M Brauer

# DROSOPHILA

## Information Service

# 49

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E. NOVITSKI

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# DROSOPHILA INFORMATION SERVICE

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## FROM THE EDITOR

Although it is not completely reflected by our Directory, DIS is found useful to biologists in more than fifty countries, who benefit mutually from this "communication system" among the world's Drosophila laboratories. Financial considerations have never been allowed to dictate the extent of this usefulness, as the benefits of this association are in direct relationship to the number of laboratories and individuals participating.

However, monetary considerations are a fact of life for the Editor, who constantly seeks ways to solve them without raising the price of an issue, which is already uncomfortably high for many subscribers and impossible to meet for individuals whose countries have strict exchange regulations.

Therefore, we are reinstituting an optional page charge for material submitted for publication, and in the future the price of reprints will be increased somewhat to more nearly reflect the actual cost of producing them.

The page charges might also serve as a reminder that DIS is not intended to be a formal publication - notes are usually reports on research in progress and should be kept as concise as possible. Stock lists should be kept up to date, but preferably in such a way as to avoid repetition when possible. To be useful, directories should be kept up to date and include only names important to communication between laboratories.

Back issues still available include DIS 35, 38 - 41 (inclusive) and 43 - 48 (inclusive).

E. Novitski, Editor

## MATERIAL REQUESTED OR AVAILABLE

Approximately 80 of the publications of Dr. M. Demerec (see Bibliography, pp. 349-361 in Advances in Genetics, Vol. 16, 1971) are available to libraries and individuals submitting requests. Also available are the Annual Reports of the Department of Genetics, Cold Spring Harbor, essentially complete from 1933 through 1962 (from volumes 32 through 61 of the Carnegie Institution of Washington YearBook). Mailing will be on a first-call basis; sets containing fewer reprints will be mailed as supplies of various articles and YearBooks dwindle. Send requests before Dec. 31, 1973, to: Dr. Philip E. Hartman, Department of Biology, The Johns Hopkins University, Baltimore, Maryland 21218, U.S.A.

The Drosophila Supply Company, formerly of Garden City, N.Y., is now located at 2412 W. Rollins Rd., Columbia, Missouri 65201 (phone 314-445-7856). We supply the following: 38 Drosophila mutants, two varieties of dry media mix, half pint cream bottles containing media, Burco polyethylene Drosophila anesthetizers, Drosophila kits suitable for classroom experiments. Our brochure listing stocks, details of materials, and prices may be obtained from us at the new address listed here.

J.L. Mensua, Autonomous University of Barcelona, Department of Basic Biology, Barcelona (Bellaterra), Spain, would be very grateful to obtain reprints on current work on Drosophila to supplement the library of this new Department.

Tester stocks of the Y chromosome fertility factors of D. melanogaster which were synthesized by George E. Brosseau, Jr., will be maintained until August 1973. Anyone wishing to obtain these stocks should write to Anne Frankel, Department of Zoology, University of Iowa, Iowa City Iowa 52240.

Request for information from Michael Ashburner, Department of Genetics, University of Cambridge Milton Road, Cambridge CB4 1XH, England. Francoise Lemeunier, of Gif-sur-Yvette, and I are making a cytotaxonomic study of the melanogaster species subgroup of the melanogaster species group. At present this subgroup comprises D. melanogaster, D. simulans, D. yakuba and D. teissieri. I feel certain that many Drosophila workers have on their files unpublished informating concerning the occurrence and distribution of inversions in natural populations of the cosmopolitan species melanogaster and simulans. Since we would like to build up a picture of the geographic distribution of these inversions, I would be very grateful if colleagues could search their files and send me any data they may have. Supplementary information concerning the populations from which the inversions were taken, and with respect to the frequencies of various inversions, would be of great help. Negative data (i.e. monomorphic standard populations) is, of course, also welcome. Full acknowledgement of any data used will be made on publication.

Mrs. H.J. Muller informs us that she still has reprints of some of Dr. Muller's publications which are available upon request. Her address is: Mrs. H.J. Muller, 916 South Mitchell, Bloomington, Indiana 47401.

## ANNOUNCEMENTS I

We deeply appreciate the responses made to our invitation to subscribers to become donors of issues of DIS and reprints to individuals and laboratories in parts of the world where the monetary exchange rules make it impossible to pay (most live in countries not represented in our Directory listing). More donors are needed, and are notified at once who their beneficiaries will be. Material is sent to a recipient with a note offering the compliments of the donor. The price of subscriptions and reprints is the same to foreign countries as to the USA.

Invertebrate Types: Drosophila by Bryan Shorrocks, Department of Zoology, University of Leeds England, was published in 1972 by Ginn & Company Limited, London. It provides a brief yet wide-ranging account of the biology of the fruit fly, somewhat biased towards ecology and genetics, with a emphasis on European information. It is illustrated in color and in black and white with photographs and line drawings and contains a key to the more common British species.

43403

## BOWLING GREEN, OHIO: BOWLING GREEN STATE UNIVERSITY
Department of Biology, Genetics Research Group
Mid-America Drosophila Stock Center

"Development of the system of nomenclature designating genetic variations of Drosophila melanogaster has been rather haphazard; consequently, the system is not a logical structure but is replete with relics, redundancies, and inconsistencies. Revision into a consistent scheme is not practicable, etc., etc...." From Lindsley and Grell "Genetic Variations of Drosophila melanogaster" (1967).

While many of our genetic "fathers" and at least some of their descendants would disagree with the sweeping generality of the above-quoted condemnation*, attempts to lessen the impact of an occasional real and/or imagined inconsistency have indicated that as with any continuously evolving system (no matter how elegant or inelegant it may be) some problems may be found. One of these can be traced back to the fact that several of the stocks currently maintained in the Center at Bowling Green represent a collection deposited here by the late H.J. Muller, J. Schultz, and others. Many of these strains, while named essentially according to the conventions established by Bridges and Brehme in their original publication, "The Mutants of Drosophila melanogaster," contain a mnemonic designation which can often serve a more useful function in conveying (in shorthand fashion) the origin of the variant (which is particularly important in the case of chromosomal aberrations), than a relatively non-specific letter or simple numerical designation [for instance, compare $sc^8$.Y with $y^+$Y, InEN with C(1) RM]. On the other hand, numerical designations also have their uses. While recognizing the need for some standardization, I would like to suggest that some nomenclatural flexibility should remain lest we end up with even more intricate inconsistencies (which could ultimately lead to the "loss" of a particular stock in the sense that its origin becomes obfuscated). This may have already happened on some rare (hopefully) occasions.

However, since Lindsley's and Grell's "Genetic Variations of Drosophila melanogaster" now represents the "Bible" for a whole new generation of Drosophilists, the Subcommittee on Insect Stocks and Dr. Herman W. Lewis, Head of the Cellular Biology Section and Program Director for Genetic Biology (National Science Foundation), have recommended that it would improve the potential usefulness of the stocks in Bowling Green for our younger colleagues if some of the more commonly-used synonyms given in Lindsley and Grell could be incorporated into our list. With this aim in mind, the current list was compiled. An alphabetical cross-referencing by variant (in order to aid in selecting a particular mutant which may be carried in a multiply-marked stock) as well as a fairly complete cross-indexing of all variants according to the format in Lindsley and Grell, has been prepared. Future copies of our list will include additional cross-references which may have been inadvertently omitted in this initial attempt.

If only to balance the prodigious effort which this has entailed, we hope that it will not have been in vain; suggestions as to any further means of improving the services provided by the Center in Bowling Green and additions to the indices will be welcomed by the undersigned.

Irwin I. Oster

* in part in view of the fact that the nomenclatural modus operandi developed in the early days of Drosophila genetics by Bridges, Morgan, Sturtevant, and Muller (and countless others) has served as a model for devising the nomenclature for most other organisms.

### Wild Stocks

a1 + Amherst 3 (homoz. Singh, 1939)
a2 + Amherst 3 (iso, 1969)
a3 + Canton-S
a4 + Canton-S, A (iso, 1952)
a5 + Canton-S, B (iso, 1970)
a6 + Crimea
a7 + Crimea (iso, 1969)
a8 + Crimea (iso, 1971)
a9 + Florida-9
a10 + Hikone-R
a11 + Lausanne-S
a12 + Lausanne-S (iso, 1970)
a13 + Oregon R
a14 + Oregon-R (iso, 1962)
a15 + Oregon-R (iso, 1970)
a16 + Oregon-R-C
a17 + Oregon-R-S
a18 + Samarkand
a19 + Samarkand (iso, 1971)
a20 + Seto, Japan
a21 + Swedish-b-6
a22 + Swedish-C
a23 + Urbana-S
a24 + Urbana-S (iso, 1971)
a25 + Wageningen
a26 + Wageningen (iso, 1969)

Chromosome 1

* Aa . . . . . (b365)
* $Aa^2$ . . . . . (b202) ($Aa^2$=Qd)
b1 ac-$sc^6$-like & y f:=
* ac . . . . . (b366,b367, etc.)
b2 $ac^3$ $w^a$
b3 $ac^3$ $w^a$.Dp($sc^{V1}$ $y^+$) & y f:=
* $ac^{-51}$ . . . . . (b380)
* $ac^{-53}$ . . . . . (b381,b382)
b4 amx/In49 $m^2$ $g^4$
b5 amx $lz^g$ v & y f:=
b6 $amx^{55}$ . . . . . (=$lz^K$)
b7 Ax
b8 B
b9 B $Bx^r$ car & y f:=
b10 B car $su^W$-f & y w f:=
* $B^3$ . . . . . (b85)
* $B^{263-20}$ . . . . . (b42)
* $B^i$ . . . . . (b86)
* $B^{M1}$ . . . . . (b48)
b11 $B^{M2}$ $f^{B27}$/C1B (mosaic in f/$f^{27}$)
* BB . . . . . (b87,b88)
* $BB^L$ . . . . . (b242)
* $BB^{36b}$ . . . . . (b89)
* $B^1B^i$ . . . . . (b90)
* bb . . . . . (b22,b196)
* $bb^{\underline{1}}$ . . . . . (b415)
* $bb^{\underline{1}158}$ . . . . . (b385)
* $bb^{\underline{1}159}$ . . . . . (b386)
* $bb^N$ . . . . . (b342)
* $bb^+$ . . . . . (b22,b196)
b12 Bg B/InAM
b13 bi $ct^6$ $g^2$
b14 bo
b15 br
b16 br $w^e$ ec rb $t^4$/FMI, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
b17 Bx
b18 $Bx^2$
b19 $Bx^3$
* $Bx^r$ . . . . . (b9)
* $Bx^{r49k}$ . . . . (b86)
b20 $Bx^J$
b21 car
* car . . . . . (b9,b10,b41)
b22 car bb
* cho . . . . . (b230)
* $cho^2$ . . . . . (b369,b483)
* C1B . . . . . (b11)
* C1B . . . . . (b25)
b23 cm
b24 cm $ct^6$
b25 cm $ct^6$ oc ptg v/y C1B v
b26 cm $ct^6$ $sn^3$ & y w f:=
b27 cm $ct^6$ su-$F1^s$/$sc^{S1}$ B In49 $sn^{X2}$ $sc^8$
b28 Co
b29 Co & y w f:=
b30 Co & $y^4$ cv v f bb
$y^2$ su-$w^a$ $w^a$ bb ) @
b31 Co rst$^2$ & y w bb.=
* $Co^2$ . . . . . (b484)
b32 $cs^{53}$ & y w bb.=
b33 $ct^6$ oc & y f:=
* $ct^6$ . . . . . (b13,b16,b24,b25,b26,b27)
b34 $ct^n$ oc/$y^{31d}$ $sc^8$ B In49 $lz^s$ $w^a$
* $ct^K$ . . . . . (b398)
b35 $ct^-$ oc ptg; $ct^i$ & $ct^-$ oc ptg/y $sc^4$ B In49 $lz^s$ v $sc^8$ ($ct^i$)
b36 $ct^-$(.$Y^S$); $ct^i$ (/$ct^i$)
b37 cv f
b38 cx
b39 $cx^{tg}$ t/FMI, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
b40 Df(1)259-4/FM4, $y^{31d}$ $sc^8$ dm B
b41 Df(1)260-1/FM4, $y^{31d}$ $sc^8$ dm B
b42 Df(1)$B^{263-20}$/$sc^7$ InAM car
b43 Df(1)$ct^{268-42}$, y/FM4, $y^{31d}$ $sc^8$ dm B

b44 Df(1)$g^{\underline{1}}$ f B/InAM
b45 Df(1)ma-1/y In49 $sn^{X2}$ $bb^{\underline{1}}$ . . . . . [= $sc^8$ Df(1)ma-1/y In49 $sn^{X2}$ $bb^{\underline{1}}$]
b46 Df(1)svr, Dp(1;f)101 (Dp. het. or hom.)
b47 dor/C1B
b48 dow $B^{M1}$/$sc^{S1}$ B In49 oc $sc^8$
b49 dow/y Hw In49 $m^2$ $g^4$
b50 dm & y f:=
* Dp($sc^{V1}$$y^+$) . . . . . (b3)
b51 Dp(1;f)3 = Del(1)3/$\underline{1}J1^{259}$ y w $Y^L$ @ $Y^S$ & y.=
b52 Dp(1;f)18 = Del(1)18/$\underline{1}J1^{259}$ y w $Y^L$ @ $Y^S$ & y v f.=
b53 Dp(1;f)52 = Del(1)52/$\underline{1}J1^{259}$ y w $Y^L$ @ $Y^S$ & y.=
b54 Dp(1;f)101 In(1)$sc^8$, Df(0+ac)$w^a$ $sc^8$ ; Dp(1;f)101
b55 Dp(1;f)107 In(1)$sc^8$, Df(0+ac)$w^a$ $sc^8$ ; Dp(1;f)107
b56 Dp(1;f)118 In(1)$sc^8$, Df(0+ac)$w^a$ $sc^8$ ; Dp(1;f)118
b57 Dp(1;f)112 = Del(1)112/$\underline{1}J1^{259}$ y w $Y^L$ @ $Y^S$ & y v f.=
b58 Dp(1;f)122 = Del(1)122/$\underline{1}J1^{259}$ y w $Y^L$ @ $Y^S$ & y v f.=
b59 Dp(1;f)135 In(1)$sc^8$, Df(0+ac)$w^a$ $sc^8$; Dp(1;f)135
b60 Dp(1;f)164 = Del(1)164/$\underline{1}J1^{259}$ y w $Y^L$ @ $Y^S$ & y v f.=
b61 Dp(1;f)1492 = Del(1)1492/$sc^{53k}$
b62 Dp(1;f)1514 = Del(1)1514/$sc^{53k}$
b63 Dp(1;f)$X^{c2}$ Dp(1;f)$X^{c2}$/y $\underline{1}$(1)7/y $\underline{1}$(1)7

b64 Dp(1;f)$z^9$ Dp(1;f)$z^9$ ; Df(1)$sc^{J4K}$ & y f:=
b65 Dp(1;1)112 y f, Dp(1;1)112 (homozygous stock)
b66 Dp(1;1)$B^S$(TMG), $sc^{8L}$, $X^{c2R}.B^S/X^D/B^S$ $Y^L$ @ $Y^S$
b67 Dp(1;1)$B^S$(TMG),y $sc^4$ m f @ $B^S/sc^7$ InAM
b68 Dp(1;$Y^L$)$sc^{S1}$ $sc^{S1}.Y^L/y,Y^S$ ; y f:= ; cn bw ; (e/+)
b69 Dp(1;3)51 = T(1;3)51/1J1$^{259}$ y w $Y^L$ @ $Y^S$ & y v f.=
b70 Dp(1;3)126 v f; Dp(1;3)126/Payne, Dfd ca
b71 Dp(1;3)$sc^{J4}$ Dp(X;3)$sc^{J4}$/Df(1)$sc^8$,$w^a$
b72 Dp(1;3)$w^{49a7}$ (Spotter)
b73 Dp(1;3)$sc^{J4}$/1J1$^{259}$ y w $Y^L$ @ $Y^S$ & y y f.= (no free Y-?)
b74 Dp(1;4)174 = T(1;4)174/1J1$^{259}$ y w $Y^L.Y^S$ & y v f.=
b75 dy
b76 ec
b77 ec $ct^6$ v $g^3$/ClB
b78 ec dx
b79 ec dx/y su-Hw $Hw^2$ In49 $m^2$ $g^4$
* eq . . . . . (f62)
b80 Ext/FM6, $y^{31d}$ $sc^8$ dm B
b81 f
b82 f B car $su^W$-f & y f:=
b83 f B os car
b84 f B os $f^{+ih}$ & y f:=
b85 f $B^3$ & y f:=
b86 f $B^i$ (Luce 436.1) & y f:=
b87 f BB & y f:=
b88 f BB/InAM
b89 f $BB^{36b}$ & y f:=
b90 f $B^iB^i$ & y f:=
b91 f $Bx^3$ mal & y f:=
b92 f $Bx^3$ mal $su^W$-f & y f:=
b93 f fu & y f:=
b94 f fu/ClB
b95 f $os^o$ car
* $f^3$ . . . . . (b308)
* $f^{3N}$ . . . . . (b297,b362)
b96 $f^5$ os $f^{+ih}$ & y w f:=
* $f^5$ . . . . . (b326)
b97 $f^5$ $su^W$-f
b98 $f^{36a}$
b99 $f^{36a}$ odsy $f^{+ih}$ & y f:=
b100 $f^{257-5}$/InAM (Demerec)
* $f^{B15}$ . . . . . (b305)
* $f^{B27}$ . . . . . (b11)
b101 $f^x$ car & y f:=
b102 fa
* $fa^n$ . . . . . (b405)
b103 fa $fa^{no}$ $sn^3$
b104 fa $N^{j24a}$/y Hw In49 $m^2$
b105 fa $N^{j24c}$ $sn^3$/y Hw In49 $m^2$
b106 fa rb
b107 fa spl $sn^3$
b108 $fa^{no}$
b109 $fa^{no}$ spl
b110 Fl oc ptg v & y f:=
b111 Fl oc ptg v/y ClB v
b112 $Fl^s$/y ClB v
b113 $Fl^s$ $ct^6$/y ClB v
b114 $Fl^s$ $sn^3$/y ClB v
b115 $Fl^s$ $sn^3$ $t^2$ v f car/y ClB v
b116 $Fl^{s1}$ $ct^6$/y ClB v
b117 flp
* Fm1 . . . . . (b16,b39)
* FM3 . . . . . (b174)
* FM4 . . . . . (b4, b41)
* FM6 . . . . . (b130,b162)
b118 fo
b119 fs Nas/FM4, $y^{31d}$ $sc^8$ dm B
b120 fu/ClB
* $fw$ . . . . . (b299,b352)
* $fw^{34e}$ . . . . . (b532)
* $fw^{51g}$ . . . . . (b407)
b121 $g_2$
b122 $g^2$
* $g^2$ . . . . . (b13)
b123 $g^2$ p1/FM3, $y^{31d}$ $sc^8$ dm B 1
b124 $g^2$ ty & y f:=
b125 $g^3$
* $g^3$ . . . . . (b77)
* $g_4$ . . . . . (b4)
b126 $g^{50e}$
b127 $g^{53d}$
b128 $g^{im}$/y $sc^{S1}$ B InS
b129 $g^w$
b130 $gg^2$/FM6, $y^{31d}$ $sc^8$ dm B
b131 $gg^3$
b132 gt $bb^{11}$/ClB
b133 gt v
b134 gt $w^a$
b135 gt $w^a$ (Oregon-R)
* $Hw$ . . . . . (b49,etc.)
* $Hw^2$ . . . . . (b449)
b136 $Hw^{49c}$/Fm1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
b137 $if^3$
b138 In49 $B^{M1}$
b139 In49 $ct^{ns}$
b140 In49 Fl v g & y w f:=
b141 In49 $lz^s$ & y f:=
b142 In49 $m_2$ v $sn^{X2}$ g/y ClB
* In49 $m^2$ . . . . . (b4)
b143 In49 ptg oc & y f:=
b144 In49 ptg oc $ct^{ns}$/y ClB v
b145 In49 ptg oc $ct^{ns}$ & y f:=
b146 In49 ptg oc $ct^{ns}$/y ClB
b147 In49 $sn^{X2}$ & y f:=
b148 In49 ty-1
b149 In49 ty-1 $bb^1$ & y v f car.=
b150 In49 v Fl g;($ct^i$)

b151 In49 v Fl g $B^{Ml}$ & y f:=
b152 In49 v Fl g/y ClB v
b153 In49 v g & y f:=
b154 In49 v ptg oc Fl $B^{Ml}$ & y f:=
b155 In49 v $sn^{X2}$ B & y f:=
b156 In49 $v^{Of}$ f
b157 InAB & y f:=
* InAM . . . . . (b42)
b158 In($X^{c2}$)$w^{vC}$/y Hw In49 $m^2$ $g^4$ $f^5$ (ring stabilized)
b159 kz
b160 l J1 $sc^{J1}$ $y^{S1}$ v f mal $su^{W}$-f bb.$y^+$ & y f:=
b161 l J1 $sc^{J1}$ $y^{S2}$ pn/y $sc^{S1}$ B InS
b162 l(1)7/FM6, $y^{34}$ $sc^8$ dm B
* l(1)451 . . . . . (b423)
* l(1)ml . . . . . (b424)
b163 lh B car bb & y f:=
b164 lz & y f:=
b165 $lz^3$ & y f:=
b166 $lz^3$ m & y w f:=
b167 $lz^{34k}$ & y f:=
b168 $lz^{37h}$
* $lz^{46f24}$ . . . . . (b255)
b169 $lz^{48f}$ & y f:=
b170 $lz^{BS}$ $lz^{46f}$ $ras^4$ v & y f:=
* $lz^{D}$ . . . . . (j14)
* $lz^{g}$ . . . . . (b5,b443)
* $lz^{K}$ . . . . . (b6)($lz^{K}$=$amx^{SS}$)
* $lz^{s}$ . . . . . (b141)
b171 m
* m . . . . . (b207)
b172 m f car & y w f:=
b173 $m_f{}^{36a}$
* $m^2$ . . . . . (b4)
b174 $m^{D}$/FM3, $y^{31d}$ $sc^8$ dm B l
b175 M(1)n/FM6, $y^{31d}$ $sc^8$ dm B
b176 M(1)o f/In AM
b177 M(1)Sp/In AM
b178 mal & y f:=
b179 $mal^{by}$
* $N^6$ . . . . . (b426)($N^6$=$N^{264-6}$)
b180 $N^8$/y Hw In49 $m^2$ $g^4$
* $N^{264-6}$ . . . . . (b426)
b181 $N^{264-10}$/FM6, $y^{31d}$ $sc^8$ dm B
b182 $N^{264-39}$ $w^{ch}$/FM4, $y^{31d}$ $sc^8$ dm B
* $N^{264-47}$ . . . . . (b427)
* $N^{264-84}$ . . . . . (b428)
* $N^{264-103}$ . . . . . (b429)
b183 $N^{264-105}$ (dm)/y Hw In49 $m^2$ $g^4$
* $N^{j24a}$ . . . . . (b104)
* $N^{j24c}$ . . . . . (b105)
b184 na & y f:=
* Nas . . . . . (b119)
b185 nd
b186 nd rb
b187 ny f/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B;(ri)
b188 oc ptg Tu/$sc^{S1}$ fu In49 $sc^8$
b189 oc $ptg^3$.Dp($sc^{S1}$ $y^+$)/ClB
* oc . . . . . (b25)
b190 os
b191 $os^o$
b192 $os^o$ Dp($f^{+ih}$) & y f:=
b193 pa/FM4, $y^{31d}$ $sc^8$ dm B
b194 peb v
b195 pn
b196 pn In49 v $B^{Ml}$ bb
b197 ("bleached") pn w rb cm $ct^6$ $sn^3$ $ras^2$ v dy $g^2$ f car/$sc^{S1}$ B In49 $sn^{X2}$ oc ptg $sc^8$
b198 ("bleached") pn w rb cm $ct^6$ $sn^3$ $ras^2$ v dy $g^2$ f car & y f:=
b199 pn, Inh l/y Hw In49 $m^2$ $g^4$
b200 $pn^2$
* $pn^3$ . . . . . (b239)
* $pn^{54c}$ . . . . . (b435, etc.)
* ptg . . . . . (b25)
b201 $ptg^2$
b202 Qd . . . . . (= $Aa^2$)
b203 $r^9$ & y f:=
b204 $r^{39k}$ f B/InAM
b205 $ras_{dy}$
b206 $ras^2$
b207 $ras^3$ m
b208 $ras^4$ m/ClB
b209 rb
* rb . . . . . (b16)
b210 rb cx
b211 $rb^{S1}$
b212 rg
b213 $rst^2$/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
b214 $rst^2$/y Hw In49 $m^2$ $g^4$
b215 $rst^3$, In & y f:=
b216 $rst^3$, In
b217 $rst^3$, In m v & y f:=
b218 $rst^{3+2}$, v f & y f:=;bw
b219 $rst^{3+17}$, v
b220 $rst^{3+88}$, v f & y f:=
b221 $rst^{3+125}$, v f & y f:=;bw
b222 $rst^{3+204}$, v f
b223 rud/$sc^{S1}$ B InS $w^a$ $sc^8$
b224 rux/FM6, $y^{31d}$ $sc^8$ dm B
b225 $rux^2$
b226 $rux^{60d}$ ct/$sc^{S1}$ B InS $w^a$ $sc^8$
b227 s
b228 sbr & y f:=
b229 sc
b230 sc cho
b231 sc $ct^6$ car & y f:=
b232 sc cv v eg
b233 sc cv v f
b234 sc cv v f B & y f:=
b235 sc ec cv $ct^6$ v $g^2$ f/FM3, $y^{31d}$ $sc^8$ dm B l
b236 sc ec cv ptg & y v f car.=
b237 sc In49 $sn^{X2}$ car/sc oc ptg sd car
b238 sc In49 v $B^{Ml}$
b239 sc $pn^3$ $g^2$ $Bx^2$ = ($g^2$ reverted)
b240 sc $t^2$ v f & y f:=
b241 sc $t^2$ v f Tu car & y f:=
b242 sc w $BB^{L}$, In $Y^{S}$ & y f:=
b243 sc z ec $ct^6$
b244 sc z $swb^{62b12}$ $ct^6$

b245 sc z $w^{17G2}$ ec $ct^6$
* $sc^2$, $sc^{3-1}$, etc. . . . (c1-77)
b246 Sc/y $sc^{S1}$ g In49 m $sc^8$ (select Sc ♀)
b247 scp t
b248 sd;(se)
b249 $sd^{62\underline{1}}$ & y f:=
b250 $Sh^2$/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
b251 $Sh^5$
b252 $shf^2$
b253 sn v $B^{M1}$ $bb^+$ & y f:=
* $sn^2$ . . . . . (b333)
b254 $sn^3$
* $sn^3$ . . . . . (b26)
b255 $sn^3$ $lz^{46}f^{24}$ $ras^4$ v & y f:=
b256 $sn^3$ v $B^{M1}$ $bb^+$ & y w f:=
b257 $sn^4$
* $sn^{5.1}$ . . . . . (c4i)
* $sn^{5S}$ . . . . . (d7)
b258 $sn^{34e}$
b259 $sn^{36a}$ ♂ & $\frac{w^{bf}\ M(1)36f}{w^{bf3}}$ )@ ♀
b260 $sn^{36a}$ & y f:=
b261 $sn^c$/y Hw In49 $m^2$ $g^4$
* $sn^{X2}$ . . . . . (b414)
b262 sp-w . . . . . (= $w^{sp}$)
b263 sp1
b264 sp1 $cho^2$
b265 sp1 rb cx & y f:=
b266 sp1 $rb^{S2}$
b267 spx & y f:=
b268 spx car
b269 sta & y f:=
b270 sta/FM3, $y^{31d}$ $sc^8$ dm B $\underline{1}$
* $su^W$-f . . . . (b10,b82, etc.)[=su(f)]
* su-$F\underline{1}$ . . . . . (b27)
* su-Hw . . . . . (b449)[=su(Hw)]
* $su\text{-}v^{51e15}$. . . . . (b493)
b271 $su^2$-s v; bw . . . . . [= $su(s)^2$]
b272 $su^2$-s $w^a$ cv t
b273 $su^3$-s cv v f & y f:= . [= $su(s)^3$]
b274 $su^{S2}$-v-pr v & f B.= . [= $(su(s)^S$]
b275 $su^{S2}$-v-pr v & y f:=
b276 $Su^x$-dx dx . . . . . [= $Su(dx)^x$]
b277 su-$w^a$ $w^a$ . . . . . [= $su(w^a)$]
b278 svr
b279 svr su-$w^a$ $w^a$
b280 svr $w^a$
b281 $svr^{poi}$
b282 sw
b283 $swb^{62b12}$
b284 sx $vb^2$ $os^s$/InAM
b285 sy . . . . . (= $os^2$)
b286 t
b287 $t^2$ v f
b288 $t^3$
* $t^4$ . . . . . (b16)
b289 Tu & y f:=
b290 tw/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
b291 un $Bx^2$ & y f:=
b292 $un^4$
b293 v
* v . . . . . (b5,b11,b25)
b294 v $B^{M1}$ (tan-like)
b295 v f $Bx^{r49k}$ car & y f:=
b296 v f $su^W$-f
b297 v $f^{3N}$ car
b298 $v_2r^{12}$
b299 $v^2$ fw
b300 $v^{36f}$
b301 $v^{48a}$
b302 $v^{51c}$
b303 $v^k$
b304 $v^+$(rev.v) $B^{M2}$
b305 $v^+$(rev.v) $B^{M2+}$(rev.B,rein) $f^{Bl5}$(mosaic)
b306 $v^+$(rev.v) $f^x$ $f^{+ih}$
b307 vb
b308 vs
b309 w
b310 w & +.=
b311 w ec
b312 w In49 $lz^s$ & y f:=
b313 w m Bx & y.=
b314 w m f
b315 w sn oc ptg/$sc^{S1}$ B In49 $lz^s$ $sc^8$
b316 $w^{22cHl}$
* $w^{34b}$ . . . . . (c17)
b317 $w^{40aHl}$/y $sc^{S1}$ B In49 v
b318 $w^a$
* $w^a$ . . . . . (b2,b3)
b319 $w^a$ $f^3$ os $f^{+ih}$ & y f:=
b320 $w^a$ $F1^s$ $sn^3$/y C1B v
b321 $w^a$ sp1
b322 $w^{a2}$
b323 $w^{a3}$
b324 $w^{a4}$
b325 $w^{a9aJ1}$
b326 $w^{bf}$ $f^5$
b327 $w^{bf2}$
b328 $w^{b1}$
b329 $w^{Bwx}$
b330 $w^{cf}$/FM4, $y^{31d}$ $sc^8$ dm B
b331 $w^{ch}$ wy
b332 $w^{co}$
b333 $w^{co}$ $sn^2$
b334 $w^{co1}$
b335 $w^{cp}$
b336 $w^e$
* $w^e$ . . . . . (b16)
b337 $w^e$ m
b338 $w^e$ sn/C1B
b339 $w^{e2}$
* $w^{e59}$ . . . . . (b375)
b340 $w^{ec3}$ (écru)
b341 $w^h$
b342 $w^i$ $f^3$ $bb^N$
b343 $w^{m4}$(3C1-2&20)

b344 $w^{m4}$ (Oregon-R autosomes; Y's extra)
b345 $w^{m4}$ v $w^{mMc}/Y^{bb-}$
b346 $w^{m4w}$
b347 $w^{m4w}$ ($Y^{Su-V}$ extra)
b348 $w^{mMc}$ & y f:=
b349 $w^{mMc}$ f $w^{m4}$
b350 $w^{mR7aH1}$
* $w^{PO}$ . . . . . (b480)
* $w^{r}$ . . . . . (c23)
b351 $w^{sat}$
* $w^{sp}$ . . . . . .(=sp-w)(b262,b480)
b352 $w^{t}$ fw
b353 wy
b354 $X^{c1}$, y & y f:=
b355 $X^{c2}$, cv v f/C1B
b356 $X^{c2}$, ec f & y f:= (ring OK 1970)
b357 $X^{c2}$, sc f & y f:=
b358 $X^{c2}$, y B & y f:= (ring OK 1971)
b359 $X^{c2}$, y $ct^{6}$ f & y f:= (ring OK 1970)
b360 $X^{c2}$, y f & y w f:= (ring OK 1971)
b361 $X^{c2}$, y v & y f:= (ring OK 1971)
b362 $X^{c2}$, y v $f^{3N}$ & y f:= (ring OK 1970)
b363 $X^{c3}$(tm-ac)$^{-}$, $sc^{8}$ $w^{a}$ Ins B & y f:=/$sc^{8}$. Y (ring from tandem X.X)
* $X^{c5}$ . . . . . (b479)
b364 y
b365 y Aa f:=♀ & B♂
b366 y ac $dvr^{(+)}$ v bb
b367 y ac pn In49 v $B^{M1}$
b368 ("tester-1") y ac pn w rb $wy^{2}$ $g^{2}$ & y f:= ; $sc^{19i}$/Cy
b369 y ac sc $cho^{2}$ & y f:=
b370 y ac sc pn & y f:=
b371 y ac sc pn B & y f:=
b372 y ac sc pn w.Dp($sc^{V1}$ $y^{+}$) & y f:=
b373 ("maple") y ac sc pn w rb cm $ct^{6}$ $ras^{2}$ v $g^{2}$ f car & y f:= ; $sc^{19i}$/Cy, InL lt
b374 y ac sc pn w spl rb cx & y f:= ; ($sc^{19i}$(b pr)/)
b375 y ac sc pn $w^{e59}$
b376 y ac sc v & y f:=
b377 y ac $t^{2}$.Dp($sc^{S1}$ $y^{+}$ $ac^{+}$) & y f:=
b378 y ac v
b379 y ac v f $su^{W}$-f
b380 (y ac)$^{-51}$ f (from y f:=) & y f:=
b381 y $ac^{-53}$ sc/y Hw In49 $m^{2}$ $g^{4}$
b382 y $ac^{-53}$ sc B car.Dp($ac^{+}$ $y^{+}$-tm)$^{53}$ & y f:=
* (y ac)$^{Sch}$ . . . . . (d41,d42)
b383 y B & y f:=
b384 y B.= & $sc^{S1}$ B InS $w^{a}$ $sc^{8}$
b385 y $bb^{l1158}$ & y w.=
b386 y $bb^{l1452}$ & $y^{2}$ su-$w^{a}$ $w^{a}$ bb.=
b387 y car $bb^{-}$, In/InAM
b388 y ct v f $os^{sH}$
b389 y $ct^{6}$ & y f:=
b390 y $ct^{6}$.Dp $sc^{V1}$ $y^{62c}$ & y f:=
b391 y $ct^{6}$ $dvr^{2}$ v f & y f:=
b392 y $ct^{6}$ f car & y f:=
b393 y $ct^{6}$ f.Dp($y^{+}$ $sc^{V1}$) & f:=
b394 y $ct^{6}$ f $mal^{2}$ $l^{120}$.Dp($sc^{V1}$ $y^{+}$)/FM6, $y^{31d}$ $sc^{8}$ dm B
b395 y $ct^{6}$ f $l^{122}$.Dp($sc^{V1}$ $y^{+}$)/FM6, $y^{31d}$ $sc^{8}$ dm B
b396 y $ct^{6}$ $ras^{2}$ f
b397 y $ct^{6}$ $t^{2}$ v f car
b398 y $ct^{K}$ ; (bw)
b399 y cv
b400 y cv v f
b401 y cv v f car
b402 y.Dp($y^{+}$ $sc^{V1}$) & y f:=
b403 y f B $f^{+ih}$ & y f:=
b404 y fa $wy^{2}$ $g^{2}$ ($g^{+}$ reversion?)
b405 y $fa^{n}$ In49
b406 y $Fl^{s}$ oc ptg v/y C1B v
b407 y $fw^{51g}$ & y f:=
b408 y Hw & RM, $sc^{8}$ B $w^{a}$ $sc^{S1}$.=
b409 y Hw In49 $B^{M1}$ & y f:=
b410 y Hw In49 $m^{2}$ $g^{4}$/$w^{e}$ sn
b411 y In49 $B^{M1}$ & y f:=
b412 y In49 f car & y f:=
b413 y In49 Fl g $B^{M1}$ & y f:=
b414 y In49 $sn^{X2}$ $B^{M1}$ & y f:=
b415 y In49 $sn^{X2}$ $bb^{l}$ & y f:=
b416 y In49 v $ct^{ns}$ B
b417 y In49 v f & y f:=
b418 y In49 v f car & y f:=
b419 y In49 v Fl g & y w f:=
b420 y In49 v Fl g $B^{M1}$ & y f:=
b421 y In49 v ptg oc Fl car $B^{M1}$ & y f:=
b422 y In49 v ptg oc Fl $g^{2}$ & y f:=
b423 y l(1)451/FM6, $y^{31d}$ $sc^{8}$ dm B
b424 y l(1)ml w In49 f.$Y^{S}$/$sc^{S1}$ B InS
b425 y $lz^{37h}$ f car $su^{W}$-f.$y^{+}$ & y f:=
b426 y $N^{264-6}$/y w dm(=$N^{6}$)
b427 y $N^{264-47}$/y Hw In49 $m^{2}$ $g^{4}$
b428 y $N^{264-84}$/FM6, $y^{31d}$ $sc^{8}$ dm B
b429 y $N^{264-103}$/y Hw In49 $m^{2}$ $g^{4}$
b430 y oc & y f:=
b431 y pn
b432 y pn v f car
b433 y pn w cm $ct^{6}$ $sn^{3}$ oc $ras^{2}$ v dy $g^{2}$ f $os^{o}$ car sw/y $sc^{S1}$ B In49 v
b434 y $pn^{54c}$spl
b435 y $pn^{54c}$ w spl
b436 y $pn^{54c}$ $w^{a}$
b437 ("tester 3") y rb cm $ras^{2}$ $g^{2}$ & y f:= ; $sc^{19i}$/Cy
b438 y $rst^{3}$, In car bb
b439 y sc
b440 y sc cv v f & w cv.=
b441 y sc In49 v g.Dp($sc^{V1}$ $y^{+}$)
b442 y sc In49 v g f
b443 y sc $lz^{g}$ v f & y f:=
b444 ("y sc bleached") y sc pn w rb cm $ct^{6}$ $sn^{3}$ $ras^{2}$ v $g^{2}$ f car/$sc^{S1}$ car f In49 v
b445 y sc v g & y f:=
b446 y $sl^{2}$ $bb^{-}$, In/FM4, $y^{31d}$ $sc^{8}$ dm B
b447 y $sn^{3}$ oc & y f:=
b448 y $sn^{3}$ bb

b449 y su-Hw $Hw^2$ In49 $m^2$ $g^4$ & y w f:=
b450 y $t^2$ v f
b451 y v & y f:=
b452 y v
b453 y v car $bb^-$, In/y InAM
b454 y v $f^x$ B & y w f:=
b455 y v $f^x$ car
b456 y v $f^x$ car $su^W$-f
b457 y v $f^x$ $f^{+ih}$ & y f:=
b458 y v mel mal
b459 y w
b460 y w B & y f:=
b461 y w bb
b462 y w Co & y f:=
b463 y w f
b464 y w In49 f
b465 y w m f & y f:=
b466 y w $sn^3$
b467 y w $sn^3$ B & y f:=
b468 y w $sn^3$ $f^{36a}$
b469 y w $sn^3$ $f^{36a}$ & y f:=
b470 y w spl
b471 y w $t^2$ v f & y f:=
b472 y $w^{-258-11}$ 1/y Hw In49 $m^2$ $g^4$
b473 y $w^{-258-11}$ $t^2$ v f/y $sc^{S1}$ B InS
b474 y $w^{-258-42}$/y Hw In49 $m^2$ $g^4$
b475 y $w^{-258-45}$/FM4, $y^{31d}$ $sc^8$ dm B
b476 y $w^a$.= & $sc^{S1}$ B InS $w^a$ $sc^8$
b477 y $w^a$ m f car
b478 y $w^{m4}$ f v $w^{mMc}$
b479 y $w^{m258-18}$ ; $Xc^5$ $w^{m4}$ & y f:=
b480 y $w^{PO}$ In49 $sn^{X2}$ $bb^1$ & y f:=
b481 y $w^{sp}$/FM6, $y^{31d}$ $sc^8$ dm B
b482 $y^2$
b483 $y^2$ $cho^2$
b484 $y^2$ $Co^2$ In49 $B^{M1}$
b485 $y^2$ cv v f
b486 $y^2$ $dvr^2$ v
b487 $y^2$ ec cv v f car
b488 $y^2$ In49 $lz^s$ & y f:=
b489 $y^2$ oc ptg $B^{M2}$ & y f:=
b490 $y^2$ oc ptg g, Inh & y f:=
b491 $y^2$ sc $w^a$ ec
b492 $y^2$ spx
b493 $y^2$ su-$v^{51e15}$ ras v
b494 $y^2$ su-$w^a$ $w^a$ lz $su^W$-f.$y^+$ & y f:=
b495 $y^2$ spx f car
b496 $y^2$ su-$w^a$
b497 $y^2$su-$w^a$ w
b498 $y^2$ su-$w^a$ $w^a$
b499 $y^2$ su-$w^a$ $w^a$ $lz^1$ $su^W$-f.$y^+$ & y f:=
b500 $y^2$ su-$w^a$ $w^a$ spl
b501 $y^2$ su-$w^a$ $w^a$ spl cv
b502 $y^2$ su-$w^a$ $w^{a2}$
b503 $y^2$ su-$w^a$ $w^{a4}$
b504 $y^2$ su-$w^a$ $w^{bf}$
b505 $y^2$ su-$w^a$ $w^{bl}$
b506 $y^2$ su-$w^a$ $w^{co}$
b507 $y^2$ su-$w^a$ $w^{col}$
b508 $y^2$ su-$w^a$ $w^h$
b509 $y^2$ su-$w^a$ $w^{sat}$
b510 $y^2$ $su^2$-$w^a$ $w^a$
b511 $y^2$ v
b512 $y^2$ v f car
b513 $y^2$ v f car $su^W$-f & y f:=
b514 $y^2$ v $mal^{bz}$
b515 $y^2$ $w^a$
b516 ("tester-2") $y^2$ $w^a$ cm $wy^2$ $g^2$ car & y f:= ; $sc^{19i}$/Cy
b517 $y^2$ $w^a$ ct f.Dp($y^+$ $sc^{S1}$)/C1B
b518 $y^2$ $w^a$ ct mw f/y $sc^{S1}$ B InS
b519 $y^2$ $w^a$ $ct^6$
b520 $y^2$ $w^a$ $ct^6$ lz v f & y f:=
b521 $y^2$ $w^a$ cv m f & y Aa f:=
b522 $y^2$ $w^a$ InS B
b523 $y^2$ $w^a$ $rb^{S1}$
b524 $y^2$ $w^a$ sn oc ptg/$sc^{S1}$ B In49 $lz^s$ $sc^8$
b525 $y^2$ $w^a$ $sn^5$ B & y Aa f:=
b526 $y^2$ $w^a$ spl
b527 $y^2$ $w^a$ v
b528 $y^2$ $w^a$ w
b529 $y^2$ $w^{cf}$
b530 $y^2$ $wy^2$ $g^2$ ($g^2$ partly reverted?)
b531 $y^{2s}$
b532 $y^{2s}$ $fw^{34e}$
b533 $y^{3d}$ & br ec.=
b534 $y^{3d}$ & y f:=
b535 $y^{3p}$, In B
b536 $y^4$, In
b537 $y^4$, In cv v f
b538 $y^4$, In $w^a$
b539 $y^{18cH1}$
b540 $y^{34c}$
b541 $y^{62a}$ sc cv & y f:=
b542 $y^{62c}$ $sc^{V1}$, Inp $t^2$ & y f:=
b543 $y^{bg}$ $ct^6$ car
b544 $y^{td}$
b545 $y^{v2}$
b546 z $w^{11E4}$

## Scute Alleles

(listed alphabetically according to scutes, regardless of position of scute in linear order)

c1 $sc^2$
c2 $sc^2$ pn & y f:=
c3 $sc^{3-1}$ w & y f:=
c4 $sc^{3B}$
c5 y $sc^4$
c6 y $sc^4$ B f InS & y f:=
c7 y $sc^4$ B InS & y f:=
c8 y $sc^4$ B $v^{41b}$/y w In49 $lz^s$
c9 y $sc^4$ InS $w^a$;S $sc^{19i}$ Bl/Cy $L^4$ sp
c10 $sc^5$ $bb^{sc5}$ ; (lt or ltd?)
c11 y $sc^5$
c12 y $sc^5$ $w^{-258-48}$ spl;Dp(1;3)$w^{vco}$;y f:=
c13 $sc^6$ $w^a$
c14 $sc^7$

c15 $sc^7$ InAm car/FM4, $y^{31d}$ $sc^8$ dm (without B)
c16 $sc^7$ oc ptg g, Inh & y f:=
c17 $sc^7$ $w^{34b}$ $B^{M1}$ & y f:=
c18 $sc^7$ $w^a$
c19 $sc^8$
c20 $sc^8$ B
c21 $sc^8$ B $f^x$ v & y f:=
c22 y $sc^8$ B InS $w^a$ & y f:=
c23 ($w^r$=reddish) $sc^8$ B InS $w^r$ & y f:=
c24 $sc^8$ bb $w^a$
c25 $sc^8$ car f In49 v & y f:=
c26 $sc^8$ f In49 v & y f:=
c27 $sc^8$ f v cv & y f:=
c28 ($w^r$=reddish) $sc^8$ InS $w^r$
c29 $sc^8$ $su^W$-f mal f v & y f:=
c30 $sc^8$ Tu $w^a$ & y f:=
c31 $sc^8$ $w^a$
c32 $y^{31d}$ $sc^8$ In49
c33 $y^{31d}$ $sc^8$ $w^a$
c34 $y^{S1}$ $sc^8$
c35 $y^{S1}$ $sc^8$ B In49
c36 $y^{S1}$ $sc^8$ B f In49 v
c37 $y^{S1}$ $sc^8$ B f In49 v $w^a$ & y f:=
c38 $y^{S1}$ $sc^8$ f InS $w^a$ & y f:=
c39 $y^{S1}$ $sc^8$ $f^3$ sd & y w f:=
c40 $y^{S1}$ $sc^8$ $sn^3$ w
c41 $y^{OX}$ $sc^8$ $sn^{5.1}$ w & y f:=
c42 $sc^9$ Bx f t $w^a$
c43 $sc^{10}$ $w^a$
c44 $sc^{10-1}$/y Hw
c45 $sc^{19-}$/$\underline{1}J1$ $sc^{J1}$;fes $sc^{19i}$ b pr/Cy $dp^{txI}$ pr $cn^2$
c46 $sc^{19-}$ & y f:=;fes $sc^{19i}$ b pr/Cy $dp^{txI}$ pr $cn^2$
c47 $sc^{28}$ $w^a$
c48 $sc^{29}$ $w^a$
c49 $sc^{260-14}$
c50 $sc^{260-15}$/y Hw In49 $m^2$ $q^4$
c51 $sc^{260-22}$
c52 $sc^C$/y $sc^{S1}$ B InS
c53 y $sc^{D1}$
c54 y $sc^{D2}$
c55 $sc^H$, TX4 & y f:=
c56 $\underline{1}J1$ $sc^{J1}$/Del($sc^7$)2 & y f:=
c57 $\underline{1}J1$ $sc^{J1}$/Del(X)24
c58 $\underline{1}J1$ $sc^{J1}$/Del($X^c$)Ag (Pontecorvo)
c59 $\underline{1}J1$ $sc^{J1}$ car.Dp($ac^+$ $y^+$-tm)53 & y f:=
c60 $\underline{1}J1$ $y^{S2}$ $sc^{J1}$ pn $t^2$ v f Tu car/y $sc^{S1}$ B InS
c61 $\underline{1}J1$ $sc^{J1}$ $y^s$ v f mal su-f bb.$y^+$ & y f:=
c62 $\underline{w}^{m5L}$;$sc^{J4R}$ & y w f:=($w^{m5L}$)
c63 $sc^{J6}$ B & y f:=
c64 $sc^{L3}$, TX4 (spoon-like)
c65 $sc^{L6}$
c66 $sc^{L8}$ car m $w^a$/y w In49 $lz^s$
c67 $sc^{Mc}$, TX3/y Hw In49 $m^2$ $g^4$
c68 $sc^{S1}$ B InS & y f:=/$Y^+$ (trans. 2:4)
c69 $sc^{S1}$ bb f In49 v w & y f:=
c70 ("plex") $sc^{S1}$ car f In49 v/y ac sc pn w rb cm $ct^6$ $sn^3$ $ras^2$ v dy $g^2$ f car
c71 $sc^{S1}$ In49 v & y f:=
c72 y $sc^{S1}$ B f In49 v & y f:=
c73 y $sc^{S1}$ B In49 $sn^{X2}$ & y f:=
c74 y $sc^{S1}$ B InS & y f:=
c75 $sc^{S2}$, T(1;2)/Cy
c76 $sc^{V1}$, Inp v/$y^{S1}$ $sc^8$ B f In49 v
c77 $sc^{V2}$, Inh

