosphoglucomutase (PGM) in D. buzzatii. The sample was halved, electrophoresed and the two gels sliced yielding four identical electrophoretograms. Different staining procedures were applied to each slice. One slice was stained for PGM using a mixture of 25 ml tris-HCl (0.2 M, pH 8.5), 25 mg glucose-1-phosphate, 0.25 mg glucose-1,6-diphosphate, 3.75 mg NADP, 5 units glucose-6-phosphate dehydrogenase, 12.5 mg nitroblue tetrazolium, 1 mg PMS and 25 ml 4% agarose in 0.025 M $MgCl_2$ at 60°C. This solution was poured onto the gel in a 14 cm diameter petri dish and incubated at 37°C in darkness.

The concentrations of the components in this mixture are far in excess of the requirements for PGM detection. Rapid band development proceeds in the agarose as it hardens. Little or no stain deposits within the acrylamide avoiding interference to bands of succeeding assays. Within 10 minutes from commencement of incubation samples were typed for PGM, the agrarose removed and the slice rinsed lightly in distilled water. The slice was then restained in the conventional manner for esterase, aldehyde oxidase or any other system. In this way the desired information was available from 4 instead of 5 slices.

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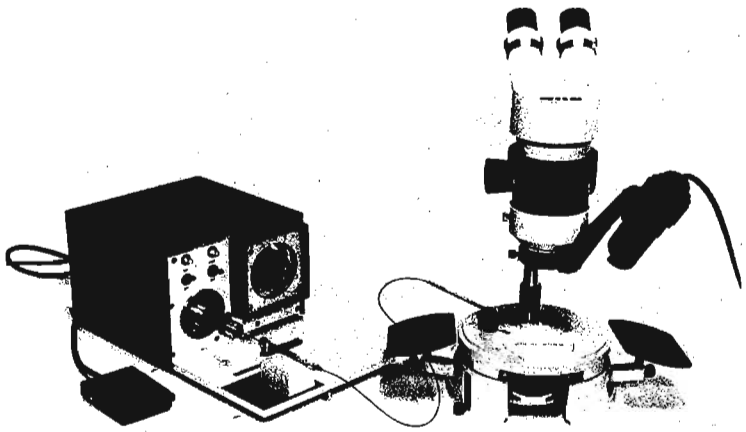
Mollet, P. and F.E. Würgler. Swiss Federal Institute of Technology, Zürich, Switzerland. An apparatus to inject large numbers of Drosophila with constant amounts of fluid within a short time.

The whole microapplication set consists of the following commercially available parts (see illustration from left to right):

1. Microapplicator ISCO Model M (Instrumentation Specialties Company, Lincoln, Nebraska, USA). A foot switch allows one to start a constant speed micrometer syringe drive. The amount of fluid to be injected is determined by a timer which stops the mechanism after a preselected time interval.

2. Microsyringe TERUMO Typ MSN-100 (Jintan Terumo Company Ltd., Tokyo, Japan). With such a 100 µl syringe quantities of 0.1 to 0.6 µl can be injected. For example the application of 0.2 µl requires a timer setting on the microapplicator of about 2.4 sec. In order to obtain high accuracy it is necessary to calibrate the timer for an individual syringe. The range of the application quantities can be extended by using syringes of other volumes.



3. Polyethylene microtube ULRICH PE 100 (Ulrich & Co., CH-9000 St. Gallen, Switzerland). In order to obtain a tight connection between the needle of the microsyringe and the polyethylene tube a metal tube of 1 mm outer diameter is placed on the needle of the microsyringe.

4. Micropipette. Capillary tubing (1 mm Ø) is drawn out by hand or with a vertical pipette puller. The base of the needle must be fire polished.

5. Micropipette holder. Instrument holder of a LEITZ-Micromanipulator (Ernst Leitz GmbH, Wetzlar, Germany). For injection the holder is kept by hand. In the illustration it is fixed for convenience only.

6. Stereo dissecting microscope WILD M 5 or M 7 (Wild Ltd., CH-9435 Heerbrugg, Switzerland).

The injection system (part 2-4) is filled with perliquid paraffin; in order to avoid the formation of bubbles a large syringe containing the paraffin is used to fill the injection system. The test material is taken up into the syringe by reversing the motor of the microapplicator. Depending on the solvent used, a suitable barrier must be used to prevent diffusion of the test material into the paraffin.