## Combinations of Scute or Similar Inversions

d1 Y $sc^4$ $sc^8$
d2 y $sc^4$ $bb^G$ $sc^8$ (Y's extra)
d3 y $sc^4$ In49 $sn^{X2}$ $sc^8$ & y f:=
d4 y $sc^4$ InS $sc^{S1}$ (extra Y in ♀)
d5 y $sc^4$ InS $sc^{S1}$ & y f:=
d6 y $sc^4$ InS $sc^{S1}$/ClB
d7 $y^{S1}$ $sc^8$ B InS $sc^{S1}$/w $sn^{5S}$ bb
d8 $y^{S1}$ $sc^8$ InS $y^{3P}$;Cy/Scd
d9 FM6, $y^{31d}$ $sc^8$ dm B & y pn.=
d10 FM7-1z, In(1)$sc^8$, d1-49, $y^{31d}$ $sc^8$ $w^a$ 1z $v^{Of}$ B & y f:=
d11 y $sc^-$ (rein. $sc^{8-4}$) $w^a$ InS B & y f:=; $sc^{19i}$/Cy 1t $cn^2$
d12 y $sc^-$ (rein. $sc^{8-4}$) $w^a$ InS bb & y f:=; $sc^{19i}$/Cy 1t $cn^2$
d13 $sc^{-17aH3}$ f car.$sc^{V1}$;Cy/$sc^{19i}$ & y f:=;Cy/$sc^{19i}$
d14 $sc^{L8}$ $g^s$ v 1z$g$ $sc^8$ & y f:=
d15 $sc^{S1}$ InS $w^a$ $sc^4$ & y f:=
d16 y $sc^{S1}$ B $v^{41b}$ $sc^4$ & y f:=
c17 y $sc^{S1}$ InS $w^a$ $sc^4$ & y f:=
c18 y $sc^{S1}$ $v^{41b}$ $sc^4$ & y f:=

## $sc^{S1}$-$sc^8$ (d19-d67)

d19 $sc^{S1}$ At In49 $sc^8$
d20 $sc^{S1}$ At In49 v $w^a$ $sc^8$ & y f:=
d21 $sc^{S1}$ B g In49 m $sc^8$ & y f:=
d22 $sc^{S1}$ B In49 $lz^s$ $sc^8$/y ac sc pn w v g f
d23 ("Binsc") $sc^{S1}$ B In49 $sc^8$
d24 ("Binsc") $sc^{S1}$ B In49 $sc^8$ & y f:=
d25 ("new Binsc, 1962") $sc^{S1}$ B In49 $sc^8$ & y f:=
d26 ("Binsc") $sc^{S1}$ B In49 $sn^{X2}$ $sc^8$ & y f:=
d27 ("Binsn") $sc^{S1}$ B In49 $sn^{X2}$ $sc^8$/oc ptg Tu
d28 $sc^{S1}$ B In49 $sn^{X2}$ $sc^8$/y sc $ct^-$
d29 $sc^{S1}$ B In49 v $sc^8$/$ct^-$ $t^2$ v & $sc^{S1}$ B In49 v $sc^8$
d30 ("Bwasc") $sc^{S1}$ B InS w $sc^8$ & y.= ($w^a$-w; W.F. Hollander)
d31 ("Basc") $sc^{S1}$ B InS $w^a$ $sc^8$
d32 ("Bosc $bb^+$") $sc^{S1}$ $bb^+$ B In49 oc ptg $sc^8$ & y w f:=
d33 $sc^{S1}$ $bb^+$ In49 oc ptg w $sc^8$ & y f:=
d34 ("Bosc $bb^1$") $sc^{S1}$ $bb^1$ B In49 oc ptg $sc^8$ & y f:=
d35 $sc^{S1}$ car B In49 v $sc^8$ & y f:=
d36 $sc^{S1}$ car dy $w^a$ $sc^8$/w In49 $lz^s$
d37 $sc^{S1}$ f In49 v $w^a$ $sc^8$ & y f:=
d38 $sc^{S1}$ In49 m w $sc^8$/y sn v

d39 ("Insc") $sc^{S1}$ In49 $sc^{8}$
d40 ("Insn") $sc^{S1}$ In49 $sn^{X2}$ $sc^{8}$ & y f:=
d41 (y ac)$^{Sch}$ $sc^{S1}$ B InS $w^{a}$ $sc^{8}$
d42 (y ac)$^{Sch}$ $sc^{S1}$ $B^{+}$ InS $w^{a}$ $sc^{8}$
d43 y $sc^{S1}$ At In49 $sc^{8}$/oc ptg
d44 ("Binscty") y $sc^{S1}$ B In49 $ct^{ns}$ $sc^{8}$
d45 ("Binscy") y $sc^{S1}$ B In49 $sc^{8}$ & y f:=
d46 ("new Binscy") y $sc^{S1}$ B In49 $sc^{8}$
d47 y $sc^{S1}$ B In49 $sc^{8}$ $\underline{l}$/oc ptg
d48 y $sc^{S1}$ B In49 $sc^{8}$ $\underline{l}$/y v car $bb^{-}$
d49 y $sc^{S1}$ B In49 $ct^{ns}$ w $sc^{8}$ & y f:=
d50 y $sc^{S1}$ B In49 $sn^{X2}$ v $sc^{8}$ & y f:=
d51 y $sc^{S1}$ B In49 $sn^{X2}$ w $sc^{8}$ & y f:=
d52 ("Binscty-v") y $sc^{S1}$ B In49 v $ct^{ns}$ $sc^{8}$
d53 y $sc^{S1}$ B In49 v $sc^{8}$ & y w f:=
d54 y $sc^{S1}$ B In49 v $w^{a}$ $sc^{8}$ & y f:=
d55 y $sc^{S1}$ B In49 w $sc^{8}$ & y f:=
d56 ("Brinscy") y $sc^{S1}$ B r In49 $sc^{8}$ y f:=
d57 y $sc^{S1}$ f ptg oc $sc^{8}$ & y f:=
d58 ("winscy Bx") y $sc^{S1}$ $Bx^{M}$ In49 w $sc^{8}$
d59 y $sc^{S1}$ car os f $sc^{8}$
d60 y $sc^{S1}$ f In49 $sc^{8}$ & y f:=
d61 y $sc^{S1}$ f In49 v $sc^{8}$ & y f:=
d62 y $sc^{S1}$ f In49 v $w^{a}$ $sc^{8}$ & y f:=
d63 ("Inscy") y $sc^{S1}$ In49 $sc^{8}$
d64 y $sc^{S1}$ In49 $ct^{ns}$ w $sc^{8}$ & y f:=
d65 y $sc^{S1}$ In49 v $sc^{8}$
d66 ("winscy Bx") y $sc^{S1}$ In49 w $sc^{8}$
d67 y $sc^{S1}$ $sc^{8}$
d68 $sc^{S1}$ f InS $y^{3P}$ & y f:=
d69 $sc^{V2}$ B $y^{3P}$
d70 (y ac)$^{-}$ $y^{3P}$ $sc^{8}$ (iso,1952)
d71 (y ac sc)$^{-}$ $y^{3P}$ InS $sc^{S1}$ & y f:=; Cy/$sc^{19i}$
d72 (y sc)$^{-}$ $ct^{6}$.$sc^{V1}$ $y^{62c}$ & y f:=

Translocations of X and 4

e1 T(X;4)(1B3$^{+}$), $sc^{8}$ B $w^{a}$
e2 T(X;4)$v^{m5}$(3C2) & y f:=
e3 T(X;4)$w^{m5}$(3C2)/$ey^{D}$
e4 T(X;4)$w^{m5}$(3C2), v f bb/$w^{m5}$ ClB
e5 T(X;4)$w^{258-18}$ (3C4), y/$ci^{D}$
e6 T(X;4)$w^{m258-18}$(3C4), y/y Hw In49 $m^{2}$ $g^{4}$
e7 T(X;4)$w^{m258-18}$(3C4), y/y w dim
e8 T(X;4)$N^{8a}$(3C5-6&7)/FM6, $y^{31d}$ $sc^{8}$ dm B
e9 T(X;4)$N^{8a}$ $w^{m5}$/ci $ey^{R}$
e10 T(X;4)$w^{m258-21}$(3E5&6)/$y^{31d}$ $sc^{8}$ B In49 $lz^{s}$ $w^{a}$
e11 T(X;4)$w^{m258-21}$, y $w^{a}$/FM4, $y^{31d}$ $sc^{8}$ dm B
e12 T(X;4)(4C3) & y f:=
e13 T(X;4)A17(8A2) & y f:=
e14 T(X;4)A17(8A2), y cv & y f:=
e15 T(X;4)(9A1) & y f:=
e16 T(X;4)"W13"(9B&20)/ClB
e17 T(X;4)"W13"(9B&20), car
e18 T(X;4)"W13"(9B&20), y w & y f:=
e19 T(X;4)"Sidky a"(13B8-9) & y f:=
e20 T(X;4)11(15A), y $\underline{l}^{11}$ car/$sc^{8}$.Y
e21 T(S;4)$B^{S}$(16A1) & y f:=
e22 T(X;4)$B^{S}$(16A1) & y w f:=
e23 T(X;4)$B^{S}$(16A1) car & y f:=
e24 T(X;4)$B^{S}$(16A1), $y^{2}$ cv v $B^{S}$ car & y f:=
e25 T(X;4)$B^{S}$(16A1).$Y^{S}$ sc.$Y^{L}$ & y w f:=
e26 T(X;4)A13(18C5)
e27 T(X;4)A19
e28 T(X;4)A20 & y f:=
e29 T(X;4)e15
e30 T(X;4)h4
e31 T(X;4)h6
e32 T(X;4)$w^{m5}$;T(X;3)$sc^{J4R}$(C1B)
e33 T(X;4)$w^{m5}L_{w}^{VD3R}/y^{2}$ $w^{a}$ Dp(1;1)Co;ci $ey^{R}$
e34 Ts(X;4)$w^{m5}$, 9A, $w^{m5}$/FM4, $y^{31d}$ w dm B
e35 Ts(X;4)$w^{m5}$ v $B^{S}$/FM4, $y^{31d}$ w dm f

Altered Y's Sometimes with Mutants in X and/or Autosomes

The presence of $Y^{S}$ and/or $Y^{L}$ attachments on X.Y chromosomes is uncertain unless they have been freshly tested or are accompanied by markers ($bb^{+}$ for $Y^{S}$ and $y^{+}$ $sc^{8}$ for $Y^{L}$) that can be followed.

f1 $y^{+}$ $Y^{S}$.X InEN y.$Y^{L}$ $y^{+}$ & $y^{2}$ su-$w^{a}$ $w^{a}$ bb.= (no free Y)
f2 $Y^{S}$.X InEN B f v y.$Y^{L}$ $y^{+}$ & y v bb.= (no free Y)
f3 $Y^{S}$.X InEN B y.$Y^{L}$ & $y^{2}$ su-$w^{a}$ $w^{a}$ bb.= (no free Y)
f4 $Y^{S}$.X InEN B y.$Y^{L}$ $sc^{8}$ $y^{+}$ & $y^{2}$ su-$w^{a}$ $w^{a}$ bb.= ; S fes Sp b/($\underline{l}^{+}$?) InCyL b (no free Y)
f5 $Y^{S}$.X InEN car f In49 v y.$Y^{L}$ & $y^{2}$ su-$w^{a}$ $w^{a}$ bb.= (no free Y)
f6 $Y^{S}$.X InEN In26 B f v Fl-like (not Fl).$Y^{L}$ $sc^{8}$ $y^{+}$ & $y^{2}$ su-$w^{a}$ $w^{a}$ bb.= (no free Y)
f7 ("snoc") $Y^{S}$.X InEN ptg oc $sn^{5}$.$Y^{L}$ & ( sc $ct^{n}$ oc ptg car / y In49 $sn^{X2}$ ) @ (no free Y)
f8 ("snoct") $Y^{S}$.X InEN ptg oc $sn^{5}$.$Y^{L}$ & ( sc $ct^{n}$ oc ptg car / y $ct\underline{l}$, In In49 $sn^{X2}$ ) @ (no free Y)
f9 $Y^{S}$.InEN ptg oc y.$Y^{L}$ $sc^{8}$ $y^{+}$ & $y^{2}$ su-$w^{a}$ $w^{a}$ bb.= (no free Y)
f10 $Y^{S}$.X InEN v cv y.$Y^{L}$ $y^{+}$ & $y^{2}$ su-$w^{a}$ $w^{a}$ bb.= (no free Y)
f11 $Y^{S}$.X InEN v ptg oc $sn^{5}$ w y.$Y^{L}$ $sc^{8}$ $y^{+}$ & y sc $t^{2}$ v f car.= (no free Y)
f12 $Y^{S}$.X InEN v y.$Y^{L}$($sc^{8}$?y)(no free Y)
f13 $Y^{S}$.X InEN v y.$Y^{L}$($sc^{8}$?y) & ( sc $ct^{n}$ oc ptg car / y In49 $sn^{X2}$ ) @ ; b pr

f14 $Y^S$.X InEN v y.$Y^L$($sc^8$?y) & ( sc $ct^n$ oc ptg car / y In49 $sn^{X2}$ ) @ ; vg bw
f15 $Y^S$.X InEN v y.$Y^L$ $sc^8$ $y^+$/v ; $bw^{VA}/L^2$ 1 (no free Y)
f16 $Y^S$.X InEN y.$Y^L$ & $y^2$ su-$w^a$ $w^a$ bb.= (no free Y)
f17 $Y^S$.X InEN y.$Y^L$ $sc^8$ $y^+$ (no free Y)
f18 $Y^S$.X InEN y.$Y^L$ $sc^8$ $y^+$ & $y^2$ su-$w^a$ $w^a$ bb.= (no free Y)
f19 $Y^S$.X y In49 v f car.$Y^L$ (no free Y)

rings produced by irrad. $y^+$ $Y^S$.X InEN y.$Y^L$ $y^+$ (f20-f45)(See also pages 32 and 34)

f20 X.$Y^{Lc}$ InEN y (#1)/Y & y f:=
f21 X.$Y^{Lc}$ InEN y (#2)/Y & y f:=
f22 X.$Y^{Lc}$ InEN y (#3)/Y & y f:=
f23 X.$Y^{Lc}$ InEN y (#5)/Y & y f:=
f24 X.$Y^{Lc}$ InEN y (#7)/Y & y f:=
f25 X.$Y^{Lc}$ InEN y (#8)/Y & y f:=
f26 X.$Y^{Lc}$ InEN y (#9)/Y & y f:=
f27 X.$Y^{Lc}$ 19-20d, y/$Y^+$ & y.=
f28 X.$Y^{Lc}$ 19-20d, y cv/$Y^+$ & y.=
f29 X.$Y^{Lc}$ 19-20d, y cv v/$Y^+$ & y.=
f30 X.$Y^{Lc}$ 19-35a, y cv/$Y^+$ & y.=
f31 X.$Y^{Lc}$ 19-35a, y cv v/$Y^+$ & y.=
f32 X.$Y^{Lc}$ InEN y (#99)/Y & y f:=
f33 X.$Y^{Lc}$ InEN y (#103)/Y & y f:=
f34 X.$Y^{Lc}$ InEN y (#104)/Y & y f:=
f35 X.$Y^{Lc}$ InEN y (#107)/Y & y f:=
f36 X.$Y^{Lc}$ InEN y (#116)/Y & y f:=
f37 X.$Y^{Lc}$ InEN w y (#400)/Y & y f:=
f38 X.$Y^{Lc}$ InEN w y (#402)/Y * y f:=
f39 X.$Y^{Lc}$ InEN w y (#403)/Y & y f:=
f40 X.$Y^{Lc}$ InEN w y (#404)/Y & y f:=
f41 X.$Y^{Lc}$ InEN w y (#405)/Y & y f:=
f42 X.$Y^{Lc}$ InEN w y (#406)/Y & y f:=
f43 X.$Y^{Lc}$ InEN w y (#407)/Y & y f:=
f44 X.$Y^{Lc}$ InEN w y (#409)/Y & y f:=
f45 X.$Y^{Lc}$ InEN w y (#410)/Y & y f:=

f46 $X^D$, $B^S$ $Y^L$.$Y^S$ & y v bb.= (no free Y)
f47 $Y^+$/y $w^{m4}$ $N^{264-84}$ sn/FM3, $y^{31d}$ $sc^8$ dm B 1 ♀ & dm sn ♂
f48 $Y^{cF}$ (H#2)/B (Filicidal ring-Y OK 1971)
f49 $Y^{cF}$ (H#2)/y $sc^{S1}$ In49 $sc^8$ (Filicidal ring-Y OK 1971)
f50 $Y^{cF}$ (#9)/B (Filicidal ring-Y OK 1971)
f51 $Y^{cF}$ (#9)/y $sc^{S1}$ In49 $sc^8$ (Filicidal ring-Y OK 1971)
f52 $Y^{cF}$ (#9)/$Y^S$.X y In49 v f car.$Y^L$ (Filicidal ring-Y OK 1971)
f53 $Y^{cF}$ (#13)/y $sc^{S1}$ In49 $sc^8$ (Filicidal ring-Y OK 1971)
f54 $Y^{cF}$ (#14 su-$f^+$)/y $sc^{S1}$ In49 $sc^8$ (Filicidal ring-Y OK 1969)
f55 $Y^{cF}$ (#20)/y $sc^{S1}$ In49 $sc^8$ (Filicidal ring-Y OK 1971)
f56 ($y^+$ ac)$^{53}$.Y/y v f $bb^+$.$Y^L$
f57 $Y^{bb}$ Su-Var 5/$w^{m4}$
f58 $Y^{bb}$/v
f59 $Y^{bb-}$/w sn bb & y v f.=
f60 $Y^{bb-}$/$w^{m4w}$
f61 $Y^{bb-}$/y $sn^5$ oc ptg.$Y^S$ ♂ & y $sc^4$ $bb^-$ B In49 $sn^{X2}$ v $sc^{S1}$/1J1 $sc^{J1(+)}$ pn w rb cm $ct^6$ oc ptg $ras^2$ v dy $g^2$ f od car sw
f62 $Y^{bb-}$/$y^2$ eq
f63 $Y^{Su-V}$ (extra)/In(1)$w^{m4w}$
f64 $Y^{Su-Var}$/$w^{m4}$ & y w bb.=
f65 Y:$bw^+$/$X^+$ ; cn bw
f66 Y:$bw^+$ ; net bw crs (iso, 1968)
f67 Y:$bw^+$/y v ; bw
f68 ("MYR") $Y^c$:$bw^+$/$X^+$ ; bw (ring-Y OK 1971)
f69 ("MYR") $Y^c$:$bw^+$/$X^{c2}$ y f ; bw (rings OK 1971)
f70 Y $mal^+$ #2/$y^2$ v mal
f71 Y.$w^+$/y $w^a$
f72 1J1$^+$.Y/$1^{59b1}$ y w/y $sc^{S1}$ B InS
f73 1J1$^+$.Y/$1^{259}$ Y (extra Y in ♀)
f74 1J1$^+$.Y/$1^{259}$ y/y $sc^{S1}$ B In49 v
f75 1J1$^+$.Y/1J1 $sc^{J1}$ (extra Y in ♀)($F1^+$)
f76 1J1$^+$.Y/1J1 $sc^{J1}$ oc ptg $B^{M1}$ & y f:=
f77 1J1$^+$.Y/1J1 $sc^{J1}$ oc ptg $B^{M1}$ & y $sc^{S1}$ B In49 $sn^{X2}$ v $sc^8$ 1-1/1J1 $sc^{J1}$ oc ptg $B^{M1}$
f78 1J1$^+$.Y/1J1 $sc^{J1}$ oc ptg $B^{M1}$ & y $sc^{S1}$ B In49 $sn^{X2}$ v $sc^8$ 1-2/1J1 $sc^{J1}$ oc ptg $B^{M1}$
f79 1J1$^+$.Y/1J1 $sc^{J1}$ y In49 v $B^{M1}$/y $sc^{S1}$ car os f $g^2$ dy v $ras^2$ $sn^3$ $ct^6$ cm rb ec w pn 1 $sc^8$
f80 1J1$^+$.Y/1J1 $sc^{J1(+)}$ & y f:=

f81 ("Maxy") $\underline{1}J1^{+}.Y/\underline{1}J1\ sc^{J1(+)}$ In49 ptg oc $B^{M1}/y\ sc^{S1}$ car os f $g^2$ dy v $ras^2$ $sn^3$ $ct^6$ cm rb ec w pn $\underline{1}\ sc^8$

f82 $\underline{1}J1^{+}.Y/\underline{1}J1\ sc^{J1(+)}$ In49 v f $B.Y^L$ & y f:=

f83 ("Maxy-v") $\underline{1}J1^{+};Y/\underline{1}J1\ sc^{J1(+)}$ In49 v ptg oc $F1^{+}$ $B^{M1}/y\ sc^{S1}$ car os f $g^2$ dy v $ras^2$ $sn^3$ $ct^6$ cm rb ec w pn $\underline{1}\ sc^8$

f84 $sc^8.Y/ac^3$

f85 $sc^8.Y/$In49 ptg oc $B^{M1}$ & y f:=

f86 $sc^8.Y/$In(1H)59(3-4), y $\underline{1}^{59}$ & y w.=

f87 $sc^8.Y/$In(1H)132(4E), y $\underline{1}^{132}$ & y w.=

f88 $sc^8.Y/$In(1H)227(1F), y $\underline{1}^{227}$ & y w.=

f89 $sc^8.Y/$In(1H)231(1C-D), y $\underline{1}^{231}$ & y w.=

f90 $sc^8.Y/$In$(X^{c2})w^{vC}$ f & y f:=

f91 $sc^8.Y/\underline{1}^{46}$ $y^{16}$ (extra Y in ♀)

f92 $sc^8.Y/\underline{1}J1\ \underline{1}^{46}$ y/y $sc^{S1}$ B In49 v

f93 $sc^8.Y/\underline{1}J1\ sc^{J1}$ & y f:=

f94 $sc^8.Y/\underline{1}J1\ sc^{J1}$ In49 dy v $ras^2$ $B^{M1}$ & y f:=

f95 $sc^8.Y/\underline{1}J1\ sc^{J1}$ In49 v $B^{M1}$ & y f:=

f96 $sc^8.Y/\underline{1}J1\ sc^{J1}$ oc ptg $B^{M1}$ & y $sc^{S1}$ B In49 $sn^{X2}$ v $sc^8$ $\underline{1}$-1/1J1 $sc^{J1}$ oc ptg $B^{M1}$

f97 $sc^8.Y/\underline{1}J1\ sc^{J1(+)}$ In49 v ptg oc $f^{S1}$ $B^{M1}/y\ sc^{S1}$ car os f $g^2$ dy v $ras^2$ $sn^3$ $ct^6$ cm rb ec w pn $\underline{1}\ sc^8$

f98 $sc^8.Y/\underline{1}$(y ac)$^-$ B In49 $sn^{X2}$ $sc^8$ & y f:= (from X-rayed oogonia $24)

f99 ("Max-Tu") $sc^8$ $Y/\underline{1}$(y ac)$^-$ Tu B In49 $sn^{X2}$ $sc^8/y$ ac pn w rb cm $sn^3$ $ct^6$ oc $ras^2$ v dy $g^2$ f $os^o$ car sw

f100 $sc^8.Y/$oc ptg & y f:= (iso, 1956)

f101 $sc^8.Y/$sc w $B.Y^S$ & y f:= ; Cy,In/S Sp $ab^2$ ltd

f102 $sc^8.Y/sc^{V1-}$ v y f:= ; $sc^{19i}$/Cy $lt^3$

f103 $sc^8.Y/X^{c2}$ $t^2$ & y f:=

f104 $sc^8.Y/X^{c2}$, y B & y f:= (ring OK 1968)

f105 $sc^8.Y/X^{c2}$, y B & y f:= ; $bw^D$ (ring OK 1967)

f106 $sc^8.Y/X^{c2}$, y f & y f:= (ring OK 1968)

f107 $sc^8.Y/X^{c2}$, y v & y f:= (ring OK 1970)

f108 $sc^8.Y/y$ ac sc $B.Dp(sc^{S1}\ ac^{+}\ y^{+})$ & y $N^W$ f:= (N with $w^{+}$)

f109 $sc^8.Y/y$ ac sc oc ptg & y f:=

f110 $sc^8.Y/y\ ac^{-53}$ sc & y f:=

f111 $sc^8.Y/y$ B & y f:=

f112 $sc^8.Y/y\ bb\underline{1}^{3a}$ & y w.=

f113 $sc^8.Y/y\ bb\underline{1}^{74}$ & y w.=

f114 $sc^8.Y/y\ bb\underline{1}^{456}$ & $y^2$ su-$w^a$ $w^a$ bb.=

f115 ("$sc^8$-Maxy") $sc^8.Y/y$ Hw In49 v ptg oc $f^{S1}$ $B^{M1}/y\ sc^{S1}$ car os f $g^2$ dy v $ras^2$ $sn^3$ $ct^6$ cm rb ec w pn $\underline{1}\ sc^8$

f116 $sc^8.Y/y$ In49 B & f:=

f117 ("Multi-♂") $sc^8.Y/y$ In49 B & y f:= ; $bw^D$

f118 $sc^8.Y/y$ In49 $B^{M1}$

f119 $sc^8.Y/y$ In49 v f

f120 $sc^8.Y/y$ In49 v f $B.Y^L$ & y f:=

f121 $sc^8.Y/y$ In49 v F1 g & y f:=

f122 $sc^8.Y/y$ In49 v F1 g & y In49 v F1 g/pn,Inh

f123 $sc^8.Y/y$ sc w In49 v g f

f124 $sc^8.Y/y$ sc w In49 v g f & y f:=

f125 $sc^8.Y/y\ sc^4$ B f InS & y f:=

f126 $sc^8.Y/y\ sc^4$ B f InS $w^a$ & y f:=

f127 $sc^8.Y/y\ sc^4$ B InS & y f:=

f128 $sc^8.Y/y\ sc^4$ B InS $w^r$ $sc^8$ & y f:=

f129 $sc^8.Y/y\ sc^4$ f InS $w^a$ & y f:=

f130 $sc^8.Y/y\ sc^4$ InS bb$^-$ $sc^{S1}$

f131 $sc^8.Y/y\ sc^4$ w $sc^8$ ($sc^8$.Y in ♂ & ♀)

f132 $sc^8.Y/y\ sc^{S1}$ B In49 $sc^8$ & y f:=

f133 ("Multipare D") $sc^8.Y/y\ sc^{S1}$ B InS/y Hw In49 $m^2$ $g^4$ ; (ci gv1 $ey^R$ $sv^n$)

f134 $sc^8.Y/y\ sc^{S1}$ In49 $sc^8$ ; dp bw ; st $p^P$

f135 $sc^8.Y/y$ sc$^-$ (rein. $sc^{8-4}$) $B.Dp(sc^{S1}\ ac^{+}\ y^{+})$ & y f:=

f136 $sc^8.Y/y$ v & y f:=

f137 $sc^8$.Y/y $w^{m4}$
f138 $sc^8$.Y/y $w^{m4}$ ; $dp^{02}$ bw
f139 $sc^8$.Y/y $w^{m4}$ ; $dp^{v2}$ bw
f140 $sc^8$.Y/$y^2$ $w^a$ $sn^5$ B bb & y $N^M$ f:= (N with w)
f141 $sc^8$.Y/$y^{S1}$ $sc^8$ B f In49 v
f142 $B^{S-}$ $v^+$ $dy^+$.Y $y^+$ #3/y $ct^6$ 1Q54 1a66 dy/FM6, $y^{31d}$ $sc^8$ dm
f143 $B^S$ $v^+$.Y $y^+$ #3/1Q54 v Df259-4/FM6, $y^{31d}$ $sc^8$ dm B
f144 $B^S$ $v^+$.Y $y^+$ #3/1ras v-1 1Q54 v/FM6, $y^{31d}$ $sc^8$ dm B
f145 $B^S$.Y/1f & y v f mal.=
f146 $B^S$.Y/T(3;X)05, D & y f:=
f147 $B^S$.Y/T(3;X)$ry^{35}$ $D^3$ & y f:=
f148 $B^S$.Y/y $sc^4$ Tu w $sc^8$ & y f:=
f149 $B^S$.Y/$y^2$su-$w^a$ $w^a$ & y f:=
f150 $B^S$.Y.$w^+$ $y^+$/y v & y f:=
f151 $y^{31d}$.Y.$B^S$/$Y^S$ y cv v f & y w f:=
f152 $y^{ah}$.Y.$B^S$/y & y w f:=
f153 $sc^8$.Y.$B^S$/$Bx^2$ ; Cy,Ins $cn^2$/Bl
f154 $sc^8$.Y.$B^S$/1J1 $sc^{J1(+)}$ In49 F1 g $B^{M1}$ & y f:=
f155 $sc^8$.Y.$B^S$/1J1 $sc^{J1(+)}$ In49 F1 g $B^{M1}$/"Maxy" y $sc^{S1}$ B os f $g^2$ dy v ras $sn^3$ $ct^6$ cm rb ec w pn 1 $sc^8$
F156 $sc^8$.Y.$B^S$/1J1 $sc^{J1(+)}$ In49 oc $B^{M1}$/ y $sc^{S1}$car os f $g^2$ dy $ras^2$ $sn^3$ $ct^6$ cm rb ec w pn 1 $sc^8$
f157 $sc^8$.Y.$B^S$/1J1 $sc^{J1(+)}$ In49 v F1 g $B^{M1}$ & y f:=
f158 $sc^8$.Y.$B^S$/Oregon-R-$X^+$ & y f:=
f159 $sc^8$.Y.$B^s$/$sc^8$ B In49 w & y f:= ; (ho ed c1/+)
f160 $sc^8$.Y.$B^S$/$sc^8$ In49 w ; (ho) ed c1
f161 $sc^8$.Y.$B^S$/$sc^{S1}$ B In49 $sc^8$ (new) & y f:=
f162 $sc^8$.Y.$B^S$/$sc^{S1}$ InS $w^a$ $sc^4$ & y f:=
f163 $sc^8$.Y.$B^S$/Urbana-S-$X^+$ & y f:=
f164 $sc^8$.Y.$B^S$/y $sc^4$ In49 v w $sc^{S1}$ & y f:=
f165 $sc^8$.Y.$B^S$/y $sc^4$ InS $sc^{S1}$ & y f:=
f166 $sc^8$.Y.$B^S$/y $sc^{S1}$ InS $w^a$ $sc^4$ & y f:=
f167 $sc^8$.Y.$B^S$/y $w^{m4}$ ; (ho) ed c1
f168 $sc^8$.Y.$B^S$/y $w^{m4}$ $ras^2$
f169 $sc^8$.Y.$B^S$/$y^2$ $ct^6$ & y f:=
f170 $sc^8$.Y.$B^S$/$y^2$ $sd^2$ & y f:=
f171 $sc^8$.Y.$B^S$/$y^2$ $w^i$ & y f:=
f172 $sc^8$.Y.$B^S$/$y^2$ $w^i$ $ct^6$ & y f:=
f173 $sc^8$.Y.$B^S$/$y^2$ $w^i$ $ct^6$ f & y f:=
f174 $sc^8$.Y.$B^S$/$y^{3PL}$ (y ac)- $sc^{8R}$
f175 $sc^8$.Y bb/y $sc^4$ B InS $w^a$ $sc^8$ & y f:=

f176 - f189

f176 $sc^{8y}$.Y.$B^S$(ga#3)/y $sc^{S1}$ In49 $sc^8$, f177 (ga#11), f178 (ga#12), f179 (ga#21) f180 (ga#81), f181 (ga#82), f182 (ga#85) f183 (ga#86), f184 (ga#87), f185 (ga#88) f186 (tids#9), 187 (x#16), 188 (sn#4), f189 (xo#7)

f190 - f199

f190 $sc^{8y}$.Y.$B^{S+}$(C#1)/y $sc^{S1}$ In49 $sc^8$ f191 (ga#10), f192 (tids#1), f193 (tids#4), f194 (tids#9A), f195 (tids#10), f196 (x#1), f197 (xo#2), f198 (xo#6), f199 (xo#8)

f200 $sc^8$.Y.$B^{SM}$(tids#41)/y $sc^{S1}$ In49 $sc^8$

f201 - f206

f201 $sc^8$.Y.$B^{S-Var}$(ga#14)/y $sc^{S1}$ In49 $sc^8$ f202 (tids#5), f203 (tids#7), f204 (tids#8), f205 (xo#1), f206 (xo#5)

f207 - f225

f207 $sc^8$.Y.$B^{S+}$(ga#2)/y $sc^{S1}$ In49 $sc^8$, f208 (ga#3), f209 (ga#4), f210 (ga#5), f211 (ga#6), f212 (ga#7), f213 (ga#8), f214 (ga#9), f215 (tids#2), f216 (tids #3), f217 (x#1), f218 (x#8), f219 (xh#1), f220 (x1#7), f221 (xn#1), f222 (xn#2), f223 (xn#3), f224 (xo#3), f225 (xo#4)
f226 $sc^8$.Y.$bw^+$/$ac^3$ ; bw
f227 $sc^8$.Y.$bw^+$/$ac^3$ : cn bw
f228 $sc^8$.Y.$bw^+$ ; dp bw
f229 $sc^8$.Y.$bw^+$/$sc^8$ B In49 w ; bw
f230 $sc^8$.Y.$w^+$/y $w^a$
f231 $y^+$.Y.$w^+$ $B^S$/y $w^a$ & y f $Bx^O$:=
f232 $y^+$.Y.$w^+$ $B^S$/y $w^a$ & y $N^M$ f:= (N with w)
f233 $y^+$ Y $w^+$ $B^S$ (YII)/y $w^a$
f234 $Y^L$/f.$Y^S$ & sc v f.=
f235 $Y^L$/f.$Y^S$ & $y^w$ $wy^2$ $g^w$ f.=
f236 $Y^L$/$ac^+$$y^+$($sc^8$ EN c.o. $Y^S$B-2)/y w.$Y^S$ & y v f.=
f237 $Y^L$.$bb^+$ $ac^+$ $y^+$($sc^8$ EN c.o. Y T-O)/y w.$Y^S$ & y f:=
f238 $Y^L$.$bb^+$ $sc^{S1}$ $ac^+$ $y^+$($sc^{S1}$ c.o. Y EY80)/y w.$Y^S$ & y v f.=
f239 $y^+$ $Y^L$.(FR-2)/$Y^S$.X InEN B y.$Y^L$ & RA, y. . . .$sc^8$
f240 $Y^{Lc}$/In49 ptg oc $B^{M1}$/$sc^{S1}$ $sn^{5.1}$ w $sc^8$ ♀ & $Y^{Lc}$/y sn oc ptg v.$Y^S$ ♂
f241 $Y^{Lc}$/InEN f.$Y^S$ & y $ct^6$ f.=
f242 $Y^{Lc}$/InEN2 f.$Y^S$
f243 $Y^{Lc}$/InEN2 f ptg oc.$Y^S$ & y $ct^6$ f.=
f244 $Y^{Lc}$/InEN2 f ptg oc w.$Y^S$ & sc v f.=
f245 $Y^{Lc}$/InEN2 f ptg oc $y^+$.$Y^S$ & $Y^{Lc}$/y $ct^6$ f.=
f246 $Y^{Lc}$/InEN2 f ptg oc $y^+$.$Y^S$ & $y^2$ su-$w^a$ $w^a$ bb.=
f247 $y^{Lc}$/InEN2.$Y^S$ & y $ct^6$ f.= (InEN2 from $X^{c2}$ opened)
f248 $Y^{Lc}$/oc ptg.$Y^S$/In49 v F1 g
f249 ("$Y^{Lc}$ snocty") $Y^{Lc}$/oc ptg $Y^S$ ♂ & ($Y^{Lc}$/y $ct^n$ oc ptg car y $ct^1$,In In49 $sn^{X2}$) @ ♀
f250 ("$Y^{Lc}$ snocty;bw") $Y^{Lc}$/oc ptg.$Y^S$ ♂ & ($Y^{Lc}$/y $ct^n$ oc ptg car y $ct^1$,In In49 $sn^{X2}$) @ ♀:bw

f251 $Y^{Lc}$/oc ptg.$Y^S$ & y v f.=;S Sp $ab^2$ ltd/Cy, Ins $cn^2$
f252 $Y^{Lc}$/y In49 v f.$Y^S$ & y sc $t^2$ v f car.=
f253 $Y^{Lc}$/y In49 v Fl g f.$Y^S$ & y $ct^6$ f.=
f254 $Y^{Lc}$/y sn oc ptg v.$Y^S$ ♂ & $Y^{Lc}$/In49 ptg oc $B^{M1}$/$sc^{S1}$ $sn^{5.1}$ w $sc^8$ ♀
f255 $Y^{Lc}$/y sn oc ptg v.$Y^S$ & $Y^{Lc}$/In49 ptg oc $B^{M1}$/y $sc^{S1}$ sl $sn^{X2}$ w $sc^8$
f256 $Y^{Lc}$/y sn oc ptg.$Y^S$ & $Y^{Lc}$/In49 ptg oc $B^{M1}$/$sc^{S1}$ w $sn^5$ $sc^8$
f257 ("new facl", 1959) $Y^{Lc}$/y sn oc ptg.$Y^S$ & $Y^{Lc}$/$y^2$ oc ptg $B^{M1}$/$sc^{S1}$ In49 $sn^{X2}$ $sc^8$
f258 ("jynd") $Y^{Lc}$/y $sn^5$ oc ptg v.$Y^S$ & $Y^{Lc}$/$sc^{J1}$ pn w rb cm $ct^6$ oc $ras^2$ v dy $g^2$ f od car sw/y $sc^{S1}$ B In49 $sn^{X2}$ $sc^8$
f259 $Y^{Lc}$/y $sn^5$ oc ptg v.$Y^S$ & $Y^{Lc}$/$sc^{S1}$ $bb^+$ $sn^{5.1}$ w $sc^8$/In49 ptg oc bb $B^{M1}$
f260 ("jynd v") $Y^{Lc}$/y $sn^5$ oc ptg.$Y^S$ & 1J1 $sc^{J1}$ pn w rb cm $ct^6$ oc $ras^2$ v dy $g^2$ f od car sw/y $sc^{S1}$ B In49 $sn^{X2}$ y sc
f261 $Y^{Lc}$/y v.$Y^S$;$bw^{VA}$.$L^2$ 1
f262 $Y^{Lc}$/y w $sn^5$ oc ptg.$Y^S$ & y v f.=
f263 $Y^{Lc}$/$y^2$ oc ptg sn.$Y^S$ & $y^2$ oc ptg $B^{M1}$/$sc^{S1}$ sn In49 $sc^8$
f264 ("facl") $Y^{Lc}$/$y^2$ oc ptg fu.$Y^S$ & $Y^{Lc}$/$y^2$ oc ptg $B^{M1}$/$sc^{S1}$ fu In49 $sc^8$
f265 $Y^{Lc}$/$y^2$ oc ptg fu.$Y^S$ & $Y^{Lc}$/y $w^a$.=

Sterilizer ("sz") stocks (f266-f285)

f266 ("sz w") $Y^{Lc}$/w.$Y^S$
f267 ("sz +") $Y^{Lc}$/X.$Y^S$
f268 ("sz bw") $Y^{Lc}$/X.$Y^S$;bw
f269 ("sz bw e") $Y^{Lc}$/X.$Y^S$;bw;e
f270 ("sz c") $Y^{Lc}$/X.$Y^S$ & y v f.=;c
f271 ("sz e") $Y^{Lc}$/X.$Y^S$ & y v f.=;e
f272 ("sz lz f") $Y^{Lc}$/$lz^3$ f.$Y^S$ & y v f.=
f273 ("sz lz m f") $Y^{Lc}$/$lz^3$ m f.$Y^S$ & y v f.=
f274 ("sz m f") $Y^{Lc}$/m f.$Y^S$ & y v f.=
f275 ("sz y w") $Y^{Lc}$/y w.$Y^S$ & y $ct^6$ f.=
f276 $sc^{S1}$.$Y^L$/1J1 $sc^{J1}$ $y^s$ In49 v f B.$Y^S$ & y f:=
f277 sc.$Y^L$/1J1 $sc^{J1}$ $y^s$ In49 v f B.$Y^S$/$sc^7$, In oc ptg g, Inh
f278 $sc^{S1}$.$Y^L$/1J1 $sc^{J1}$ $y^s$ In49 v f B.$Y^S$/$sc^8$ 1 B
f279 sc.$Y^L$/1J1 $sc^{J1(+)}$ $y^s$ In49 v f B.$Y^S$/$sc^7$, In oc ptg g, Inh
f280 sc.$Y^L$/oc ptg.$Y^S$ & y f:=;Cy, Ins $cn^2$/S Sp $ab^2$ ltd
f281 sc.$Y^L$/sc w B.$Y^S$ & y f:=;Cy, In/S Sp $ab^2$ ltd
f282 sc.$Y^L$/sc w $BB^L$, In.$Y^S$ & y f:=
f283 sc.$Y^L$/sc w $ct^6$ f.$Y^S$ & y f:=;Cy, In/S Sp $ab^2$ ltd
f284 sc.$Y^L$/y ac sc $ct^6$ v f.$Y^S$
f285 sc.$Y^L$/y $ct^6$ f.$Y^S$/$sc^8$ B InS $w^a$

f286 sc.$Y^L$/y In49 v f.$Y^S$
f287 sc.$Y^L$/y In49 v f.$Y^S$;e
f288 sc.$Y^L$/y sc⁻(rein. $sc^{8-4}$).$Y^S$ & y f:=
f289 sc.$Y^L$/y w v f.$Y^S$;ell
f290 sc.$Y^L$/y.$Y^S$ & y f:=;cn bw;(e)
f291 sc.$Y^L$/$y^2$ v f.$Y^S$ & y w f.=
f292 sc.$Y^L$/$y^2$ $w^a$ $ct^6$ f.$Y^S$ ♂ & $y^2$ $X^+$.$sc^8$ $w^a$ InS B ♀ (tandem X.X giving rings)
f293 $y^3$.$Y^L$/1J1 $sc^{J1(+)}$ In49 v f B.$Y^S$ & f:=
f294 $y^3$.$Y^L$/sc w oc f.$Y^S$ & y f:=
f295 $y^3$.$Y^L$/$sc^{V1-}$ oc $lz^3$.$Y^S$ & y f:=
f296 $y^3$.$Y^L$/$sc^{V1-}$ w.$Y^S$ & y f:=
f297 $y^3$.$Y^L$/y $ct^6$ oc lz.$Y^S$ & y f:=
f298 $y^3$ $Y^L$/$y^2$ oc lz.$Y^S$ ♂ & $sc^{S1}$ bb B In49 $lz^s$ v $sc^{S1}$/y ac pn w rb cm $ct^6$ $sn^3$ oc ras v dy $g^2$ Tu-1 f od car sw ♀ ("plond v")
f299 $y^3$.$Y^L$/$y^2$ oc lz.$Y^S$ ♂ & y ac pn w rb cm $ct^6$ $sn^3$ oc $ras^2$ v dy $g^2$ Tu-1 f od car sw/$sc^{S1}$ B In49 $lz^s$ v $sc^8$ ♀
f300 ("plond") $y^3$.$Y^L$/$y^2$ oc lz.$Y^S$ ♂ & $y^3$.$Y^L$/y ac sc pn w rb cm $ct^6$ $sn^3$ oc ras v m $g^2$ f car/$sc^{S1}$ B In49 $lz^s$ $sc^8$ ♀
f301 $Y^S$/$g^2$ B.$Y^L$ & y f:= (Stern) ($dp^T$)
f302 $Y^S$/In49 v B.$Y^L$ & y f:=
f303 $Y^S$/In49 v f B.$Y^L$ & y f:=
f304 $Y^S$/y $ct^6$ f.$Y^L$ & y f:=
f305 $Y^S$.$Y^S$/$y^+$ $sc^{S1}$ InEN f ptg oc $\overset{?}{y}$.$Y^L$/1J1 $sc^{J1}$ $y^S$ In49 v f B.$Y^L$
f306 $Y^S$.$Y^S$#2/y v f $bb^-$.$Y^L$ & y f:=
f307 Y"/$y^+$ $sc^8$ car f v cv y EN.$Y^L$ & y.=
f308 Y"/y cv v f car.$Y^L$ & y.=

f309 $sc^{V1}.Y^S/\underline{1}J1\ sc^{J1}\ y^S$ In49 v f B.$Y^L$ & y f:=
f310 $sc^{V1}.Y^S/\underline{1}J1\ sc^{J1}\ y^S$ In49 v f B.$Y^L$ & $\underline{1}J1\ sc^{J1}\ y^S$ In49 v f B.$Y^L$/y $sc^{S1}$ InEN ptg oc.$Y^L$
f311 $sc^{V1}.Y^S/\underline{1}J1\ sc^{J1}\ y^S$ In49 v f B.$Y^L$ & $\underline{1}J1\ sc^{J1}\ y^S$ In49 v f B.$Y^L$/y $w^{m4}$ ptg oc
f312 $sc^{V1}.Y^S$/y ac oc ptg.$Y^L$ & y ac oc ptg. $Y^L$/y $sc^{S1}$ B In49 $ct^{ns}$ v $sc^8$
f313 $sc^{V1}.Y^S$/y $ct^6$ f.$Y^L$ & y f:=
f314 $sc^{V1}.Y^S$/y In49 $sn^{X2}$ B.$Y^L$ & y f:=
f315 $sc^{V1}.Y^S$/y In49 v B.$Y^L$ & y f:=
f316 $sc^{V1}.Y^S$/y In49 v f B.$Y^L$ & y f:=
f317 $sc^{V1}.Y^S$/y In49 v f B.$Y^L$/y $w^{m4}$
f318 $sc^{V1}.Y^S$/y In49 v ptg oc f B.$Y^L$ & y f:=
f319 $sc^{V1}.Y^S$/y In49 v.$Y^L$ & y f:=
f320 $sc^{V1}.Y^S$/y $sc^{S1}$ InEN f ptg oc y.$Y^L$/$\underline{1}J1\ sc^{J1}\ y^S$ In49 v f B.$Y^L$
f321 $sc^{V1}.Y^S$/y $sc^{S1}$ InEN ptg oc.$Y^L$ & f:=
f322 $sc^{V1}.Y^S$/y v f $bb^+.Y^L$ & f:=
f323 $sc^{V1}.Y^S$/y w v f $bb^+.Y^L$;$e^{11}$
f324 $sc^{V1}.Y^S$/y w v f $bb^+.Y^L$ & y v f.=;$e^{11}$
f325 v.Y.$y^+$#1/1Q54 $N^{166}$;$bw^D$ & y f:=
f326 $y^+$.Y.$mal^{106}$/ot/FM6, $y^{31d}$ $sc^8$ dm B
f327 T(X;$B^SY^L$)3C #107/FM4, $y^{31d}$ w dm f
f328 " " #111/ " " "
f329 " " #114/ " " "
f330 " " #128/ " " "
f331 " " #140 (homozygous)
f332 " " #164/FM4, $y^{31d}$ w dm f
f333 " 3D #104/$sc^8$.Y & y f:=
f334 " 5D #108/FM4, $y^{31d}$ w dm f
f335 " 6E #149/FM4, w dm f
f336 " 7D #102 & RM, y
f337 " " #156/FM4, $y^{31d}$ w dm f
f338 " 11A #129/ " " "
f339 " 12A + T(1;3)3F;69C #143/FM4, $y^{31d}$ w dm f
f340 " 13A #152 & y f:=
f341 " 13E #142/FM4, $y^{31d}$ w dm f
f342 " 14A #240/ "
f343 " 15A #112/ "
f344 " 15D #125/ "
f345 " 16A #106/ "
f346 " 16E #118 & y f:=
f347 T(X;$B^SY^S$)3F #150/FM4, $y^{31d}$ w dm f
f348 " 6E #131/ "
f349 " 7B #155/ "
f350 " 8F #115/ "
f351 T(X;$B^SY^S$)8F #147/FM4, $y^{31d}$ w dm f
f352 " 11B #145/ "
f353 " 12E + T(1;3)19E;62A #134/FM4, $y^2$ w dm f
f354 T(X;$B^SY^S$)14D + T(1;4)9C:101 #116/FM4, $y^{31d}$ w dm f
f355 T(X;$B^SY^S$)19E #101/FM4, $y^{31d}$ w dm f
f356 " " #133 & y f:=
f357 " " #141/FM4, $y^{31d}$ w dm f
f358 " 19F #103/ "
f359 " " #105 & y f:=
f360 " " #119/FM4, $y^{31d}$ w dm f
f361 " " #132/ "
f362 " " #137/ "
f363 " " #151
f364 " 20A #122
f365 T(X;$y^+Y^L$)3E #10 & y f:=
f366 " 4C #16/FM6, $y^{31d}$ dm B
f367 " 11A #4 & y f:=
f368 " " #20/FM4, w dm f
f369 T(X;$y^+Y^L$)11D + T(X;3)14F;72 #5/FM6, $y^{31d}$ dm B
f370 T(X;$y^+Y^L$)16F #1/FM6, $y^{31d}$ dm B
f371 T(X;$y^+Y^S$)3E #3/FM6, $y^{31d}$ dm B
f372 T(X;$y^+Y^S$)5E, 11F, 19F #2/FM6, $y^{31d}$ dm B (free Y?)
f373 T(X;$y^+Y^S$)11D #6/FM6, $y^{31d}$ dm B
f374 " 19F #14/ "
f375 T(Y;X)1E, y $sc^+$/y sc & y f:=;cn bw
f376 T(Y;X)2E/v car $\underline{1}$ (Stern #64) & y f:=; cn bw
f377 T(X;2;$y^+Y^L$)7B;39 #17/FM6, $y^{31d}$ dm B
f378 T(X;2H?;$B^SY^L$)3C #109/FM4, $y^{31d}$ w dm f
f379 T(X;3;$B^SY^S$)10A;97A #154/FM4, w dm f
f380 T(X;3;$B^SY^L$)15E;74 #144/FM4, w dm f
f381 " 17A;94 #161/ "
f382 T(X;3;$B^SY^S$)19F;85 #127/FM4, $y^{31d}$ w dm f
f383 T(XY$^L$.$Y^S$;4)$B^S$(16A1), $X^D$, $B^S$ $Y^L$.$Y^S$ & y v bb.= (no free Y)
f384 T(Y;3)(II4A$a^3$) $\underline{1}$/ru h D InsCXF ca [T(Y;3) in ♀ and ♂]
f385 T(Y;3)Su-Var 3; Cy/$bw^{VDe3}$; ru h st ca
f386 Tp4:Y (2 Y:4's in both ♂ and ♀) (Transpos. Edmondson)
f387 Tp4:Y/Basc & y.=;ci $ey^R$
f388 Tp4:Y/Cat/Cat ♂ & Cat/M4 ♀ (unselected)
f389 Tp4:Y/$ci^D$
f390 Tp4:Y/$ey^D$ or Cat ♂ & $ey^D$/Cat ♀
f391 Tp4:y/$X^+$ & y f:=

## Attached X.Y chromosomes (f392-f421)

f392 X.$Y^S$(P-8b), $sc^8$ EN, $y^+$ f y @ $Y^S$ & y v f.=
f393 $B^SY^S$X.(FR-1, $3a^R$), $B^SY^S$ y cv v f @/$Y^{Lc}$ & y v f.=
f394 (FR-1,$3a^R$), $y^+Y^S$ X y cv v f @ & y f:=
f395 $y^{31d}Y^S$X(FR-1,$3a^R$), $y^{31d}$ $Y^S$ y cv v f @ & y f:=
f396 $Y^S$X.(P-o), InEN, $Y^S$ y @ & y w.=
f397 $Y^S$X.(FR-1),$Y^S$ y cv v f @ & y f:=
f398 $Y^S$X.(FR-$1^L$, Inp(1)$sc^{V1R}$), $Y^S$ y cv v f @ $y^+/Y^{Lc}$
f399 $Y^S$X.(FR-1), $Y^S$ y m f car @/$Y^{Lc}$ & y v f.=
f400 X.$Y^L$(k-7), $sc^8$ InEN $y^+$ f v cv y @ $Y^L$ & y.=/Y"

## Ring X;Y (of Novitski)(f422-f432)

See page 34

f401 X.$Y^L$(k-7), $sc^8$ InEN(Rein.), $y^+$ y f @ $Y^L$ & y.=
f402 X.$Y^L$(P-8b), $sc^8$ InEN, $y^+$ f y @ $Y^L$/Y" & y.=
f403 X.$Y^L$(c-2), y cv v f car $bb^-$ @ $Y^L$ & RA, (ND-27) v f/Y"
f404 X.$Y^L$(c-2), y w $bb^-$ @ $Y^L$ & y.=/Y"
f405 X.$Y^L$(U-8e), y w @ $Y^L$/Y" & y.=
f406 $Y^S$X.$Y^L$(FR-$1^L$,c-$2^R$), $Y^S$ y $bb^-$ @ $Y^L$ & $y^2$ su-$w^a$ $w^a$ bb.= (no free Y)
f407 $Y^S$X$Y^L$.(FR-$1^L$,174-$13^R$), $Y^S$ y cv v bb $Y^L$ @ & RA, $y^2$ su-$w^a$ $w^a$....M-5/
f408 $Y^S$X.$Y^L$(FR-$1^L$,U-8$d^R$), $Y^S$ y $w^a$ cv v f @ $Y^L$ & $y^2$ su-$w^a$ $w^a$ bb.= (no free Y)
f409 $Y^S$X($Y^L$.)(FR-$1^L$,3-$18^R$), $Y^S$ y($Y^L$ @ $bb^+$)/RA, $y^2$ su-$w^a$ $w^a$....M-5
f410 $Y^S$X($Y^L$.)(FR-$1^L$,118-2$b^R$ #1), $Y^S$ y ($Y^L$ @ $bb^+$) & $y^2$ su-$w^a$ $w^a$ bb.= (no free Y)
f411 $XY^S$.$Y^L$ $y^+$ (Parker), $y^2$ su-$w^a$ $w^a$ $Y^S$ @ $Y^L$ $y^+$ & y v bb.= (no free Y)
f412 $XY^S$.$Y^L$(110-8 Parker), $y^2$ su-$w^a$ $w^a$ $Y^S$ @ $Y^L$ $y^+$ & y v bb.= (no free Y)
f413 $XY^S$.$Y^L$(115-9 Parker), $y^2$ su-$w^a$ $w^a$ $Y^S$ @ $Y^L$ $y^+$ & y v bb.= (no free Y)
f414 $XY^S$.$Y^L$(129-16 Parker), $y^2$ su-$w^a$ $w^a$ $Y^S$ @ $Y^L$ $y^+$ & y v bb.= (no free Y)
f415 X.$Y^L$.$Y^S$(Parker), $y^2$ su-$w^a$ $w^a$ $Y^L$ @ $Y^S$ & y.=
f416 $XY^L$.$Y^S$(2-10T13 Parker), $y^2$ su-$w^a$ $w^a$ $Y^L$ @ $Y^S$ & y.=
f417 $XY^L$.$Y^S$(2-10T15 Parker), $y^2$ su-$w^a$ $w^a$ $Y^L$ @ $Y^S$ & y.=
f418 $XY^L$.$Y^S$(112-17 Parker), $y^2$ su-$w^a$ $w^a$ $Y^L$ @ $Y^S$ & y v bb.= (no free Y)
f419 $XY^L$.$Y^S$(129-11 Parker), $y^2$ su-$w^a$ $w^a$ $Y^L$ @ $Y^S$
f420 $XY^L$.$Y^S$(108-9 Parker), $y^2$ su-$w^a$ $w^a$ $Y^L$ @ $Y^S$ & y v bb.= (no free Y)
f421 $Y^S$X$Y^S$.$Y^L$(FR-$1^L$,115-$9^R$), $Y^S$ y cv $Y^S$ @ $Y^L$ $y^+$ & $y^2$ su-$w^a$ $w^a$ bb.= (no free Y)

## Chromosome 2*

* $S^2$ and/or Cy are to be understood always to be accompanied by InCyL and $cn^2$ by InCyR even where not so designated. When cn is present InCyR is absent. Ins following $S^2$ or Cy after a comma refers to both of these inversions, but InL only to the left-arm one. If either of these inversions is designated in a chromosome without the other, the latter should be understood to be absent.

g1 a px or
g2 a px or If
g3 a px sp
g4 ab
g5 $ab^2/S^2$ Ins(2L+2R)Cy, 1c $cn^2$
g6 $ab^2$/T(Y;2)E
g7 $ab^2$ $cn^4$ $Pm^1$/Cy pr Bl $cn^2$ $L^4$ $sp^2$
g8 $ab^2$ $ix^2$ bw $sp^2$/Cy, $dp^{Th}$ Bl $L^4$ $sp^2$
g9 $ab^2$ InCyR $L^4$ $sp^2$/b InsNS mr
g10 $ab^2$ (ms) ta crs/Cy pr Bl $cn^2$ $L^4$ $sp^2$
* $ab^{51g}$ . . . . . (g24)
* abb . . . . . (g479)
g11 abr/SM5, $al^2$ Cy $lt^v$ $sp^2$
g12 ad
g13 al
g14 al b c sp
g15 al b cn sp (iso)
g16 al dp b bw l(2)ax/SM5, $al^2$ Cy $lt^v$ $sp^2$
g17 al dp b pr
g18 al dp b pr Bl c px sp/SM1, $al^2$ $Cy^{rev}$ $sp^2$
g19 al dp b pr blt bw/SM5, $al^2$ Cy $lt^v$ $sp^2$
g20 ("apl") al dp b pr c px sp
g21 ("twelvepl") al dp b pr cn vg c a px bw mr sp/$S^2$ Cy $lt^3$ $pr^+$ Bl $cn^2$ $L^4$ $sp^2$
g22 al ex ds S ast/SM1, $al^2$ Cy $sp^2$
g23 al S ast ho/SM1, $al^2$ Cy $sp^2$
g24 $al^2$ Cy $ab^{51g}$ pr Bl $cn^2$ $L^4$ $sp^2$/S Sp cn bw sp
g25 $al^2$ Cy pr Bl $cn^2$ InCyR c vg $sp^2$/InsNS px sp
g26 $al^2$ Cy pr Bl $cn^2$ $L^4$ bw $sp^2$/InsNS px sp
g27 $al^2$ Cy,InL $lt^3$/b pr Bl $lt^3$ $cn^2$ InCyR $L^4$ $sp^2$
g28 $al^2$ InCyL E(S) InCyR $sp^2$ (homozygous)
g29 $al^2$ InMis $dp^{txI}$ Cy $cn^2$ $L^4$ $sp^2$/S Sp U, InLR bw
g30 $al^2$ InMis Cy,Ins (but $Cy^+$) b pr $cn^2$ $sp^2$ (l)/$dp^{tx}$ Sp b
g31 $al^2$ InMis $dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ $sp^2$/S Sp U,InLR bw
* $al^2$ . . . . . (g11,g16,g18,etc.)
* alpha-1 . . . . . (= tyr-1)(g65)
g32 Alu
* Alu . . . . . (g281)
g33 an/SM5, $al^2$ Cy $lt^v$ $sp^2$
g34 ang
g35 ant;(ro)
g36 $ap^4$/$dp^{txI}$ Cy, Ins0 pr $cn^2$
g37 $ap^4$/Rvd,In2LR
g38 $ap^4$/SM5, $al^2$ Cy $lt^v$ $sp^2$
g39 $ap^{56f}$
g40 $ap^{56f}$ cn
g41 arch ch1/SM5, $al^2$ Cy $lt^v$ $sp^2$
g42 ast ho
g43 ast ho ed $dp^{ov1}$ c1/Cy Bl L
* $ast^3$ . . . . . (g306)
g44 $ast^4$ dp c1
g45 $ast^4$ dp c1 sp
* $ast^X$ . . . . . (g280)
g46 b
g47 b cn bw
g48 b cn B

g49 b cn $vg^{-B}$/InsNS px sp
g50 b el $rd^{s}$ pr cn
g51 b gp
g52 b Go/Gla, InLR
g53 b If
g54 b j
g55 b l(2)Bld pr c px sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
g56 b lt bw
g57 b lt l cn mi sp/b $bw^{VDel}$
g58 b lt $wx^{wxt}$ bw
g59 b nub pr
g60 b pr
g61 b pr Bl tk/$S^2$ Cy $cn^2$ $L^4$ $sp^2$
g62 b pr c px sp
g63 b pr tk/T(Y;2)G
g64 b sf
g65 b tyr-1
g66 b vg
g67 b $vg^{-B}$/$S^2$ Cy Bl $cn^2$ $L^4$ $sp^2$
* ba . . . . . (g82)
g68 Bl/Cy, $bw^{45a}$ $sp^2$ $or^{45a}$
g69 Bl/esc
g70 Bl/In(2LR)dp
g71 Bl $bw^{VA}$ T(2;3)/Cy,InL $L^2$
g72 Bl $L^2$/Cy, $dp^2$
g73 Bl $stw^{48}$ blt tuf/SM5, $al^2$ Cy $lt^v$ $sp^2$
g74 Bla/SM5, $al^2$ Cy $lt^v$ $sp^2$
g75 blo
g76 blt . . . . . (= $ap^{blt}$)
g77 bran
g78 bri
g79 $bs^2$
* $bs^3$ . . . . . (g321)
g80 bur fs2.1/Cy $cn^2$ $L^4$ $sp^2$
g81 bw (iso 2, 1959)
g82 bw ba
g83 bw sp (iso, 1954)
g84 bw sp $Px^{-2}$/SM1, al Cy $sp^2$
g85 $bw^{2b}$
g86 $bw^4$
* $bw^{45a}$ . . . . . (g68)
g87 $bw^{75}$
g88 $bw^{81}$
g89 $bw^{-5}$/Cy $cn^2$ $L^4$ $sp^2$
g90 $bw^{-5}$/Cy, $dp^2$
g91 $bw^{-5}$ $sp^2$/Xa
g92 $bw^D$
* $bw^{V1}$ . . . (g7,g141,g408,g474)($bw^{V1}$=$Pm^1$)
* $bw^{V32g}$ . . . . . (g409,g410)($bw^{V32g}$=$Pm^2$)
g93 $bw^{V4}$/SM1, $al^2$ Cy $sp^2$
g94 $bw^{V5}$/Cy
g95 $bw^{V5}$/SM5, $al^2$ Cy $lt^v$ $sp^2$
g96 $bw^{VDe2}$/Rev, l
g97 $bw^{VDe2L}$ $Cy^R$/Gla
g98 $bw^{VDe4}$/SM5, $al^2$ Cy $lt^v$ $sp^2$
g99 c
g100 c bw
g101 c px $bw^D$ sp
g102 c px M(2)33/$al^2$ InMis Cy $cn^2$ $sp^2$
g103 c wt px
g104 cg c/U,InLR
g105 ch
g106 chl
g107 chl en/SM5, $al^2$ Cy $lt^v$ $sp^2$
g108 chl l(2)bw $bw^{2b}$ $mr^2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
g109 chy
g110 ck/SM5, $al^2$ Cy $lt^v$ $sp^2$
g111 cl
g112 $cl^2$ px/T(Y;2)E
g113 cn (iso, 1960)
g114 cn (iso, 1962)
g115 cn bw
g116 cn bw (sensitive to S.D. #72)
g117 cn bw Kr/Pm
g118 cn bw sp
g119 cn l(2)crc/SM5, $al^2$ Cy $lt^v$ $sp^2$
g120 cn px,InLR crs/$S^2$ $dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ $sp^2$
g121 cn sp (iso, 1954)
g122 $cn^2$ InCyR cg $sp^2$/InsNS px sp
g123 $cn^3$/T(Y;2)C
g124 $cn^3$ cg $bw^{-5}$ mr/Cy $cn^2$ $L^4$ $sp^2$
g125 $cn^{35k}$
g126 Coi
* cq . . . . . (g441)(= $rk^4$)
g127 cr-u/Cy;($w^e$)
g128 Cy $Stw^{DC}$ $sp^2$/$ds^{33k}$ $Pm^1$ (Lobe?)
g129 Cy (no Ins) ed/$ds^{33k}$ $Pm^1$
g130 db/SM5, $al^2$ Cy $lt^v$ $sp^2$
g131 da/SM1, $al^2$ Cy $sp^2$
g132 Df(2)42 en/$al^2$ Cy $lt^3$ $L^4$ $sp^2$
g133 Df(2)al/Cy, E(S)
g134 Df(s)Px/Df(2)P;Dp(2;3)P/In(3R)Mo, sr;$w^e$
g135 Df(2)$rl^{10a}$ lt cn/Cy
g136 Df(2)$rl^{10b}$ lt cn/$al^2$ Cy $lt^3$ $L^4$ $sp^2$
g137 dke c
g138 $dil^2$ hv bw sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
g139 dp . . . . . (= $dp^{ov}$)
g140 dp b cn sp/$al^2$ Cy pr Bl $cn^2$ $L^4$ $sp^2$
g141 dp b $L^4$ $Pm^1$/IndpT23 b
g142 dp b $bw^{-5}$ mr/$al^2$ InMis Cy $cn^2$ $sp^2$
g143 dp cn bw
g144 dp ho (mutant eye)
g145 dp lt $bw^{amherst}$
g146 dp ltd bw
g147 $dp^{cm2}$ cl
g148 $dp^D$/SM1, $al^2$ Cy $sp^2$
g149 $dp^{l-DG82}$/Cy $Stw^{DC}$ ($L^4$) $sp^2$
g150 $dp^{l-DG83}$/Cy $Stw^{DC}$ ($L^4$) $sp^2$
* $dp^{lv2}$ . . . . . (g90,g262)(=$dp^2$)
g151 $dp^{LM}$/Cy Bl L
* $dp^{lv1}$ . . . . . (g254)

g152 - g164

g152 $dp^{lv-CS38}$/Cy $Stw^{DC}$ ($L^4$) $sp^2$
g153 $dp^{lv-CS43}$..., g154 $dp^{lv-CS51}$...,
g155 $dp^{lv-CS58}$..., g156 $dp^{lv-CS67}$...,
g157 $dp^{lv-CS78}$..., g158 $dp^{lv-CS105}$...,
g159 $dp^{lv-DG7}$..., g160 $dp^{lv-DG12}$...,
g161 $dp^{lv-DG13}$..., g162 $dp^{lv-DG14}$...,
g163 $dp^{lv-DG16}$..., g164 $dp^{lv-DG23}$...

g165 - g176

g165 $dp^{lv\text{-}DG24I}/Cy\ Stw^{DC}\ (L^4)\ sp^2$
g166 $dp^{lv\text{-}DG38}$..., g167 $dp^{lv\text{-}DG39}$...,
g168 $dp^{lv\text{-}DG49}$..., g169 $dp^{lv\text{-}DG50}$...,
g170 $dp^{lv\text{-}DG51}$..., g171 $dp^{lv\text{-}DG57}$...,
g172 $dp^{lv\text{-}DG65}$..., g173 $dp^{lv\text{-}DG66}$...,
g174 $dp^{lv\text{-}DG67}$..., g175 $dp^{lv\text{-}DG72}$...,
g176 $dp^{lv\text{-}DG81}$...

g177 $dp^{o}$
g178 $dp^{o2}$
g179 $dp^{o2}\ dp^{lvl}$ b/Cy Bl L
g180 $dp^{o3}$ ta sp/Cy pr Bl $cn^2\ L^4\ sp^2$ (iso 2)
g181 $dp^{obm}$/Cy Bl L
g182 $dp^{obm}\ dp^{v2}$
g183 $dp^{oA2}$
g184 $dp^{oA4}$

g185 - g196

g185 $dp^{ol\text{-}CS27}/Cy\ Stw^{DC}\ (L^4)\ sp^2$
g186 $dp^{ol\text{-}CS48}$..., g187 $dp^{ol\text{-}CS69}$...,
g188 $dp^{ol\text{-}CS88}$..., g189 $dp^{ol\text{-}CS110}$...,
g190 $dp^{ol\text{-}DG20}$..., g191 $dp^{ol\text{-}DG25}$...,
g192 $dp^{ol\text{-}DG36}$..., g193 $dp^{ol\text{-}DG52}$...,
g194 $dp^{ol\text{-}DG53I}$..., g195 $dp^{ol\text{-}DG54}$...,
g196 $dp^{ol\text{-}DG74}$...

g197 $dp^{ols}\ dp^{v2}$/Cy Bl L
g198 $dp^{olvl}$/Cy Bl L

g199 - g219

g199 $dp^{olv\text{-}CS3}/Cy\ Stw^{DC}\ (L^4)\ sp^2$
g200 $dp^{olv\text{-}CS8}$..., g201 $dp^{olv\text{-}CS28}$...,
g202 $dp^{olv\text{-}CS39}$..., g203 $dp^{olv\text{-}CS91}$...,
g204 $dp^{olv\text{-}CS93}$..., g205 $dp^{olv\text{-}CS100}$...,
g206 $dp^{olv\text{-}CS112}$..., g207 $dp^{olv\text{-}DG}$...,
g208 $dp^{olv\text{-}DG10}$..., g209 $dp^{olv\text{-}DG27}$...,
g210 $dp^{olv\text{-}DG32}$..., g211 $dp^{olv\text{-}DG34}$...,
g212 $dp^{olv\text{-}DG44}$..., g213 $dp^{olv\text{-}DG45}$...,
g214 $dp^{olv\text{-}DG46}$..., g215 $dp^{olv\text{-}DG59}$...,
g216 $dp^{olv\text{-}DG62}$..., g217 $dp^{olv\text{-}DG63}$...,
g218 $dp^{olv\text{-}DG75}$..., g219 $dp^{olv\text{-}HC3}$...,

g220 $dp^{olv\text{-}HC39}$/SM1, $al^2$ Cy $sp^2$
g221 $dp^{olv\text{-}HC59}/Cy\ Stw^{DC}\ (L^4)\ sp^2$
g222 $dp^{olv\text{-}HC69}/Cy\ Stw^{DC}\ (L^4)\ sp^2$
g223 $dp^{olv\text{-}HC234}/Cy\ Stw^{DC}\ (L^4)\ sp^2$
g224 $dp^{olvbm}$/Cy Bl L
g225 $dp^{olvr}$, $In/ds^{33k}\ Pm^1$
g226 $dp^{olXAS\text{-}1}$/SM1, $al^2$ Cy $sp^2$
* $dp^{ov}$ . . . . . (g139,140,141,etc.)
g227 $dp^{ov\text{-}DG37}/Cy\ Stw^{DC}\ (L^4)\ sp^2$
g228 $dp^{ov(1)\text{-}DG55}/Cy\ Stw^{DC}\ (L^4)\ sp^2$
g229 $dp^{ov(1)\text{-}DG2}/Cy\ Stw^{DC}\ (L^4)\ sp^2$
g230 $dp^{ovh}$ Sp cn bw/$dp^{lv}$ Cy cn bw
g231 $dp^{ovN}$
g232 $dp^{ovN}$ or
g233 $dp^{Rf}$/SM5, $al^2$ Cy $lt^v\ sp^2$
g234 $dp^{T}\ ab^2$ pr Bl mT23 InNSR mr/$al^2$ Cy $cn^2\ L^4\ sp^2$
g235 $dp^{T}$ Sp $ab^2$ cn bw sp/$S^2$ ls Cy pr $cn^2$ bw sp
g236 $dp^{T}$ Sp cn bw sp/$S^2$ $(ls^+)$Cy,InL cn bw sp
g237 $dp^{T}$ Sp cn InNSR mr/$S^2$ ls Cy pr Bl $cn^2\ L^4$ bw $sp^2$
g238 $dp^{tx}$ b/Cy,Ins $cn^2$
g239 $dp^{tx}$ b/SM5, $al^2$ Cy $lt^v\ sp^2$
g240 $dp^{tx}$ Sp $ab^2/S^2$ ls Cy,InCyL
g241 $dp^{tx}$ Sp $b/S^2$ ls Cy,InCyL
g242 $dp^{tx}$ Sp $b/S^2$ ls Cy,InCyL b
g243 $dp^{tx}$ Sp $cn/S^2$ Cy,InCyL cn
g244 $dp^{tx}$ Sp cn bw/$S^2$ Cy,InCyL cn bw
g245 $dp^{tx}$ Sp $cn^2/S^2$ Cy $cn^2$ (homoz. InCyR)
g246 $dp^{txI}$ Cy Bl $cn^2\ L^4\ sp^2$/InsNS px sp
g247 $dp^{txI}$ Cy,Ins04 pr $cn^2$/InsNS px sp
g248 $dp^{txI}$ Cy,Ins06 pr $cn^2$/InsNS px sp
g249 $dp^{v}$;vo-3 (2;3)
g250 $dp^{v1}$/SM5, $al^2$ Cy $lt^v\ sp^2$
g251 $dp^{v2}$
g252 $dp^{vm}$/Cy
g253 $dp^{vM}$/SM5, $al^2$ Cy $lt^v\ sp^2$
g254 $dp^{w1}/dp^{lv1}$ Cy,InCyL
g255 $dp^{w2}$/ed dp cl
g256 $dp^{w3}$
* $dp^{w18}$ . . . . . (g309)
g257 Dp(2;f)1/sp $Pin^2/Px^4$
g258 Dp(2;2)S, (S ast)(S $ast^4$) net dp cl/Cy,E(S)
g259 ds dp
g260 ds ft $dp^{v2}$ l(2)M b pr/SM5, $al^2$ Cy $lt^v\ sp^2$
g261 ds S G b pr/Cy, $al^2\ lt^3\ L^4\ sp^2$
g262 $ds^{38k}$/Cy,InCyL, $dp^2$ b pr
g263 $ds^{w}$/In(2L)Cy-t, Su(S) $sp^2$ pr
g264 dsr
g265 dw-24F cl/SM5, $al^2$ Cy $lt^v\ sp^2$
* E(S) . . . . . (g28)
g266 ed dp cl
g267 ed dp cl bw
g268 ed dp cl lt
g269 ed dp cl lt bw
g270 ed $dp^{o2}\ dp^{olv101}$ cl/SM1, $al^2$ Cy $sp^2$
g271 ed $dp^{lvl}$ cl/Cy $Bl^+\ L^+$
g272 ed $dp^{ols}$ cl/Cy Bl L
g273 ed $dp^{ovl}$ cl
g274 ed $dp^{ovl}$ cl lt bw
g275 ed $dp^{o2}$ cl
g276 ed $dp^{v2}$ cl
g277 ed $Su^2$-dx
g278 el
* en . . . . . (g107)
g279 ex
g280 ex ds S ast/SM1, $al^2$ Cy $sp^2$
g281 fes Alu lt/SM5, $al^2$ Cy $lt^v\ sp^2$
g282 fes $dp^{tx}$ Sp/$al^2$ Cy $lt^3$ $(L^4)\ sp^2$
g283 fes $dp^{tx}$ Sp ms bw mr crs/$al^2$ InMis Cy $cn^2\ L^4\ sp^2$
g284 fes IndpT23 b sp/$al^2$ Cy $cn^3\ L^4\ sp^2$

g285 fes ms b cn sp/al$^2$ InMis dp$^{txI}$ Cy pr Bl cn$^2$ lt$^3$ L$^4$ sp$^2$ (iso 1960)
g286 fes ms cn sp/net dp$^{txI}$ Cy b pr Bl lt$^3$ cn$^2$ L$^4$ sp$^2$ (iso, 1957)
g287 fes pr rnT23/al$^2$ Cy b cn$^2$ L$^4$ sp$^2$
g288 fj l(2)Su(H)/SM5, al$^2$ Cy lt$^v$ sp$^2$
g289 fj wt/SM5, al$^2$ Cy lt$^v$ sp$^2$
g290 fr/Cy, sp$^2$
g291 fr$^2$ wt/SM5, al$^2$ Cy lt$^v$ sp$^2$
g292 Frd/Cy, sp$^2$
* fs2.1 . . . . . (g80)
g293 ft
* G . . . . . (g261)
g294 G$^{rv}$/SM5, al$^2$ Cy lt$^v$ sp$^2$
g295 Gla,InLR/S$^2$ Cy cn$^2$ bw sp
g296 Gla,InLR cn/al$^2$ Cy pr Bl cn$^2$ c vg sp$^2$
g297 Gla,InLR-Cy,Ins0 dp$^{txI}$/S bw$^D$
* Go . . . . . (g52)
* gp . . . . . (g51)
* gt-4 . . . . . (g496)
* Hia . . . . . (g526,527)
g298 hk
g299 hk pr
g300 ho
g301 ho dp$^{ovl}$
g302 hv/SM5, al$^2$ Cy lt$^v$ sp$^2$
g303 Hx
g304 hy/SM5, al$^2$ Cy lt$^v$ sp$^2$
* If . . . . . (g2)
g305 In(2)41.59, bw$^D$/Cy
g306 In(2L)Cy, al$^2$ ast$^3$ b pr (Cy not present)
g307 In(2L)Cy b pr cn$^2$ In(2R)Cy
g308 In(2L)Cy cn$^2$ In(2R)Cy (l?)/dp$^{tx}$ Sp b
g309 In(2L)dp$^{wl8}$/SM, al$^2$ Cy sp$^2$
g310 In(2L)t, esc c sp/SM5, al$^2$ Cy lt$^v$ sp$^2$
g311 In(2L)t, lt l L$^4$ sp$^2$/ds$^{33k}$ Pm$^1$
g312 In(2LR) lt$^{m37}$/SM5, al$^2$ Cy lt$^v$ sp$^2$
g313 In(2LR)lt$^{mr}$/SM3, al$^2$ Cy lt$^v$ sp$^2$
g314 In(2LR)102, ds$^W$ sp$^2$/SM1, al$^2$ Cy sp$^2$
g315 InNSL InNSR/al$^2$ Cy,InL lt$^3$ L$^2$
* ix$^2$ . . . . . (g8)
g316 j
g317 j-l ab$^2$ InNSR mr/S$^2$ dp$^{txI}$ Cy cn$^2$ bw sp
g318 J Bl/In(2L)NS
g319 J$^{34e}$
g320 kn
* Kr . . . . . (g117)
g321 l(2)a bs$^3$, In(2L)t/ds$^{33k}$ Pm$^1$
* l(2)ax . . . . . (g16)
g322 l(2)ay b c sp/SM5, al$^2$ Cy lt$^v$ sp$^2$
* l(2)Bld . . . . . (g55)
* l(2)bw . . . . . (g108)
* l(2)C . . . . . (g476)
* l(2)crc . . . . . (g119)
g323 l(2)gl cn bw/al$^2$ Cy lt$^3$ L$^4$ sp$^2$
g324 l(2)gl cn bw/SM5, al$^2$ Cy lt$^v$ sp$^2$
g325 l(2)H L$^2$/SM5, al$^2$ Cy lt$^v$ sp$^2$
* l(2)M . . . . . (g260)
g326 l(2)mat/SM5, al$^2$ Cy lt$^v$ sp$^2$
g327 l(2)me/SM1, al$^2$ Cy sp$^2$
* l(2)R . . . . . (g488)
* l(2)Su(H) . . . . . (g288)
g328 l(2)39a px slt sp/SM5, al$^2$ Cy lt$^v$ sp$^2$
g329 L
g330 L$^2$
g331 L$^4$
g332 L$^5$
g333 L$^G$
g334 L$^r$
g335 L$^{si}$
g336 ll$^2$
g337 lm/S$^2$, Cy dp$^2$ En-S
g338 ls dp$^T$/al$^2$ Cy cn$^2$ L$^4$ sp$^2$
g339 lt/T(Y;2)A
g340 lt bw
g341 lt bw$^{Amherst}$
g342 lt std/SM1, al$^2$ Cy sp$^2$
g343 lt stw$^3$
g344 lt$^3$ Dp(2;2)41 L$^4$ sp$^2$ In(2R)Cy/ds$^{33k}$ Pm$^1$
* lt$^{mr}$ . . . . . (g312)
* lt$^{m3}$ . . . . . (g313)
* lt$^v$ . . . . . (g11,16,19)
g345 ltd
g346 ltd bw
g347 lw
g348 lys rc;(ss)
g349 M(2)33a/al$^2$ InMis Cy cn$^2$ sp$^2$
g350 M(2)33a/Pm$^2$
g351 M(2)173/SM5, al$^2$ Cy lt$^v$ sp$^2$
g352 M(2)B/In(2L)t, l(2)B
g353 M(2)B/SM1, al$^2$ Cy sp$^2$
g354 M(2)B/SM5, al$^2$ Cy lt$^v$ sp$^2$
g355 M(2)l$^2$/ds$^{33k}$ Pm$^1$
g356 M(2)l$^2$/SM1, al$^2$ Cy sp$^2$
g357 M(2)l$^2$ sp/al$^2$ InMis Cy cn$^2$ sp$^2$
g358 M(2)S2/Cy, E(S)
g359 M(2)S3/SM1, al$^2$ Cy sp$^2$
g360 M(2)S3; Dp(2;2)a, Cy, E(2)
g361 M(2)S4/SM1, al$^2$ Cy sp$^2$
g362 M(2)S5/al$^2$ Cy lt$^v$ sp$^2$
g363 M(2)S6/SM5, al$^2$ Cy lt$^v$ sp$^2$
g364 M(2)S6/Cy L$^4$ sp$^2$
g365 M(2)S6/Cy pr
g366 M(2)S7/SM5, al$^2$ Cy lt$^v$ sp$^2$
g367 M(2)S8/Cy al$^2$ lt$^3$ Dp(2;2)41 L$^4$ sp$^2$
g368 M(2)S8/SM1, al$^2$ Cy sp$^2$
g369 M(2)S9/SM5, al$^2$ Cy lt$^v$ sp$^2$
g370 M(2)S10/Cy cn$^2$ L$^4$ sp$^2$
g371 M(2)S10/Ins(2L+2R)Cy, Cy pr Dp(2;2)41$^2$
g372 M(2)S10/SM1, al$^2$ Cy sp$^2$
g373 M(2)S11/Cy, gw$^{v34}$
g374 M(2)S11/SM5, al$^2$ Cy lt$^v$ sp$^2$
g375 M(2)z/SM5, al$^2$ Cy lt$^v$ sp$^2$
g376 M(2)z Sk b/Cy(2L)dp$^2$ b pr
* mi . . . . . (g408)
g377 mn/Cy cn$^2$ L$^4$ sp$^2$
g378 mr bs$^2$/Cy sp$^2$
g379 mr bs$^2$/ds$^{33k}$ Pm$^1$
g380 mr$^2$/Bld, In(2R)Cy
g381 ms b cn sp/dp$^{txI}$ Cy pr Bl lt$^3$ cn$^2$ L$^4$ sp$^2$ (iso, 1957)

g382 ms bw/Cy pr Bl $cn^2$ $L^4$ $sp^2$ (iso, 1955)
g383 ms cn bw/$dp^{txI}$ Cy pr Bl $lt^3$ $cn^2$ $L^4$ $sp^2$
g384 ms cn mr/Cy $cn^2$ $L^4$ $sp^2$
g385 ms cn sp/$dp^{txI}$ Cy pr Bl $lt^3$ $cn^2$ $L^4$ $sp^2$
g386 msf/SM5, $al^2$ Cy $lt^v$ $sp^2$
g387 net
g388 net al ex ds S ast shv ho rub/SM1, $al^2$ Cy $sp^2$
g389 net b cn crs/$dp^{txI}$ Cy pr Bl $lt^3$ $cn^2$ $L^4$ $sp^2$ (iso, 1955)
g390 net bw crs/$dp^{txI}$ Cy pr Bl $lt^3$ $cn^2$ $L^4$ $sp^2$ (iso, 1955)
g391 net bw mr crs/$al^2$ InMis $dp^{txI}$ Cy Bl $cn^2$ $L^4$ $sp^2$ (iso, 1963)
g392 net bw sp
g393 net dp
g394 net dp b pr cn/$dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ $sp^2$
g395 net ed $Su^2$-dx
g396 net ta $vg^{S2}$ sp/$dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ sp
* nub . . . . . (g59)
g397 $nub^2$
g398 $nw^2$/Cy-RNS
* or . . . . . (g2)
g399 $or^{45a}$ $sp^2$
g400 $or^{49h}$
g401 pd
g402 pd $ll^2$ sp
g403 Pfd/SM5, $al^2$ Cy $lt^v$ $sp^2$
g404 pi/SM5, $al^2$ Cy $lt^v$ $sp^2$
g405 pi l(2)301/SM5, $al^2$ Cy $lt^v$ $sp^2$
g406 Pin
* $Pin^2$ . . . . . (g257)
g407 pk cn
g408 $Pm^1$/T(Y;2)G
g409 $Pm^2$/mi
g410 $Pm^2$/mi $sp^2$
g411 $po_{vg}^2$
g412 $po^2$
g413 pr
g414 pr cn/T(Y;2)C
g415 pr cn ix/SM5, $al^2$ Cy $lt^v$ $sp^2$
g416 $pr^{bw}$
g417 pu
g418 $Pu^2$/SM1, $al^2$ Cy $sp^2$
g419 puf
g420 pw-c/SM5, $al^2$ Cy $lt^v$ $sp^2$
g421 px
g422 px bl (old Berlin stock of Goldschmidt) . . . . . (bl=bs?)
g423 px bw mr $Px^{-2}/al^2$ InMis Cy $cn^2$ $sp^2$
g424 px bw mr sp/$ds^{33k}$ $Pm^1$
g425 px bw ($mr^+$) $Px^{-2}/al^2$ InMis Cy $cn^2$ $sp^2$
g426 px bw sp/T(Y;s)J
g427 px slt sp
g428 $Px^{-2}$/Cy $cn^2$ $L^4$ $sp^2$
g429 $Px^{-2}$, bw sp/SM1, $al^2$ Cy $sp^2$
* $Px^4$ . . . . . (g257)
g430 pys
g431 Q
g432 Qn(2;2)S, (+ast)5, al ho/$S^2$ Cy E(S)
g433 rc pr
* $rc^2$ . . . . . (g495)
g434 rd/SM5, $al^2$ Cy $lt^v$ $sp^2$
* $rd^s$ . . . . . (g50)
g435 rdo
g436 rdo (iso, 1963)
g437 rdo hk pr
g438 $rdo^2$ pr
g439 rh
g440 rk cn bw (iso, 1969)
g441 $rk^4$ . . . . . (= cq)
* $rk^5$ . . . . . (g508)
g442 $rk^{crm}$
g443 rl
* $rl^{10a}$ . . . . . (g135)
* $rl^{10b}$ . . . . . (g136)
g444 rnT23/Cy Bl $cn^2$ $L^4$ $sp^2$
g445 rub
g446 Ruf/$ds^{33k}$ $Pm^1$
* Rvd,In2LR . . . . . (g37)
g447 S/Cy, E(S)
g448 S $dp^T/al^2$ Cy $cn^2$ $L^4$ $sp^2$
g449 S fes Alu lt/$al^2$ Cy $cn^2$ $L^4$ $sp^2$
g450 S fes Sp b/Cy b $lt^3$ $cn^2$ $L^4$ $sp^2$
g451 S fes Sp ms cn bw mr crs/$dp^{txI}$ Cy,InsO pr $cn^2$
g452 S fes Sp ms cn crs/$al^2$ InMis $dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ $sp^2$ (iso, 1957)
g453 S fes Sp ms ta cn mr crs/$al^2$ InMis $dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ $sp^2$
g454 S fes Sp ms ta cn mr crs/$dp^{txI}$ Cy,InsO pr $cn^2$
g455 S Sp $ab^2$ $ap^4$ InNSR px sp/$al^2$ Cy Bl $cn^2$ $L^4$ $sp^2$
g456 S Sp Bl $bw^D$/Cy $cn^2$ lc
g457 S Sp Bl $L^2$ $Px^-$/$dp^{txI}$ Cy,InsO pr $cn^2$
g458 S Sp Bl $L^{rm}$ $bw^D$/$dp^{txI}$ Cy,InsO pr $cn^2$
g459 S Sp Bl Pfd $bw^D$/$dp^{txI}$ Cy,InsO pr $cn^2$
g460 S Sp cn bw/$dp^{txI}$ Cy cn bw
g461 S Sp crs/$al^2$ Cy pr Bl $L^4$ $sp^2$
g462 S Sp InNSR mr/$dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ $sp^2$
g463 S Sp (ls?) cn/$dp^{txI}$ Cy cn
g464 S Sp ($ls^+$?) cn bw sp/$dp^{txI}$ Cy,InL cn bw sp
g465 S Sp ms ta cn crs/$al^2$ InMis $dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ $sp^2$
g466 S Sp ms ta cn crs/$dp^{txI}$ Cy,InsO pr $cn^2$
g467 S Sp pr Bl rnT23 InNSR mr/$dp^{txI}$ Cy pr $cn^2$
g468 S Sp pr $cn^2$ InCyR/$dp^{txI}$ Cy pr $cn^2$
g469 S Sp rdo hk pr cn/$dp^{txI}$ Cy,InsO pr $cn^2$
g470 S Sp rnT23/$dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ $sp^2$
g471 $S^2$ $ab^{51b}$ InCyL/dp b $L^4$ $Pm^1$
g472 $S^2$ Cy $lt^3$ $pr^+$ Bl $cn^2$ $L^4$ $sp^2$/InNSL InNSR px sp
g473 $S^2$ $dp^{txI}$ InCyL/ls Sp b
g474 $S^r$/$ds^{33k}$ $Pm^1$
* $S^X$ . . . . . (g280)
g475 sca
g476 sca l(2)C/SM5, $al^2$ Cy $lt^v$ $sp^2$
g477 $sca^{65131}$

* sf . . . . . (g64)
g478 $sf^2$
g479 shr $bw^{2h}$ abb sp/SM5, $al^2$ Cy $lt^V$ $sp^2$
g480 shv
g481 shv ho
* Sk . . . . . (g376)
* slt . . . . . (g328)
g482 sm px/SM5, $al^2$ Cy $lt^V$ $sp^2$
g483 sm px pd/SM5, $al^2$ Cy $lt^V$ $sp^2$
g484 so
g485 $so^2$ b cn
* sp . . . . . (g3,14,15)
g486 $sp^2$ $bs^2$
g487 $spd^{fg}$
g488 Sp/In(2L)t, l(2)R
g489 Sp/SM5, $al^2$ Cy $lt^V$ $sp^2$
g490 Sp J/In(2L)Cy-t, Su(S) $dp^2$ pr
g491 Sp J $L^2$ Pin/SM5, $al^2$ Cy $lt^V$ $sp^2$
g492 Sp lys d/SM1, $al^2$ Cy $sp^2$
g493 Sp lys rc J/SM1, $al^2$ Cy $sp^2$
g494 Sp ms cn mr crs/Cy pr Bl $cn^2$ $L^4$ $sp^2$
g495 Sp $rc^2$/Cy Bl L
g496 spd gt-r/SM5, $al^2$ Cy $lt^V$ $sp^2$
g497 sple
g498 spt
g499 std/SM5, $al^2$ Cy $lt^V$ $sp^2$
g500 stw
g501 $stw^2$
g502 $stw^3$
g503 $stw^3$/T(Y;2)B
g504 $stw^5$
g505 $stw^{48}$ blt tuf
g506 Su(Pm)/Cy cn vg Pm
* Su(S) . . . . . (g263)
g507 T(Y;2)B/b
g508 ta cn $rk^5$ bw/$al^2$ Cy pr Bl $cn^2$ $L^4$ $sp^2$
g509 Tft/SM1, $al^2$ Cy $sp^2$
* tk . . . . . (g63)
g510 tkd/SM5, $al^2$ Cy $lt^V$ $sp^2$
g511 tkv
g512 toothed (cut-like in 2R)
g513 tri $vg^{No2}$/SM5, $al^2$ Cy $lt^V$ $sp^2$
g514 tuf ltd
* U,InLR . . . . . (g29,31,104)
g515 Uf
g516 vg (iso 2,3)
g517 vg bw
g518 $vg^{-B}$/SM5, $al^2$ Cy $lt^V$ $sp^2$
g519 $vg^{-B}$ bw crs/$dp^{txI}$ Cy b pr Bl $L^4$ ($sp^2$?)
g520 $vg^{-C}$/Rvd,In2LR
g521 $vg^{-C}$/SM5, $al^2$ Cy $lt^V$ $sp^2$
g522 $vg^{-D}$/SM5, $al^2$ Cy $lt^V$ $sp^2$
g523 $vg^{-D}$ $sp^2$/Cy cn $L^4$ $sp^2$
g524 $vg^{ni}$
* $vg^{No2}$ . . . . . (g513)
g525 $vg^{np}$
g526 $vg^{nw}$ Hia/SM5, $al^2$ Cy $lt^V$ $sp^2$
g527 $vg^{nw}$ Hia/T(2;3)$S^M$ Cy
g528 $vg^U$/Roi bw sp or
g529 vst/SM5, $al^2$ Cy $lt^V$ $sp^2$
g530 whd
g531 wt