The etherised flies are laid on their wings in a row on a slide covered by a double-faced Scotch tape. During injection the abdomen of the fly is held firm with a brush. As soon as the point of the micropipette has been introduced into the abdominal cavity the foot switch is depressed and the fluid is injected automatically. The same microapplication set can be used for injection of larvae and pupae or for topical application.

Acknowledgements: For the illustration we thank Dr. R. Camenzind. The work was supported by Schweizerischer Nationalfonds zur Förderung der wissenschaftlichen Forschung.

Anxolabehere, D. and G. Periquet. University of Paris 7, France.

Experimental and simulated evolutions in teaching population genetics: An approach through computer assisted instruction.

The purpose of this paper is to describe an educational process in which students are able to get a concrete approach to theoretical models of population genetics using both computer simulation and laboratory experiment on Drosophila populations.

Teaching is divided into three steps:

1°) Students analyse the evolution of the allelic frequencies of different genes by studying in cages Drosophila melanogaster populations in various environments. For each generation, allelic frequencies are estimated on flies hatching from egg samples put on a rich nutrient medium to avoid competition. Two cases are generally observed: elimination of one of the alleles or maintenance of the two forms. After 15 generations complementary experiments provide estimations of the selective values for each genotype.

2°) Simultaneously students learn an elementary programing language (OPE 1970) and write programs using classic recurrent formulae in population genetics; application of the Hardy Weinberg law, sex linked genes, two pairs of genes, selfing, sib mating, gene mutation and selection. These simulations allow students to weigh the relative influence of each factor introduced in these mathematical models.

3°) With the selective values observed experimentally each student conducts a simulated experiment based on the elementary constant fitness selective model.

Generally this theoretical evolution does not fit the experimental data well, so students discuss the obtained results criticizing precision of estimated fitness values and validity of the elementary model. New simulated experiments are performed using a linear frequency dependent selective model in which the rare types are at an advantage. The student's program changes the parameters' values to build families of theoretical evolution which are then compared and discussed with the observed evolutions.

The same educational process is carried on for the study of small populations: 1) experimental studies on genetic drift at the laboratory with Drosophila melanogaster; 2) program writing on random numbers and Monte-Carlo simulation model; 3) comparison between observed and expected data and discussion with classic experiments of Wright and Kerr, and Buri.

This constant feed-back between laboratory and computer simulated experiments helps students to avoid a purely speculative manipulation of the mathematical models in population genetics.

COMPUTER ASSISTED INSTRUCTION. The experiment of the Faculty of Science in Paris. Technical report Conference of use of Computer in Higher Education Center for Education research and Innovation. OECD 1970.

Hedgley, E.J. and M.J. Lamb. Birkbeck College, University of London, England.

An alternative to ether.

Although diethyl ether is traditionally used for anaesthetising Drosophila in genetics experiments, it is highly inflammable and subject to possible abuse. It is therefore a serious potential hazard in teaching laboratories. Chloroform is a possible alternative anaesthetic but it has both acute and chronic toxic effects. We have found that methylene chloride (dichloromethane) is a cheap and adequate non-inflammable substitute for ether. Methylene chloride is ten times less toxic than chloroform and, unlike the latter, it appears not to generate chronic ill effects, although of course it is still necessary to ensure that, as with ether or chloroform, the laboratory in which it is used is adequately ventilated.

For anaesthetic purposes one may use methylene chloride in exactly the same way as ether. The length of time that the flies need to be left in the vapor and the time taken for recovery are similar to those for ether. However, the behaviour of the flies while anaesthetised is rather different. Initially the wings of the flies may be held vertically above the body, i.e., they may appear to be "over-etherised", but this effect is temporary. The flies also tend to twitch in a way which may be slightly disconcerting to Drosophila workers who are used to handling etherised flies. Although this twitching might possibly detract from the use of methylene chloride for some experiments, e.g., those involving bristle counting, we have in general found no difficulty in training students to use it for work involving the mutants

commonly used in elementary genetics courses. We have seen no effects on the fecundity or fertility of the flies. An Oregon-R stock which has been anaesthetised with methylene chloride in each generation still shows no apparent differences from our normal stock after 70 generations.