Chromosome 3
(containing genes of 2 in a few cases)

h1 a(3)26
* a-3 . . . . . (h1)
h2 aa h
h3 abd
* $Adh^S$ . . . . . (h231)
h4 $Antp^B$/TM1, Me ri sbd1
* $Antp^R$ . . . . . (h206)
h5 app
h6 $as^{hg}$
h7 bar-3(Ives)
h8 $Bd^G$/In(3R)C, l(3)a
h9 bf/In(3R)C, Sb e l(3)e
* bod . . . . . (h146)
h10 bp/TM1, Me ri sbd1
h11 bul
h12 bv
* bx . . . . . (h44)
h13 $bx^3$ Cbx Ubx bxd pbx/T(2;3)$ap^{Xa}$
h14 $bx^{34e}$
* $bx^D$=Ubx . . . . . (h13,241)
* bxd . . . . . (h13,200)
* $bxd^{107}$ . . . . . (h232)
* by . . . . . (h168)
h15 $c_3G$ (Pasadena)
* $c_3G$ . . . . . (h170,173,210)
h16 ca
h17 ca bv
h18 ca K-pn
h19 $ca^2$
* $ca^2$ . . . . . (h39)
h20 $ca^{572}$ jIIIa3/TM1, Me ri sbd1
h21 $ca^{nd}$/M(3)1 $Ubx^{130}$ $e^s$
h22 $ca^{nd}$/TM3, Sb Ser
h23 Cbx
* Cbx . . . . . (h13)
h24 cd
h25 cmp ca/In(3R)C, e
h26 Cor/ru h D InsCXF
h27 cp
h28 cp in ri $p^p$
h29 cu
h30 cu $kar^2$
h31 cur
h32 cv-c
h33 cv-c $sbd^2$
h34 cv-d
* Cyd . . . . . (h50)
h35 D/Gl
h36 D InsCXF/Tri
h37 D tra ca/InLP Dfd InRP ca
h38 $D^3$ H/In(3L+3R)P
h39 $D^3$ Sb $ca^2$/In(3L+3R)P
h40 det
h41 Df(3R)ry/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$
h42 Df(3)$ry^k$/$Ubx^{130}$
h43 Df(3)$sbd^{105}$/T(2;3)$ap^{Xa}$

h44 Df(3)sbd$^{105}$, p$^{p}$ sbd$^{105}$ bx sr e$^{s}$/LVM
h45 Dfd/In(3LR)Cx
h46 Dfd$^{r}$
h47 Dl H e$^{s}$ cd/In(3R)spr, spr
h48 Dl$^{3}$/In(3R)C, e
h49 Dl$^{5}$/In(3R)C, l(3)a
h50 Dl$^{14}$/In(3R)Cyd, Cyd
h51 Dl$^{x}$/In(3L+3R)P
* dn . . . . . (h182)
h52 Dp(3:4)ry$^{+}$/st ry$^{2}$ e$^{s}$/st ry$^{2}$ e$^{s}$
* Dr . . . . . (h137,138)
* Dr$^{Mio}$ . . . . . (h125)
* Dr$^{We}$ . . . . . (h256,257)
h53 drb
h54 dwh/InLP Dfd InRP ca
h55 e
h56 e p$^{i}$/ru h D InsCXF e
h57 e tx
* e$^{2}$ . . . . . (h151)
h58 e$^{4}$ wo ro
* e$^{4}$ . . . . . (h66)
h59 e$^{11}$
h60 e$^{s}$
h61 eg/In(3LR)Cx
h62 eg$^{2}$/In(3LR)Cx
h63 eyg
h64 fz
h65 gl
h66 gl$^{2}$ e$^{4}$
h67 gl$^{3}$
h68 gl$^{60j9}$
h69 Gl bx$^{D}$/InsLVM
h70 Gl Sb H/In(3L+3R)P
* gm . . . . . (h141)
h71 gs
h72 h
h73 h ri
h74 h ri ca (iso, 1953)
h75 h ri e$^{s}$ (iso, 1957)
h76 h$^{2}$
h77 H/In(eR)hp, hp
h78 H Pr/In(3R)C, e
h79 H$^{2}$/T(2;3)ap$^{Xa}$
h80 H$^{3}$/In(3R)C, Sb e l(3)e
* Hn$^{r}$ . . . . . (h91)
h81 Hn$^{r3}$ sr
* Hu . . . . . (h88)
h82 in
* in . . . . . (h28)
* In(3)a . . . . . (h149)
* In(3)b . . . . . (h160)
* In(3)c . . . . . (h150)
h83 In(3L)p$^{mot-36e}$/R
h84 In(3L)P, In(3R)P18, Me Ubx e$^{4}$/l(3)tr Sb
h85 InLP Dfd InRP ca/ru h D InsCXF ca
h86 In(3LR)4833.10, bx$^{34e}$/TM1, Me ri sbd$^{1}$
h87 In(3R)Dl$^{B}$, st Dl$^{B}$/In(3R)P$^{W}$, st l(3)W ca
h88 In(3R)Hu, Hu Sb$^{Spi}$/In(3L+3R)P
h89 In(3R)Mo, sr/T(2;3)ap$^{Xa}$, ca
* In(3R)P$^{F1A}$ (homozygous) . . . (a9)
h90 jv
h91 jv Hn$^{r}$ h
h92 jvl
* k . . . . . (h185,200)
* K-pn . . . . . (h18)
h93 kar$^{2}$
* kar$^{2}$ . . . . . (h30)
h94 kar$^{2}$ ca
h95 kar$^{2}$ Sb/ru h D InsCXF ca
h96 ki
* l(3)e . . . . . (h9, etc.)
* l(3)a . . . . . (h8)
h97 l(3)ac e$^{s}$ M(3)w/LVM
h98 l(3)36d10/In(3LR)Cx, D
h99 l(3)tr Sb/In(3LR)Ubx$^{130}$, Ubx$^{130}$ e$^{s}$
h100 l(3)tr Ubx/TM1, Me ri sbd$^{1}$
* l(3)W . . . . . (h87)
h101 ld
* lxd . . . . . (h168)
h102 Ly/D$^{3}$
h103 Ly Sb/LVM
h104 M(3)1/In(3R)C, l(3)e
h105 M(3)36e/In(3R)C, l(3)a
h106 M(3)40130/InLP Dfd InRP ca
h107 M(3)B/In(3R)C, e l(3)e
h108 M(3)B$^{2}$/In(3R)C, Sb e l(3)e
h109 M(3)h$^{S37}$/In(3L)P, Me
h110 M(3)h$^{y}$/In(3L)P, Me
h111 M(3)S31/T(2;3)Me
h112 M(3)S32/T(2;3)Me
h113 M(3)S34/T(2;3)Me
h114 M(3)S36/T(2;3)Me
* M(3)S37 . . . . . [=M(3)h$^{S37}$]
h115 M(3)w/In(3R)C, e l(3)e
* M(3)x . . . . . (h167
* M(3)y . . . . . [=M(3)h$^{y}$]
h116 ma
h117 ma fl
h118 ma ry/TM1, Me ri sbd$^{1}$
h119 mah
h120 Mas/Me,Ins ♂ & Me,Ins/B$^{S3i}$ ♀
h121 Mc/T(2;3)ap$^{Xa}$
h122 Me,InL bx$^{D}$/ru h D InsCXF Sb
h123 Me,InL InRC e l3e/ru h D InsCXF Sb e$^{s}$
h124 Me,InL Sb/ru h D InsCXF
* Me,Ins . . . . . .(=TM1, Me ri sbd$^{1}$)
h125 Mio . . . . . (=Dr$^{Mio}$)
h126 NX/T(2;3)ap$^{Xa}$
h127 obt
h128 p
h129 p$^{p}$
h130 p$^{p}$ bx sr e$^{s}$
h131 p$^{p}$ cu
h132 p$^{p}$ Ki
h133 pb/In(3LR)Cx
h134 pbx/T(2;3)ap$^{Xa}$
h135 Pc/TM1, Me ri sbd$^{1}$
h136 Pr/In(3R)C, e
h137 Pr Dr/TM3, y$^{+}$ ac$^{+}$ ri p$^{p}$ sep bx$^{34e}$ e$^{s}$
h138 Pr$^{K}$ Dr/In(3L+R)P (Krivshenko)
h139 Pt/T(2;3)ap$^{Xa}$, ca
h140 pyd

h141 R Ly/In(3L)P, gm
h142 ra
h143 red (Malpighians)
h144 red (Malpighians) e
h145 ri
h146 ri bod $e^s$/Me, In(3R)C, Sb e 1(3)e
h147 ri e
h148 ri $p^p$
h149 ri $p^p$ Ina (/ru h D InsCXF ca)
h150 ri $p^p$ Inc 1/ru h D InsCXF ca
h151 ri sbd $e^2$
h152 ro
h153 ro Bd ca/In(3R), 1(3)a
h154 $rs^2$
h155 rsd
* $rt^2$ . . . . . (h183)
h156 ru
h157 ru h
h158 ru h $e^s$
h159 ru h ri
h160 ru h ri $p^p$ Inb
h161 ru h th ss bx
h162 ru h th st cp cu sr $e^s$ Pr ca/ru h D InsCXF ca
h163 ("threepl") ru h st $p^p$ ss $e^s$
h164 ("rucuca") ru h th st cu sr $e^s$ ca
h165 ("ruPrica") ru h th st cu sr $e^s$ Pr ca/T(2;3)Me
h166 ("rupes") ru h th st $p^p$ cu sr $e^s$
h167 ru h th st $p^p$ H $e^s$ ro/C(3)x, M(3)x $e^x$ C(3)x=In(3L+3R)P
h168 ru lxd by
h169 ru $p^p$ e Pr ca/TM1, Me ri sbd1
h170 ru st $c_3G$ $e^s$ (iso 3)(b sp)
h171 ru st $c_3G$ sr $e^s$
h172 ru tra p/ru h D InsCXF e
h173 rug jv se by
h174 ry
h175 $ry^2$
h176 Sb H/In(3R)C, cd
h177 Sb Ubx/T(2;3)$ap^{Xa}$
h178 $Sb^{Spi}$/In(3LR)Cx
* sbd . . . . . (h216)
h179 $sbd^2$ $bx^3$
* $sbd^{105}$ . . . . . (h43,44)
* abd1 . . . . . (h4,86,etc.)
h180 se
h181 se h
h182 se Ly dn/LVM
h183 se $rt^2$ th/Me,InL
h184 se ss
h185 se ss k $e^s$ ro
h186 $se^{51j}$
* sed = $Hn^{r3}$ . . . . . (h81)
h187 ("separated arms of 3" Dubinin) T3L.4L; 4R.3R/1 InLP Dfd InRP 1
h188 sep,InLR ri $p^p$
h189 sep,InLR ri $p^p$ Sb/Me,InL Dfd InRC e 13e
h190 sep,InLR ri $p^p$ Sb/Me,InL InRC e 13e
h191 Ser/In(3R)C, e 1(3)e
h192 $sf^2$ cd
h193 $sf^2$ $kar^2$
h194 snb
h195 sr
h196 sr gl
h197 ss
h198 ss bx
h199 ss bx $Su^2$-ss
h200 ss bxd k $e^s$/T(2;3)$ap^{Xa}$
h201 ss ca (iso, 1953)
h202 ss e tx
h203 ss $e^s$ (iso, 1953)
h204 ss tx
h205 $ss^a$
h206 $ss^A$,In3/Sb $bx^D$ . . . . . ($ss^A$=$Antp^R$)
h207 $ss^{a-40a}$
h208 $ss^a$-B
h209 st
h210 st $c_3G$ ca/TM1, Me ri sbd1 ($sp^2$)
h211 st in ri $p^p$
h212 st Ki $p^p$
h213 st $ry_2$
h214 st $ry^2$
h215 st $ry^2$ sr $e^s$
h216 st sbd $e^s$ ro ca
h217 st sr $H^2$ ca/In(3R)$P^W$, st 1(3)W ca
h218 st W cu ry ss
h219 $st^{54i}$ ri $p^p$
h220 $st^{sp}$
h221 su-ve ru ve bv (h? th?)
h222 su-ve ru ve h th
* $Su^2$-ss . . . . . (h199)
h223 $Su^2$-Hw bx bxd/TM1, Me ri sbd1 ($sp^2$)
h224 th
h225 th st cp
h226 th st pb $p^p$/In(3LR)Cx
h227 th st pb $p^p$ kar $su^2$-Hw jvl ss bx sr gl/TM1, Me ri sbd1
* T(2;3)$ap^{Xa}$ . . . . . (h13,79,etc)
h228 TM1, Me ri sbd1/$D^3$ st ri InRC e 1(3)e
h229 TM1, Me ri sbd1/ru h D InsCXF ca
h230 TM1, Me ri sbd1 e/InLP Dfd InRP
h231 TM3 Sb/W $Adh^S$
h232 Tp(3)$bxd^{107}$, bx $bxd^{107}$ sr $e^s$/$bx^{34e}$ Mc
h233 Tp(3R)Vno/$H^2$
h234 tra/T(2;3)Me
* Tri . . . . . (h36)
h235 tt wo
h236 Tub (=Tubby pupa)
h237 tx
h238 tx bar-3 (Ives)
h239 Ubx $e^4$/InLP Dfd InRP ca . . (Ubx=$bx^D$)
h240 $Ubx^{101}$/Sb
h241 $Ubx^{130}$/T(2;3)$ap^{Xa}$
h242 ve
h243 ve bv (iso, 1957)
h244 ve ca (iso, 1953)
h245 ve ca (iso, 1961)
h246 ve h th
h247 ve h th gl
h248 ve h th ss bx
h249 ve R/In(3L)P, gm
h250 ve R $D^3$ st $bx^D$ ($e^4$?) Pr ca/InLP Dfd InRP ca

h251 ve R $D^3$ $Sb^{Spi}$ $Bd^G$/In(3L+3R)P
h252 ve st (iso, 1964)
h253 ve st sbd
* Vno . . . . . (h233)
h254 W
h255 W Sb/InsCX
h256 We/Me,InL InRC e 1(3)e . (We=$Dr^{We}$)
h257 We/TM1, Me ri $sbd^1$
h258 wk/InLP Dfd InRP ca
h259 wo
h260 Xa/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$
. . . . . [Xa=T(2;3)$ap^{Xa}$]

Chromosome 4

i1 ar/$ey^D$
i2 bt
i3 bt $ey^R$ $sv^n$
i4 $bt^D$/$ci^D$ . . . . . [$bt^D$=1(4)$2^{btD}$]
i5 $Ce^2$/$spa^{Cat}$
i6 ci $ey^R$
i7 ci $ey^R$ $sv^n$
i8 ci gvl bt
i9 ci gvl $ey^R$ $sv^n$
i10 ci gvl $spa^{Cat}$/$ci^D$
i11 $ci^{361}$
i12 $ci^D$/4-simulans
* $ci^D$ . . . . . (i20,21,etc.)
i13 $ci^W$
i14 Df(4)3/$ci^D$
i15 Df(4)G/$ci^D$
i16 Df(4)$M^{62f}$/$ey^D$
i17 $ey$
i18 $ey^2$
i19 $ey^4$
i20 $ey^D$/$ci^D$
i21 ("cid") $ey^D$/$ci^D$ $spa^{pol}$
* $ey^R$ . . . . . (i3,6,ect.)
i22 gvl
i23 gvl $ey^R$
i24 gvl $ey^R$ $sv^n$
i25 gy
i26 1(4)1a/$ci^D$ . . . . . [=1(4)27]
i27 1(4)2/$ci^D$
i28 1(4)$2^b$/$ci^D$ $spa^{pol}$
* 1t(4)$2^{btD}$ . . . . . (=$bt^D$)
i29 1(4)$2^f$/$ci^D$ (Hochman). . . [=1(4)Pt-2]
i30 1(4)4c/$ci^D$ (Hochman). . . [=1(4)PT-3]
i31 1(4)5/$ci^D$
i32 1(4)6b/$ci^D$ (Hochman). . . [=1(4)PT-1]
i33 1(4)10/$ci^D$ . . . . . [=1(4)33]
i34 1(4)13/$ey^D$
i35 1(4)$14^b$/$ci^D$ (Hochman) . . [=1(4)ST-2]
i36 1(4)15/$ci^D$ . . . . . [=1(4)ST-4]
i37 1(4)$15^a$/$ci^D$ . . . . . [=1(4)SLC-1]
i38 1(4)21/$ci^D$ (Hochman). . . [=1(4)ST-1]
i39 1(4)22/$ci^D$ (Hochman). . . [=1(4)AM-1]
i40 1(4)25/$ci^D$ (Hochman). . . [=1(4)ST-3]
* 1(4)27 . . . . . [=1(4)1a/$ci^D$]
* 1(4)33 . . . . . [=1(4)10/$ci^D$]
* 1(4)AM-1 . . . . . [=1(4)22/$ci^D$]
* 1(4)PT-1 . . . . . [=1(4)6b/$ci^D$]
* 1(4)PT-2 . . . . . [=1(4)$2^f$/$ci^D$]
* 1(4)PT-3 . . . . . [=1(4)4c/$ci^D$]
* 1(4)SLC-1 . . . . . [=1(4)15 /$ci^D$]
* 1(4)ST-1 . . . . . [=1(4)21/$ci^D$]
* 1(4)ST-2 . . . . . [=1(4)$14^b$/$ci^D$]
* 1(4)ST-3 . . . . . [=1(4)25/$ci^D$]
* 1(4)ST-4 . . . . . [=1(4)15/$ci^D$]
i41 M(4)$ey^D$
i42 M(4)$^{57g}$/$ci^D$ $spa^{pol}$
i43 M(4)$^{63a}$/$ci^D$
i44 Mal;(pr)
i45 spa
i46 $spa^{Cat}$/$ci^D$
i47 $spa^{pol}$
* $spa^{pol}$ . . . . . (i21)
i48 $spa^{pol65}$ (Y contains bb)
i49 $sv^{35a}$
i50 $sv^{de}$/$ey^D$
i51 $sv^n$
i52 Tp4:Y(Edmondson)/4-sim & 4 sim

Multiple Chromosomes

X,2 (j1-79)

j1 $ac^3$;1t $stw^3$
j2 Bld, $Tx^2$ InCyR/ClB
j3 $bx^3$ $dx^{st}$;ed $Su^2$-dx
j4 Bx/Hw;CyL/Pm
j5 car;$ap^{56f}$ cn
j6 car;ltd
j7 cm;cl
j8 cm:dp bw
j9 $dx^{st}$;Su-dx
j10 $en^x$-S;S/Cy
j11 $f^{56e}$ & y f:=;cn bw
j12 gt v;b
j13 gt v;vg
j14 In(1)$w^{m4}$;Cy/T(Y;2)G
j15 $lz^D$/In49 $m^2$ $g^4$;Cy/Pm
j16 $os^s$;tet
j17 pn;$ap^{56f}$ cn
j18 ras;ltd
j19 rb;ca
j20 rb;ltd
j21 sc cho;$ap^{56f}$ cn
j22 $sc^8$ f In49 v;$bw^{VA}$/$L^2$ 1 (iso Y,X,2)
j23 $sn^3$;ed dp cl
j24 T(X;2)459, y $1^{459}$/FM6, $y^{31d}$ $sc^8$ dm B
j25 T(X;2)1t/Cy (carries eq., Possibly $su^3$-s)
j26 T(X;2H)25(20), y $1^{25}$/FM6
j27 T(X;2LH)150(16-17), y $1^{150}$/FM6
j28 T(X;2LH)219(10A), y $1^{219}$/FM6
j29 T(X;2RH)75(20), y $1^{75}$/FM6
j30 T(X;2RH)135(18-19), y $1^{135}$/FM6
j31 v;bw
j32 v;$bw^D$
j33 $w^a$;$ap^{56f}$ cn
j34 $w^{e2}$;$ap^{56f}$ cn

j35 $w^{i}$ $f^{3}$ $bb^{N}$;$ap^{56f}$ cn
j36 $w^{m4}$;En-Var 5/Cy
j37 " " 7/Cy
j38 " " 8/Cy
j39 " " $8^{46g}$/Cy
j40 ("vicn") $y^{+}$ $Y^{S}$.X InEN y.$Y^{L}$ $y^{+}$ & $y^{2}$ su-$w^{a}$ $w^{a}$ bb.=;$dp^{T}$ Sp cn/$S^{2}$ ($1s^{+}$) Cy,InL cn bw sp (no free Y)
j41 y;ed $dp^{ov7}$ cl
j42 y;S Sp cn/$dp^{tx}$ Cy cn
j43 (y ac)- $y^{3P}$ $sc^{8}$;lt $stw^{3}$
144 y ac;ltd
j45 y ac;$sc^{19i}$/$S^{2}$ Cy
j46 y f:=;al b cn sp
j47 " ;$bw^{VA}$.$L^{2}$ 1
j48 " ;Cy,Ins $cn^{2}$/Gla,InLR
j49 " ;dp
j50 " ;dp bw
j51 " ;$dp^{tx}$ Sp cn bw/$S^{2}$ Cy cn bw
j52 " ;ed dp cl
j53 " ;ed dp cl lt bw
j54 " ;net bw sp
j55 " ;pr
j56 " ;S Sp Bl $L^{rm}$ $bw^{D}$/$dp^{txI}$ Cy,InsO pr $cn^{2}$
j57 " ;$S^{2}$ Cy $cn^{2}$/$dp^{txI}$ Sp pr $cn^{2}$
j58 " ;vg-B/SM5, $al^{2}$ Cy $lt^{v}$ $sp^{2}$
j59 y Hw In49 m g/y $sc^{S1}$ B Ins;net bw sp
j60 y $sc^{4}$ $sc^{8}$;Cy $bw^{V34k}$/ds rl (Y's extra)
j61 ("scute twelvep1") y $sc^{5}$;al dp $sc^{19i}$ b pr cn vg c a px bw mr sp/$al^{2}$ Cy pr Bl $cn^{2}$ $L^{4}$ $sp^{2}$
j62 y $sc^{S1}$ B In49 $ct^{ns}$ $sc^{8}$;dp
j63 y $sc^{S1}$ B In49 $sc^{8}$;dp
j64 y $sc^{S1}$ In49 v $sc^{8}$;$dp^{h}$ b bw/$dp^{txI}$ Cy pr Bl $cn^{2}$ $L^{4}$ $sp^{2}$
j65 y $t^{2}$ v f;Bl $bw^{"VA"}$ T(2;3)/Cy,InL $L^{2}$
j66 y v $f^{x}$:$f^{+ih}$;$bw^{"VA"}$ T(2;3)/$L^{2}$ 1
j67 y w f;dp
j68 y w f.Dp($sc^{S1}$ $y^{+}$);dp
j69 y w In49 f;$dp^{v2}$
j70 y w m f;dp
j71 y w m f & y f:=;dp
j72 y w $sn^{3}$;dp
j73 y w $sn^{3}$ B;dp
j74 y w $sn^{3}$ $f^{36a}$;dp
j75 $y^{2}$ $cho^{2}$;$ap^{56f}$ cn
j76 $y^{2}$ oc ptg $B^{M2}$/y $sc^{4}$ Ins $sc^{S1}$ T(X;2)Cl; Cy/Sp
j77 $y^{2}$ $t^{2}$ bb;cn bw
j78 $y^{44d}$ $w^{m4}$ ;Cy/+ (Y's extra)
j79 $Y^{S}$.X InEN v y.$Y^{L}$;Sp dp Sp cn/$dp^{tx}$ Cy cn (no free Y)

X;3 (80-127)

j80 ct- $t^{2}$ v;$ct^{i}$/TM1, Me ri sbd1
j81 en-bx,In(1);$bw^{3}$ +/+ pbx
j82 In49 ptg oc $ct^{ns}$ & y f:=;ri $p^{p}$
j83 In49 v $ct^{ns}$;ru h D InsCXF/Me,Ins
j84 In49 Fl v g;$ct^{i}$/ru h D InsCXF e
j85 In49 v $ct^{ns}$;ru h $p^{p}$ Pr ca/Me,Ins
j86 ma-l;st $ry^{2}$
j87 sc w $B^{S3}$.$Y^{S}$ & y f:= ($B^{S3}$ Del.-Inser, into 3)
j88 $sc^{S1}$ B In49 $ct^{ns}$ v $sc^{8}$;ri $p^{p}$
j89 $sc^{S1}$ $bb^{+}$ B In49 $ct^{ns}$ v $sc^{8}$;ri $p^{p}$
j90 ("Tam X3 sn") sn T(X;3), red & y f:=
j91 $sn^{3}$;Mw/1 InLP InRP 1
j92 T(X;3) $D^{3}$ Sb & y w f:=
j93 ("Tam X3") T(X;3), red & y f:=
j94 T(X;3)$B^{581}$/InAM
j95 T(X;3)"Del 143" ru $e^{s}$ ca/CXF, ru h ca
j96 T(X;3)05, D & y f:=
j97 T(X;3)$ras^{V}$ & y f:=
j98 T(X;3)sta & y f:=
j99 T(X;3)v, v/FM6, $y^{31d}$ $sc^{8}$ dm B
j100 T(X;3)$w^{vco}$, v f/C1$B^{36d}$
j101 T(X;3H)361(20), y 1(1)361/FM6
j102 " 453(12D), y 1(1)453/FM6
j103 " 463(20), y 1(1)463/FM6
j104 T(X;3LH)163(17A-B), y 1(1)163/FM6
j105 " 455(3C), y 1(1)455/FM6
j106 T(X;3RH)3(3-4), y 1(1)3/FM6
j107 " 129(18B), y 1(1)129/FM6
j108 T(3;X)$ry^{35}$, cu $ry^{35}$ kar/Xa
j109 v;$bw^{VDel}$/SM1, $al^{2}$ Cy $sp^{2}$
j110 w;red
j111 $w^{a}$ & y v f.+;tra/D InsCXF
j112 $w^{m4}$;En-Var 12/Cx,D
j113 $w^{m4}$;En-Var 13/Cx,D
j114 $X^{c2}$ y B & y f:=;$e^{11}$ (ring OK 1971)
j115 $X^{c2}$ y v & y f:=;$e^{11}$
j116 y;mwh
j117 y;red
j118 ("Tam Tester 1") y f:=;$D^{3}$ Sb/InLP Dfd InRP ca
j119 y f:=;$e^{11}$
j120 " ;ru h D InsCXF/InLP Dfd InRP
j121 " ;ru $p^{p}$ $e^{s}$ Pr ca/ru h D InsCXF
j122 " ;su-ve ru ve bv (h? th?)
j123 y Hw In49 m g/y $sc^{S1}$ B InS;ru bv
j124 y w;red (Malpighians)
j125 y w;red e
j126 $y^{2}$ $en^{2}$-bx $w^{bf}$/FMA3, $y^{2}$;$Sb^{r2}$ ss $bx^{34e}$/TM1, Me ri sbd1
j127 $y^{2}$ oc ptg bb $B^{M2}$/1 y $sc^{4}$ InS $sc^{S1}$ $bb^{-}$ T(X;3)-78

X,4 (j128-129)

j128 $w^{a56}$;$spa^{Cat}$/$ci^{D}$
j129 X.Y InEN In49 y;(st/+);ci $ey^{R}$(no free Y)
j130 " " " " ;ci $ey^{R}$ $sv^{n}$ "
j131 " " " " ;gvl $ey^{R}$ "
j132 " " " " ;$spa^{pol}$
j133 " " " " ;$spa^{pol}$(no free Y)
j134 " " " " ;$spa^{pol165}$
j135 " " " " ;$spa^{pol165}$(no free Y)
j136 y;$spa^{pol}$
j137 y f:=;spa

j138 y f:=;spa$^{Cat}$ ci$^{D}$
j139 y sc$^{S1}$ InS sc$^{8}$ & y.=;ci ey$^{R}$

2,3 (j140-j302)

j140 a px or;st
j141 al;ss$^{a}$
j142 al b cn sp/al$^{2}$ Cy Bl cn$^{2}$ L$^{4}$ sp$^{2}$;ru
j143 al dp b Bl c px sp/Cy;D/C(3)X
j144 al T(2;3)101 sp$^{2}$/Cy L$^{4}$ sp$^{2}$
j145 " " 108 c sp$^{2}$/Cy, al$^{2}$ lt$^{3}$ L$^{4}$ sp$^{2}$
j146 " " B sp$^{2}$/Cy, L$^{4}$ sp$^{2}$
j147 al$^{2}$ InMis Cy cn$^{2}$ L$^{4}$ sp$^{2}$/net bw mr crs; ru h D InsCXF ca/ve ca
j148 al$^{2}$ InMis Cy cn$^{2}$ L$^{4}$ sp$^{2}$/net bw mr crs; ru h D InsCXF/ve ca
j149 ap$^{56f}$ cn;ca
j150 " ;gl
j151 " ;ma
j152 " ;p
j153 " ;red
j154 " ;rs$^{2}$
j155 " ;ry
j156 " ;ry$^{2}$
j157 " ;se
j158 " ;snb
j159 " ;st
j160 b;e$^{11}$
j161 b;p$^{p}$
j162 Bl bw$^{D}$;Su-283/T(2;3)ap$^{Xa}$
j163 Bl T(2;3)A;ru h D TA ss e$^{s}$/In(3L+3R)P
j164 bw;e
j165 bw;ru h ri
j166 bw;ru h D$^{3}$ st ri InRC e 13e/TM1, Me ri sbd$^{1}$
j167 bw;ss
j168 bw;st
j169 bw;st (iso, 1970)
j170 bw$^{4}$;st
j171 bw$^{D}$ T(2;3)289/Cy;In(3R)Mo, Sb sr
j172 bw$^{VDe3}$;Ubx bxd/In(3LR)Cx
j173 bw$^{VDe3}$;Ubx bxd/In(3R)C
j174 c;e
j175 cl;cd
j176 cn;ca
j177 cn;ca$^{2}$
j178 cn;se$^{51j}$
j179 cn;st
j180 cn;st (iso, 1968)
j181 cn bw;ri e
j182 cn bw;ru h th ri e$^{s}$
j183 cn crs/al$^{2}$ Cy lt$^{3}$ pr Bl cn$^{2}$ L$^{4}$ sp$^{2}$;e$^{2}$
j184 cn crs/Cy pr Bl cn$^{2}$ L$^{4}$ sp$^{2}$;ve (iso)
j185 cn;ve (iso, 1960)
j186 Cy/ds$^{33k}$ Pm$^{1}$;H/sr Mo,In(3R)
j187 Cy/Pm$^{1}$;ru h D InsCXF ca/InLP Dfd InRP ca
j188 Cy/Pm$^{1}$(dp b);D/Sb
j189 Cy/Pm ;st (iso X,2,3)
j190 dp;cp
j191 dp;cu
j192 dp;e$^{11}$
j193 dp;e$^{11}$ (iso, 1970)
j194 dp;e$^{s}$
j195 dp;red
j196 dp;sbd$^{2}$ bx$^{3}$
j197 dp;tx
j198 dp;tx (iso, 1969)
j199 dp;ve
j200 ("iser 2b") dp b cn c P$^{-}$/al$^{2}$ Cy lt$^{3}$ pr Bl cn$^{2}$ L$^{4}$ sp$^{2}$;ru h D InsCXF Dl H e P$^{1}$
j201 ("Pale e") dp b cn c P$^{-}$/Cy cn$^{2}$;e P$^{1}$/e P$^{i}$
j202 ("Pale H") dp b cn c P$^{-}$/Cy cn$^{2}$;p$^{56}$ Dl H e P$^{i}$/p$^{56}$ In3R 1
j203 dp b cn P$^{-}$/dp$^{txI}$ Cy,InsO pr cn$^{2}$;e P$^{i}$/TM1, Me ri sbd$^{1}$
j204 dp b Pm$^{1}$/Cy sp$^{2}$;Sb/D InsCXF (ru h ca?)
j205 dp b (L$^{4}$) Pm$^{1}$/dp$^{txI}$ Cy,InsO pr cn$^{2}$;ru h D InsCXF ca/TM1, Me ri sbd$^{1}$
j206 dp bw;st p$^{p}$
j207 dp bw;st p$^{p}$ (iso, 1970)
j208 dp cn bw/Cy Bl cn$^{2}$ L$^{4}$ sp$^{2}$;h ri e$^{s}$
j209 dp$^{03}$ cn bw;ru h D$^{3}$ ri In3RC e 13e/Me, Ins ri sbd$^{1}$
j210 dp$^{T}$ Sp cn/S$^{2}$ Cy cn;ri e
j211 dp$^{T}$ Sp cn/S$^{2}$ Cy cn;ru h D$^{3}$ ri InC e 13e/Me,Ins ri sbd$^{1}$
j212 dp$^{T}$ Sp cn bw/S$^{2}$ Cy cn bw;ri e
j213 dp$^{tx}$ Sp cn/Cy cn$^{2}$ sp$^{2}$;Me,InL InC e 1(3)e/InsCXF
j214 dp$^{tx}$ Sp cn/Cy cn$^{2}$ sp;Me,InL InRC e 1(3)e/ru h D InsCXF Sb
j215 dp$^{tx}$ Sp cn/S$^{2}$ Cy cn;ru h D Ins CXF/Me, Ins ri sbd$^{1}$
j216 dp$^{tx}$ Sp cn/S$^{2}$ Cy cn;ru h D InsCXF Sb/Me,InL InC e 13e
j217 dp$^{tx}$ Sp cn/S$^{2}$ Cy cn;sep ri p$^{p}$ Sb/Me, InL InC e 13e
j218 dp$^{tx}$ Sp ms ta cn crs/S$^{2}$ Cy pr Bl cn$^{2}$ L$^{4}$ sp$^{2}$;e$^{s}$
j219 dp$^{tx}$ Sp pr cn$^{2}$/S$^{2}$ Cy cn$^{2}$;Me,InL InC e 13e/ru h CXF Sb
j220 dp$^{tx}$ Sp pr cn$^{2}$/S$^{2}$ Cy cn$^{2}$;Me,InL InC e 1(3)e/ru h D InsCXF Sb e
j221 dp$^{txI}$ Cy,InsO pr cn$^{2}$;Me,Ins ri e Sb$^{1}$/T(2;3)B cn bw InC e 1(3)e
j222 dp$^{txI}$ Cy,InsO pr cn$^{2}$;ru h D InsCXF/T(2;3) B cn bw InC e 1(3)e
j223 dp$^{txI}$ Cy, InsO pr cn$^{2}$/S Sp cn;Me,Ins/e p$^{i}$ Pr ca
j224 dp$^{txI}$ Cy,InsO pr cn$^{2}$/S Sp T(2;3)B ru h Pr ca
j225 dp$^{txI}$ Cy,InsO pr cn$^{2}$ T(2;3) Me,Ins ri Sb$^{1}$/("twelvep1") al dp b pr cn vg c a px bw mr sp;("rucuca") ru h th st cu sr e$^{s}$ ca
j226 dp$^{txI}$ Cy,InsO pr cn$^{2}$ T(2;3) Me,Ins ri Sb$^{1}$/("twelvep1") al dp b pr cn vg c a px bw mr sp;("threep1") ru h st p$^{p}$ ss e$^{s}$

j227 ("TIn") $dp^{txI}$ Cy,InsO pr $cn^2$ T(2;3) Me,Ins ri $Sb\underline{1}$/S Sp cn;ru h $D^3$ st InRC e $\underline{1}3e$ (contains $sc^8$.Y in Y)
j228 $dp^v$;$vo^3$
j229 $dp^{v2}$ cn bw;h ri $e^2$ (iso, 7/57)
j230 ed dp cl;tx
j231 fes ms cn sp/$dp^{txI}$ Cy,InsO pr $cn^2$ h ri $e^s$/Me,Ins ri $Sb\underline{1}$ (iso, 7/57)
j232 Gla,InLR-Cy,InsO $dp^{txI}$/Me,Tp
j233 hk pr;st in ri $p^p$
j234 ho dp;ri $p^p$
j235 ho dp;tx
j236 ("Pale Indp") IndpT23 b $P^-$ .Dl H e $P^i$/dp b $Pm^1$.Sb In3R
j237 InNSL InNSR mr/$al^2$ Cy pr Bl $lt^3$ $cn^2$ $L^4$ $sp^2$;$ri^{53j}$
j238 lt;$rs^2$
j239 lt;se
j240 lt bw;st $p^p$
j241 ltd;ca
j242 ltd;ma
j243 ltd;ry
j244 ltd;se
j245 ltd;st
j246 ltd;$st^{sp}$
j247 M(2)33a/$al^2$ Cy pr Bl $cn^2$ $L^4$ $sp^2$;ru
j248 ms sp/Cy pr Bl $cn^2$ $L^4$ $sp^2$;$ri^M$ (iso)
j249 Ms bw sp/$dp^{txI}$ Cy,InsO pr $cn^2$ bw ♀ & Fs/$dp^{txI}$ Cy,InsO pr $cn^2$ bw ♂
j250 net;ri
j251 net bw mr crs/$dp^{txI}$ Cy,InsO pr $cn^2$;ve bv/Me,Ins ri $Sb\underline{1}$
j252 $P^G$/$dp^{txI}$ Cy,InsO pr $cn^2$
j253 $P^{Gr}$/SM1, $al^2$ Cy $sp^2$
j254 pr;cd
j255 $pr^{bw}$;st
j256 px;ri
j257 px pd;Prd H dp (2;3)F/Prd
j258 ru h D T(2;3)B ss $e^s$/Payne
j259 ru h D T(2;3)C ss $e^s$/Payne
j260 ru h T(2;3)101 $e^4$ ro ca/Payne, Dfd ca
j261 ("STin") S fes Sp ms ta cn mr crs;$D^3$ ri InC e $\underline{1}$(3)e/$dp^{txI}$ Cy,InsO pr $cn^2$ T(2;3) Me,Ins ri $Sb\underline{1}$
j262 S fes Sp T(2;3)01 ms cn mr crs $D^3$ st ri InC e $\underline{1}$(3)e/$al^2$ InMis Cy $dp^{txI}$ Bl $cn^2$ $L^4$ $sp^2$; Me,Ins ri $Sb\underline{1}$
j263 S fes Sp ms b T(2;3)B cn bw mr crs InC e $\underline{1}$(3)e/$al^2$ InMis Cy $dp^{txI}$ $cn^2$ $L^4$ $sp^2$;Me,Ins ri $Sb\underline{1}$
j264 S fes Sp T(2;3)B $D^3$ ri Sb/Cy $cn^2$ $sp^2$;InsCXF
j265 S fes Sp T(2;3)01 ms cn mr crs $D^3$ st ri InC e $\underline{1}3e$/$dp^{txI}$ Cy,InsO pr $cn^2$;Me,Ins ri $Sb\underline{1}$
j266 S Sp/$dp^{txI}$ Cy,InsO pr $cn^2$;Mas/Me,Ins ♂ & Me,Ins/$B^{S3i}$ ♀
j267 S Sp cn/$dp^{txI}$ Cy cn;Me, InL InRC e $\underline{1}3e$/ru h D Sb InsCXF
j268 " " " " ";h ri $e^s$
j269 " " " " ";ru h $D^3$ ri InRC e $\underline{1}3e$/Me,Ins ri $Sb\underline{1}$
j270 " " " " ";ru h $e^{11}$
j271 " " " " , InsO pr $cn^2$;h $D^3$ ri InC e $\underline{1}3e$/Me,Ins ri $Sb\underline{1}$
j272 " " " " , InsO pr $cn^2$;ru h D InsCXF ca/Me,Ins ri $sb\underline{1}$
j273 S Sp cn bw/$dp^{txI}$ Cy cn bw;ru h $D^3$ ri InRC e $\underline{1}3e$/Me,Ins ri $Sb\underline{1}$
j274 S Sp cn bw/$dp^{txI}$ Cy cn bw;h ri $e^s$
j275 ("iser 1") S Sp (crs)/Cy InL $lt^e$;Me,Ins ri $Sb\underline{1}$/$Bd^G$
j276 S Sp ms ta cn crs/$dp^{txI}$ Cy pr Bl $cn^2$ $L^4$ $sp^2$;$e^2$
j277 ("sifter O") S Sp $P^-$ T(2;3), InsCXF/$dp^{txI}$ Cy,InsO pr $cn^2$;Dl H e $P^i$
j278 S Sp T(2;3)B/$dp^{txI}$ Cy,InsO pr $cn^2$;Me,Ins ri $Sb\underline{1}$
j279 S Sp T(2;3)B e $P^i$/$dp^{txI}$ Cy pr $cn^2$;Me,Ins
j280 $S^M$ Cy;C3G Sb Ubx/st C3G ca
j281 $sbd^2$ $bx^3$ pbx/T(2;3)$Ubx^{105}$
j282 stw;e
j283 Su-er tu bw;st er su-tu

j284 T(2;3)109 $p^p$/Payne, Dfd ca
j285 T(2;3)Antp$^{Yu}$/Cy;Sb
j286 T(2;3)At, At/In(2R)Mo$^k$
j287 T(2;3)B cn bw $D^3$ ri/dp$^{txI}$ Cy,InsO pr cn$^2$;Me,ri Ins Sb$^1$
j288 T(2;3)B cn bw InC e l3e/al$^2$ InMis dp$^{tx}$ Cy cn$^2$ $L^4$ sp$^2$;Me,Ins ri Sb$^1$
j289 T(2;3)B cn bw InC e l(3)e/dp$^{txI}$ Cy,InsO pr cn$^2$;ru h D InsCXF
j290 T(2;3)B cn bw InC e l(3)e/Gla;ru h D InsCXF
j291 T(2;3)B cn bw InC e l(3)e/Gla;ru h D InsCXF e
j292 T(2;3)B $D^3$ ri/dp$^{txI}$ Cy,Ins pr cn$^2$;Me, Ins ri Sb$^1$
j293 T(2;3)dp$^{w8}$/SM1, al$^2$ Cy sp$^2$
j294 T(2;3)Dp-s;ho/Cy, E(S) (hom. viable)
j295 ta/Cy Bl cn$^2$ $L^4$ sp$^2$;ru ri (iso)
j296 ta sp/Cy Bl cn$^2$ $L^4$ sp$^2$;jv (iso)
j297 ta sp/Cy Bl cn$^2$ $L^4$ sp$^2$;ru (iso)
j298 vg;bv
j299 vg;bv (iso, 1970)
j300 vg;e$^{11}$
j301 vg;ss$^a$
j302 Xa/l(3)XaR

2,4 (j303-321)

j303 al dp T(2;4)d px sp/Cy,pr;ey$^2$
j304 ap$^{56f}$ cn;spa$^{pol}$
j305 ap$^{56f}$ cn;spa$^{pol65}$
j306 "apl" l/Cy cn$^2$ sp$^2$;IV-sim.ci ey
j307 bw;ci$^D$/IV-sim
j308 ed dp cl lt bw;ci ey$^R$
j309 ed dp cl lt bw;spa$^{pol}$
j310 ed dp cl lt bw;spa$^{pol65}$
j311 ho;ci ey$^R$
j312 ho;sv$^n$
j313 pr;ey$^2$
j314 pr;spa$^{pol}$
j315 pr;spa$^{pol65}$
j316 ri;ci ey$^R$
j317 T(2;4)a/Cy, pr;ey$^2$
j318 T(2;4)d/Cy, pr
j319 T(2;4)ast$^v$/al$^2$ Cy lt$^3$ $L^4$ sp$^2$
j320 T(2;4)b/Cy, pr;ey$^2$
j321 dp;ci

3,4 (j322-346)

j322 bv;spa$^{Cat}$/ci$^D$
j323 e;ey
j324 h th st T(3;4)e cu sr e$^s$ ca/Payne, Dfd ca
j325 net or$^{48a}$ sp$^2$;ru bv
j326 ri;ci ey$^R$
j327 ry$^2$;spa
j328 ry$^2$;spa$^{pol65}$
j329 se ss;spa$^{pol65}$
j330 st ry;spa$^{pol}$
j331 st ry;spa$^{pol65}$
j332 st ry$^2$;spa$^{pol}$
j333 st ry$^2$;spa$^{pol65}$
j334 se$^{51j}$;spa$^{pol}$
j335 T(3;4)89E, ss bs bxd/ey$^D$
j336 " A2/ci$^D$
j337 " A2/Me, ca
j338 " Al2/Cx, D
j339 " Al3, ve ca/Me, ca
j340 " A28, ve ca (homozygous)
j341 " c/Payne, Dfd ca
j342 " e/ci$^D$
j343 " f, h th st cu sr e$^s$ ca/Payne, Dfd ca
j344 T(3;4)f/Me
j345 tx;ci ey$^R$
j346 tx;sv$^n$

X,Y,2 (j347-349)

j347 Y$^{st}$/w$^e$ bb$^1$/w$^e$ bb$^1$/ & w$^e$ bb$^1$/Y$^+$;InsNs px sp/l mr$^2$ (Bridges)
j348 Y$^{bb}$ Su-Var 5/w$^{m4}$;En-Var $8^{48g}$/Cy
j349 Y$^{Lc}$/y w.Y$^S$ & y v f.+;S Sp cn/dp$^{txI}$ Cy, InsO pr cn$^2$

X,Y,3 (j350-354)

j350 ("multi- ♀") X.Y InEN y;st (no free Y)
j351 X.Y InEN In49 y;st (no free Y)
j352 X.Y y;st (no free Y, no In)
j353 sc$^8$.Y/y f:=;ca K-pn
j354 sc$^8$.Y:bw$^+$/X$^+$;Mas/Me,Ins ri Sb$^1$ & Dl H e P$^i$/Me,Ins ri Sb$^1$

X,Y,4 (j355-358)

j355 sc$^8$.Y/X$^+$ & y f:=;ci gvl ey$^R$ sv$^n$
j356 Tp4:Y/X.Y InEN In49 v g;ci gvl ey$^R$ sv$^n$
j357 Y$^S$.InEN y.Y$^L$:4 & y.=;ci ey$^R$ (no free Y)
j358 Y$^S$.InEN y.Y$^L$ sc$^8$ y$^+$;ci ey$^R$ (no free Y)

X,2,3 (j359-378)

j359 In49 v ct$^{ns}$;b;p$^p$
j360 In49 ct$^{ns}$;b;ri p$^p$
j361 ptg;px pd;su-pd
j362 T(X;2;3)zzO(14A;50A;75), y l$^{220}$/FM6
j363 v;ap$^{56f}$ cn;st
j364 X$^-$.Y$^S$;S fes Sp ms cn mr crs T(2;3)MI B$^{Si3}$ & y f:=
j365 y f:=;cn bw;e
j366 y f:=;dp;ve
j367 y In49 v;bw;e
j368 y sc$^{S1}$ f In49 y sc$^8$;bw;e
j369 y sc$^{S1}$ In49 sc$^8$;bw;st p$^p$
j370 y sc$^{S1}$ In49 sc$^8$;dp bw;st p$^p$
j371 y sc$^{S1}$ In49 sc$^8$ & y f:=;dp bw;st p$^p$
j372 y sc$^{S1}$ In49 sc$^8$;ho dp;ri p$^p$
j373 y sc$^{S1}$ In$^{49}$ w sc$^8$;dp;e$^{11}$
j374 y$^{Si}$ sc$^8$ B f In49 v;bw;e

j375 ("MI") $y^{Si}$ $sc^{8}$ InS $y^{3P}$;$al^{2}$ Cy $lt^{3}$ $cn^{2}$ $sp^{2}$/dp b $Pm^{1}$;ru h D InsCXF ca/Sb In3R
j376 y w $sn^{3}$;dp bw;st $p^{p}$
j377 y w $sn^{3}$ $f^{36a}$;dp bw;st $p^{p}$
j378 T(X;3) +T(2;3)Dinty & y f:=

X,2,4 (j379-381)

j379 ("scar") sc $t^{2}$ v f car;Cy/bw;ey
j380 $w^{m4}$;T(2;4)Su-Var 2/Cy
j381 X.Y InEN In49 y;st $spa^{pol}$

Y,2,3 (j382-387)

j382 Y:$bw^{+}$;bw;h ri
j383 Y:$bw^{+}$;$Px^{-2}$/Me,Tp
j384 Y:$Bw^{+}$;S Sp T(2;3)B/$dp^{txI}$ Cy,InsO pr $cn^{2}$;Me,Ins
j385 $sc^{8}$.Y:$bw^{+}$;fes ms cn sp/$dp^{txI}$ Cy,InsO pr $cn^{2}$;h ri $e^{s}$/Me,Ins ri $Sb^{1}$ (iso 7/57)
j386 $sc^{8}$.Y:$bw^{+}$;net bw mr crs/$dp^{txI}$ Cy,InsO pr $cn^{2}$;Me,Ins ri $Sb^{1}$ (iso, 1969) (ve bv)
j387 $sc^{8}$.Y:$bw^{+}$;net dp sp/$dp^{txI}$ Cy,InsO pr $cn^{2}$;ve bv/Me,Ins ri $Sb^{1}$

2,3,4 (j388-394)

j388 bw;e;ci $ey^{R}$
j389 T(2;3;4)295, $bw^{D}$/Cy;H/+
j390 dp;ve;$spa^{pol}$
j391 dp;ve tx;$spa^{pol}$
j392 dp lt bw;st $p^{p}$;$spa^{pol}$
j393 dp lt bw;st $p^{p}$;$sv^{n}$
j394 ho dp;ve;$spa^{pol}$

X,Y,2,3 (j395-406)

j395 ("Multipare R") $sc^{8}$.Y/$X^{c2}$, y & y f:=;twl bw;$st^{54i}$
j396 $sc^{8}$.Y/y Hw In49 $B^{M1}$ & y f:=;twl bw;$st^{54i}$
j397 ("Multipare") $sc^{8}$.Y/y sc In49 $B^{M1}$;twl bw;$st^{54i}$
j398 $sc^{8}$.Y/y $sc^{S1}$ In49 $sc^{8}$;dp bw;st $p^{p}$
j399 " " " " $sc^{8}$ & y f:=;dp bw;st $p^{p}$
j400 " " " " $sc^{8}$;dp;ve;$spa^{pol}$
j401 " " " " $sc^{8}$;ed dp cl lt bw;st $p^{p}$
j402 " " " " w $sc^{8}$;dp;ve;$spa^{pol}$
j403 $sc^{8}$.Y/y w $sn^{3}$ $f^{36a}$;dp bw;st $p^{p}$
j404 $Y^{Lc}$/y.$Y^{S}$;cn bw;e
j405 ("y s cn bw e") $Y^{Lc}$/y s.$Y^{S}$/y $sc^{S1}$ B f In49 v $sc^{8}$;cn bw;e
j406 $sc^{VI}$.$Y^{S}$/y In49 v f.$Y^{L}$;bw;e

X,2,3,4 (j407-414)

j407 $sc^{S1}$ B InS $w^{a}$ $sc^{8}$;SM1, $al^{2}$ Cy $sp^{2}$/dp $ds^{33k}$ $Pm^{1}$;C Sb/$Ubx^{130}$ $e^{s}$;$spa^{pol}$
j408 $sc^{S1}$ InS $w^{a}$ $sc^{8}$;SM1, $al^{2}$ Cy $sp^{2}$/dp $ds^{33k}$ $Pm^{1}$;C Sb/$Ubx^{130}$ $e^{s}$;$spa^{pol}$
j409 T(X;2;3;4)454, y 1(1)454/FM6
j410 y;bw;e;ci $ey^{R}$
j411 y f:=;bw;e;ci $ey^{R}$
j412 y f:=;bw;e;$spa^{pol}$
j413 y $sc^{S1}$ In49 $sc^{8}$;dp;ve;$spa^{pol}$
j414 y $sc^{S1}$ In49 w $sc^{8}$;dp;ve;$spa^{pol}$

X,Y,2,3,4 (j415-430)

j415 $sc^{8}$.Y/$Y^{S}$.X InEN In49 y.$Y^{L}$;dp bw;st $p^{p}$;$spa^{pol}$
j416 $sc^{8}$.Y/y $sc^{S1}$ In49 $sc^{8}$;dp bw;st $p^{p}$;$spa^{pol}$
j417 $sc^{8}$.Y/y $sc^{S1}$ In49 $sc^{8}$;dp lt bw;st $p^{p}$;$spa^{pol}$

j418 $sc^8$.Y/y $sc^{S1}$ In49 $sc^8$;ed dp cl lt bw;st $p^p$;$sv^n$
j419 sc8.Y/y scS1 In49 $sc^8$;ed dp cl lt bw;st $p^p$;$sta^{pol}$
j420 $sc^8$.Y.$B^S$/y $sc^{S1}$ In49 $sc^8$;dp;ve;$spa^{pol}$
j421 $Y^S$.X InEN y.$Y^L$;bw;e;ci $ey^R$ (no free Y)
j422 $Y^S$.X InEN In49 y.$Y^L$ & "snocty";cn bw;e;ci $ey^R$
j423 " " " " ;ed dp cl lt bw;st $p^p$;$spa^{pol}$ (no free Y)
j424 " " " " ;lt $stw^3$;$p^p$;$sv^n$ (no free Y)
j425 " " " " ;lt $stw^3$;st;$sv^n$ (no free Y)
j426 " " " " ;lt $stw^3$;ve;$spa^{pol}$ (no free Y)
j427 " " y.$Y^L$;bw;e;ci $ey^R$ (no free Y)
j428 " " " ;bw;e;$spa^{pol}$ (no free Y)
j429 " " " :4/Y & y f:=;bw;e;ci ey
j430 " " In49 y.$Y^L$;ho dp;ve;$spa^{pol}$

Lethal Stocks (l)

226 sex-linked recessive lethal stocks (EMS-induced)
124 sex-linked recessive lethal stocks (X-ray-induced)
242 sex-linked recessive lethal stocks (U.V.-induced)
151 recessive lethals in Chromosome III (X-ray-induced)

Miscellaneous Stocks

m1 $CO_2$-sensitive (Baumiller)
m2 $CO_2$-sensitive (Seecof's No. 13)
m3 SD-$72/dp^{txI}$ Cy, Ins O pr $cn^2$
m4 SD-bw/$dp^{txI}$ Cy, Ins O pr $cn^2$
m5 Sd-(NH)$2/dp^{txI}$ Cy,Ins O pr $cn^2$

Attached (compound) chromosomes (m6-26)

m6 RA, In(1)AB, y.....$sc^8$ & $y^+$ $ac^+$ $Y^L/Y^S$X InEN B y.$Y^L$
m7 RA, lJl.....$sc^8$ & y $sc^8$.Y/$lJl^{259}$ y w $Y^L$.$Y^S$
m8 RA, y.....$sc^8$ InEN.$Y^L$ & $y^+$ $ac^+$.$Y^L/Y^S$.InEN B y.$Y^L$
m9 RA, $Y^L$, +.....$sc^8$ InEN, y.$Y^L$ $y^+$ & $y^+$ $ac^+$.$Y^L/Y^S$.X InEN B y.$Y^L$
m10 RM(TAX), In(1)$y^4$, $w^a$ $y^4$/y v f & $Y^S$.X InEN B y.$Y^L$ (no free Y)
m11 RM, InEN $Y^S$ & $Y^S$.X InEN car f In49 v y.$Y^L$ (no free Y)
m12 RM, pn & $Y^S$.X y B.$Y^L$
m13 RM(13-0-15=$XY^L$.X), $y^2$ su-$w^a$ $w^a$ bb $Y^L/y^2$ su-$w^a$ $w^a$ bb? $bb^+$ & $Y^S$.X InEN B y.$Y^L$
m14 RM(15-DRP=$XY^L$.$Y^L$X), $y^2$ su-$w^a$ $w^a$ bb $Y^L/y^2$ su-$w^a$ $w^a$ bb? Y? $bb^+$ & $Y^S$.X InEN B f v y.$Y^L$ $y^+$
m15 TM(Hw f), originally y Hw v f.$y^+$ cv f $y^+$/X.Y, y B
m16 RM2L;RM2R (attached 2L" & 2R")
m17 RM2L, b;RM2R, cn
m18 RM3L;RM3R
m19 RM3L;RM3R (Rasmussen no. 26)
m20 RM3L, ri;RM3R sr
m21 RM3L, se $h^2$ $rs^2$;RM3R, sbd gl $e^s$ (Rasmussen no. 44)
m22 AF1, ci $ey^R$/gvl $sv^n$
m23 AF2, ci $ey^R$/gvl $sv^n$
m24 C(1)DX, y f;C(4)RM, ci $ey^R$ & $sc^{S1}$ B InS $w^a$ $sc^8$
m25 $sc^{S1}$ B InS $w^a$ $sc^8$;C(3L)RM, se $h^2$ $rs^2$;C(3R), sbd gl $e^s$
m26 w;C(4)RM;ci $ey^R$

Non-autonomous sex-linked lethals (E. Novitski) (m27-43)

m27 (alaful-1) y w spl sn/$sc^{S1}$ $bb^+$ B In49 oc ptg $sc^8$
m28 (gluful-1) y w spl sn bb/$sc^{S1}$ $bb^+$ B In49 oc ptg $sc^8$
m29 (gluful-2) y w spl sn bb/$sc^{S1}$ $bb^+$ B In49 oc ptg $sc^8$
m30 (gluful-3) y w spl sn bb/$sc^{S1}$ $bb^+$ B In49 oc ptg $sc^8$
m31 (gluful-4) y w spl sn/$sc^{S1}$ $bb^+$ B In49 oc ptg $sc^8$
m32 (glufulproless-1) y w spl sn bb/$sc^{S1}$ $bb^+$ B In49 oc ptg $sc^8$

m33 (glufultyrless-1) y w spl sn/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$
m34 (glufultyrless-2) y B/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$;(bw)/+;(st p$^{p}$)/+
m35 (glufultyrless-3) y w spl sn bb/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$
m36 (glufultyroproless-1) y w spl sn bb/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$
m37 (glyful-1) y w spl sn bb/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$
m38 (phenyfultyrless-1) y w spl sn bb/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$
m39 (tyrless-1) y w spl sn bb/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$
m40 (tyrless-2) y w spl sn/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$
m41 (tyrproless-1) y w spl sn/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$
m42 (tyrproless-2) y w spl sn bb/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$
m43 (tyrproless-3) y w spl sn bb/sc$^{S1}$ bb$^{+}$ B In49 oc ptg sc$^{8}$

Non-lethal "tumorous" stocks (m44-52)

m44 bw tu
m45 tu vg bw
m46 tu$^{36a}$ st sr e$^{s}$ ro ca
m47 tu$^{48a}$
m48 tu$^{50j}$
m49 tu$^{g}$
m50 tu$^{51m}$
m51 tu$^{h}$
m52 vg tu

## CROSS INDEX TO CHROMOSOME ABERRATIONS AND SPECIAL CHROMOSOMES

### DEFICIENCIES

#### Deficiencies-X

* Df(1)0-sc,LVM . . . . . (see Df(1)260-1)
* Df(1)259-4 . . . . . (b40)
* Df(1)260-1 . . . . . (b41)
* Df(1)B$^{263-20}$ . . . . . (b42)
* Df(1)bb . . . . . (b387,b446,b453)
* Df(1)bb$^{G}$ . . . . . (d2)
* Df(1)bb$^{l}$-3a . . . . . (f112)
* Df(1)bb$^{l}$-74 . . . . . (f113)
* Df(1)bb$^{l}$-158 . . . . . (b385)
* Df(1)bb$^{l}$-452 . . . . . (b386)
* Df(1)bb$^{l}$-456 . . . . . (f114)
* Df(1)ct . . . . . (b35,b36)
* Df(1)ct$^{268-42}$ . . . . . (b43)
* Df(1)f$^{257-5}$ . . . . . (b100)
* Df(1)g$^{l}$ . . . . . (b44)
* Df(1)mal . . . . . (b45)
* Df(1)N$^{8}$ . . . . . (b180)
* Df(1)N$^{264-39}$ . . . . . (b182)
* Df(1)N$^{264-105}$ . . . . . (b183)
* Df(1)rst$^{2}$ . . . . . (b213.b214)
* Df(1)sc$^{J1}$ . (=lJ1 sc$^{J1}$)(b160,b161,c56,etc.)
* Df(1)sc$^{4L}$ sc$^{8R}$ . . . . . (d1-d3)
* Df(1)sc$^{8}$ . . . . . (b71)
* Df(1)sc$^{10-1}$ . . . . . (c44)
* Df(1)sc$^{19}$ . . . . . (c45,c46,etc.)
* Df(1)svr . . . . . (b46)
* Df(1)w$^{258-11}$ . . . . . (b472,b473)
* Df(1)w$^{258-42}$ . . . . . (b474)
* Df(1)w$^{258-45}$ . . . . . (b475)
* Df(1)w$^{258-48}$ . . . . . (c12)

#### Deficiencies-2

* Df(2)M33a . . . . . (g349,g350)
* Df(2)MB . . . . . (=Df(2L)M-z$^{B}$)
* Df(2)MS4 . . . . . (g361)
* Df(2)MS8 . . . . . (g367,g368)
* Df(2)MS10 . . . . . (g370-g372)
* Df(2)rl$^{10a}$ . . . . . (g135)
* Df(2)rl$^{10b}$ . . . . . (g136)
* Df(2L)al . . . . . (g133)
* Df(2L)M-z$^{B}$ . . . . . (g352-g354)
* Df(2L)S2 . . . . . (g358)
* Df(2L)S3 . . . . . (g359,g360)
* Df(2R)42 . . . . . (g132)
* Df(2R)bw$^{5}$ . . . . . (g89,g90,g91)
* Df(2R)bw$^{VDe2L}$ Cy$^{R}$ . . . . . (g97)
* Df(2R)Px . . . . . (g457)
* Df(2R)Px$^{2}$ . . . . . (g84,g428,g429)
* Df(2R)Px$^{4}$ . . . . . (g257)
* Df(2R)vg$^{B}$ . . . . . (g49,g518,g519)
* Df(2R)vg$^{C}$ . . . . . (g520,g521)
* Df(2R)vg$^{D}$ . . . . . (g522,g523)

#### Deficiencies-3

* Df(3L)Ly . . . . . (h102,h103,h141)
* Df(3R)M-S31 . . . . . (h111)
* Df(3R)ry . . . . . (h41)
* Df(3R)ry$^{K}$ . . . . . (h42)
* Df(3R)sbd$^{105}$ . . . . . (h43,h44)

#### Deficiencies-4

* Df(4)3 . . . . . (i14)
* Df(4)G . . . . . (i15)
* Df(4)M . . . . . (i41)
* Df(4)M$^{62f}$ . . . . . (i16)
* Df(4)M$^{63a}$ . . . . . (i43)

### Duplications

* Dp(1;1)B$^{S}$ TMG . . . . . (b66,b67)
* Dp(1;1)sc$^{V1}$ . . . . . (b3)

* Dp(1;3)51 . . . . . (b69)
* Dp(1;3)126 . . . . . (b70)
* Dp(1;3)sc$^{J4}$ . . . . . (b71)
* Dp(1;3)w$^{vco}$ . . . . . (c12)
* Dp(1;3)w$^{49a7}$ . . . . . (b72)
* Dp(1;4)174 . . . . . (b74)
* Dp(1;f)3 . . . . . (b51)
* Dp(1;f)18 . . . . . (b52)
* Dp(1;f)52 . . . . . (b53)
* Dp(1;f)101 . . . . . (b46,b54)
* Dp(1;f)107 . . . . . (b55)
* Dp(1;f)112 . . . . . (b57)
* Dp(1;f)118 . . . . . (b56)
* Dp(1;f)122 . . . . . (b58)
* Dp(1;f)135 . . . . . (b59)
* Dp(1;f)164 . . . . . (b60)
* Dp(1;f)1492 . . . . . (b61)
* Dp(1;f)1514 . . . . . (b62)
* Dp(1;f)R . . . . . (b63)
* Dp(1;f)X$^{c2}$ . . . . . (=Dp(1;f)R)
* Dp(1;f)z$^{9}$ . . . . . (b64)
* Dp(2;2)41A . . . . . (g344)
* Dp(2;2)S . . . . . (g258)
* Dp(2;f)1 . . . . . (g257)
* Dp(2;3)P . . . . . (g134,j257)
* Qn(2;2)S . . . . . (g432)
* Dp(3;4)ry$^{+}$ . . . . . (h52)

## INVERSIONS

It should be noted that several other combinations, particularly involving the scute inversions, can be found in the c's and d's of the preceding list.

### Inversions-X

* In(1)26 . . . . . (f6)
* In(1)AB . . . . . (b157)
* In(1)ac$^{3}$ . . . . . (=sc$^{10}$)(b2,b3)
* In(1)AM . . . . . (b12,b44)
* Ins(1)At, sc$^{S1R}$, dl-49, sc$^{8R}$ . . (d19,d20)
* In(1)B$^{M1}$ . . . . . (b48,b151,b154)
* In(1)B$^{M2}$ . . . . . (b11,b304)
* In(1)bb . . . . . (b387,b446)
* In(1)ClB . . . . . (b11,b25,b47,etc.)
* In(1)ct . . . . . (f8)
* In(1)dl-49 . . . . . (b4,b49,etc.)
* Ins(1)dl-49 B$^{M1}$ . . . . . (b138)
* In(1)e(bx) . . . . . (j81)
* In(1)EN . . . . . (f1,f2,etc.)
* In(1)EN2 . . . . . (f242-f247)
* In(1)FM1 . . . . . (b16,b136)
* In(1)FM3 . . . . . (b123,b174)
* In(1)FM4 . . . . . (b43,b446)
* In(1)FM6 . . . . . (b80,b130)
* In(1)FM7 . . . . . (d10)
* In(1)FMA3 . . . . . (j126)
* In(1)g . . . . . (b490)
* In(1)N$^{264-84}$ . . . . . (b428)
* In(1)pn . . . . . (b199)
* In(1)rst$^{3}$ . . . . . (b215-b217,b438)
* In(1)S . . . . . (b522)
* In(1)sc$^{4}$ . . . . . (c5)
* In(1)sc$^{4L}$ sc$^{8R}$ . . . . . (d1-d3)
* In(1)sc$^{7}$ . . . . . (c14,c18)
* Ins(1)sc$^{7}$, AM . . . . . (c15)
* Ins(1)sc$^{7}$, B$^{M1}$ . . . . . (c17)
* In(1)sc$^{8}$ . . . . . (c19,c20)
* Ins(1)sc$^{8}$, dl-49 . . . . . (c25,c26)
* Ins(1)sc$^{8}$, S . . . . . (c22,c28)
* In(1)sc$^{9}$ . . . . . (c42)
* In(1)sc$^{260-14}$ . . . . . (c49)
* In(1)sc$^{260-22}$ . . . . . (c51)
* In(1)sc$^{J1}$ . . . . . (c56-c61)
* In(1)sc$^{J1(+)}$ . . . . . (f80-f83)
* Ins(1)sc$^{S1}$, dl-49 . . . . . (b433,b444)
* Ins(1)sc$^{S1L}$, dl-49, sc$^{8R}$ . (b27,b48,d23)
* In(1)sc$^{S1L}$, S, sc$^{8R}$ . . . . . (d30,d31)
* In(1)sc$^{V2}$ . . . . . (c77)
* In(1)spx . . . . . (b267,b268,b492)
* In(1)w$^{m4}$ . . . . . (b343,b344)
* In(1)w$^{m4R}$, w$^{mMcL}$ . . . . . (b345)
* In(1)w$^{mMc}$ . . . . . (b348)
* In(1)w$^{mMcL}$, w$^{m4R}$ . . . . . (b349)
* In(1)y$^{3P}$ . . . . . (b535)
* Ins(1)y$^{3PL}$, sc$^{8R}$ . . . . . (d70)
* Ins(1)y$^{3PL}$, S, sc$^{S1R}$ . . . . . (d71)
* In(1)y$^{4}$ . . . . . (b536-b538)
* Inp(1LR)sc$^{V1}$ . . . . . (b542)

### 2L Inversions

* In(2L)Cy (Cy$^{+}$) . . . . . (g306)
* In(2L)Cy . . . . . (g27,g240,g241)
* In(2L)dp$^{olv\ R}$ . . . . . (=dp$^{Rf}$)(g233)
* In(2L)dp$^{w18}$ . . . . . (g309)
* In(2L)Cy$^{L}$ t$^{R}$ . . . . . (g263)
* In(2L)NS . . . . . (g318)
* In(2L)t . . . . . (g488)

### 2L + 2R Inversions

* In(2L + 2R)Cy (Cy$^{+}$) . . . . . (g28)
* In(2L + 2R)Cy . . . . . (g7,g8,g9)
* Ins(2L + 2R)Cy, bw$^{V34k}$ . . . . . (g373)
* Ins(2L)Cy, (2R)NS . . . . . (g398)
* In(2L + 2R)NS . . . . . (g9,g25)
* Ins(2L)t, (2R)Cy . . . (=In(2L)t, Roi, In (2R)Cy, bw sp$^{2}$ or)(g528)
* In(2LR)102 . . . . . (g314)
* In(2LR)bw$^{V1}$ . . . . . (=Pm)(g7,g408)
* In(2LR)bw$^{V32g}$ . . . . . (=Pm$^{2}$)(g409,g410)
* In(2LR)O . . . . . (g36,g454,g457,etc.)
* In(2LR)dp . . . . . (g70)(see T(2;3)dp)

* In(2LR)Gla . . . . . (g52,g97)
*Ins(2LR)Gla, CyO . . . . . (g297)
* In(2LR)lt$^{m3}$ . . . . . (g313)
* In(2LR)lt$^{mr}$ . . . . . (g312)
* In(2LR)Pm . . . . . (see In(2LR)bw$^{V1}$)
* In(2LR)Pm$^{2}$ . . . . . (see In(2LR)bw$^{V32q}$)
* In(2LR)px . . . . . (=In(2LR)px$^{52q}$)(g120)
* In(2LR)Rev . . . . . (g96)
* In(2LR)Rev$^{B}$ . . . . . (=Rvd)(g37)
* In(2LR)Rvd . . . . . (see In(2LR)Rev$^{B}$)
* In(2LR)SM1 . . . . . (g22,g23,g148,etc.)
* In(2LR)SM3 . . . . . (g312)
* In(2LR)SM5 . . . . . (g11,g38,g41,etc.)
* In(2LR)U . . . . . (g29,g31)

2R Inversions

* In(2R)bw$^{V34k}$ . . . . . (g373)
* In(2R)bw$^{VDel}$ . . . . . (g57)
* In(2R)bw$^{VDe2}$ . . . . . (g96)
* In(2R)Cy . . . . . (g344)
* In(2R)Mo$^{k}$ . . . . . (j286)
* In(2R)NS . . . . . (g237)
* In(2R)vg$^{U}$ . . . . . (g528)

3L Inversions

* In(3L)D . . . . . (h35)
* In(3L)D$^{3}$ . . . . . (h38,h39)
* In(3L)P . . . . . (h141,h249,etc.)

3L + 3R Inversions

* In(3L + 3R)LVM . . . . . (h44,h69,h97,etc.)
* In(3L + 3R)P . . . . . (h37,h38,h39)
* Ins(3L)P, (3R)P18 . . . . . (h84)

3LR Inversions

* In(3LR)4833.10 . . . . . (h86)
* In(3LR)Cx . . . . . (h45)
* In(3LR)DcxF . . . . . (h36,h56)
* In(3LR)a . . . . . (h149)
* In(3LR)b . . . . . (h160)
* In(3LR)c . . . . . (h150)
* In(3LR)sep . . . . . (h188-h190)
* In(3LR)TM1 . . . . . (h4,h10,h20,etc.)
* In(3LR)TM3 . . . . . (h22)
* (In(3LR)Ubx$^{101}$ . . . . . (h240)
* In(3LR)Ubx$^{130}$ . . . . . (h21,h41,h42)

3R Inversions

* In(3R)Antp$^{B}$ . . . . . (h4)
* In(3R)Antp$^{R}$ . . . . . (=ss$^{A}$, In)(h206)
* In(3R)C . . . . . (h8,h9)
* In(3R)Cyd . . . . . (h50)
* In(3R)D1$^{B}$ . . . . . (h87)
* In(3R)hp . . . . . (h77)
* In(3R)Hu . . . . . (h88)
* In(3R)Mo . . . . . (h89,j186)
* In(3R)P . . . . . (h47)
* In(3R)P18 . . . . . (h84)
* In(3R)P$^{Fla}$ . . . . . (a9)
* In(3R)P$^{W}$ . . . . . (h87,h217)
* In(3R)spr . . . . . (=In(3R)P)

RINGS(See also pages 10 and 34)

* R(1)1 . . . . . (=X$^{c}$)(=X$^{O}$)(b354)
* R(1)2 . . . . (=X$^{c2}$)(b355-b362,f103-f107)
* R(1)3 . . . . . (b363)
* R(1)w$^{vC}$ . . . . . (=In(X$^{c2}$)w$^{vC}$)(b158,f90)
* R(Y) . . . . . (see Y derivatives)

TRANSLOCATIONS

Translocations (1;Y)(see also f327-374)

* T(1;Y)E . . . . . (f375)
* T(1;Y)2E . . . . . (f376)

Translocations (1;2)(see also j24-30)

* T(1;2)459 . . . . . (j24)
* T(1;2;y$^{+}$Y$^{L}$)7B; 39 #17 . . . . . (f377)
* T(1;2H?;B$^{S}$Y$^{L}$)3c #109 . . . . . (f378)
* T(1;2)Bld . . . . . (j2)
* T(1;2)Cl . . . . . (j76)
* T(1;2)lt . . . . . (j25)
* T(1;2)N$^{264-10}$ . . . . . (b181)
* T(1;2)sc$^{S2}$ . . . . . (c75)
* T(1;2)sc$^{19}$ . . . . . (c45,c46)

Translocations (1;3)
(see also j90-93 and j101-108)

* T(1;3)19E;62A #134 . . . . . (f353)
* T(1;3)-78 . . . . . (j127)
* T(1;3;B$^{S}$Y$^{S}$)10A;97A #154 . . . . . (f379)
* T(1;3;B$^{S}$Y$^{L}$)15E;74 #144 . . . . . (f380)
* T(1;3;B$^{S}$Y$^{L}$)17A;94 #161 . . . . . (f381)
* T(1;3;B$^{S}$Y$^{S}$)19F;85 #127 . . . . . (f382)
* T(1;3)B$^{581}$ . . . . . (j94)
* T(1;3)"Del-143" . . . . . (j95)
* T(1;3)Din . . . . . (j378)
* T(1;3)N$^{264-6}$ . . . . . (b426)
* T(1;3)05 . . . . . (j96)
* T(1;3)ras$^{v}$ . . . . . (j97)
* T(1;3)ry$^{35}$ . . . . . (f147)
* T(1;3)sc$^{J4}$ . . . . . (c62)
* T(1;3)sc$^{Mc}$ . . . . . (c67)
* T(1;3)sc$^{260-15}$ . . . . . (c50)
* T(1;3)sta . . . . . (j98)
* T(1;3)v . . . . . (j99)
* T(1;3)w$^{vco}$ . . . . . (j100)

Translocations (1;4)(see also e1-35 for other T(1;4)'s and those produced by M. Lieb, 1946)

* T(1;4)9c;101 #116 . . . . . (f354)
* T(1;4)sc$^{H}$ . . . . . (c55)

* T(1;4)sc$^{L3}$ (spoon-like) . . . . . (c64)
* T(XY$^{L}$.Y$^{S}$;4)B$^{S}$ (16A1) . . . . . (f383)

Translocations (Y;2)(Y;3)(Y;4)
(see also f176-225)

* T(Y;2)A . . . . . (g339)
* T(Y;2)B . . . . . (g503,g507)
* T(Y;2)C . . . . . (g123,g414)
* T(Y;2)E . . . . . (g6,g112)
* T(Y;2)G . . . . . (g63)
* T(Y;2)J . . . . . (g426)
* T(Y;3)(II4Aa$^{3}$) . . . . . (f384)
* T(Y;3)Su-Var 3 . . . . . (f385)
* T(Y;4) . . . . . (see Y derivatives)
(f386-f391,j356-j358)

Translocations (2;3)

* T(2;3)101 . . . . . (j144)
* T(2;3)108 . . . . . (j145)
* T(2;3)109 . . . . . (j284)
* T(2;3)289 . . . . . (j171)
* T(2;3)A . . . . . (j163)
* T(2;3)ap$^{Xa}$ . . . . . (h13,j162)
* T(2;3)Antp$^{Yu}$ . . . . . (j285)
* T(2;3)Ata . . . . . (=T(2;3)At)(j286)
* T(2;3)B $^{"VA"}$ . . . . . (j146,j222)
* T(2;3)bw$^{VA}$ . . . . . (g71)
* T(2;3)bw$^{V4}$ . . . . . (g93)
* T(2;3)bw$^{V5}$ . . . . . (g94,g95)
* T(2;3)bw$^{VDe3}$ . . . . . (j172,j173)
* T(2;3)bw$^{VDe4}$ . . . . . (g98)
* T(2;3)C . . . . . (j289)
* T(213)Din . . . . . (j378)
* T(2;3)dp . . . . . (j236)
* T(2;3)dp$^{D}$ . . . . . (g148)
* T(2;3)dp$^{wl}$ . . . . . (g254)
* T(2;3)dp$^{w8}$ . . . . . (j293)
* T(2;3)Me . . . . . (h111-h114)
* T(2;3)P . . . . . (j257)
* T(2;3)p$^{Gr}$ . . . . . (=T(2;3)Pu$^{Gr}$)
* T(2;3)Pu$^{Gr}$ . . . . . (j253)
* T(2;3)rn . . . . . (g444,g467,g470)
* T(2;3)Dp-S . . . . . (j294)
* T(2;3)S$^{M}$ . . . . . (g527,j280)
* T(2;3)Xa . . . . . (=T(2;3)ap$^{Xa}$)

Translocations (2;4)

* T(2;4)a . . . . . (j317)
* T(2;4)ast$^{v}$ . . . . . (j319)
* T(2;4)b . . . . . (j320)
* T(2;4)d . . . . . (j303,j318)

Translocations (3;4)

* T(3;4)89E . . . . . (j335)
* T(3;4)A2 . . . . . (j336,j337)
* T(3;4)A12 . . . . . (j338)
* T(3;4)A13 . . . . . (j339)
* T(3;4)A28 . . . . . (j340)
* T(3;4)c . . . . . (j341)
* T(314)e . . . . . (j324,j342)
* T(3;4)f . . . . . (j343,j344)

Transpositions

* Tp(3)bxd$^{107}$ . . . . . (h232)
* Tp(3)Vno . . . . . (h233)

COMPOUND CHROMOSOMES

Attached-X
(.= is a synonym for C(1)RM)
(:= is a synonym for C(1)DX)

* C(1)RA . . . . . (f239,m6-m9)
* C(1)RM (b30-b32,b51,b53,b57,b58,b149,b236
b259,b274,b310,b384-b386,b408,b440
b476,b533,d9,f1-f3,f7,f8,f13,f14
f145,f234,f235,f241,f243,f244,f249
f250,f252,f270,m11-m14)
* C(1)RM(TAX) . . . . . (m10)
* C(1)DX . . . . (b1,b3,b5,b10,b26,b29,b365
b521,b525,f140,f235)

Attached Autosomal Arms

* C(2L)RM;C(2R)RM . . (=RM2L;RM2R)(m16,m17)
* C(3L)RM;C(3R)RM . . . . . (m18,m19)
* C(3L)RM;C(3R)RM . . . . . (m20,m21,m25)
* C(4)RM . . . . . (m22,m23,m24,m26)

X.Y COMBINATIONS

* X.Y$^{L}$ . . . . . (=XY$^{1}$)(f301,f302,etc.)
* X.Y$^{L}$.Y$^{S}$ . . . . . (f416-f420)
* X.Y$^{L}$.Y$^{S}$ 129-16 . . . . . (f414)
* X.Y$^{L}$C2 . . . . . (f403,f404)
* X.Y$^{L}$, In(1)EN . . . . . (f307)
* X.Y$^{S}$ . . . . . (f266-f275)
* XY$^{S}$.Y$^{L}$ . . . . . (f411)
* Y$^{S}$X., In(1)EN . . . . . (f396)
* Y$^{S}$X.Y$^{L}$ . . . . . (f406-f408)
* Y$^{S}$X.Y$^{L}$ In(1)d1-49 . . . . . (f19)
* Y$^{S}$X.Y$^{L}$, In(1)EN . . . . . (f1)
* Y$^{S}$X.Y$^{L}$, Ins(1)EN, d1-49 . . . . (f5,j351)

Y-DERIVATIVES
(Other cases may be found under f's)

* 4Y . . . . (=Tp 4.Y)(f386-f391;j356-j358)
* B$^{S}$w$^{+}$Yy$^{+}$ . . . . . (f231,f232)
* B$^{S}$v$^{+}$Yy$^{+}$ . . . . . (f143,f144)
* B$^{S}$Y . . . . . (=YB$^{S}$)(f145-f147)
* B$^{S}$Yw$^{+}$y$^{+}$ . . . . . (f150)
* B$^{S}$Yy$^{+}$ . . . (=sc$^{8}$.Y.B$^{S}$)(f153,154,155,etc.)
* B$^{S}$Yy$^{31d}$ . . . . . (=y$^{31d}$.Y.B$^{S}$)(f151)
* B$^{S}$Yy$^{ah}$ . . . . . (=y$^{ah}$.Y.B$^{S}$)(f152)
* B$^{S}$Y(y ac)$^{-}$ . . . (=sc$^{8y}$.Y.B$^{S}$)(f176,etc.)
* B$^{S+}$Yy$^{+}$ . . . . . (=sc$^{8}$.Y.B$^{S+}$)(f207,etc.)

* $B^{S+}Y(yac)^-$ . . . . ($=sc^{8y}.Y.B^{S+}$)(f190,etc.)
* $B^{S-}v^+dy^+Yy^+$ . . . . . (f142)
* $B^{S-Var}Y(yac)^-$ . ($=sc^8.Y.B^{S-Var}$)(f201,etc.)
* $Bw^+Y$ . . . . . ($=Y:bw^+$)(f65,66,67)
* $Bw^+Yy^+$ . . . . . ($=sc^8.Y:bw^+$)(f226,227,228)
* $l(\underline{1})J1^+Y$ . . . . . (f72-83,etc.)
* $R(Y)bw^+$ . . . . ("MYR")($=Y^c:bw^+$)(f68,69)
* $R(Y)^F$ . . ($=Y^{cF}$)("Filicidal ring")(f48-55)
* $R(Y)L$ . . . . . ($=Y^{Lc}$)(f240-275,etc.)
* $sc^{V1}.Y^S$ . . . . . (f309-324,etc.)
* $w^+y^+Y$ . . . . . ($=sc^8.Y.w^+$)(f230)
* $w^+Y$ . . . . . ($=Y.w^+$)(f71)
* $y^+Y$ . . . . . ($=sc^8.Y$)(f84-141,etc.)
* $y^+Y^L$ . . . . . (=FR2)(f239)
* Ybb . . . . . (f57,58)
* $Ybb^-$ . . . . . (b345,f59-62)
* $Y\ mal^+2$ . . . . . (f70)
* $Y\ mal^{106}$ . . . . . ($=y^+.Y.mal^{106}$)(f326)
* $Y^L.sc^{S1}$ . . . . . ($=sc.Y^L$)(b68)
* $Y^L.sc^{S1}3$ . . . . ($=sc^{S1}$ c.o. Y EY80)(f238)
* $Y^L.y^+B2$ . . . ($=sc^8$ EN c.o. Y B-2)(f236)
* $Y^L.y^+T0$ . . . ($=sc^8$ EN c.o. Y T-0)(f237)
* $Y^L.y^{3M}$ . . . . . ($=y^3.Y^L$)(f293-300)
* $Y^S$ . . . . . (f301-304)
* $Y^S.Y^S$ . . . . . (="Y")(f305,307,308)
* $Y^S.Y^{S2}$ . . . . . ($=Y^S.Y^S$ #2)(f306)

Ring X;Y (of Novitski)(f422-432, continued from page 15)(See also pages 10 and 32)

f422 R(X;Y)1, y/FM6
f423 R(X;Y)A5-11A, $y/y^2$ su-$w^a$ $w^a$ bb/0
f424 R(X;Y)A5-15A, $y/y^2$ su-$w^a$ $w^a$ bb/0
f425 R(X;Y)A5-19A, $y/y^2$ su-$w^a$ $w^a$ bb/0
f426 R(X;Y)B7-3A, $y/y^2$ su-$w^a$ $w^a$ bb/0
f427 R(X;Y)D5-11C, $y/y^2$ su-$w^a$ $w^a$ bb/0
f428 R(X;Y)D7-2D, $y/y^2$ su-$w^a$ $w^a$ bb/0
f429 R(X;Y)E5-19G, $y/y^2$ su-$w^a$ $w^a$ bb/0
f430 R(X;Y)F5-19H, $y/y^2$ su-$w^a$ $w^a$ bb/0
f431 R(X;Y)F7-2H, $y/y^2$ su-$w^a$ $w^a$ bb/0
f432 R(X;Y)8, y/FM6

PLEASE NOTE: Because of the inclusion of the extensive stock list of the Mid-America Drosophila Stock Center in Bowling Green in this issue, other stock lists are being reserved for DIS 50, now in preparation.

## MELANOGASTER - NEW MUTANTS

Report of M. Ashburner
University of Cambridge, England

The following chromosome aberrations were induced in Canton-S males by 3000r X rays. Homozygous lethal unless stated.

T(2;3)A2 T(2;3)47F;78A + In(2R)46B;47F New orders 60-47F/78A-100; 61-78A/46B-47F/46B-21
T(2;3)HR17 T(2;3)26A;69F
T(2;3)HR30 T(2;3)34E;70B Homozygous viable
T(2;3)HR28 T(2;3)25A;91D + T(2;3)42A;71AB
T(2;3)HR26 T(2;3)28A;64D
In(3LR)HR5 New order 61-67F/88E-75C/98C-88E/67F-75C/98C-100
In(3LR)HR6 In(3LR)69F;83E
In(3L)HR10 In(3L)64C;75C
In(3LR)HR8 New order 61-70B/78C-83A/72C-70B/78C-72E/83A-100
In(3L)HR2 In(3L)62E-78B Homozygous viable
In(3L+R)HR13 In(3R)81;98E + In(3L)75F;80
In(3L)HR15 In(3L)64D;68C Homozygous viable
In(3LR)HR20 In(3LR)64C;84D
In(3LR)HR32 In(3LR)66E;81
In(3LR)HR38 In(3LR)68F;96D Homozygous viable
In(3LR)HR33 In(3LR)61A;87B Homozygous viable
In(3L)HR27 In(3L)76A;80B Homozygous viable

The following two aberrations were found in 'wild'stocks

In(3L)AM44 In(3L)61A;62C In a stock collected by P. Ives, South Amherst, Mass., 1965 Inversion detected Aug. 1972; homozygous viable

Ins(2LR)BEL In(2LR)36D;46F + In(2LR)30C;47E In a stock collected in Belinga, Gabon, June 1970 by J. David; inversion detected January 1972; homozygous lethal

Report of G. Trippa
Universita di Roma, Italia

Pgm$^{C}$: phosphoglucomutase$^{C}$ Trippa, G., C. Barberio and A. Loverre 1972.

Pgm$^{D}$: phosphoglucomutase$^{D}$ Trippa, G., C. Barberio and A. Loverre 1972.

Pgm$^{E}$: phosphoglucomutase$^{E}$ Trippa, G., C. Barberio and A. Loverre 1972.

Pgm$^{F}$: phosphoglucomutase$^{F}$ Trippa, G., C. Barberio and A. Loverre 1972. These four new electrophoretic forms of phosphoglucomutase (PGM) have been found during a study of the distribution of PGM polymorphism in natural populations, collected in the South of Italy, using the method of Spencer et al. (Nature 204:742, 1964) modified for single fly homogenates.

Each variant exhibits a single anodal band of activity.

In comparison to the more frequent alleles, Pgm$^{A}$ and Pgm$^{B}$ (Trippa, G., DIS 46:42, 1971) these new variants migrate as follows: Pgm$^{E}$ more slowly than does Pgm$^{B}$; Pgm$^{F}$ intermediate between Pgm$^{B}$ and Pgm$^{A}$, but nearer to Pgm$^{B}$; Pgm$^{C}$ more rapidly than Pgm$^{A}$; and Pgm$^{D}$ more rapidly than Pgm$^{C}$.

In the samples studied different gene frequencies are observed (Trippa et al., Research Notes, this issue). These variants are always found in the heterozygous state. All types of heterozygotes exhibit two bands and no hybrid bands. RKl.

Report of M. Bownes
University of Sussex, Brighton, England

Temperature sensitive eye colour mutant X-unmapped. Recovered in a screen of X chromosomes for temperature sensitivity, from a cross of EMS treated Oregon males with attached X females. The flies have a maroon-like eye colour at 25°C and an orange eye colour at 29°C. The mutant compliments with ma-1$^{F2}$ to give normal red eye coloured adults.

Report of J.B. Whitney III and J.C. Lucchesi
University of North Carolina, Chapel Hill, N.C.

$w^{71e}$: white$^{71e}$ Whitney 71e. 1-1.5. Spontaneous on the dor-bearing chromosome of a dor/FM3 stock. Like white.

$y^{71f}$: yellow$^{71f}$ Whitney 71f. 1-0.0. Spontaneous as $y^{71f}$ dor $w^{71e}$ sons of a dor $w^{71e}$/+ + by dor $w^{71e}$ mass mating. Like yellow.

$w^{bw71g}$: white-brownish Whitney 71g. Arose spontaneously as a single $w^{bw71g}$/dor $w^{71e}$ female from a dor $w^{71e}$/+ + by dor $w^{71e}$ mass mating. $w^{bw71g}/w^{71e}$ eyecolor resembles aged dor and darkens with age. $w^{bw71g}$-chromosome male lethality and eyecolor effect fully covered by $w^{+}$.Y and Dp(1;3)$^{vco}$. Variegated eyecolor in presence of T(1;3)$w^{vco}$, v f. y cv v f/$w^{bw71g}$ females produced 9 y non-cv sons and no non-y sons among 428 male progeny. Probably associated with a chromosome rearrangement.

srw: short round wings Whitney 71h. 1-probably distal to w. Spontaneous in chromosome sampled from isogenic Samarkand. Semilethal. Wings short, rounded, usually cupped upward, though sometimes unexpanded, warped, crumpled, or curved downward. Veins frequently malformed. Body slightly darkened. Front femurs usually kinked, legs infrequently gnarled. Males sometimes fertile. Females sterile, rarely lay eggs. srw/br is wiid type.

kf$^{2}$: kinked femur-2 Whitney 70. 1-20.3 ± 0.2. Spontaneous in Muller-5 chromosome. Removed and located relative to y, cv, rux$^{2}$, bis, sn, v, and f. Wings unexpanded or rumpled. Front femurs frequently kinked. Presumed allelic to the extinct kf, though less extreme. 4% of genetically-kf$^{2}$ males and no females are phenotypically normal. Male phenotypic escape is independent of temperature (18°, 25°, 29°), though more flies have fully unexpanded wings at lower temperatures in both sexes.

Report of A. Schalet
University of Leiden, The Netherlands

The mutants listed below are in the proximal region of the X chromosome between mal-64.8 and bb-66.0. They have been positioned by complementation mapping as follows: lf, vao, unc, (lfl, l(1)B-214), l(1)W-2, l(1)W-4, (l(1)LB20, l(1)A112), eo, (wap, l(1)Q56), uncl, l(1)114, l(1)X-3, l(1)P-19, l(1)Q463, sph, ($su(f)^{pb}$, $su(f)^{3DES}$). The left-right orientation of bracketed mutants has not been determined. The mutants rfr positioned at 67.9 in Lindsley and Grell, and gr positioned at 72.0 by Roberts and Hewitt (DIS 44:49), are not covered by $y^+$ Y $mal^{106}$ and must be to the left of ot the distal-most visible mutant in the proximal region thus far found to be covered by $y^+$ Y $mal^{106}$ (DIS 43:129).

vao: varied outspread 1- between lf and unc. A mutant, now lost, was originally described by Fahmy (Lindsley and Grell) as having outspread wings and a mottled brown eye color. It was associated with In(1)vao which had a proximal breakpoint at 19E7-8. We have adopted the symbol vao for the phenotype displayed by females such as Df(1)A118/Df(1)Q539 heterozygotes which lack 19E7-8 (Schalet and Finnerty, DIS 45:77). Df(1)B-57/Df(1)Q539 heterozygotes are vao and unc at 24-25°C. At 17-18°C such females are unc but the eyes appear normal even though the other vao characteristics mentioned by Schalet and Finnerty show.

unc: uncoordinated 1- between vao and lfl or vao and lethal B-214. Four alleles, R-10-1, W-1, W-5, LB17. When these alleles are compounded with deficiencies such as Df(1) D43L1 and/or Df(1)Q539, up to 25% or more of the expected mutant/deficiency females hatch provided overcrowding is prevented. In crosses which yield W-5/W-5 or W-5/W-1 females, a higher % of such individuals may hatch. LV7 (DIS 44:96), which was an allele of LB17, has been lost.

uncl: uncoordinatedlike 1- between wap and lethal 114 or between lethal Q56 and lethal 114. Three alleles, R-10-10, Q456, B83. Most flies die just before eclosion, but those which do hatch have uncoordinated leg movements and die soon afterward. Mutant/deficiency females have the same phenotype as mutant males and females. Viability of various compounds is low (approximately 1-12%).

lfl: littlefly-like 1- between unc and lethal B-214 or between lethal B-214 and lethal EN3. Originally described as lethal B-56. When overcrowding is prevented emergence is delayed until at least the third day after non-lfl individuals have begun to hatch, so that more than 90% of the non-lfl flies have hatched by the time lfl flies begin to emerge. Viability about 20-30% normal. lfl individuals are small like the mutant little fly (located to the left of vao) but without a narrow abdomen. Compounds of lfl and deficiencies such as Df(1)D43L1 or $Df(1)mal^3$ are phenotypically lfl. FM6/lfl is normal.

$lfl^2$: littlefly-like$^2$ Originally described as lethal B-96. Compounds $lfl/lfl^2$, $lfl^2/Df(1)mal^3$, $lfl^2/Df(1)DCB1-35b$, $lfl^2/Df(1)D43L1$ are not particularly small, but they lack thoracic hairs, head and thoracic bristles, eyes are often roughened, wings are abnormal with posterior crossveins often interrupted. $lfl^2/Y$ males hatch only very rarely, approximately less than 1%. Such males and males which die just prior to eclosion have a phenotype similar to the above described females. $FM6/lfl^2$ females and $y^+$ Y $mal^{106}/lfl^2$ males frequently lack ocellar, postvertical and humeral bristles, posterior crossveins may be interrupted and vein L5 usually interrupted, some thoracic hairs absent. $Df(1)B-57/lfl^2$, lethal $B-214/lfl^2$ are normal.

eo: extra organs 1- between lethal A112 and wap or between lethal A112 and lethal Q56. Phenotype shows in males carrying $y^+$ Y $mal^{126}$/lethal A7 or lethal E54 and other tested alleles of this locus (Schalet, DIS 44:96). In extreme cases a leg may be branched or completely duplicated, an antenna or arista may be duplicated or triplicated. Usually only the eyes are malformed, less frequently the wings are malformed.

wap: wings apart 1- between eo and lethal Q56 or between lethal Q56 and uncl. Three alleles, A200, Q464, Q217. Generally, most flies die just before eclosion, but flies which do hatch usually have wings set slightly apart, a somewhat darker than normal thorax and one or more extra crossveins. Mutant/deficiency females have the same phenotype as mutant males

and females. Viability of various compounds usually low (approximately 2-9%). However, about 50% of the expected Q217/Df(1)su(f)$^5$ females were observed at 24-25°C, whereas only about 15% hatched at 29-30°C. At the higher temperature the mutant phenotype was more extreme.

sph: sparse hairs 1- between lethal Q463 and su(f). Phenotype shows in compounds of Df(1)17-87 or Df(1)22 with lethals X-4, R-9-5 or 4P1. (These deficiencies do not interact with mutants to the left of sph and are broken proximally to the right of su(f)). Thoracic hairs are sparse, eyes show variable roughening, wings are extended and margins are incised. Overlaps wild-type at 24-25°C, but when compounds with 17-87 were tested at 29-30°C, all flies show the mutant phenotype.

su(f)$^{pb}$: suppressor of forked-pale bristles 1- most proximal "ordinary" X-linked gene distal to the bobbed locus. (This placement attributed to su(f) complex locus and not position of su(f)$^{pb}$ within the locus). Originally described as lethal R-9-18. Lethal at 24-25°C and 29-30°C. Fully viable (but weak) at 17-18°C provided overcrowding is prevented. Bristles and hairs are pale yellow and thread-like, dark pigment on thorax usually concentrated at dorsal anterior region, wings curled or wrinkled. f su(f)$^{pb}$ males and females are f$^+$ at 17-18°C. f su(f)$^{pb}$/f su(f) females are f$^+$, like f su(f)/f su(f), at all temperatures. f su(f)$^{pb}$/f su(f)$^{3DES}$ females are fully viable, show a good but not complete suppression of f, and are normal for the su(f)$^{pb}$ phenotype at all temperatures. su(f)$^{pb}$/su(f)$^{X-2}$ females are fully viable and normal at 17-18°C, vary in viability, show a broad streak of dark pigment on thorax which sometimes is concentrated at dorsal anterior region, have wings which sometimes is concentrated at dorsal anterior region, have wings which sometimes stick up and out, at 24-25°C; die sometime prior to puparium formation at 29-30°C. su(f)$^{pb}$/su(f)$^{D13}$ females die about the time of puparium formation at 24-25°C, but are fully viable with some bristles and hairs of all individuals showing the su(f)$^{pb}$ phenotype and the texture of the wings appearing abnormal at 17-18°C. su(f)$^{pb}$/Df(1)D43L1 females die about the time of puparium formation at 24-25°C, but die just prior to eclosion at 17-18°C. su(f)$^{pb}$/su(f)$^{ts}$ females (ts = temperature sensitive allele of T.R.F. Wright) are fully viable and normal at 17-18°C and 24-25°C, but fully viable and Minute-like at 29-30°C. w su(f)$^{pb}$/w$^a$ su(f) females are phenotypically w at 24-25°C. w$^a$ su(f)$^{pb}$ males have an eye color intermediate between w and w$^a$ at 17-18°C. lz$^{37h}$ su(f)$^{pb}$ males show an enhancement of the lz$^{37h}$ phenotype at 17-18°C.

su(f)$^{3DES}$: suppressor of forked$^{3DES}$ Originally described as lethal 3DES. f su(f)$^{3DES}$/ f su(f) females are f$^+$ at 24-25°C. (Schalet and Singer DIS 46:131). f su(f)$^{3DES}$/f$^5$ su(f) shows f suppression similar to f su(f)/f$^5$ su(f) at 24-25°C. At 20-30°C f su(f)$^{3DES}$/f$^5$ su(f) shows f suppression but bristles are Minute-like. (T.R.F. Wright has found and we can confirm that at 29-30°C f su(f) males and females show the Minute-like effect earlier reported for various su(f)/deficiency genotypes at normal temperatures by Schalet (1968) and Lindsley and Grell (1968).)

su(f)$^{X-2}$: suppressor of forked$^{X-2}$ Earlier assumed to be an aberration because it was originally reported as lethal in compounds with lethal R-9-18 and lethal 3DES, but see su(f)$^{pb}$ above. su(f)$^{X-2}$/su(f) is Minute-like at 24-25°C, lethal at 29-30°C.

su(f)$^{D-13}$: suppressor of forked$^{D-13}$ Earlier assumed to be an aberration because it was originally reported as lethal in compounds with lethal R-9-18 and lethal 3DES, but see su(f)$^{pb}$ above. su(f)$^{D-13}$/su(f) is Minute-like at 24-25°C, lethal at 29-30°C.

su(f)$^1$: suppressor of forked The interaction of su(f) and w$^a$ reported by Green (Lindsley and Grell) to produce nearly white eyes does not occur at 29-30°C. At this temperature the eyes are apricot. At 24-25°C the eyes are near white, at 17-18°C the eyes are indistinguishable from white.

Report of W.J. Morrison
Cornell University, Ithaca, New York

In(3R)C13: X-ray induced in a normal 3rd carrying Pr (Prickly) and Dr (Drop). Reduces crossing over between Pr (3-90.0) and Dr (3-99.2) to < .1%. Breakpoints 94E;98D.

Report of E. Bahn
University of Copenhagen, Denmark

su(r): suppressor of rudimentary 1-27.7. EMS induced recessive mutation acting as a metabolic suppressor of all rudimentary alleles. It is extremely sensitive to exogenous supply of pyrimidines. Biochemical studies have shown that the suppressing effect on the pyrimidine requiring mutant rudimentary is due to a block in the catabolic pathway of pyrimidines. su(r) has little or no dihydrouracil dehydrogenase activity, so the degradation of uracil as well as thymine is greatly hampered. (See research note this issue). Other phenotypic expressions of the suppressor are enhancement of the wing mutants dp and net.

Report of P.T. Ives
Amherst College, Amherst, Massachusetts

bd: bald 2-56.1. P.T. Ives, 71j25. From a +/bd ♂ in the 71i21 Porch collection, South Amherst natural population. A medial bald spot, semi-circular in shape, and about 1/3 the width of the thorax, extends back slightly from the anterior dorsal edge of the thorax which is somewhat reduced in size. Very little variation at 25°C. Locus from test with Bl $L^2$, 1003 flies giving 13 crossovers between Bl and bd, 175 between Bl and L, and 539 $bd^+$ to 464 bd in all classes combined. Not tested for locus with respect to the many other recessives in this region. Rk 1.

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

$g^{tuh-1}$: garnet from tumorous-head, #1 1-44.4. A spontaneous mutation observed in a single male from a tumorous-head strain originally maintained at the California Institute of Technology (tuh(CT)). Tests for allelism place the mutation at the garnet locus. The site within the pseudoallelic locus was not determined. Viability and fertility are good.

$g^{tuh-2}$: garnet from tumorous-head, #2 1-44.4. A spontaneous mutation observed in several males taken from a tuh(CT) strain in which Urbana 2nd chromosomes had been placed. Phenotypically indistinguishable from $g^{tuh-1}$. Tests for allelism place the mutation at the garnet locus. The site within the pseudoallelic locus was not determined. Viability and fertility are poor.

Report of I. Schwinck and L. Schwinck
University of Connecticut, Storrs, Conn.

$g^{70k}$: $garnet^{70k}$ I. Schwinck and L. Schwinck, 1970k10. The spontaneous new eye color mutant appeared in an inbred cinnabar-rosy stock (cn; $ry^2$) which had been made isogenic in 1960. In combination with cinnabar and rosy the eye color is an extremely light yellow; the biochemical characteristics of rosy are epistatic; the effect on the eye color is additive like in the previously described new mutant $orange^{66k}$ (DIS 44:46). The viability of the original $g^{70k}$;cn;$ry^2$ stock was very poor. By outcrossing the new mutant with a coisogenic cinnabar and later with a wild-type Canton-S stock, a $g^{70k}$;cn stock and a $g^{70k}$ stock were obtained, both of excellent viability. The allelism to garnet (1-44.4, Lindsley and Grell, 1968) was established by crossing to a g;cn stock and to a $g^{50e}$ stock, both from D.F. Poulson's stock cultures. The eye color phenotype of $g^{70k}$ is similar but not identical with these g and $g^{50e}$ stocks - in all alleles the reddish brown eyes age to a more dark brown color. Two-dimensional chromatography on cellulose plates shows the presence of five separable drosopterins with a large fraction of a new orange-fluorescent spot chromatographically separable from drosopterin and isodrosopterin (I. Schwinck, Genetics 69:s59, 1971; I. Schwinck and M. Mancini, in press). This chromatographic drosopterin pattern indicates a certain similarity between the garnet alleles g, $g^{50e}$, and $g^{70k}$; however, some quantitative differences are found. Viability and fecundity of $g^{70k}$ excellent, RK1.

Report of M. Gans, M. Masson and D. Thierry-Mieg
Centre de génétique Moléculaire, C.N.R.S., Gif-sur-Yvette, France

$gs^{87}$: grandchildless 87 EMS-induced; recessive; thermosensitive. The homozygous females born from heterozygous mothers have normal viability and fertility, regardless of temperature.

When these homozygous females are raised and mated at 16°, their own progeny seem to be normal. But, when mated at 29°, either with $gs^{87}$ or with wild type males, their progeny male and female are often sterile. This sterility, as shown by dissection, is related to the absence of germ-line cells in the ovaries or testes. The average frequency of flies having two agametic gonads is about 0.50, and that of flies having one agametic and one gametic gonad is about 0.10. These frequencies are of the same order for both sexes.

The proposed interpretation is that most of the eggs laid at 29° by the homozygous females bear an anomaly in the region of the posterior pole, where the polar cells differenciate, and become the germ-line cells; thus the progeny are agametic and sterile.

This mutant is very similar to the grandchildless mutant of Drosophila subobscura.

Report of P. De Angelis
University of Milan, Italy

depilated Spontaneous, found in a wild stock of unknown origin. Thorax and head bristles are gradually lost during the first week after emergence. Hairs normal. Recessive located in the 3rd chromosome. Penetrance 100%, viability excellent.

Report of E.R. Vyse and A. James
Montana State University, Bozeman, Montana

$sd^{UCI}$: scalloped 1-51.5. A new EMS induced male sterile allele. This new allele has an extreme vestigal-like wing phenotype (similar to $sd^{58d}$) with erect postscutellar bristles. Halteres are greatly reduced in size. In some flies, outgrowths of tissue are found on one side of the metanotum. In extreme cases, these outgrowths can be recognized as duplications of the scutellum with some duplication of the wing bases and the reduction of the wing blade. The wing margins of $sd^{UCI}$/M-5 females are frequently excised. Heterozygous M-5/$sd^{UCI}$ females produce a significant number of patroclinus (XO)? males. Attempts to separate the mutation(s) which cause(s) these pleiotrophic effects have not been successful.

$ss^{a-UCI}$: spineless aristapedia 3-58.5. Mutation arose spontaneously in a C(1)Dx, y f/ y w sn $r^9$ stock. C(1)Dx, y f and the y w sn $r^9$ X-chromosomes have been replaced with an Ore R X-chromosome. Penetrance of this allele is 100% and expressivity is high. The 3rd antennal segment contains both leg and antennal bristles and the claw is usually abnormal.

Report of L. Gerace and W.H. Sofer
Johns Hopkins University, Baltimore, Maryland

$Adh^n$: Alcohol Dehydrogenase Negative Mutants EMS induced. These mutants were selected using the pentenol procedure of Sofer and Hatkoff (1). $Adh^{n6}$ to $Adh^{n10}$ were induced in b, cn, vg flies (originally carry the $Adh^F$ allele). All these lack detectable alcohol dehydrogenase activity as judged by a spot test assay, and by susceptibility to ethanol poisoning. They are homozygous viable. In addition, all of these are non-complementing and are non-complementing with $Adh^{n1}$ to $Adh^{n5}$ of Grell et al. (2). Mutants $Adh^{nA}$ and $Adh^{nB}$ were induced in the balancer chromosome CyO and are carried as the stock Df(2L)64j/CyO. They too show no detectable alcohol dehydrogenase activity.

| | |
|---|---|
| $Adh^{n6}$ | Df(2L)64j/CyO, $Adh^{nA}$ |
| $Adh^{n7}$ | Df(2L)64j/CyO, $Adh^{nB}$ |
| $Adh^{n8}$ | |
| $Adh^{n9}$ | |
| $Adh^{n10}$ | |

References: (1) Sofer, W.H. and M. Hatkoff 1972, Genetics (Nov.); (2) Grell, E., K.B. Jacobson and J.B. Murphy 1968, Ann. N.Y. Acad. Sci. 151:411.

Report of J.L. Ménsua
Universidad Autónoma, Barcelona, Spain

pyd$^{66}$: polychaetoid$^{66}$ Appeared in a stock selected for high number of dorsocentral macrochaetae. Allele of pyd. Very good expression. Extra macrochaetae especially in dorsocentrals, scutelars, verticals and orbitals. Enhance the number of abdominal, sternopleural and interocellar microchaetae also. (See Ménsua, 1967). RK1

## MELANOGASTER - LINKAGE DATA

Report of R.H. Maddern
University of Adelaide, S.A., Australia

brc: brachymacrochaetae Fahmy 1958 DIS 32:68, has been localised to a position between su(s) and tw by the use of duplicated fragments of the X chromosome carried on a Y chromosome. Although brc results in the loss of some thoracic bristles, in the presence of a particular $y^2$ su(s)$^+$ Y duplication (from Professor M.M. Green's laboratory) which also has its break point between tw and su(s), extra scutellar and dorsocentral bristles are present in all flies. This effect does not occur in combination with other such duplicated fragments.

Report of A. Robertson and J.M. Malpica
Institute of Animal Genetics, Edinburgh, Scotland

Lindsley and Grell give eyg 3-35.5, Est-6 3-36.8, though Franklin (DIS 47:113) has revised the latter to 35.9 ± 0.5. A 3-point test, segregating from h eyg st/+ + + ♀ in two backgrounds and h + st/+ eyg + ♀ in one, gave heterogeneity between backgrounds, the position of eyg being 39.3, 36.1 and 38.1 respectively, each with a standard error of 0.5. In a further test with segregation from h eyg Est-6S st/+ + Est-6F + ♀, crossovers between eyg and st were homogeneous for Est-6. Of 44 crossovers between h and eyg, 4 were between Est-6 and eyg. eyg is therefore to the right of Est-6, probably between 3-37 and 3-38.

## DROSOPHILA SPECIES - STOCKS

See note concerning stock lists on page 34

## ANNOUNCEMENTS II

The 15th Drosophila Research Conference will meet at Northern Illinois University, DeKalb, Ill. 60115, on May 4, 5 and 6, 1973. If you are interested in attending but not listed in the most recent DIS Directory, write to Dr. S. Mittler or Dr. J. Bennett, Dept. of Biological Sciences.

The Isozyme Bulletin: Contributions, orders, and requests for information should be sent to Ross MacIntyre, Section of Genetics, Development & Physiology, Cornell University, Ithaca, New York, 14850. Orders should be paid in advance ($3.00 USA, $3.50 foreign). Contributions should follow the DIS format.

Tribolium Information Bulletin-16 - call for contributions from investigators of Tribolium and other Coleoptera. Deadline March 1, 1973. Departments are stock lists (up to date please!), new mutants reports, technical, research and teaching notes and announcements, current bibliography, directory information and orders. Until the subscription list grows, the price will continue to be $10.00. Please ask your library to subscribe also. Send material to Dr. A. Sokoloff, School of Natural Sciences, California State College, San Bernardino, California 92407. Late contributions will be included in TIB-17.

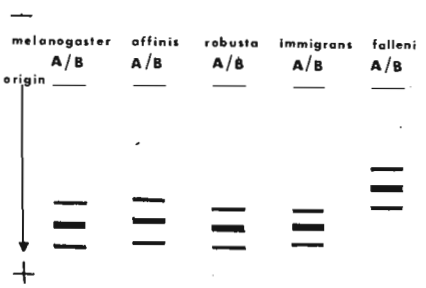
Report of D. Policansky
University of Oregon, Eugene, Oregon

w: white Discoverer D. Policansky. Sex-linked and undoubtedly homologous to white of melanogaster. No other information available.

Report of G.E. Collier and R.J. MacIntyre
Cornell University, Ithaca, New York

The following electrophoretic variants of soluble α-glycerophosphate dehydrogenase were detected by cellulose acetate electrophoresis using a Beckman Microzone Cell. A continuous buffer system of one part Tris-Barbital-NaBarbital, 0.05 ionic strength, pH 8.8; one part 0.2M Tris-HCl, pH 8.3; two parts water was used. Electrophoresis was carried out for 1 hour at 300 v. The isozymes were visualized using the procedure of O'Brien and MacIntyre (Am. Nat. 103:97-113, 1969) with the modification of solidifying the staining mixture in 1 1/2% agar to reduce the effects of diffusion.

All of the variants are naturally occurring, autosomal, co-dominant alleles presumably of the structural gene for the soluble enzyme (see O'Brien and MacIntyre, Biochem. Gen. 7:141-161, 1972). All of the variants were isolated from populations found in the Ithaca, N.Y. area.




falleni

αGPDH-$^{A}$: α-glycerophosphate dehydrogenase-$1^{A}$ Collier, 1972.

αGPDH-$^{B}$: α-glycerophosphate dehydrogenase-$1^{B}$ Collier, 1972. The A allele is the more common allele. Eighteen individuals from four different collecting sites were homozygous for this allele. One of five females from a fifth collecting site was heterozygous for the B allele. αGPDH-$1^{A}$ migrates with a mobility of 0.74 relative to melanogaster αGPDH-$1^{A}$. αGPDH-$1^{B}$ migrates with a mobility of 0.50 relative to melanogaster αGPDH-$1^{A}$.

robusta

αGPDH-$1^{A}$: α-glycerophosphate dehydrogenase-$1^{A}$ Collier, 1972.

αGPDH-$1^{B}$: α-glycerophosphate dehydrogenase-$1^{B}$ Collier, 1972. The A allele is the more common allele. It occurred with a frequency of 0.97 in a sample of seventeen flies. αGPDH-$1^{A}$ migrates with a mobility of 0.77 relative to melanogaster αGPDH-$1^{A}$. αGPDH-$1^{B}$ migrates with a mobility of 1.00 relative to melanogaster αGPDH-$1^{A}$.

immigrans

αGPDH-$1^{A}$: α-glycerophosphate dehydrogenase-$1^{A}$ Collier, 1972.

αGPDH-$1^{B}$: α-glycerophosphate dehydrogenase-$1^{B}$ Collier, 1972. In a sample of 53 flies the allele occurred with a frequency of 0.98. αGPDH-$1^{A}$ migrates with a mobility of 0.77 relative to αGPDH-$1^{A}$ of melanogaster. αGPDH-$1^{B}$ migrates with a mobility of 0.95 relative to αGPDH-$1^{A}$ of melanogaster.

affinis

αGPDH-$1^{A}$: α-glycerophosphate dehydrogenase-$1^{A}$ Collier, 1972.

αGPDH-$1^{B}$: α-glycerophosphate dehydrogenase-$1^{B}$ Collier, 1972. The A allele occurred with a frequency of 0.96 in a sample of 33 flies. αGPDH-$1^{A}$ migrates with a mobility of 0.72 relative to αGPDH-$1^{A}$ of melanogaster. αGPDH-$1^{B}$ migrates with a mobility of 0.95 relative to αGPDH-$1^{A}$ of melanogaster.

Trippa, G., C. Barberio, A. Loverre and P. Santolamazza. Istituto di Genetica, Universita di Roma, Italia. Phosphoglucomutase (PGM) polymorphism in natural populations of Drosophila melanogaster.

The distribution of phosphoglucomutase (PGM) polymorphism has been investigated in seven natural Italian populations. The samples were collected in Puglia (Castellaneta, Otranto and Corato) and Sicily (Ranna, Pedalino, Vittoria and Archi) during September-October 1971. The zymograms of single fly homogenates, following the technique by Spencer et al. (1964) and modified by Trippa et al. (1970) for single fly homogenate, show that the wild populations contain six anodic forms of PGM, each showing different electrophoretic mobility.

In addition to the more common and already described alleles $Pgm^A$ and $Pgm^B$ (Trippa, G. DIS 46:42, 1971) four new alleles have been found. They have been called $Pgm^C$, $Pgm^D$, $Pgm^E$ and $Pgm^F$ (Trippa, G., New Mutants, this issue). The corresponding forms migrate from the cathode to the anode in this order: $Pgm^E$, $Pgm^B$, $Pgm^F$, $Pgm^A$, $Pgm^C$ and $Pgm^D$.

Before PGM phenotype determination all males were crossed with females homozygous for a known Pgm allele. The frequencies of the PGM phenotypic classes were the expected ones in all the offspring of those males who turned out to be carriers of new Pgm alleles.

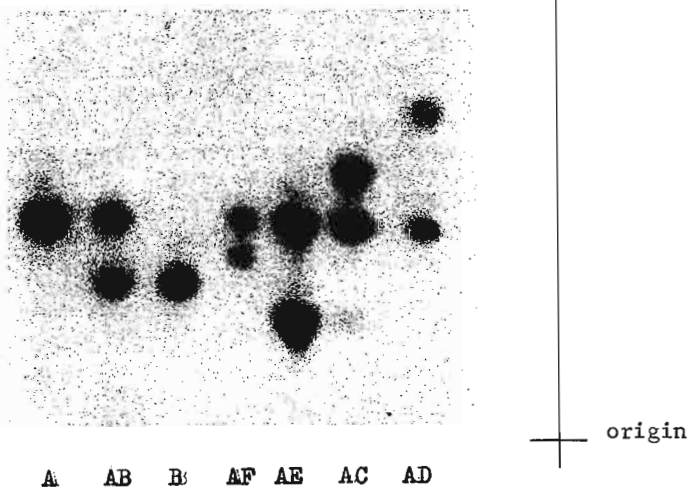



Fig. 1. The phosphoglucomutase patterns of the different genotypes observed in the present survey.

The observed PGM phenotypic values and the allele frequencies found in the seven natural populations are shown in Table 1. The chi-square test for the Hardy-Weinberg equilibrium in the populations, where it was possible, showed no significant difference between the values for the observed against the

| Populations | PGM phenotypes A | AB | B | AC | AD | BC | AE | TOT | Gene Frequencies |
|---|---|---|---|---|---|---|---|---|---|
| Castellaneta | 177 | 28 | 4 | 3 | 1 | - | - | 213 | $Pgm^A$=90.6 ± 1.38 $Pgm^B$= 8.5 ± 1.34 $Pgm^C$= 0.7 ± 0.40 $Pgm^D$= 0.2 ± 0.21 |
| Otranto | 258 | 29 | 1 | 2 | 5 | 1 | - | 296 | $Pgm^A$=93.2 ± 1.03 $Pgm^B$= 5.4 ± 0.93 $Pgm^C$= 0.5 ± 0.29 $Pgm^D$= 0.8 ± 0.37 |
| Corato | 207 | 24 | - | 1 | - | - | 1 | 233 | $Pgm^A$=94.4 ± 1.06 $Pgm^B$= 5.2 ± 1.03 $Pgm^C$= 0.2 ± 0.20 $Pgm^E$= 0.2 ± 0.20 |
| Ranna | 184 | 17 | - | 4 | 1 | - | - | 206 | $Pgm^A$=94.7 ± 1.10 $Pgm^B$= 4.1 ± 0.97 $Pgm^C$= 1.0 ± 0.48 $Pgm^D$= 0.2 ± 0.22 |
| Pedalino | 143 | 19 | - | - | - | - | - | 162 | $Pgm^A$=94.1 ± 1.31 $Pgm^B$= 5.9 |
| Vittoria | 31 | 11 | 4 | - | - | - | - | 46 | $Pgm^A$=79.3 ± 4.22 $Pgm^B$=20.7 |
| Archi | 197 | 3 | - | - | - | - | - | 200 | $Pgm^A$=99.2 ± 0.44 $Pgm^B$= 0.8 |

Table 1. Distribution of PGM phenotypes and allele frequencies in the seven wild populations.

expected genotypes.

It is interesting to note that all the populations are polymorphic for the Pgm gene and that in all samples the more frequent alleles are the same, i.e., $Pgm^A$ and $Pgm^B$. The other alleles, when present, show very low values.

A more detailed paper, discussing the possible mechanisms for the interpretation of these data is in preparation.

References: Spencer, N., D.A. Hopkinson and H. Harris 1964, Nature 204:742-745; Trippa, G., C. Santolamazza and R. Scozzari 1970, Biochem. Genet. 4:665-667.

Dübendorfer, A. and G. Shields. University of Sussex, Brighton, England. Proliferation in vitro and in vivo of a cell line originally derived from imaginal disc cells.

I. Schneider (1972) reported that the in vitro imaginal disc cell lines she had obtained from old embryos of Drosophila melanogaster had lost the ability to metamorphose when transplanted into larval hosts, although cells from primary cultures ("spheres") could do so. We confirmed these results using cells of her line 3 which had been maintained in our laboratory for 1 1/2 years in Schneider's medium. When injected into late third instar larvae these cells continued multiplication throughout metamorphosis and subsequent adult life of the host.

The same cells, when transplanted into young adult females, proliferated rapidly and killed the host after 9 to 10 days. The implants were very similar in appearance to atelotypic imaginal disc lines (Hadorn, 1969) and neoplastic cell lines derived from l(2)gl brain tissue (Gateff and Schneiderman, 1969). They could be returned to in vitro culture after 3 transfer generations in vivo (3 weeks) without apparent change in cell form and multiplicative ability.

Recently, we also obtained a fast growing cell line in vivo which appears to be similar to atelotypic imaginal disc tissue but originated from a cell culture set up in vitro after the method of Shields and Sang (1970) from 6-8$^h$ old embryos. After 3 weeks of culture in vitro, a sample has been transplanted into an adult female where rapid cell multiplication took place. We are now maintaining this line in vivo at 17$^o$C.

References: Gateff, E. and H.A. Schneiderman 1969, Nat. Cancer Inst. Monogr. 31:365; Hadorn, E. 1969, Nat. Cancer Inst. Monogr. 31:351; Schneider, I. 1972, J. Embryol. Exp. Morph. 27:353; Shields, G. and J.H. Sang 1970, J. Embryol. exp. Morph. 23:53.

Paterson, H.E. and N. Monzu. University of Western Australia, Nedlands, W.A., Australia. New records of Drosophila nicholsoni Malloch and D. nitidithorax Malloch from Perth, Western Australia.

Malloch (1927) described two new species of Drosophila from Perth, Western Australia, D. nitidithorax and D. nicholsoni. There are no later records in the literature.

In July 1970 students of the Department of Zoology, University of Western Australia, surveyed the Drosophila species occurring within a 50 km radius of Perth. Fourteen sites were sampled using banana and fermenting wheat bran baits. At eight of the sites D. nitidithorax was recorded, usually at low density, but commonly at Quinn's Rock (40 km N. of Perth, on the coast). Banana bait proved attractive to this species, but it has been found subsequently on liver bait in blowfly traps. It is a member of the subgenus Scaptodrosophila and has been found to have typical six filament eggs and "skipping" larvae. It can be maintained on standard medium.

D. nicholsoni is an unusual species having only one dorsal ray on the arista. A specimen was caught indoors in Nedlands, a Perth suburb, during June 1971. Its relationships are not certain.

The type of D. nitidithorax has been destroyed, but the description agrees well with the specimen collected. The type of D. nicholsoni is still in existence though somewhat damaged. However, there is no question about the conspecificity of the new material with the type.

Reference: Malloch, J.R. 1927, Proc. Linn. Soc. N.S.W. 52:1-15.

Note added in proof: Since the above was written a further five specimens of D. nicholsoni were recovered from a large collection of insects taken by sweeping at York, Western Australia, about 60 miles inland from Perth.

David, J., R. Allemand and P. Fouillet.
University Claude Bernard, Lyon, France.

On the duration of the fertilization and of the waterproofing process of the egg in the genital duct of Drosophila female.

When the egg (mature oocyte) migrates from the ovaries to the uterus, it is fertilized by a spermatozoon coming from the ventral receptacle of the female. At the same time, a waterproofing of the external surface takes place. This can be easily demonstrated by the fact that, in the ovarioles, mature eggs are always highly permeable to distilled water, dyes or fixatives. However, eggs taken from the uterus are always strictly impermeable, so that the chorion can be removed by hypochloride without affecting the developing embryo.

The impermeability is determined by the deposition of a thin waxy layer below the chorion on the surface of the vitellin membrane. This is not a consequence of fertilization, because it occurs in the same way in virgin females. The origin of this wax is still a matter of speculation, as is also the duration of the waterproofing process. Concerning the latter, some details may be obtained by studying the kinetics of oviposition.

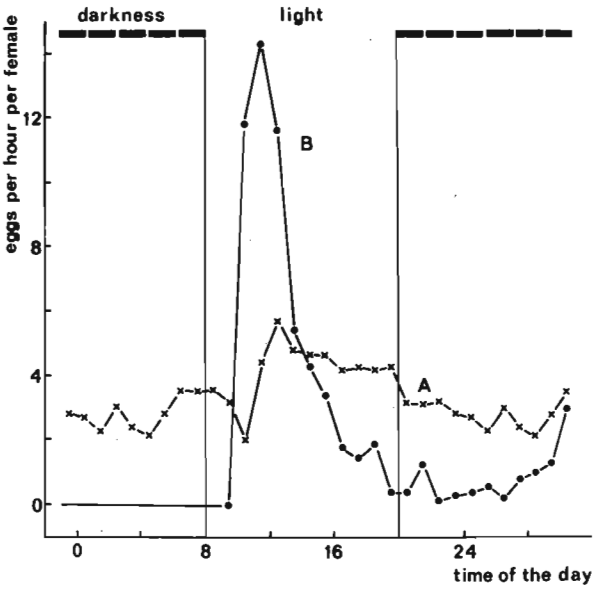



Figure: Hourly record of egg production by D. melanogaster at 25°C. A: curve of normally fed females (control). B: females starved for 24 hours, having mature oocytes in their ovaries, transferred to fresh food at 10 o'clock.

Under the optimal conditions, a female of Drosophila melanogaster can lay a little more than 120 eggs per day. In this species the circadian rhythm of oviposition is slight (David and Fouillet, 1972); so that the duration of fertilization and waterproofing can be estimated as being no more than 10 minutes. However it is possible to obtain more accurate data by inducing the deposition of a great number of eggs in a minimum period of time.

For this purpose, well-fed females, producing about 90 eggs per day, were deprived of food during 24 hours. Under these conditions, oviposition stopped completely, although the developing oocytes in the ovaries reach maturity. Many mature eggs in retention, sometimes more than 100 for a female, were thus observed in the ovarioles. After starvation, the flies were transferred to fresh food and the hourly egg production was recorded, using the technics of David and Fouillet (1972). Under such conditions, the females quickly laid the mature oocytes and it is probable that fertilization and waterproofing were the only limiting processes of the rate of oviposition.

The data (hourly curves of egg laying) are presented in the figure. Oviposition clearly reached a maximum of about 15 eggs per hour. Therefore it may be concluded that the minimum duration of the fertilization and of the waterproofing process at 25°C is only 4 minutes.

Moreover, when mature oocytes are in retention in the ovaries for some time, their volume decreases and their surface appears shrunk. This is probably the consequence of an osmotic phenomenon. The internal osmotic pressure of the oocyte cytoplasm is probably lower than that of the hemolymph. But, when such eggs are laid, they always appear normal and swollen. Therefore, it must be concluded that penetration of water, restoring a normal turgescence, takes place probably in the uterus, before the deposition of the waxy impermeable layer.

These observations illustrate the complexity and the rapidity of the changes taking place in the oocyte when it enters the uterus.

Reference: David, J., P. Fouillet, Enregistrement continu de la ponte chez D.m. et étude du rythme circadien d'oviposition. Rev. Comp. anim. (in press).

Picard, G., A. Bucheton, J.M. Lavige, A. Fleuriet, J.C. Bregliano and Ph. L'Heritier.
Université de Clermont-Ferrand, Aubière, France. Further data on non-Mendelian female sterility in D. melanogaster.

In previous notes (DIS 46:54, C.R. Acad. Sc. Paris 272:2484, 1971 and 275:933, 1972) we reported the discovery of a female sterility consequent to the cross between two laboratory strains of Drosophila melanogaster; the first one bears the gene sepia (se), the other one is the well known wild type Oregon strain. Most of the eggs laid by the $F_1$ females from the cross se ♀ by Oregon ♂ fail to hatch, not going beyond the blastoderm stage. This sterility can be characterized by two peculiar physiological features. First, more eggs hatch when females grow older. Second, the sterility vanished nearly completely, albeit reversibly, when egg-laying females were put at 30°C. $F_1$ females from the reciprocal cross, Oregon ♀ by se ♂, and males from both crosses, are normally fertile.

We are reporting here the first chief points which have come out from the work on this phenomenon in our laboratory.

I - The three groups of strains:

Quite a number of strains, some being laboratory strains from "Le laboratoire de Génétique des virus du C.N.R.S., Gif-s/Yvette, France" and others coming from flies caught in the wild, were assayed for their behaviour in the crosses with se and Oregon strains. They could be separated into three groups:

a) Inducer strains. They behave like the Oregon strain. They can intercross with this strain, without any sterility in the offspring, and sterile $F_1$ females arise when they are used as males in crosses with sepia females. All the strains (30) from the wild are inducer.

b) Reactive strains. They behave like the sepia strain. They breed with it, without bringing out any sterility, but produce sterile females when crossed with any inducer male.

c) Neutral strains. They breed freely either with the Oregon or the sepia strain. The tested laboratory strains are distributed in the three groups.

II - Genetic investigations:

The sterility is met again in the generations which follow the original cross, allowing genetic investigations to be carried on.

Sterile females arise when two factors, which have been called R and I (reactive and inducer factors) meet in the same fly. They are propagated in isolation, in the original strains. The two factors appear to be inherited in quite different ways.

A) Inheritance of the R factor. R is transmitted normally by maternal inheritance, and is independent of chromosomal segregations. An important complication arises, however, from the existence of a large variability in the R factor. A reactive female may be characterized by the mean hatching frequency of the eggs laid by its daughters by an inducer male. On this basis, it is observed that most of the reactive strains hold a mixture of "strong reactive" females whose daughters have but a very low fertility, and "weak reactive" females, which lead to a nearly normal fertility. Every kind of intermediate is apparently found.

The character responds to selection and "strong" as well as "weak" families were built artificially or found to have arisen spontaneously. Long lasting genetic stability seems however difficult, or perhaps impossible, to obtain, and spontaneous genetic drifts were frequently observed. These observations leave open the possibility that so called "neutral strains" are but very "weak reactive" strains, which may drift occasionally towards strongness.

Crosses were made between "strong" and "weak" reactive families with the following results. The character is inherited chiefly from the maternal side. A slight, but significant deviation towards the paternal side is however regularly observed. This implies that in crosses within the reactive strains group at least, the R factor may be transmitted by males.

B) Inheritance of the I factor. From but preliminary investigations, it appears that the I factor, though in some circumstances it follows segregation of the chromosomes, does not behave like a regular Mendelian gene (or genes). Indeed, chromosomes from reactive strains appear liable to acquire the inducer potentiality, independently of any specific recombination.

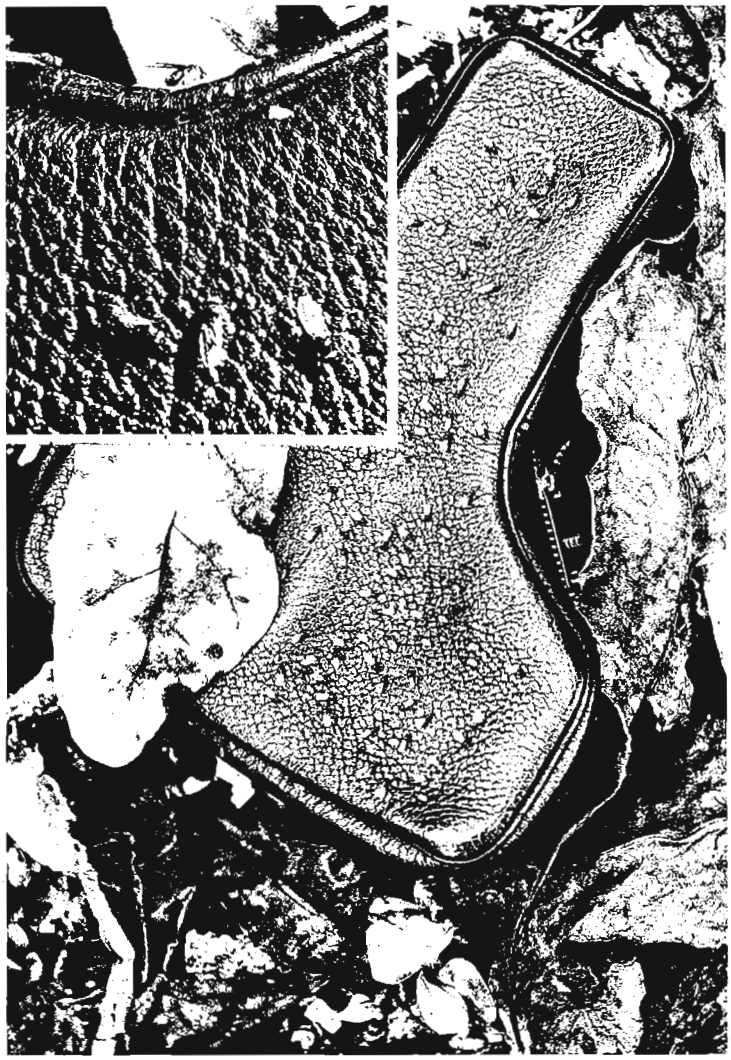
Rules of transmission of the two factors allow the possibility of introducing both of them in the same strain. Thus, "autosterile" strains are built in which females are more or less sterile and, with any kind of males, breed daughters with the same kind of sterility. Males of these autosterile strains are inducer.

In our current working hypothesis, we assume that the death of the eggs arises from a complementation between inherited symbionts.

Johnston, J.S. and R.H. Richardson.
University of Texas at Austin, Texas.

A unique sex-specific, species-specific attractant for a Hawaiian Drosophila.

Kipuka Puaula (Bird Park) on the island of Hawaii supports large numbers of 3 species of the "modified mouth-parts" group: D. imperasetae (Hardy 1965) and the sibling species D. mimica and D. kambysellisi (Hardy 1965, Hardy and Kaneshiro 1969). The males of D. kambysellisi were attracted in large numbers to a black leather case of a Honeywell Pentax 2° Spot Meter. The attractiveness was limited to this particular case and, to a lesser extent, to the case of a Honeywell Spotmatic Camera. Other leather cases, notably that on a Canon FT camera, a Topcon R.E. Super, and a ukelele case did not attract flies.



On August 10, 1972 at 3:00 p.m. the light meter case was placed in an area which had been collected with a net daily for 5 days. A sweep of the net for 1 meter in all directions along the ground produced an average collection of 7.6 D. mimica (6 ♂, 1.6 ♀), 5.4 D. imperasetae (2.6 ♂, 2.8 ♀) and 24.2 D. kambysellisi (8.0 ♂, 16.2 ♀). A collection at 5:00 p.m., however, made by aspirating flies off the spot meter case yielded 135 flies, all males and all D. kambysellisi. A separate collection in the same area was made by placing a net over the case and capturing the flies that crawled into the net. Three such collections in rapid succession produced 433 D. kambysellisi males and no other flies. When the case was moved 100 meters to an area where numerous D. mimica had been netted, but where D. kambysellisi had never been collected, a total of 7 D. kambysellisi males were attracted to the case. In light of the very restricted habitat of D. kambysellisi (Richardson and Johnston 1972) this represents a large departure from the normal distribution of the fly.

D. kambysellisi breeds in rotting leaves of Pisonia brunonianum (Heed, personal communication). Males are normally concentrated in the leaves above the ground (Speith 1966). In this instance, the males were attracted to the leather case placed directly on the ground, avoiding, in fact, Pisonia leaves placed on the case (see Figure). The nature of the attractant is not yet known. The sex-limited nature of the attraction suggests that the case has a pheromone-mimetic odor. A unique odor was evident and research to purify the responsible element is underway.

References: Hardy, D.E. 1965, Insects of Hawaii, Vol. 12, Diptera: Cyclorrhapha II, Series Shizophora, Section Acalypterae I, Family Drosophilidae, 814 pp. (Univ. of Hawaii Press); Hardy, D. Elmo and Kenneth Y. Kaneshiro 1969, Univ. of Texas Pub. 6918:39-54; Richardson, R.H. and J.S. Johnston 1972, Habitat selection and competition in Hawaiian Drosophila I. Description of behavior (In manuscript); Spieth, H.T. 1966, Univ. of Texas Pub. 6615:243-313.

Sreerama Reddy, G. and N.B. Krishnamurthy.
University of Mysore, Manasagangotri, India.

Ovoviviparity in Drosophila anomelani.

In insects fertilization and oviposition follow rapidly after ovulation. But in many instances the terminal oocytes of each ovariole may be ovulated and spend some time in the pedicel of the ovary or the oviducts before they are fertilized and oviposited. In certain others, the phenomenon of egg retention accompanied by ovariolar fertilization has resulted in the development of ovoviviparity, a method of reproduction common among muscoid flies (King 1963).

Such a departure from oviparity has been observed in Drosophila anomelani, a new species described by the authors (in press). When some females from the laboratory stock were dissected to study the structure of female reproductive organs, one female was shown to contain an active first instar larva in one of the ovarioles and the egg capsule in the oviduct (Fig. 1). Actually the authors observed under the binocular stereozoom the larva coming out of the egg retained in the oviduct. By the time the photograph (Fig. 1) was taken the larva had moved into one of the ovarioles and was trying to come out of the ovariole. Later this larva died when transferred to a food vial. This clearly shows that the fertilized egg has been retained in the oviduct and hatched liberating the larva.

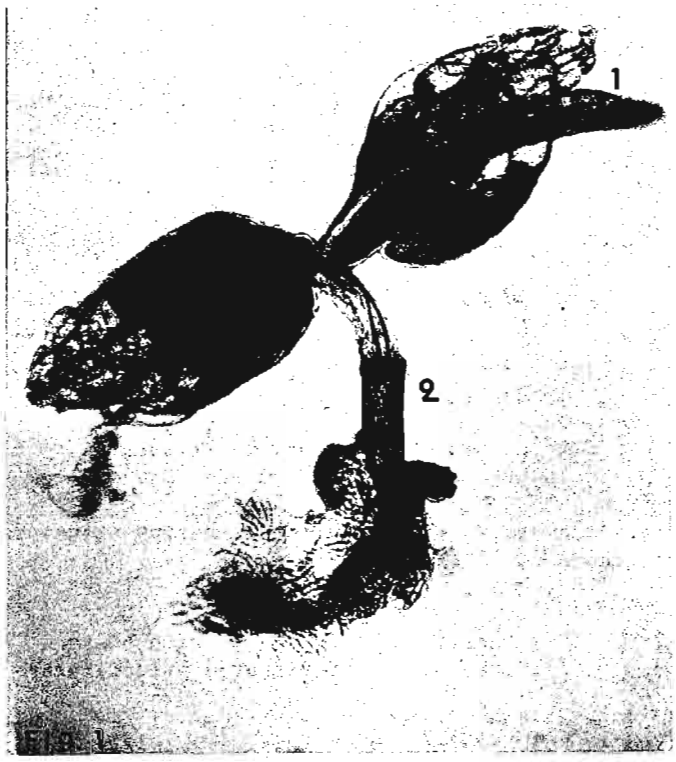



Fig. 1. Female reproductive organs with the first instar larva and the egg capsule.
1. first instar larva within the ovariole
2. egg capsule

The two conditions (a) egg retention and (b) ovariolar fertilization suggested by King (1965) are necessary for the development of ovoviviparity and has been observed in certain strains of D. melanogaster. Further he suggested that the inter-strain variation in egg retention in D. melanogaster virgin females gives some evidence of the existence of genotypes for the establishment of high egg retention and the development of ovoviviparity. It may be pointed out that departures from oviparity has also been observed in certain species like D. hydei, where a type of egg retention is established. This phenomenon of egg retention observed in D. hydei explains the capacity of these females to lay large numbers of eggs in short bursts from the reserve pools at the peak egg laying time (Gregg and Day 1965).

The present finding of an active first instar larva in one of the ovarioles in D. anomelani is an additional evidence in support of the existence of genotypes tending towards the development of ovoviviparity by egg storage and oviolar fertilization.

Acknowledgements: We are highly grateful to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology, University of Mysore, for his constant help and constructive criticisms. One of us (G. Sreerama Reddy) is thankful to University Grants Commission for financial aid.

References: Gregg, T.G. and J. Day 1965, DIS 40:85; King, R.C. 1963, DIS 38:96.

Zhimulev, I.F. and V.A. Lytchev.
Institute of Medical Radiology, Obninsk, Kaluga Region, U.S.S.R.

Some additional characteristics of the phenotype of ltl (lethal tumorous larvae) of D. melanogaster.

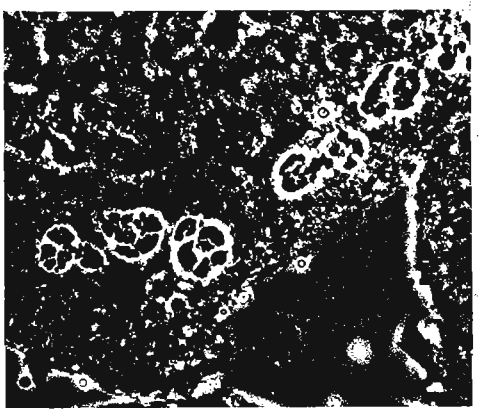
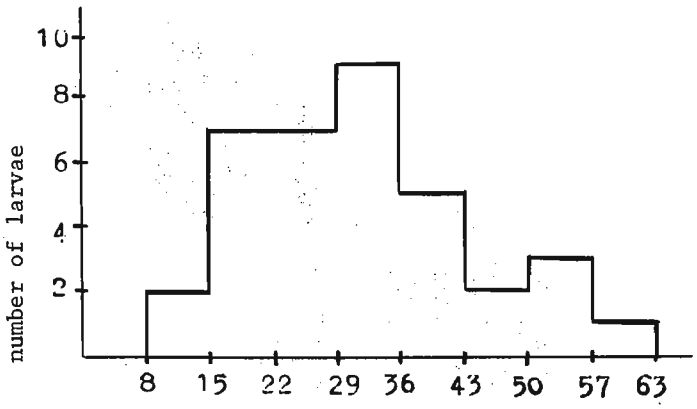


Fig. 1. Structures in the cell of salivary gland (phase contrast).

Alongside the great changes of chromosomes (see Kobel and Breugel, Genetica 38:305-327) in cytoplasm of some cells of salivary glands of nonpupating larvae of the ltl strain, unknown structures are detected (Fig. 1). These structures were observed in all studied larvae of 5, 7, 9, 11, 13 and 17 days old. In the cell, more frequently one such structure is observed, more




The number of cells having structures in one hundred cells of one larvae.

Fig. 2. Average quantity of cells having structures in glands of 11 days old larvae.

Fig. 3. Vacuoles in the salivary gland cell of 20 days of age larvae (phase contrast).

rarely up to 7. Cells with these structures are situated primarily in the proximal part of the gland, near the duct. The number of cells having such structures in the gland varies even in the larvae of one and the same age (Fig. 2).

In some larvae of 17 days of age and all the larvae of 20 days of age and older, in the cytoplasm big vacuoles are observed (Fig. 3).

The authors are very grateful to Prof. H.D. Berendes for sending flies of the ltl strain.

Maddern, R.H. University of Adelaide, S.A., Australia. Presence of a number of lethal mutants at the extreme tip of the X chromosome.

Many stocks of $dor^1$ (1(1)7) have accumulated a second lethal or semi-lethal (personal observation, and also noted by Dr. Rayle, personal communication), and in the stock in this laboratory a second lethal allelic to, or exposing the distal lethal in the balancer FM3 has been found. In addition three non-autonomous lethals 1403, 152 and 139 (kindly made available by Prof. E. Novitski) are also allelic to or expose this lethal. None of these lethals overlaps with 1(1)J1 or an ethyl methane sulphonate induced mutant which is allelic to it, but not associated with a visible cytological change. Both groups of lethals are covered by $1(1)J1^{+}Y$ and their relative order is not known.

Berger, E.M. State University of New York, Albany, N.Y. Isolation and dissociation of Drosophila ribosomes.

Drosophila virilis ribosomes isolated from adult homogenates (1) were found to have a sedimentation coefficient in sucrose gradients of about 80S; with respect to parallel standards including E. coli monosomes, and monosomal subunits. Drosophila ribosomes were completely dissociated after incubation in 0.01 M Tris, pH 7.3, 0.1 M KCL into subunits having $S_{20}$ coefficients of 40S and 60S. Proteins isolated from small and large subunits by the acetic acid method (2) were analyzed in 10% acrylamide, 0.1% SDS gels (3). In the small subunit 21 bands of staining were observed, while large subunits yielded 31 bands (Figure 1a and b).

One interesting effect on ribosome dissociation was noted when $Na_2$ EDTA, a chelating agent,

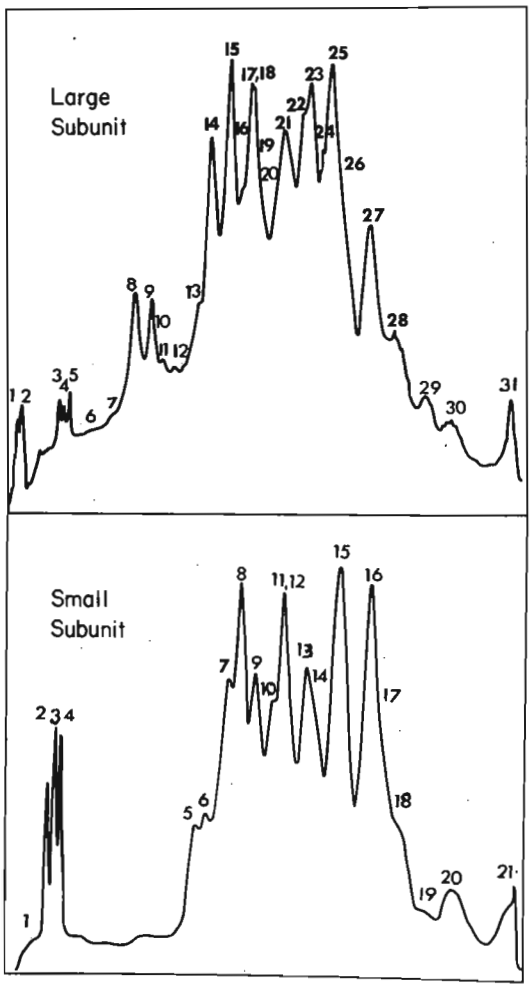



Figure 1. Densitometric scan of 40 S and 60S ribosomal proteins following SDS acrylamide gel electrophoresis. Migration is toward the right.

was included in the monosome dissociating buffer. With increasing EDTA concentration, the apparent sedimentation velocity of both subunits decreased (Figure 2). This reduction appears to involve a conformational change in the subunit structure induced by cation ($M_g$++ probably) removal since:

(1) The 260/280 ratios of the altered subunits are not affected.

(2) The protein spectra of altered subunits are indistinguishable from the OmM EDTA controls.

(3) The alteration can be reversed by adding $M_g$++ back to isolated subunits, and resedimenting them in sucrose, including in parallel tubes subunits dissociated in the absence of EDTA.

This work was supported by a grant from the NIH (GM 18910).

References: 1. Lambertson, A., S. Rasmuson and G. Bloom 1970, Molec. Gen. Genetics 108:349; 2. Leboy, P., E. Cox and J.G. Flaks 1964, PNAS, U.S. 52:1367; 3. Weber, K. and M. Osborn 1969, M.J. Biol. Chem. 344:4406.

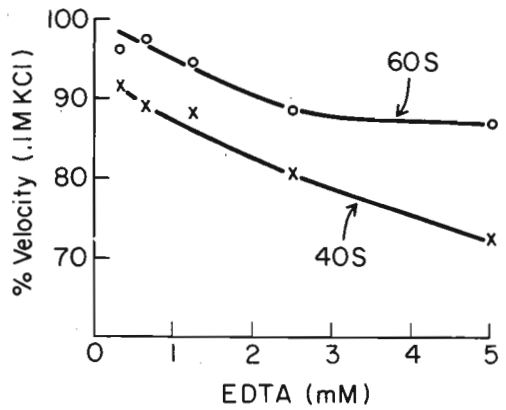



Figure 2. Reduction of ribosomal subunit sedimentation velocity following dissociation in varying concentrations of EDTA. Percent velocity is with respect to subunits dissociated in the absence of EDTA and sedimented in parallel gradient tubes.

Wargent, J.M. University of Sheffield, England. Position-effect variegation in the Revolute of Bridges strain of Drosophila melanogaster.

The strain Revolute of Bridges contains an inversion with breakpoints in section 52D5 of the euchromatin and section 40F of the heterochromatin in the salivary second chromosome. The wings of the flies show a variable mutant phenotype, expressed as upward or downward curling and variation in the angle at which they are held to the body.

The effect of temperature on mutant expression was determined by phenotypic comparison between 24 hour old adult females of genotype $Rev^B/vg^C$, bred entirely at 25°C, or bred at 14°C and allowed to eclose at 25°C. The phenotype was assessed on an arbitrary scale, ranging from Grade 0, representing no mutant expression, through to Grade 5, the maximal mutant expression observed. The results, shown in Table 1, indicate that low culture temperature enhances the expression of $Rev^B$.

Table 1

| | 25° | | 14° | |
|---|---|---|---|---|
| Grade of expression | No. of flies examined | % | No. of flies examined | % |
| 0 | 223 | 26.87 | 12 | 1.93 |
| 1 | 197 | 23.73 | 25 | 4.02 |
| 2 | 166 | 20.00 | 86 | 13.85 |
| 3 | 130 | 15.66 | 145 | 23.35 |
| 4 | 73 | 8.79 | 176 | 28.34 |
| 5 | 41 | 4.94 | 177 | 28.50 |
| Total No. of flies observed | 840 | | 621 | |
| | $\chi^2 = 455.74$ | n = 5 | $p < 0.001$ | |

The effect of the Y chromosome on the mutant phenotype was observed in 24 hour old progeny of the cross: XX; $Rev^B/vg^C$ ♀♀ x $\overline{XY}$; +/+ ♂♂. Grade 0 mutant expression, which is indistinguishable from wild type, was not scored since flies not carrying the $Rev^B$ chromosome segregate from this cross. It can be seen from the results in Table 2 that $Rev^B$ expression is

Table 2

| | XX; $Rev^B$ ♀♀ | | $\overline{XXY}$; $Rev^B$ ♀♀ | |
|---|---|---|---|---|
| Grade of expression | No. of flies examined | % | No. of flies examined | % |
| 1 | 261 | 45.63 | 112 | 40.29 |
| 2 | 126 | 22.03 | 107 | 38.49 |
| 3 | 103 | 18.01 | 47 | 16.91 |
| 4 | 53 | 9.26 | 10 | 3.60 |
| 5 | 29 | 5.07 | 2 | 0.72 |
| Total No. of flies observed | 572 | | 278 | |
| | $\chi^2 = 37.59$ | n = 4 | $p < 0.001$ | |
| | XY; $Rev^B$ ♂♂ | | XO; $Rev^B$ ♂♂ | |
| 1 | 302 | 46.97 | 128 | 23.10 |
| 2 | 171 | 26.59 | 185 | 33.39 |
| 3 | 101 | 15.71 | 133 | 24.00 |
| 4 | 43 | 6.91 | 72 | 13.50 |
| 5 | 26 | 4.04 | 36 | 6.50 |
| Total No. of flies observed | 643 | | 554 | |
| | $\chi^2 = 78.07$ | n = 4 | $p < 0.001$ | |

(Continues at bottom of next page)

Schäfer, U. University of Düsseldorf, Germany. Some observations on bobbed in Drosophila hydei.

There is good cytological evidence that in addition to the nucleolus organizer (N.O.) in the X chromosome, in both D. melanogaster (Cooper, 1959) and D. hydei (van Breughel, 1970), a second one is located in the short arm of the Y chromosome. However, contrary to the situation in D. melanogaster, XO males of D. hydei with a normal $bb^+$ X chromosome show the bobbed phenotype (Hess and Meyer, 1963; van Breughel, 1970). It seemed, therefore, possible to genetically confirm the localization of the N.O. in D. hydei by the use of different Y chromosome fragments.

Males of the following 6 different sex chromosome constitutions were used:
1) $X(bb^+)/T(A;Y^{TCN})$. The latter is an autosome-Y translocation chromosome which carries the entire short arm and a proximal part of the long arm of the Y chromosome. Cytologically this chromosome includes the sites for the lampbrush loops "tubular ribbons" (T), "clubs" (C), and "nooses" (N) (Hess and Meyer, 1968).
2) $X(bb)/T(A;Y^{TCN})$.
3) $X(bb^+)/T(A;Y^{ThP})$. Here the reciprocal half of the translocation chromosome described in 1) was used. It carries a distal part of the long arm of the Y chromosome with the loci for the lampbrush loops "threads" (Th) and "pseudonucleolus" (P).
4) $X(bb)/T(A;Y^{ThP})$.
5) Nondisjunction males with $X.Y^S$, the Y fragment containing only the short arm with the loop forming sites for the "nooses".
6) Nondisjunction males carrying $X.Y^{Th}$, in which the Y fragment comprises only the tip of the long arm including the site for the "threads".

Contrary to the expectation that only those males carrying fragments of the short arm of the Y chromosome would show the $bb^+$ phenotype, in all 6 cases the males were found to be wild-type.

Ritossa (1968) and Tartof (1971) have shown that the lack of a certain number of the ribosomal RNA cistrons (rDNA) could be compensated for in XO males of D. melanogaster. The results from the present experiments seem to indicate that such "compensation" does not occur in XO males of D. hydei, but does occur in D. hydei in the presence of various - even very small - fragments from different parts of the Y chromosome.

The situation becomes considerably more complex, however, in view of Hennig's report (1968) that XX and XXY females of D. hydei have 0.25% and 0.08% rDNA, respectively, in their total complements of DNA. Both types of females are, furthermore, phenotypically $bb^+$. On the basis of these measurements one would expect that, at a minimum, 0.125% of the total DNA in the XO males would be rDNA. Under such conditions, however, the observation that XO males in D. hydei show the bobbed phenotype while XXY females with less rDNA do not show bb is difficult to reconcile with the explanation of the bobbed mutation (Ritossa, Atwood, and Spiegelman, 1966).

A biochemical analysis of this problem is currently in progress.

References: van Breughel, F.M.A. 1970, Genetica 41:589; Cooper, K.W. 1959, Chromosoma 10:535; Hennig, W. 1968, J. mol. Biol. 38:227; Hess, O. and G.F. Meyer 1963, J. Cell Biol. 16:527; Hess, O. and G.F. Meyer 1968, Advanc. Genet. 14:171; Ritossa, F.M. 1968, P.N.A.S. 60:509; Ritossa, F.M., K.C. Atwood and S. Spiegelman 1966, Genetics 54:819; Tartof, K.D. 1971, Science 171:294.

(Continued from preceding page)

suppressed in females with a Y chromosome, and males lacking this chromosome show enhanced mutant expression.

Examination of the Malpighian tubules from third instar female larvae of genotype $Rev^B/lt$ revealed that the tubules are composed of yellow (wild type) cells and colourless cells similar to those of the light mutant.

These results suggest that the variable $Rev^B$ phenotype occurs as a result of position effect at the relavent locus, induced by the $Rev^B$ inversion, and that the same inversion is also responsible for position effect at the light locus.

Hanks, G.D. Indiana University Northwest, Gary, Indiana. Low level radiation as one causative component in sex ratios.

Some results observed in 1965 can now be interpreted. Studying humans and other vertebrate populations helped the author to gain understanding. Table 1 shows a peak percentage of females obtained in March of 1965 (61.8) when the male tested was Oregon-R. That was the highest value ever obtained for Oregon-R males. During March of 1965 a high value (56.2) was obtained for sepia males. Also during March of 1965 a high percent female value (55.7) was obtained for aristaless roughoid. In 1957 and 1958 at Oak Ridge National Laboratory the same stock gave a percent female value of 47.8 and 49.0. For the Oregon-R males the values obtained in Feb. 1960, Jan., March, May and June 1965 were all higher than typical. Typical values for males from the Oregon-R stock are about 53.5 to 54.5% females. Unusually high values were not obtained for the Canton-S stock in March of 1963 or dumpy, ebony males in April of 1965. Apparently, males from some stocks do not respond to the environment in the same way. Table 2 shows the observed accumulation and annual deposition of strontium-90 in the Northern Hemisphere from 1958 to 1970. It can be seen that the peak annual deposition comes in 1963 with 1964 in second place. The peak in cumulative deposition was almost reached in 1965 and was reached in 1966 and 1967. The rather close association of peak percentages of females in three stocks with cumulative deposition of strontium-90 suggests a cause and effect relationship. If one takes into account the well known effects of low level radiation on the sex ratio in

Table 1. Observed percentages of females at various times when different males were tested. Each male was tested thoroughly by mating with 3 to 5 test females.

| Date ♂♂ were first mated | % ♀♀ | No. ♂♂ | Type of ♂ tested | Location where tested |
|---|---|---|---|---|
| Feb. 1, 1960 | 55.4 | 18 | Oregon-R | Eugene, Oregon |
| Mar. 21, 1963 | 53.5 | 15 | " | Salt Lake City, Utah |
| June 8, 1964 | 54.5 | 24 | " | " |
| June 11, 1964 | 53.8 | 27 | " | " |
| Jan. 19, 1965 | 57.0 | 30 | " | " |
| Mar. 12, 1965 | 61.8 | 17 | " | " |
| May 14, 1965 | 56.6 | 16 | " | " |
| May 14, 1965 | 56.5 | 16 | " | " |
| June 9, 1965 | 58.2 | 10 | " | " |
| June 9, 1965 | 56.3 | 11 | " | " |
| Aug. 24, 1966 | 53.6 | 16 | " | " |
| Aug. 24, 1966 | 52.6 | 14 | " | " |
| Mar. 21, 1963 | 51.3 | 15 | Canton-S | " |
| 1957 | 47.8 | | aristaless, roughoid | Oak Ridge |
| Jan. 7, 1958 | 49.0 | | " " | " |
| Mar. 20, 1965 | 55.7 | 18 | " " | Salt Lake City |
| Mar. 12, 1965 | 56.2 | 16 | sepia | " |
| Apr. 11, 1965 | 49.9 | 13 | dumpy, ebony | " |

Table 2. Observed accumulation and annual deposition of Strontium-90 in megacuries in the Northern Hemisphere from 1958 to 1970.

| Cumulative deposition | | Annual deposition | |
|---|---|---|---|
| 1958 | 1.5 | 1958 | 0.66 |
| 1959 | 2.1 | 1959 | 0.97 |
| 1960 | 3.0 | 1960 | 0.23 |
| 1961 | 3.2 | 1961 | 0.31 |
| 1962 | 3.4 | 1962 | 1.26 |
| 1963 | 4.6 | 1963 | 2.31 |
| 1964 | 6.9 | 1964 | 1.46 |
| 1965 | 8.0 | 1965 | 0.69 |
| | | Predicted | |
| 1966 | 8.5 | 1966 | 0.2 |
| Predicted | | | |
| 1967 | 8.5 | 1967 | 0.1 |
| 1968 | 8.4 | 1968 | 0.05 |
| 1969 | 8.2 | 1969 | 0.02 |
| 1970 | 8.0 | | |

Note: Data came indirectly from the U.S. Weather Bureau, ESSA, and the Health and Safety laboratory of the AEC. The values were decay corrected.

man and considers other possible causes of the high percentages of females it is strongly suggested that fallout radiation (perhaps mainly beta radiation in the water supply) was the main cause of the unusually high percentage female values in Drosophila. It is recommended that only distilled or reconstituted distilled water (with only the helpful chemicals added) should be used for Drosophila experiments. There may be important implications of this finding for all living organisms.

Parádi, E.* University of Milano, Italy.
Establishment of a new cell line from Drosophila mutant embryo.

I made the primary cultures from 12-14$^{h}$ old T(Y;2)C, cn$^{3}$ Drosophila melanogaster mutant embryos, which contain a translocated Y chromosome (1). When younger embryos are used for the primary cultures, the cells are fragile during the dissection; on the other hand, with the older embryos the tissue fragments are too large and don't attach to the bottom of the vessel which is essential for the tissue cultures (2).

The embryos were kept at 25 ± 0.5°C. The Drosophila eggs were washed several times with sodium chloride, after it with 1% sodium chloride which contained a few drops of soap solution (to reduce the floating of the eggs). Then 10 min. treatment with 2.5% sodium hypochlorite solution to sterilize and remove the chorion. The embryos treated in this way were mechanically dissected by conical glass homogenizer. The cell groups settled on the bottom of the homogenizer, while the injured cells remained in the liquid.

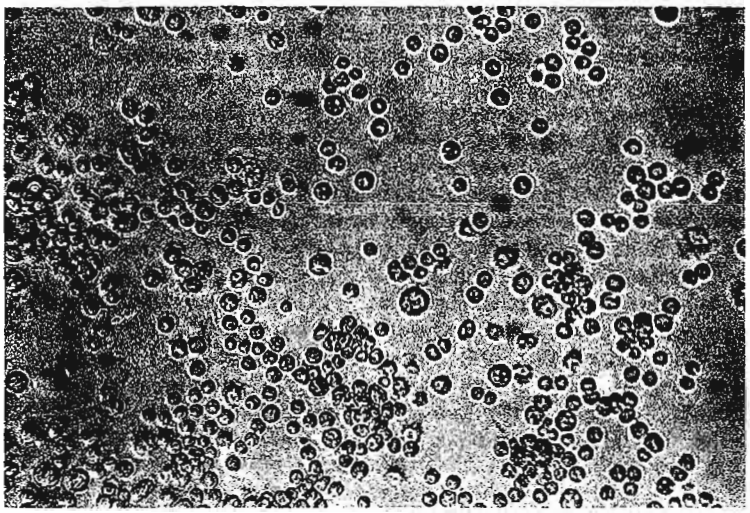


Fig. 1. Embryonic cells derived from T(Y;2)C, cn$^{3}$ Drosophila mutant. Living cells, x 280.

I used for the primary cultures the small flasks described by Vago and Flandre (3). The vessels containęd 0.3 ml D 225 medium (2) which was supplemented with 20% fetal calf serum.

The cultures were incubated at 25 ± 0.5°C. The medium was changed weekly; in this manner the cell aggregates which didn't attach to the bottom of the vessel were removed. For the first two weeks an intensive cell multiplication started. After it a rest period followed; in this period only some of the cell aggregates showed convulsions.

Three months later relatively small, round, light cells grew up from the cell aggregate because of the intensive cell division. First they were together, after it the cells dispersed and in one week they filled the flask. First the cells appeared in a single layer, then when they filled the flask they were inclined to aggregate.

The cells kept their mitotic activity in T-30 vessel, where they were transferred. The cell population is heterogeneous as to the cell size, but only round cells can be seen (Fig. 1).

According to the first preliminary examinations the obtained $IH_1$ cells are mainly tetra and octoploids.

The importance of this cell line is that the second autosome which contains the translocated Y chromosome can be detected readily, e.g. in fluorescence way, so thus the fate of this chromosome is readily detectable, e.g. in cell fusion experiments.

References: (1) Dobzhansky, Th. 1930, Biol. Zentr. 50:671; (2) Echalier, G., A. Ohanessian 1970, In vitro 6:162; (3) Vago, C. and P. Flandre 1963, Ann. Epiphyties 14:127.

* Present address: Dept. of Evolution and Genetics, Eötvös Lóránd University, Budapest, Hungary.

Chung, Y.J. Ewha Womans University, Seoul Korea. Study of alcohol dehydrogenase in the Korean natural populations of Drosophila melanogaster.

Since Johnson and Denniston (1964) and Grell et al. (1965) independently discovered the strains differing in the electrophoretic mobility of the alcohol dehydrogenase in Drosophila, a number of investigations have been done on this isozyme in D. melanogaster (Ursprung and Leone 1965, Rasmuson et al. 1966, Courtright et al. 1966, Jacobson 1968, Ursprung and Carlin 1968, Dunn et al. 1969). The present investigation was undertaken to study the alcohol dehydrogenase (ADH) isozyme in the natural populations of Drosophila melanogaster from various localities of Korea.

The flies were collected from the natural populations of D. melanogaster in 18 localities of Korea and kept in the constant temperature room (25 ± 1°C) until they were used in the experiment. The food used was a standard corn meal, yeast agar type with 0.5% propionic acid as a mold inhibitor. Six to seven flies were homogenized in a drop of a 1/20 dilution of the stock buffer EBT in a glass pestile homogenizer. The homogenate was absorbed on to a 1 x 8 mm piece of Whatman No. 1 filter. This filter paper was placed on the original (spotting) in the cellulose acetate strip (Nakai, EPI-4 5.7 x 14.5 cm). It was cut into a piece of 1.5 x 5.7 cm when used. This strip was immersed in Barbitone buffer for about 20 minutes and dried, and then was used so that the sample could be electrophoresed evenly on the surface of the strip. As electrode buffer, Barbitone buffer solution (pH 8.6) was filled in the tank and a voltage of about 250 V was applied, with a current of 0.8 mA/cm passing through the cellulose acetate strip. Electrophoresis was completed in about one hour. For detection of the ADH activity, the strip was placed in the staining mixture and the staining was completed in about 15 minutes. For fixation, the strip was left in a fixative for about five minutes at 37°C. They were then covered with filter paper and dried. The activity of the ADH isozymes was measured by densitometer and histogrammed, resulting in the figure.

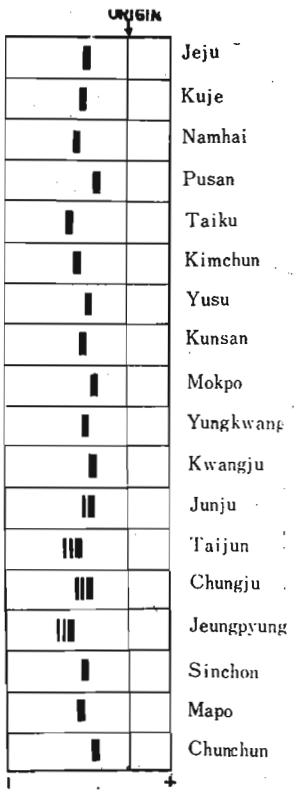



Figure 1. Alcohol dehydrogenase zymograms of D. melanogaster from 18 localities of Korea.

The results obtained from the present investigation show the difference in the ADH patterns of electrophoresis in various Korean natural populations of D. melanogaster as seen in Figures 1 and 2. The results are summarized as below:

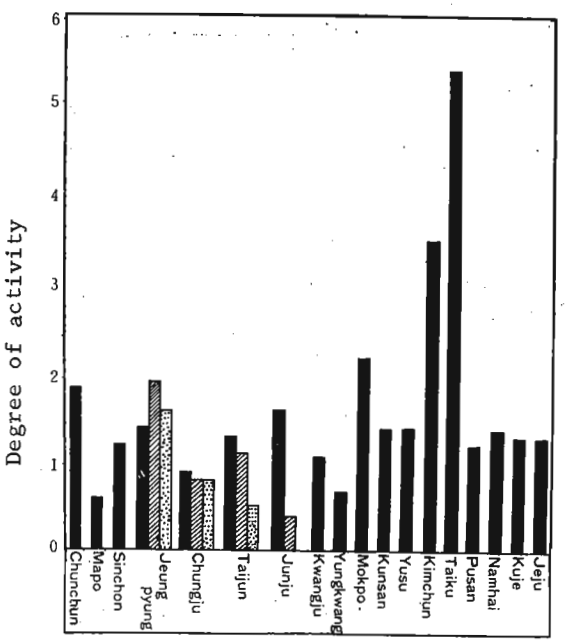



Figure 2. Activity of alcohol dehydrogenase isozyme of D. melanogaster from 18 localities of Korea.

1. The electrophoretic patterns of the ADH isozymes of Drosophila melanogaster is observed to be different among the strains from various localities (Chungju, Jeungpyung, and Taijun) show three bands, the Junju strain shows two bands and the remaining strains from 14 localities exhibit only one band in the zymograms.
2. The electrophoretic mobility is slower in the five strains (Mokpo, Yusu, Pusan, Chunchun,

and Kwangju-Chunnam), faster in the three strains (Taiku, Taijun, and Jeungpyung), and intermediate between them in the remaining strains from ten localities.
3. The activity of the ADH isozyme is extremely strong in the Taiku strain.
4. Such a difference of the ADH isozyme patterns, mobility, and activity among strains of Drosophila melanogaster from various localities of Korea implies that the genetic constitutions of the ADH isozymes is different among the strains from various localities of Korea.
5. The genetic analysis on this difference of the ADH isozyme patterns is going on now.

Baker, B.S. University of Washington, Seattle, Washington. Tests for chromosome breakage in the meiotic mutant paternal loss.

The recessive male meiotic mutant paternal loss (2-35.7) (pal = mei-W5 of Sandler, 1971) when homozygous in males causes frequent loss of paternal chromosomes (Baker, 1972). Loss may be either complete (nullisomic exceptions) or somatic during the early cleavage divisions of progeny of pal males. To examine whether chromosome loss might be the result of chromosome breakage the following tests were performed. (1) Muller-5 tests: 9 lethals were found among 1312 chromosomes recovered from pal/pal fathers in a Muller-5 test. From $pal^+/pal^+$ control males 4 lethals were recovered among 1299 chromosomes. An $F_2$ Muller-5 test gave 4 lethals/1154 chromosomes from pal males and 4 lethals/997 chromosomes from $pal^+$ males. (2) Translocation tests: $y/\underline{y^+YB^S}$;pal/pal males were crossed to $\underline{XY}$, EN(1)y/y, dl-49, Hw $m^4$ $g^2$;bw;st females and 495 $\underline{XY}/\underline{y^+YB^S}$ $F_1$ males were crossed to females of their mothers' genotype and one T(Y;3) was found. $\underline{XY}/y^+YB^S$;+/bw;+/st sons of the $F_1$ males were similarly tested for the presence of translocations and none found (454 males tested). (3) Male recombination. No recombinants between Pr(3-90.0) and Ly(3-40.5) were found among 5548 progeny of pal/pal;Pr Ly/++ males. (4) Isochromosomes. No new compound third chromosomes were found in a cross of 210 pal/pal;+/+ males to +/+;C(3L)RM, se $h^2$ $rs^2$;C(3R)RM, sbd gl $e^S$ females. (5) Dominant lethality. Egg hatch experiments revealed that there was 12-17% more dominant lethality in crosses involving pal/pal males than in crosses using females of the same stocks to SM1/pal or +/+ males (Table 1).

Table 1. Effect of pal on egghatch. Flies premated 3 days and only those matings that showed larvae in the premating vial were used to obtain eggs.

| | male | female | total eggs | % unhatched |
|---|---|---|---|---|
| 1. | pal/pal | Canton-S | 814 | 30.1 |
| | pal/SM1 | " | 985 | 12.4 |
| 2. | pal/pal | Canton-S | 1254 | 40.6 |
| | pal/SM1 | " | 1066 | 28.6 |
| | +/+ | " | 807 | 24.9 |
| 3. | pal/pal | $y/y;spa^{pol}/spa^{pol}$ | 561 | 21.0 |
| | pal/SM1 | " | 665 | 3.9 |
| | +/+ | " | 714 | 3.5 |

That this dominant lethality is probably due to sperm exceptional for the major autosomes and not chromosome breakage is suggested by the following considerations. Between 0.3 and 0.75 sperm nondisjunctional for one major autosome are recovered per male in cross of pal/pal; +/+ males to $X/X/B^SY$ females bearing either compound second or third chromosomes. Only about 1/6 of the ova produced by such females are complimentary to a given type of exceptional sperm (Grell 1970). Thus considering both major autosomes there are 3.6 to 9 autosomal exceptional sperm produced per pal male. As 120 progeny are produced per male in crosses to free autosome females using the same mating regime 3 to 7.5% of the recoverable sperm produced by a pal male are exceptional for one major autosome. Hence 18% (3/17) to 63% (7.5/12) of the dominent lethality results from aneuploidy for a major autosome. As these calculations do not take into account somatic loss of the major autosomes or the concomitant loss of both major autosomes it seems likely that all of the dominant lethality caused by pal is the result of aneuploidy for the major autosomes. In summary, all tests for chromosome breakage in pal males have been negative.

References: Baker, B.S. 1972 (in preparation); Grell, E.H. 1970, Genetics 65:65-74; Sandler, L. 1971, DIS 47:68.

Wright, C.P. Western Carolina University, Cullowhee, North Carolina. Oxygen consumption in glufultyrless-1, 1(1)EN7, a lethal mutant of Drosophila melanogaster.

Glufultyrless-1, 1(1)EN7 is a sex-linked lethal mutant of Drosophila melanogaster which was X-ray induced by Novitski (1963). Death in this mutant usually occurs in the pupal stage. Both weight and oxygen consumption measurements were made on individual male larvae and pupae from the first instar larval stage until the time when oxygen uptake ceased. Oxygen consumption measurements were made with small respirometers in a 25° 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 or pupa was determined. Control larvae and pupae 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 weights and rates of oxygen consumption in 1(1)EN7 larvae (Table 1) were significantly less than those in controls (Table 2). Control larvae formed puparia at 110 ± 17 hours after oviposition. About 60% of 1(1)EN7 formed puparia at 195 ± 60 hours. The remaining

Table 1. Average weights and rates of oxygen consumption for 1(1)EN7 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 | 0.0033 ± 0.0004 | 8 | 0.103 ± 0.006** | 8 | 31.174 ± 1.847 |
| 48 hr. | | did not weigh | 4 | 0.014 ± 0.001 | 8 | 0.237 ± 0.025** | 8 | 16.947 ± 1.786* |
| 72 hr. | 8 | 0.17 ± 0.01** | 8 | 0.05 ± 0.01** | 8 | 0.780 ± 0.076** | 8 | 15.426 ± 1.204** |
| 96 hr. | 9 | 0.39 ± 0.06** | 9 | 0.09 ± 0.01** | 9 | 1.244 ± 0.159** | 9 | 14.687 ± 1.828 |
| 120 hr. | 6 | 0.76 ± 0.06 | 6 | 0.14 ± 0.01 | 6 | 1.881 ± 0.056 | 6 | 13.848 ± 1.039 |
| 144 hr. | 4 | 0.89 ± 0.08 | 4 | 0.17 ± 0.03 | 4 | 2.064 ± 0.230 | 4 | 12.411 ± 0.895 |

* Significant at .05 level ** Significant at .01 level

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 |

1(1)EN7 larvae stayed in the larval stage showing increasing deterioration until death. Willis and Wright (1972) reported that histological abnormalities are observable in 1(1)EN7 larvae.

Throughout pupal development both fresh and dry weights of 1(1)EN7 (Table 3) were significantly less than in controls (Table 4). Dry weight was maintained at a fairly constant level, but fresh weight decreased steadily until at 152 hours after puparium formation 1(1)EN7 pupae were almost desiccated. Rates of oxygen uptake in 1(1)EN7 pupae (Table 3) followed to a certain extent the U-shaped curve of oxygen uptake in control pupae (Table 4). Controls emerged as adults at about 90 hours after puparium formation. Oxygen consumption in 1(1)EN7 decreased from 3 to 40 hours after puparium formation and then increased slightly until 80 hours, after which it decreased until none could be detected at 152 hours. Morphological development in 1(1)EN7 pupae reached its peak at 80 hours, with all pupae showing some adult structures such as head, legs or wings, but none of the pupae were completely developed. After this point a complete developmental breakdown occurred which resulted in death.

(Tables 3 and 4 on next page)

Table 3. Average weights and rates of oxygen consumption for 1(1)EN7 pupae

| Age in hr. after puparium formation | Fresh weight/ pupa in mg. | | Dry weight/ pupa in mg. | | $O_2$ consumption in cu. mm./ pupa/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. |
| 3 hr. | 8 | 0.68 ± 0.04** | 8 | 0.18 ± 0.01** | 8 | 0.971 ± 0.112** | 8 | 5.442 ± 0.548** |
| 20 hr. | 8 | 0.68 ± 0.02** | 8 | 0.23 ± 0.01** | 8 | 0.276 ± 0.071** | 8 | 1.243 ± 0.324** |
| 40 hr. | 8 | 0.57 ± 0.07** | 8 | 0.19 ± 0.01** | 8 | 0.130 ± 0.017** | 8 | 0.715 ± 0.131** |
| 60 hr. | 8 | 0.53 ± 0.04** | 8 | 0.18 ± 0.01** | 8 | 0.177 ± 0.015** | 8 | 1.001 ± 0.094** |
| 80 hr. | 8 | 0.50 ± 0.05** | 8 | 0.15 ± 0.01** | 8 | 0.230 ± 0.057** | 8 | 1.426 ± 0.274** |
| 104 hr. | 8 | 0.30 ± 0.04 | 8 | 0.14 ± 0.01 | 8 | 0.062 ± 0.021 | 8 | 0.465 ± 0.150 |
| 128 hr. | 8 | 0.22 ± 0.02 | 8 | 0.16 ± 0.01 | 8 | 0.033 ± 0.013 | 8 | 0.208 ± 0.072 |
| 152 hr. | 8 | 0.13 ± 0.01 | 8 | 0.11 ± 0.01 | 8 | 0.000 ± 0.000 | 8 | 0.000 ± 0.000 |

* Significant at .05 level ** Significant at .01 level

Table 4. Average weights and rates of oxygen consumption for y, w, spl, sn control pupae

| Age in hr. after puparium formation | Fresh weight/ pupa in mg. | | Dr. Weight/ pupa in mg. | | $O_2$ consumption in cu. mm./ pupa/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. |
| 3 hr. | 10 | 1.38 ± 0.02 | 10 | 0.38 ± 0.01 | 10 | 3.092 ± 0.076 | 10 | 8.244 ± 0.187 |
| 10 hr. | 10 | 1.19 ± 0.03 | 10 | 0.36 ± 0.01 | 10 | 2.213 ± 0.066 | 10 | 6.261 ± 0.211 |
| 20 hr. | 10 | 1.16 ± 0.02 | 10 | 0.34 ± 0.01 | 10 | 1.114 ± 0.037 | 10 | 3.340 ± 0.162 |
| 40 hr. | 10 | 1.17 ± 0.04 | 10 | 0.35 ± 0.01 | 10 | 0.842 ± 0.083 | 10 | 2.433 ± 0.097 |
| 60 hr. | 10 | 1.16 ± 0.02 | 10 | 0.35 ± 0.01 | 10 | 1.266 ± 0.049 | 10 | 3.620 ± 0.098 |
| 80 hr. | 10 | 1.14 ± 0.02 | 10 | 0.34 ± 0.02 | 10 | 1.864 ± 0.070 | 10 | 5.542 ± 0.194 |

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; Willis, D.E. and C.P. Wright 1972, DIS 48:32.

Thomasson, W.A. Chicago College of Osteopathic Medicine, Chicago, Illinois.

Juvenile hormone does not inhibit protein granule accumulation in larval fat body.

The fat body of many holometabolous insects, including D. melanogaster (1), accumulates large granules of protein during the hours before pupation. In the butterfly Calpodes ethlius this accumulation has been shown to be under the control of the molting hormone, ecdysone (2). Thomasson and Mitchell (3) have shown that in D. melanogaster, while ecdysone can induce granule accumulation, the granules appear too early for ecdysone to be the natural inducer. Furthermore, fat bodies incubated in vitro can form granules in the absence of ecdysone.

It was therefore speculated that the natural control might be an inhibition by juvenile hormone, with the granules appearing as soon as the juvenile hormone titer drops below a critical level. However, further experiments have now shown that addition to the cultures of "synthetic juvenile hormone (Calbiochem)" in concentrations as high as 0.06 mg/ml had no effect on granule formation.

The natural inducer in D. melanogaster therefore remains unknown. It is conceivable that the fat body responds to increasing protein concentrations in the hemolymph. Certain results reported in reference (3) are compatible with this possibility, though it was excluded as a possible mechanism in C. ethlius.

References: (1) von Gaukecker, 1963, Z. Zellforsch. 61; (2) Collins, 1969, J. Insect Physiol. 15; (3) Thomasson and Mitchell, 1972, J. Insect Physiol. 18.

Woodruff, R.C. University of Texas, Austin, Texas. A new sex-linked mutator gene in Drosophila melanogaster.

A new sex-linked mutator gene (Mu-$f^{3n}$) has been identified in Drosophila melanogaster. This gene significantly increases the frequency of reversion of the mutationally unstable mutant forked-3n ($f^{3n}$, 1-56.7) in females. The spontaneous reversion frequency of $f^{3n}$ in females in the presence of Mu-$f^{3n}$ is $8.7 \times 10^{-5}$ (13 revertants/149,074 $f^{3n}$ loci scored), whereas, the frequency is $2.0 \times 10^{-5}$ (2/100,499) in the absence of Mu-$f^{3n}$. Furthermore, the reduction in $f^{3n}$ reversion frequency in the absence of Mu-$f^{3n}$ in females ($2.0 \times 10^{-5}$) is similar to the $f^{3n}$ reversion frequency in males which contain the gene ($2/101,303 = 2.0 \times 10^{-5}$) (Woodruff, Bowman and Simmons, 1972). This suggests that Mu-$f^{3n}$ does not affect $f^{3n}$ reversion events in males. Green (1970) has reported a third chromosome mutator gene in Drosophila melanogaster which also functions only in females.

Other genetic properties of Mu-$f^{3n}$ are as follows: (1) It is a dominant mutant. (2) It is located on the X chromosome between $f^{3n}$ and Beadex-2 ($Bx^2$, 1-59.4). (3) Since Mu-$f^{3n}$ does not increase the spontaneous sex-linked-lethal frequency, it does not seem to be a general mutator. (The influence of Mu-$f^{3n}$ on the reversion frequencies of other mutants is currently being tested). (4) Preliminary data indicate that Mu-$f^{3n}$ reduces recombination on the proximal half of the X chromosome. The negative influence of Mu-$f^{3n}$ on recombination is shown in the accompanying table. The relationship between the influence of Mu-$f^{3n}$ on recombination and mutation is unknown.

Influence of the mutator gene, Mu-$f^{3n}$, on X-chromosome recombination

| Region | Expected recombination frequency[a] | Observed recombination frequency | |
|---|---|---|---|
| | | Mu-$f^{3n}$ present | Mu-$f^{3n}$ absent |
| un-$f^{36a}$ | 2.3% | ----- | 48/2,068 = 2.32% |
| un-$f^{3n}$ | 2.3% | 28/2,183 = 1.28%** | ----- |
| $f^{36a}$-B | 0.3% | ----- | 6/3,052 = 0.20% |
| $f^{3n}$-B | 0.3% | 4/7,240 = 0.06%**@ | 5/2,036 = 0.25%@ |
| $f^{36a}$-$Bx^2$ | 2.7% | ----- | 58/2,068 = 2.81% |
| $f^{3n}$-$Bx^2$ | 2.7% | 6/1,235 = 0.49%** | ----- |
| v-m | 3.1% | 65/3,206 = 2.03%**# | 100/3,010 = 3.32%# |

a Lindsley and Grell, 1968
** Significantly different from the expected frequency at the 1% level.
@ Frequencies are significantly different at the 5% level.
# Frequencies are significantly different at the 1% level.

References: Green, M.M. 1970, Mutation Res. 10:353-363; Woodruff, R.C., J.T. Bowman and J.R. Simmons 1972, Mutation Res. 15:86-89.

Research supported by NIH Genetics Training Grant 1029 and NIH Training Grant No. HD-00268 from the National Institute of Child Health and Human Development.

Tartof, K.D. Institute for Cancer Research, Philadelphia, Pennsylvania. A novel virginator stock.

I have constructed a temperature sensitive stock that will yield only female larvae, pupae or adults in large quantity, that permits the recovery of homozygous recessive markers for any chromosome and that does not involve the use of balancer chromosomes. This has been achieved by utilizing the second chromosome dominant lethal heat temperature sensitive mutant DTS-L7 discovered by Suzuki and Procunier (Proc. Nat. Acad. Sci. U.S. 62:369, 1969) which, by recombination, has then been placed onto a translocation involving the Y and second chromosomes, T(Y;2)C. Thus, the DTS-L7 mutation is maintained only in the male and since there is essentially no crossing over here, the need for balancers is obviated. In addition, recessive markers for the X chromosome or autosomes can be recovered in a homozygous condition in females at the non-permissive temperature (29°C; permissive temperature is 25°C or less). Since DTS-L7 exerts its lethal effect in the egg stage, exclusively female larvae, pupae or adults (necessarily virgin) can be obtained. DTS-L7 was kindly supplied by David Suzuki. Supported by grants from the NSF (GB-32487) and NIH (GM-19194).

Voelker, R.A. North Carolina State University, Raleigh, N.C. On the possible identity of the factor(s) giving rise to male recombination and to unique chromosome aberrations in Drosophila melanogaster.

Hiraizumi reported that 20% or more of the second chromosomes extracted from a Harlingen, Texas population of Drosophila melanogaster exhibited male recombination. Further work (in MS) by Hiraizumi has revealed that this phenomenon is associated with the following features: (a) clustering of recombinants; (b) relatively high frequency of recombination around the centromere; (c) mosaics among flies carrying this factor, and (d) a comparable amount of recombination in the third chromosome.

Mettler (in preparation) has reported "unique" chromosome aberrations (found once or only a few times in a given locality, but not in other localities) which were discovered while surveying the salivary gland inversion polymorphism in Drosophila melanogaster populations from various localities, among them a Jacksonville, Florida sample in which second and third chromosomes were simultaneously extracted.

Since both of these phenomena could be the end products of some type of chromosome breakage, the following experiment was conducted to determine if male recombination could be detected in lines in which unique inversions had been observed. Males simultaneously heterozygous for the extracted Jacksonville chromosomes and triply marked second and third chromosomes (+++/al b sp;+++/ve st ca) were derived and backcrossed to al b sp;ve st ca females and their progeny were scored for recombinants. Five such matings were prepared from each of six Jacksonville lines in which "unique" inversions had been observed. Because of low fertility, very few progeny and no recombinants were recovered from line 5; thus it is omitted from the following table.

Table 1. Recombinants recovered from lines in which "unique" inversions had been observed.

| | II | | III | |
|---|---|---|---|---|
| Line | al-b | b-sp | ve-st | st-ca |
| 1 | 1 | 6 | 4 | 9 |
| 2 | 0 | 5 | 2 | 7 |
| 3 | 1 | 0 | 1 | 12 |
| 4 | 0 | 15 | 1 | 18 |
| 6 | 2 | 1 | 1 | 3 |
| TOTALS | 4 | 27 | 9 | 49 |

Since parental type totals were not scored, the frequency of recombination cannot be computed, but it appeared to be of the same order of magnitude as that reported by Hiraizumi (Ca 0.5 - 1.0%). In both the second and third chromosomes, there is a tendency for the greater number of crossovers to have occurred in the region which included the centromere. This and the tendency for recombinants to occur in clusters are in agreement with Hiraizumi's results. Thus, it appears that male recombination and the occurrence of unique aberrations may be different manifestations of the same or, at least, related events.

Attempts were made to extract recombinant chromosomes by mating recombinant males to females carrying appropriate balancer chromosomes. Approximately half of these recombinant males exhibited slight fertility or sterility, such as is frequently associated with deficiencies. This suggests that the mechanism of exchange may be rather imprecise. Such an imprecise exchange mechanism may explain the unequal recovery of recombinant classes and the clustering of recombinants around the centromere found by Hiraizumi. Duplications for a given region may survive while their corresponding deficiencies may not; moreover, larger duplications and larger deficiencies may survive if they involve the centric heterochromatin. Salivary gland chromosomes of recombinant males which were fertile are being examined for the presence of duplications or deficiencies.

Baker, B.S. University of Washington, Seattle, Washington. "Suppressors" of the Minute phenotype.

The following observations suggest that some balancer chromosomes may carry suppressors of, or duplications for, the + allele of specific Minute mutants. (1) In crosses of $M(2)1^2$/+ by SM5/+ no SM5 phenotypically Minute progeny could be obtained. However, from pair matings of the progeny of this cross, stocks could be established that were SM5/? x SM5/?, non-Minute, and which bred true. Outcrosses of males of these stocks to Canton-S females gave only SM5 (197) and Minute (102) progeny. (2) a stock of M(2) $e^S$/In(2L+2R)Cy, In(2R)bw was obtained from the Pasadena Stock Center and found to contain all Cy non-Minute flies. Outcrosses of this stock to Canton-S gave only Cy and a few (ca. 1%) extremely Minute flies that were too sterile to test further.

Nirmala Sajjan, S. and N.B. Krishnamurthy. University of Mysore, Manasagangotri, India. Drosophila albomicans - a race of Drosophila nasuta.

Biological species are genetically closed systems. D. nasuta subgroup of the immigrans species group includes 3 major morphophenotypic complexes - one with frontal sheen, other with orbital sheen and another without any such sheen on frons. The frontal sheen complex includes 4 species - D. nasuta, D. albomicans, D. kohkoa and D. kepulauana; orbital sheen complex includes 4 species - D. neonasuta, D. sulfurigaster, D. pulaua and D. nixifrons; and the third complex includes only one species - D. pallidifrons. The present report discusses the status of D. albomicans (received from Mrs. F.D. Wilson, Genetics Foundation, Austin, Texas) in the light of the results obtained from the genetic tests between D. albomicans and D. nasuta.

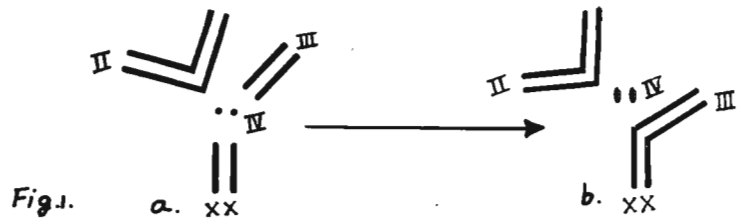


Fig. 1.

Figure 1. a. Karyotype of D. nasuta
b. Karyotype of D. albomicans

D. nasuta was originally described by Lamb (1914) from Seychelles Islands. Later Wakahama and Kitagawa (1972) reported the karyotype of this species - a pair of sex chromosomes, the X was rod-shaped, while the Y was J-shaped, a pair of V's (chromosome 2), a pair of rods (chromosome 3) and a pair of dots (chromosome 4). This karyotype is similar to the one reported earlier for Indian D. nasuta by the authors (1971). In all probabilities, both Seychelles island nasuta and Indian nasuta are one and the same.

D. albomicans (Duda 1923) is morphologically similar to D. nasuta, but differs cytologically valid species by Wilson et al. (1969) only because of the difference encountered in the karyotype. However, the crosses between D. nasuta and D. albomicans have yielded very interesting results. All the $F_1$, $F_2$ and $F_3$ generations are fertile. Males and females are produced in equal proportions. The karyotype of the $F_1$ consists of $2n = 7$, while that of $F_2$ and $F_3$ revealed only $2n = 8$.

As D. albomicans crosses freely with D. nasuta and has not yet reproductively isolated, it cannot be considered to be a full fledged species. The authors opine that D. albomicans certainly is nothing but a chromosomal race of D. nasuta. The karyotype of D. albomicans might have originated from that of D. nasuta by centric fusion of sex chromosomes to the third chromosome and involving also an addition of heterochromatin to the dot (Fig. 1). In the authors' opinion D. albomicans could as well be called D. nasuta albomicana.

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References: Lamb, C.G. 1914, Trans. Linn. Soc. London 16:307-372; Sajjan, S.N. and N. B. Krishnamurthy 1971, DIS 47:121; Wakahama, K. and O. Kitagawa 1972, Jap. J. Genet. Vol. 47 No. 2; Wilson, F.D., M.R. Wheeler, M. Harget and M. Kambysellis 1969, Univ. Tex. Publ. 6918: 207-254.

Mosna, G. University of Milan, Italy. Medium for Drosophila cells in vitro without serum.

Echalier and Ohanessian's medium for Drosophila cells in vitro contains 20% of bovine foetal serum. In order to eliminate from the medium ingredients, the chemical composition of which is not exactly known, an attempt has been made to grow cells without serum. Three established cell lines obtained two years ago in our Laboratory (GM1, GM2 and GM3) have been submitted to a gradual elimination of the amount of serum. Each step consisted in a 2% decrease of serum, the duration of a step being nearly 20 days.

During this process the cells did not show any serious indication of damage. Cell line GM2 has been, up to now, in a medium without serum for two months. The cells look perfectly healthy and multiply normally. Lines GM1 and GM3 are on the way to complete the same process.

Novitski, E. University of Oregon, Eugene, Oregon. Strengths of autosomal centromeres.

The distinction between weak vs. strong centromeres of the X chromosome depends upon the elimination of dicentric chromosomes from the meiotic products with the consequent production of a nullo-X egg, vs. the delivery of fractured pieces of a dicentric to the products, followed by the lethality of those products. Such a test is not so simply carried out on one of the major autosomes since the first possibility (nullo-autosome) is not as easily detectable, but would ordinarily appear as a dominant lethal.

Males carrying compounds for an autosome produce a sizable number of sperm with both components, i.e., are diplo-autosome, and tests suggest that about 25%, the number expected if the compounds for the two arms segregate at random, are of this type. This makes it possible to set up a test for the production of nullo-autosome eggs by a female heterozygous for an inversion, since the absence of the autosome in the egg will be compensated for by its presence in duplicate from the male.

In(2R)bw$^{VDe2}$ is a large inversion running from the heterochromatin of 2R to the brown locus, and therefore should allow a small number of four strand doubles: Tests of some 48 females heterozygous for this inversion and b vg gave 8.7% crossing over between b and the inversion, and 0.6% (31/5391) detectable doubles within the inversion. Since 3/8 of all chromosomes recovered from double exchange tetrads are double crossovers, and 2/8 from doubles are nullo-chromosome, the ratio of doubles to nullos should be 3:2 (discounting the small number of doubles undetected because they occurred on the same side of vg). The expected number of nullo-autosome eggs is then 0.6% x 2/3 or 0.4%. If only 1/4 are recoverable, because only 1/4 of the sperm are diplo-autosome, the expected frequency of exceptions is about 0.1%.

Since matings of diploid females to males with compound autosomes usually produce no offspring, an estimate of the productivity of the females must be made by making a parallel run of such females to ordinary diploid males. Sixteen females, handled similarly to the experimental females, produced 2391 progeny, or 149.4 per female. 780 females mated to compound second chromosome males, produced only 33 progeny carrying the two compounds from the male. The expected number of progeny from this number of females is 116,561, so that the calculated frequency of exceptions 33/116,561 or .028%. This is about one quarter of the number expected, a fraction about the same as some of the strong X centromeres previously tested. From this, we can reasonably conclude that autosomal centromeres are strong.

With a suitable translocation-inversion combination, where the X (or Y) centromere is attached to a substantial part of an autosomal arm, with the corresponding autosomal segment of the homolog inverted, it should be possible to measure the relative strengths of the X, Y and autosomal centromeres.

Puijk, K. and G. de Jong. Genetisch Instituut, Utrecht, The Netherlands. $\alpha$-amylases in a population of D. melanogaster from Dahomey.

Doane (J. Exp. Zoöl. 164:363, 1967) described isoenzymes of $\alpha$-amylase in Drosophila melanogaster. They were designated amylase 1, 2, 3, 4 and 6, 1 being the fastest anodically migrating enzyme and 6 the slowest.

In a population collected in March 1972 in Dahomey (West Africa) a further band was found between bands 4 and 6. This band was designated band 5.

By inbreeding, stocks were obtained with band 5 alone, (presumably homozygous Amy$^{5}$), or in combination with band 6 (homozygous Amy$^{5,6}$). Amy$^{5}$ and Amy$^{5,6}$ behave in crosses as alleles

| amylase phenotypes | 1 | 5 | 1,4 | 1,5 | 1,6 | 3,6 | 4,6 | 5,6 | 1,3,6 | 1,4,6 | 1,5,6 | 1,4,5 | 3,4,6 | 3,5,6 | 4,5,6 | 1,4,5,6 | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| number of flies found | 1 | 5 | 3 | 8 | 2 | 15 | 9 | 10 | 9 | 5 | 3 | 2 | 2 | 15 | 10 | 1 | 100 |

of Amy$^{1,4}$ of Doane. In population cages started with the Dahomey flies a larger number of Amy phenotypes was found than in any other population (e.g. McCune, DIS 44:77, 1969). The frequency of band 5 is rather high, band 2 has not been found (see Table).

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

The production of diploid gametes by female D. melanogaster.

In a series of experiments designed to identify male meiotic mutants, evidence has been found for the occurrence of diploid, or unreduced, gametes from females. In the tests, males with structurally normal second chromosomes were crossed to C(2L);C(2R) females. Only two classes of offspring were expected; those resulting from a diplo-2 sperm fertilizing a nullo-2 egg, and those resulting from a nullo-2 sperm fertilizing a diplo-2 [C(2L): C(2R)] egg. However, in addition to these two diploid classes, two unexpected classes of offspring were recovered. These were triploid females and intersexes. Three types of crosses were made (see footnote, Table 1), and exceptional progeny were recovered from all crosses. In the first set

Table 1

| Cross 1 number | Number ♀ tested | 3N ♀ | Ix | Σ | Frequency |
|---|---|---|---|---|---|
| 1 | 8161 | 148 | 92 | 240 | 0.029 |
| 2 | 708 | 12 | 5 | 17 | 0.024 |
| 3 | 956 | 6 | 10 | 16 | 0.017 |

Cross #1: X X $B^{S}Y$;C(2L)RM4, dp;C(2R)RM4, px ♀ X cn;$e^{s}$ ♂
Cross #2: C(1)RM, y/$B^{S}Y$;C(2L)RM, +;C(2R)RM, + ♀ X $Y^{S}X.Y^{L}$, $y^{2}$ su($w^{a}$)$w^{a}$/$y^{+}$ Y ♂
Cross #3: C(1)RM, y/$B^{S}Y$;C(2L)RM4, dp;C(2R)RM, + ♀ X cn ♂

of crosses, the triploids and intersexes were phenotypically Bar-eyed. In cross #2, the triploids were $y^{2}$ B and the intersexes were B. In cross #3, the triploids were B and the intersexes were y B.

From crosses 2 and 3, it is clear that these progeny received both a compound-X and $B^{S}Y$ from their mother. The exceptional offspring also received the compound autosomes, since in crosses 1 and 3 they are phenotypically $cn^{+}$. This means that in a diploid oocyte, all four of these elements would have segregated to the same pole at the first meiotic division, a highly infrequent event. This also requires nondisjunction of the third chromosomes in one of the two parents. Because of the relative frequent recovery of these two classes (there were approximately three times as many triploids and intersexes as there were diploid exceptions), the explanation that these are the result of nondisjunction in a diploid cell seems highly unlikely. Alternatively, these two classes could be the result of a normal haploid sperm fertilizing a diploid, or unreduced, egg. An X bearing sperm would give rise to a triploid female and a Y bearing sperm would give rise to an intersex.

Such diploid eggs could be the result of either a failure of the first meiotic division, or a failure of a pre-meiotic mitotic division which would result in tetraploid gonial cells. In the latter, one would predict clusters of triploid and intersex progeny, whereas in the former, no large clusters would be expected. In cross #1, a total of 1313 crosses were made. Of these, 1112 produced no triploid or intersex progeny, 168 crosses gave one exception, 26 crosses produced two exceptions, six produced clusters of three, and one cross yielded four exceptional offspring. The lack of any large clusters suggests that the diploid eggs are most likely the result of a failure of the first meiotic division.

Crosses of compound autosome males to structurally normal females have not yielded any triploid or intersex progeny, indicating that males do not produce diploid gametes, or if they do, they are produced at a much lower frequency. Approximately 600 males have been tested in this type of a cross. Diploid gametes from a male in these crosses would yield intersex progeny, and any diploid gametes from the females would produce either haplo-2 or tetra-2 offspring, and thus would not be recovered.

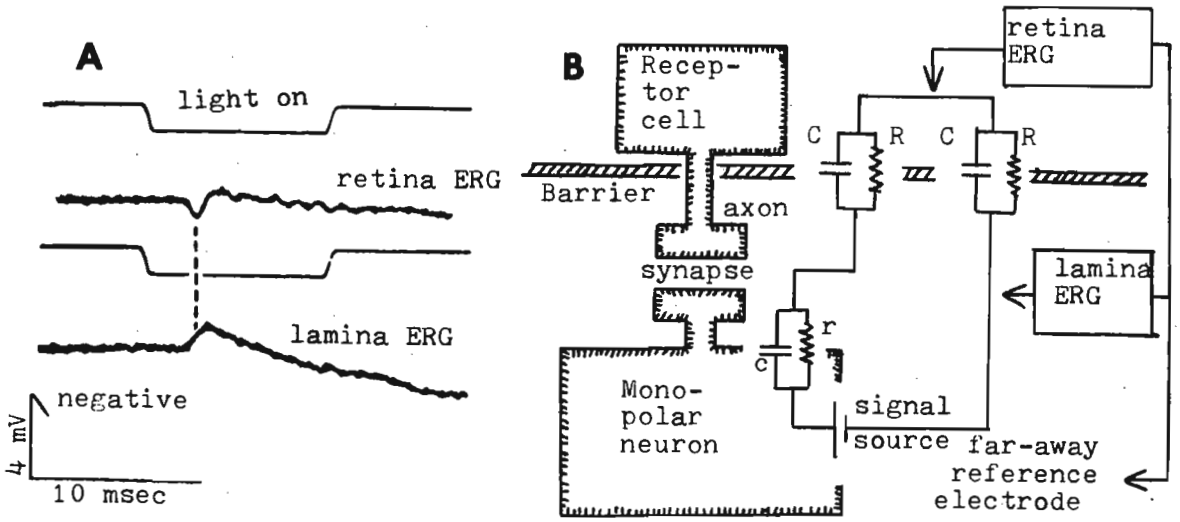
Presumably, the production of diploid gametes is a characteristic of female meiosis, and would explain the occurrence of rare triploid females and intersexes in structurally normal Drosophila stocks. Since the effect has been found with two unrelated stocks of compound autosomes, it is felt that the effect is not due to any meiotic mutant induced when the compound stocks were constructed. It is, however, possible that the production of these diploid gametes could be due, in some way, to the structural rearrangements involved in these stocks.

Stark, W.S. and G.S. Wasserman. University of Wisconsin, Madison, Wisconsin.

Temporal properties of the ERG on-transient recorded in the retina and lamina.

Recent intracellular recording work on Drosophila by Alawi and Pak and on Calliphora by Autrum, Zettler, and Järvilehto indicate that monopolar neurons in the lamina ganglionaris generate the on-transient in the ERG. A reversal in the polarity of the components of the extracellularly recorded ERG recorded in the retinular layer as opposed to the lamina was described by Pak, Grossfield, and White. The accompanying figure shows oscillographic tracings of the ERG for a 10 msec flash with the electrode in the retina and in the lamina. For both tracings white light of the same intensity was used on the same white-1 Drosophila melanogaster preparation. While the on-transient from both locations begins at the same time, it consistently peaks sooner in the retina. This evidence would seem to contradict the lamina origin of the on-transient since it makes sense that the signal arises where it is seen earliest.

Part b of the accompanying figure shows a model which may reconcile the presently reported finding with the convincing evidence of the origin in the lamina of the on-transient.




It takes into account the high resistance "receptor barrier" between the retina and lamina proposed by Heisenberg. There is a clear basement membrane associated with a barrier (through which receptor axons pass) made up of end feet of cells in the retina layer which may cause this high resistance barrier. If we assume that this barrier also has a high electrical capacitance, then the barrier might be expected to act as a high-frequency-pass-filter. Such a filter would preferentially pass rapid changes and reject slower changes. A signal, generated across the cell membrane of a cell in the lamina, must pass through resistors and capacitors in the barrier (R and C) and in the lamina cell membrane (r and c) in order to be recorded in the retina. Additional cytoplasmic and extracellular resistances are not shown since they would be relatively small. The resistances would divide the voltage between the source at one point on a lamina cell and the point where the circuit is completed at another point on the same cell. If the barrier resistance were high relative to the laminar cell membrane resistance, this sort of voltage divider might be expected to reverse the potential of the signal as it is reversed when it is recorded in the retina. And the high-pass-filter would make the signal peak earlier in the retina than in the lamina. This model can reconcile the laminar origin of the on-transient, the reversal of the on-transient as recorded in the retina, and the fact that the on-transient peaks earlier in the retina.

References: Alawi, A.A. and W.L. Pak 1971, Science 172:1055-1057; Autrum, H., F. Zettler and M. Järvilehto 1970, Z. vergl. Physiologie 70:414-424; Heisenberg, M. 1971, J. exp. Biol. 55:85-100; Pak, W.L., J. Grossfield, and N.V. White 1969, Nature 222:351-354.

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Schalet, A. University of Leiden, The Netherlands. Lethal, semilethal, and ♂ sterile mutants in the proximal region of the X chromosome of D. melanogaster.

The construction of a reasonably accurate and comprehensive genetic map of the proximal X region in Drosophila melanogaster requires the collation of mutants originally collected and analyzed at the University of Connecticut with the collection of mutants generated at The Hebrew University in Jerusalem and analyzed in the collaborative and independent publications of Lifschytz and Falk. For this purpose we have worked with some 40 mutants from the Israeli collection. In a number of instances our analysis differs from previously presented data. The results of our findings for 16 mutants are summarized in Table 1. These results bear upon the genetic extents and positions of additional mutants to be found in the papers of Lifschytz and Falk. To the best of our knowledge the phenotypes of the mutants listed in the table and in the new mutants section (aside from the "mal" mentioned in Table 1) have been previously described by these authors simply as lethal. In connection with the analysis of mutants in the proximal X region, we wish to take issue with some of the previously drawn conclusions.

Is the observation that most X-ray-induced mutations in the proximal X region are aberrations applicable to the nature of X-ray-induced mutations in the genome as a whole (Lifschytz and Falk 1968)? It was observed that 86% (30 out of 35) of the X-ray-induced lethals in post-meiotic male germ cells exposed to 3,000 R affected two or more functional units. Such a result, a very high proportion of aberrations, in the segment adjacent to the proximal heterochromatic region in a normal chromosome, was to be expected on the basis of earlier analyses of mutations at the y, w and N loci in normal chromosomes as compared to inverted chromosomes where these loci were brought close to the proximal heterochromatic region. For example, as compared to the frequency obtained in its normal position, y "mutations" in the $sc^8$ inversion chromosome were increased 10-fold, and the increase was mainly due to the rise in lethals (Belgovsky, 1938; Muller, 1940). Again, as compared to the frequency obtained in its normal position, N mutations in the $rst^3$ and $w^{m4}$ inversions were increased 3 to 4-fold (Lefevre, Ratty and Hanks, 1953). For $w^{m4}$ it was shown that the excess number of Notches resulted from deficiencies extending into the heterochromatic region and not from a position effect on mutability of $N^+$. Thus, the conclusion that "proximity to heterochromatin facilitates deletions" is applicable to the segment normally adjacent to the proximal heterochromatic region in an uninverted chromosome. The enrichment due to the proximity to heterochromatin is indicated by the fact that 16 of the 35 mutants have a proximal breakpoint at or in the proximal heterochromatic region. Furthermore, as noted in the 1968 paper, the distal portion of the X chromosome segment covered by Y $mal^+$ was not included in the published analysis. Genetically, this portion is at least 0.5 map units in length and includes the ot, sw, mel and mal loci; cytologically it comprises at least 18 distal bands of salivary Section 19. Despite the above considerations, which we consider as weighting the data in favor of aberrations, our analysis would add at least 5 additional mutants (B214, B56, A200 and B83) to give a total 10 out of 35 (28%) mutants affecting single functional units.

As for the nature of X-ray-induced mutations, the following observations should be considered: 1) The 1968 paper cites Alikhanyan (1937) for the method of mapping, but does not mention that Alikhanyan's cytogenetic analysis of 22 lethals induced in the left end of the X chromosome following the exposure of male germ cells to X-ray doses of 3,500 R, 4,000 R and 5,000 R showed that 12 out of 22 (54%) affected single functional units and were without visible chromosomal changes in the polytene analysis. 2) The 1968 paper cites Chovnick et al. (1964) for evidence that a deficiency for ry is not lethal, but fails to note that all of the essentially unselected sample of 14 X-ray-induced ry mutants used in this study proved to involve lesions that must have been considerably smaller than the entire cistron since all were capable of intracistronic recombination. (Admittedly, we did not point out the relevance of this observation to the nature of X-ray-induced mutations.) 3) Recently, Judd et al. (1972) have observed that the large majority of X-ray-induced changes in the polytene 3A2-3C2 region are single site mutations not associated with visible chromosome rearrangement.

Are "nearly-whole X-chromosome deletions" having one break in the proximal euchromatic region induced by X-rays twice as frequently in X·Y chromosomes and 7-10 times as frequently in ring-X chromosomes than in a regular rod X chromosome (Falk 1970)? Falk's interpretation of the data is subject to reservation, if only because the scheme used to detect deletions required the coverage of lethals carried by maternally derived chromosomes, and one scheme used B-56 whereas a second scheme used both B-56 and Q464. Neither of these mutants are complete lethals (see Table 1). Data from unirradiated males were not presented.

Are deficiencies for both su(f) and bb "invariably male sterile" as stated by Lifschytz

Table 1

| | Mutant | Functional units 1969 map | Position 1969 map | Characteristics which differ from previously presented data of Lifschytz and Falk |
|---|---|---|---|---|
| 1 | W-1 | 3 | 1-3 | similethal unc allele, unc locus located immediately to the right of unit 10* |
| 2 | W-5 | 3 | 3-5 | semilethal unc allele, " " " " " " " " " " |
| 3 | R-10-1 | 2 | 3-4 | semilethal unc allele, " " " " " " " " " " |
| 4 | B-214 | 2 | 10-11 | not allelic to unit 10, B-214/A-118 is wild-type; B-214/B-82 is lethal; located to right of unc. |
| 5 | B-56 | 2 | 12-13 | similethal lfl allele, 20-30% viability; developmental time is 13 days or longer |
| 6 | Q464 | 2 | 19-20 | semilethal wap allele |
| 7 | A200 | 2 | 20-21 | semilethal wap allele, A200/Q217 is mutant |
| 8 | B-83 | 2 | 24-25 | semilethal unc allele |
| 9 | R-10-10 | 2 | 25-26 | semilethal unc allele |
| 10 | X-2 | 2 | 28-29 | su(f) allele, X-2/R-9-18 is fully viable and normal at 17-18$^{o}$C, viable and mutant at 24-25$^{o}$C, lethal at 29-30$^{o}$C |
| 11 | D-13 | 2 | 28-29 | su(f) allele, D-13/R-9-18 is lethal at 24-25$^{o}$C, fully viable and mutant at 17-18$^{o}$C |
| 12 | Q217 | 2 | 19 & 22 | semilethal wap allele |
| 13 | B-96 | 1 | 13 | semilethal allele of B-56, B-96/B-56 flies show mutant phene affecting bristles, eyes, wings |
| 14 | X-1 | 5 | 26-30 | not allelic to R-10-10; since R-9-5 and X-4 are alleles, X-1 involves only 3 functional units to the left of bb; X-1/sc$^{4}$ sc$^{8}$ is lethal, therefore X-1 is deficient for su(f) and bb; ♂ fertile |
| 15 | B-57 | 7 | 2-8 | extends to the right to include unit 10 and unc locus located immediately to the right of unit 10 |
| 16 16 | Q539 | 8 | 3 & 10-16 | does not have independent lethal at unit 3 which is allelic to B-57 and W-1, W-5, R-10-1; allelism occurs because deficiency includes unc locus. B-57/Q539 shows vao* unc |

* unit 10 identified as mal locus in 1969 map actually contains a completely unrelated mutant (Schalet & Finnerty, 1970)

and Lindsley (1972)? But according to Lifschytz (1971), "Df(1)A209 is a partial bb deficiency and is semi-sterile". Furthermore, figure 1 of the 1971 paper shows A209 to be deficient for su(f) and to extend well to the left. Our analysis indicates that A209 is deficient for both su(f) and bb, but we can find no evidence for bb locus activity. $y^+$ Y $mal^{126}$/A209 males appear to have reduced fertility and could be described as semi-sterile. Likewise Df(1)X-1, somewhat shorter on the left than A209, deficient for both su(f) and bb with no evidence for bb locus activity, is male fertile when provided with a $y^+$ Y $B^S$. The fertility of individual $y^+$ Y $B^S$/X-1 males is reduced in some cases, but appears to be normal in others. In addition we have previously (DIS 43:65) described Df(1)$mal^{12}$ as broken distally between 1(1)34 and ot, proximally in bb, and male fertile with $y^+$ Y $mal^+$. Finally, we have observed a number of additional proximal X deficiencies for both su(f) and bb which are male fertile when covered by a Y $mal^+$ (Munoz and Schalet, unpublished).

Is recombination per complementation unit in the distal segment of the region studied by Lifschytz (Lifschytz and Falk 1970; Lifschytz 1971) about twice that in the proximal segment? Recombination in the distal segment (units 2-17 of the 1969 and 1971 maps) as a whole was reported to average 0.1% per complementation unit; the same value was found for subsegments. Our analysis of the genetic extents and positions of mutants W-1, W-5, R-10-1, B-214 and B-56 presented in Table 1 as well as additional mutants used to construct the 1969 and 1971 maps (AA33, E81, R-9-28, R-9-29) indicates that the number of functional units from AA33 (unit 2) through vao (unit 10) is not 9 but 5, and B-56 can not be used to define more than 1 functional unit to the right of unit 10. Consequently, the figures given for recombination per complementation unit in Table 1 of Lifschytz (1971) are subject to revision. Likewise our analysis indicates a revision is required for the recombination per complementation unit in the proximal segment which is given as 0.047 in cross 2 of Lifschytz's table. This is because we find that the number of functional units from E54 (unit 17) through Q463 (unit 34) is reduced to 8 functional units. Our analysis reduces the number of functional units defined by mutants Q464, Q217 and A200 from 4 to 1, and the number of functional units defined by mutants B-83, R-10-10 and Q456 from 3 to 1. Allelism of R-9-5 and X-4 reduces 2 units to 1 unit. Q463 is located to the left of R-9-5, R-9-18 and 3DES, so that the segment encompassed by E54 and Q463 contains no more than 8 functional units defined by allelism tests. Lifschytz (1971) correctly places su(f) proximal to Q463, but it should also be noted that we have found 3DES, R-9-18, D-13, X-2 and X-1, which are all placed distal to Q463 (Lifschytz and Falk, 1969; Lifschytz, 1971), are all allellic to su(f).

Does the complementation analysis provide a good correlation between the number of bands and functional units in the segment from unit 10 through unit 17 (Lifschytz, 1971)? According to Lifschytz, 8 bands correspond to 7 genes. But no evidence is provided that the region is saturated. Indeed, on the 1969 map, units 11 and 12 are not represented by mutants restricted to single functional units. Our analysis indicates that mutants B-214 and B-56, which appear in the 1969 map as two unit deficiencies, that helped to define units 11 and 12, involve only single functional units. Furthermore, the unc locus, represented by the mutants W-1, W-5 and R-10-1 which had been mapped to the left of unit 10, is located immediately to the right of unit 10. Consequently, the evidence presented by Lifschytz concerning a possible 1:1 relationship between bands and genes is insecure.

(This work was carried out within the framework of the Association between Euratom and the University of Leiden, Contract 052-65/2 BIAN, and it also received support from the J.A. Cohen Institute for Radiopathology and Radiation Protection.)

## QUOTABILITY OF NOTES

Bennet-Clark, H.C. 49:
Elens, A. 48:108; 48:108
Elens, A. and J.M. Wattiaux 39:118; 45:110; 46:81
Ives, P. 49:
Mather, W.B. and P. Thongmeearkom 49:
Pulvermacher, C. and W. Köhler 48:65
Seecof, R.L. 41:185-187; 42:121; 46:113
Seecof, R., W. Kaplan, P. Wong, W. Trout, and J. Donady 46:154
Wattiaux, J.M. and A. Elens 45:97

For previous listings see DIS 38, 42, 43, 44, 45, 47 and 48.

Wood, D.D. and F.M. Butterworth. Oakland University, Rochester, Michigan. Mating behavior and reproductive tract morphology of male-sterile mutants.

In an attempt to ascertain the cause of sterility, the mating behavior, reproductive tract morphology, sperm motility, and ultrastructure of spermiogenesis of certain male-sterile mutants have been studied. Previous work in this general area has been carried out by Kiefer (1969), Romrell, et al., (1972) and Tokuyasu et al., (1972).

Analysis of courtship patterns was carried out on males homozygous for tapered (ta), shaven-depilate ($sv^{de}$) and scute$^{260\text{-}15}$ ($sc^{260\text{-}15}$)(See Lindsley and Grell, 1968). From one to five sterile males (2-7 days old) were paired with one to five wild-type, virgin females (2-3 days old). In addition to normal females, wild-type virgin females whose antennae were removed and wingless, fertile apterous virgins were also employed. The flies were placed in small, 1" x 1" x 3/8" clear plastic breeding boxes and were observed from 15 minutes to two hours. Some were left up to 24 hours in which cases the females were isolated and the eggs were checked for fertization for as long as five days. Approximately 100 tests were made for each mutant and the mating behavior was compared to the wild type in accordance with Spieth (1952). Normal courtship pattern begins with the 360° circling of the female by the male; next the male vibrates his wings holding one at a 90° angle to his body; then the male intermittently touches the female with fore legs and/or proboscis; next the female expresses acceptance by spreading her wings and/or vaginal plates; the male then mounts female and completes intromission; following insemenation the female begins rejection of the male by kicking; and finally the male rotates 180°, dismounts and withdraws. When a female is non-receptive she exhibits the kicking phenomenon and other avoidance techniques. Presumably the non-receptivity is caused by a previous mating (Merle, 1968) or the courtship by the male is unsatisfactory.

It was found that ta and $sv^{de}$ cannot mate. Vague interest in the form of circling and approaching the female was displayed by the ta and $sv^{de}$ males, up to 15 min. in many cases. Both mutants displayed wing vibrations periodically at 90° and proboscis movements. No intermittent touching or any other type of behavioral steps were observed. Using the kicking motions, the females always prevented the males from coming close to them. Presumably the courtship steps employed by the mutants were insufficient to induce acceptance by the female. It is interesting to note that non-receptivity was also exhibited by the antennaless and wingless females. Furthermore it was shown that females made receptive by wild-type males would not accept the mutants. In this experiment 7-day Oregon-R wild-type males were placed in with one or more females, and just prior to intromission the normal males were replaced with mutant ones. With regard to the sterile mutant $sc^{260\text{-}15}$ the normal courtship pattern was displayed, mating was completed, but fertilization did not take place.

In order to examine the reproductive tract morphology and sperm motility the complete tract was dissected out in Drosophila Ringer's solution placed on a slide with fresh Ringer's solution, and examined under the light microscope. Spermatozoan motility was then checked in flies whose seminal vesicles had been dissected open.

In ta there were a frequent number of abnormalities in the reproductive tract, the most common was atrophy of one of the accessory glands, or both in some cases. Accompanying these abnormalities club-like structures usually were present on the apical ends of either of the testes. Frequently either the right or left testis was atrophied, and occasionally both. Out of 300 flies examined the ones with two or more of the malformations had non-motile spermatozoa. This number amounted to about 20% of the total number of flies studied. Preliminary electron microscopic studies revealed a large number of degenerating spermatids and in numerous instances double and triple axonemes per cell. There were also cases where several axonemes share one major mitochondrial derivative.

In $sv^{de}$ abnormalities in the reproductive tract morphology were more common. In many cases either one of the accessory glands were shrivelled and in a few instances, both. Either one or both of the testes in several flies were completely atrophied. Similar to ta the percentage of flies with non-motile spermatozoa increases greatly with the number of abnormalities present. Out of 300 flies examined approximately 50% had non-motile spermatozoa when containing more than two of the above abnormalities. Preliminary fine-structural analysis showed normal development within the testes. Both pre and post individualization stages appear consistant with wild type.

At the light microscope level the testes of $sc^{260\text{-}15}$ appeared normal in most cases, but in a few flies the accessory glands were constricted toward the apical end. Spermatozoa were motile but somewhat less than normal. About 15 minutes following copulation the vaginal tract

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Chen, P.S. and C. Brugger. Zoologisches Institut der Universität, Zürich, Switzerland. The ultrastructure of the anal organs of Drosophila larvae.

We reported previously the presence of a pair of anal organs at the posterior end of Drosophila larvae (Gloor and Chen 1950). These can easily be demonstrated by immersing the larvae briefly in a 0.5% $AgNO_3$ solution. Subsequent to a short exposure to light, two symmetrical dark-brown plates located on each side of the anus are visible. From their analysis of the ionic concentration of larval hemolymph in solutions of different salinities it was concluded that these organs absorb chloride and sodium ions from the environmental medium. In connection with our studies on the lethal mutant 1(3)tr, which accumulates an enormous amount of hemolymph of very low osmotic concentration (Hadorn 1949 and unpublished data), we undertook an electron microscope analysis of the anal organs of both the wild type and the ltr-homozygous larvae of D. melanogaster.

By using a scanning electron microscope (Cambridge Stereoscan S4) it was observed that the cuticular surface in the anal organ region has a porous structure, due to infoldings of the epicuticle. Studies of thin sections with a Hitachi transmission electron microscope revealed that underneath the cuticle the plasma membrane of the giant hypodermal cells show numerous folds which are oriented perpendicular to the cuticular surface. On the other hand, the plasma membrane on the basal surface appears quite smooth. The hypodermal cells are rich in mitochondria and other organelles such as free ribosomes, endoplasmic reticulum, microtubules, lysosomes, vacuoles of heterogeneous sizes and electron dense bodies have also been noticed.

Of particular interest are the profound changes of the fine structure of the hypodermal cells in larvae treated with solutions of different salt concentrations. When the larvae were immersed for one hour in a hypotonic medium (distilled water), the folds of the plasma membrane become greatly increased and penetrate more deeply into the cell. Mitochondria are also increased in number and many of them move into the interspace between the folds. Conversely, in a hypertonic medium (1.5-5.85% NaCl) there is a distinct reduction of both the folds and mitochondria. At still higher salinity (10% NaCl) the folds of the plasma membrane nearly disappear. Similar to that already reported for the anal papillae of the mosquito larvae (Copeland 1964, Sohal and Copeland 1966) the present results indicate that in Drosophila larvae the uptake of inorganic ions through the anal organs can be regulated by variations of the surface area of the plasma membrane of the hypodermal cells on the cuticular surface. Furthermore, alterations in the number and distribution of mitochondria at different salinities suggest that the ionic absorption must be an energy-consuming process.

We have so far detected no morphological difference in the ultrastructure of the anal organs between the wild type and mutant larvae. If osmoregulation is involved in the ltr lethal mutation, the effect must occur at some other level.

Literature cited: Copeland, E. 1964, J. Cell Biol. 23:253-264; Gloor, H. and P.S. Chen 1950, Rev. suisse Zool. 57:570-576; Hadorn, E. 1949, Rev. suisse Zool. 56:271-280; Sohal, R.S. and E. Copeland 1966, J. Insect Physiol. 12:429-439.

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of the female was dissected out and the spermatozoa in the tract did not appear as active as wild-type. Most spermatozoa were confined to the region just distal to the vaginal plug and none were observed in the spermathecae. Electron microcroscopy of the testis showed a large percentage of degenerating sperm bundles. In many individualized spermatids the axial filament was attached to the major mitochondrial derivative lateral to the normal position. This abnormality may be a symptom of the underlying cause of sterility.

With regard to $sc^{260\text{-}15}$ the cause of sterility must be associated with the spermatozoa or the seminal fluid. With regard to ta and $sv^{de}$ the interesting problem lies in the causal relationship of the two reproductive phenomena studied: courtship behavior and reproductive tract morphology. Are the two abnormalities related or do they occur independently? Does one give rise to the other or are they both induced by a third cause? (Supported by N.S.F. Research grant GB12969 and N.I.H. Biomedical Institutional Research Funds).

References: Kiefer, B.I. 1969, Genetics 61:157-166; Lindsley, D.L. and E.H. Grell 1968, Genetic Variations in Drosophila melanogaster. Carnegie Inst., Washington, D.C.; Merle, J. 1968, Insect Physiol. 14:1159-1168; Romrell, L.J. et al. 1972, J. Ultrastr. Res. 38:563-577; Spieth, H.T. 1952, Bul. Am. Museum Nat. His. 99:7-473; Tokuyasu, K.T. et al. 1972, Z.F. Zellforsch. 124:479-506.

Stark, W.S. University of Wisconsin, Madison, Wisconsin. Light-elicited wavelength-specific shifts in the ERG visual sensitivity of Drosophila.

Cosens and Briscoe have reported that in white-eyed Drosophila blue light causes a long-term decrease in visual (ERG) sensitivity accompanied by diminished transients and continuous receptor cell depolarization. Normal sensitivity returns following exposure to long-wavelength light.

Cosens and Briscoe performed their experiments using broad-band-pass color filters; a more precise description of the wavelengths responsible for setting the sensitivity up and down can be obtained using interference filters. Stimulation with ultraviolet (375 nm) is nearly as effective as stimulation with blue (475 nm) in lowering the sensitivity from the maximally sensitive state. While 500 nm lowers the sensitivity, 525 nm raises it. Only intense blue or ultraviolet light above a certain threshold intensity will cause the decreased sensitivity. Perhaps a summated stimulation of the whole eye is needed to decrease the sensitivity; this would explain the discovery of the decreased sensitivity phenomenon in white-eyed Drosophila which have no screening pigments.

Since Cosens and Briscoe excluded the possibility of extensive bleaching of a photopigment, it would seem necessary to explain the sensitivity changes in terms of neural interactions between photoreceptors with different peak sensitivities. Burkhardt's single cell spectral sensitivities from the blowfly Calliphora show that most cells have peaks at 350 nm and 490 nm with a small proportion of cells having peaks at 350 nm and either 450 nm or 520 nm. If Drosophila had similar single cell spectral sensitivities, it would seem probable that a 350-490 nm receptor could mediate the decreased sensitivity while a 350-520 nm receptor reinstates normal sensitivity. Ultraviolet would lower sensitivity since there would be many more 350-490 nm receptors than 350-520 nm receptors.

The altered ERG following blue light and the ERG's of the blind mutants tan and ebony are similar. Both have reduced transients. The receptor cell polarization of normal white-eyed flies' eyes is altered following blue light; Hotta and Benzer suggested that the physiological lesion in the nonphototactic mutants may be altered polarization of receptor cells. However, both tan and ebony (made white-eyed with w-1) show shifts in sensitivity similar to those described by Cosens and Briscoe for normal white-eyed flies. Changes in DC level indicate continuous receptor cell depolarization while there is a smaller ERG receptor component in both blind mutants after intense blue light. The spectral parameters of the sensitivity changes are the same for the normal and blind white-eyed flies.

References: Burkhardt, D. 1962, Symp. Soc. Exp. Biol. 16:86-109; Cosens, D. and D. Briscoe 1972, J. Insect Physiol. 18:627-632; Hotta, Y. and S. Benzer 1969, Nature 222:354-356.

Supported by NSF grant GB-8581, an NSF predoctoral fellowship, and by the Wisconsin Alumni Research Foundation.

Hunter, A.S. and L. Waterman. University of the Pacific, Stockton, California. Effects of delayed mating.

Although it has been established for D. melanogaster that the time of mating affects the productivity of females, there has been very little work done on other species with regard to such factors. In order to test the generalization that delayed mating decreases the productivity of Drosophila females, a variety of other species are being studied.

The data obtained from 60 D. immigrans females as controls and 60 each of 2 days of virginity, of 4 days etc. are shown in Table 1.

Table 1. Mean total productivity.

| Days of virginity | Mean S. D. |
|---|---|
| 0 | 431 ± 53 |
| 2 | 371 ± 43 |
| 4 | 360 ± 26 |
| 6 | 233 ± 61 |
| 8 | 149 ± 30 |
| 10 | 100 ± 42 |
| 12 | 87 ± 39 |
| 14 | 58 ± 42 |
| 16 | 56 ± 28 |
| 18 | 41 ± 23 |
| 20 | 38 ± 22 |

It is obvious that a delay in mating of only a few days markedly decreases productivity in this species. At present we are testing D. virilis and D. busckii.

Portin, P. and M. Ruohonen. University of Turku, Finland. A new type of allelic interaction at the Ax locus in Drosophila melanogaster.

A sex linked mutant showing an Abruptex phenotype was found in April, 1971, in a "Muller-5" test of $F_1$ females of X-irradiated (1000 r) wild-type males. The mutant is dominant and homozygous viable and fertile both in females and males. The mutant gene was localized at or close to the Ax locus by means of crossing-over test. To test the allelism the mutant was crossed to the original Ax. The cross yielded only male offspring, i.e. the heterozygous females were lethal. Thus, the results suggest that the new mutant is allelic to Ax, and accordingly, it was named $Ax^{71d}$.

The lethality of the heterozygous combination of two viable alleles ($Ax/Ax^{71d}$) shows a new type of allelic interaction which has been recently observed also by Foster (1972, Genetics 71 Suppl. s:18) likewise at the Ax locus. According to Foster, $Ax^{E1}/Ax^{E2}$, $Ax^{E1}/Ax^{16172}$, $Ax^{9B2}/Ax^{E2}$, and $Ax^{9B2}/Ax^{16172}$ combinations are lethal. From the alleles involved, at least three ($Ax^{9B2}$, $Ax^{16172}$, and $Ax^{E2}$) are homozygous viable and fertile (Welshons, personal communication). It is suggested that the lethality of the heterozygotes is due to an active process, probably some kind of interaction between two different mutant gene products, but not due to lack of function.

This type of allelic interaction is tentatively called contracomplementation.

Lefevre, G.Jr. California State University, Northridge, California. The distribution of X-ray-induced sex-linked lethals.

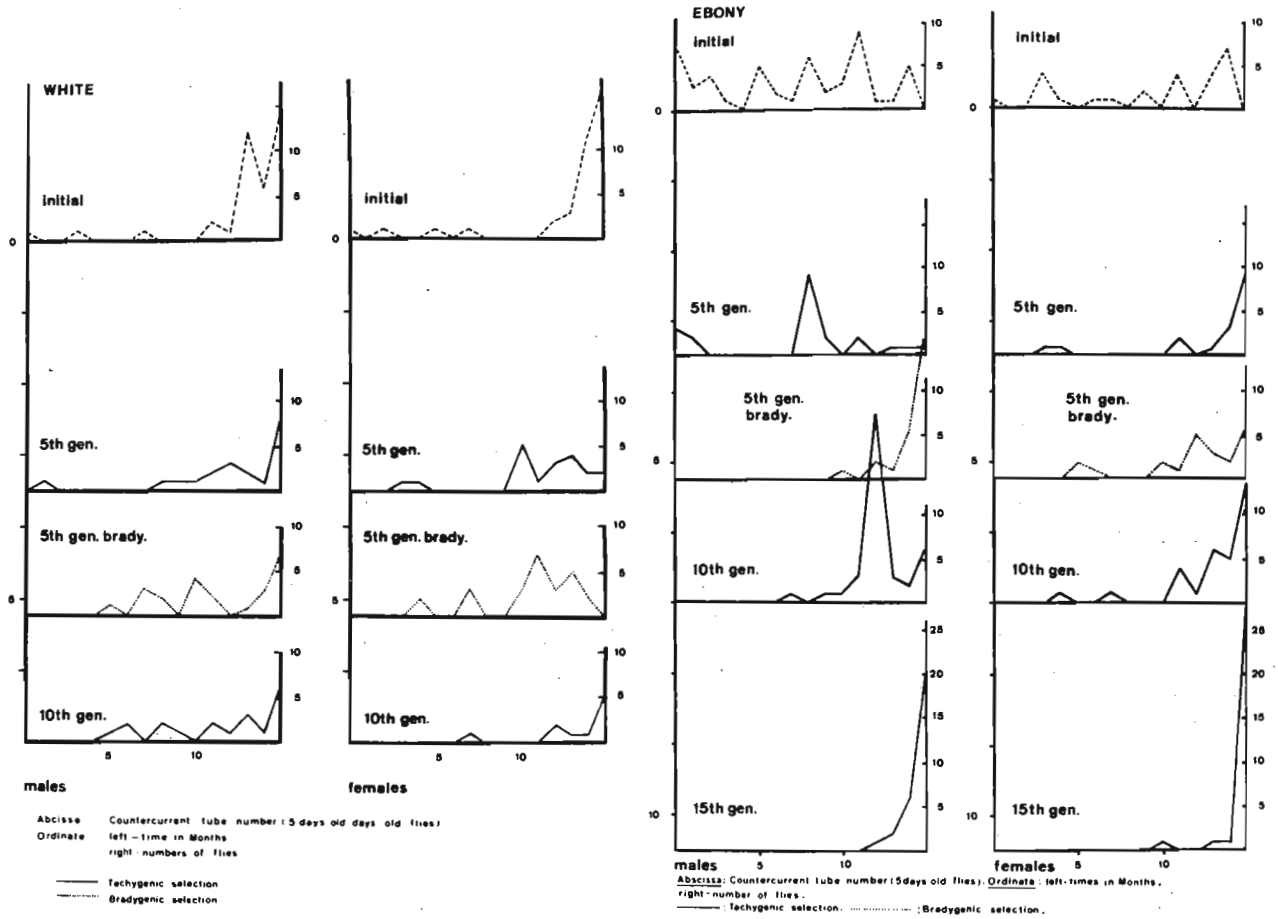
The distribution of X-ray-induced sex-linked lethals among ten cytologically defined intervals of the X chromosome has been investigated by using a series of duplications to "cover" random lethals. The results are recorded in the accompanying table. Each interval is characterized by a specific band length, which can also be expressed as a percent of the total number of X-chromosome bands. The percent of tested lethals falling in each interval can be compared with the percent of bands. In general, the

| Region | # of bands | % of bands in interval | % lethals covered | Coefficient of mutation: % lethals/% bands |
|---|---|---|---|---|
| 1A1 - 1B2 | 10 | 0.99% | 6/778 = 0.77% | 0.78 |
| 1B3 - 2C2 | 54 | 5.34 | 42/522 = 8.05 | 1.51 |
| 2C1 - 3C5 | 44 | 4.35 | 34/522 = 6.51 | 1.50 |
| 3C2 - 3E8 | 25 | 2.47 | 13/778 = 1.67 | 0.68 |
| 6C12 - 7C9 | 52 | 5.14 | 13/228 = 5.70 | 1.11 |
| 9E1 - 9F13 | 23 | 2.27 | 9/778 = 1.16 | 0.51 |
| 10A1 - 10A11 | 11 | 1.09 | 8/1098 = 0.73 | 0.67 |
| 10B1 - 11A7 | 59 | 5.83 | 58/1098 = 5.28 | 0.91 |
| 18F1 - 20A2 | 39 | 3.85 | 39/778 = 5.01 | 1.30 |
| 20A3 - 20F4 | 17 | 1.68 | 33/778 = 4.24 | 2.52 |
| TOTALS | 334 | 33.00% | 39.12% | 1.19 |

correlation is rather poor. Band number appears to be an inappropriate measure for estimating the frequency of induced lethal mutations, as it is also for estimating the distribution of chromosome breaks. A more useful measure is the relative DNA content of a region, which is reflected in the size of bands, not simply their number. Allelism tests of lethals falling in each different interval must be completed before a final assessment of the results can be made and should permit the identification of mutational "hot" and "cold" spots. Cytological analysis of all lethals, covered or not, is being carried out in order to permit a comparison of the distribution of lethals arising from mutation with that of those associated with chromosome rearrangement.

Elens, A. Facultés Universitaires N.D. de la Paix, Namur, Belgium. Parental age and efficiency of selection for phototactism.

Using the "countercurrent distribution" method of S. Benzer (1967) it is possible to fractionate populations of D. melanogaster according to their phototactic response, which changes with age and differs between strains (DIS 46:81; DIS 48:108). On the other hand, it is known that after a few generations, behavioural differences can result from the selection of "tachygenetic" and "bradygenetic" lines, i.e. by systematically choosing young or old genitors respectively (Wattiaux, J.M. 1968). The present paper concerns an attempt to combine the selection for positive phototactism with a selection for "tachygenetic" and "bradygenetic" lines.




Male and female flies were separated after eclosion and tested separately for phototactism, at 5 days of age for the "tachygenetic" line and at 30 days for the "bradygenetic" one; after the test they were allowed to mate. For the 5th and 10th generations of both "bradygenetic" and "tachygenetic" lines, flies were tested at the 5th and the 30th days after hatching, in order to permit a comparison. The figures here presented concern the behaviour of flies of 5 days, for "bradygenetic" as for "tachygenetic" lines of the white and ebony strains.

The selection for positive phototactism did not succeed in the white strain as in the ebony one. But the most interesting fact seems to be that, for the ebony males at least, the selection has been much more efficient in the "bradygenetic" line than in the "tachygenetic" one, as it can be seen by comparing the data concerning both 5th generations. It needs 15 generations of "tachygenetic" selection to obtain the same effect as 5 generations of "bradygenetic" selection, i.e., 10 months instead of 5 months.

Analogous results have been reported previously for an experiment in which the mice

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Nirmala Sajjan, S. and N.B. Krishnamurthy. University of Mysore, Manasagangotri, India. Structural variability in natural populations of Drosophila nasuta.

Natural populations of D. nasuta are highly polymorphic with regard to gene arrangements (Sajjan and Krishnamurthy 1970) Population studies revealed the presence of a total of 27 gene arrangements. These include one overlapping inversion in X-chromosome, 6 inversions in second chromosome and 19 inversions in the third chromosome (Fig. 1). Thus the third chromosome is the highly variable one and is comparable to the third chromosome of D. pseudoobscura.

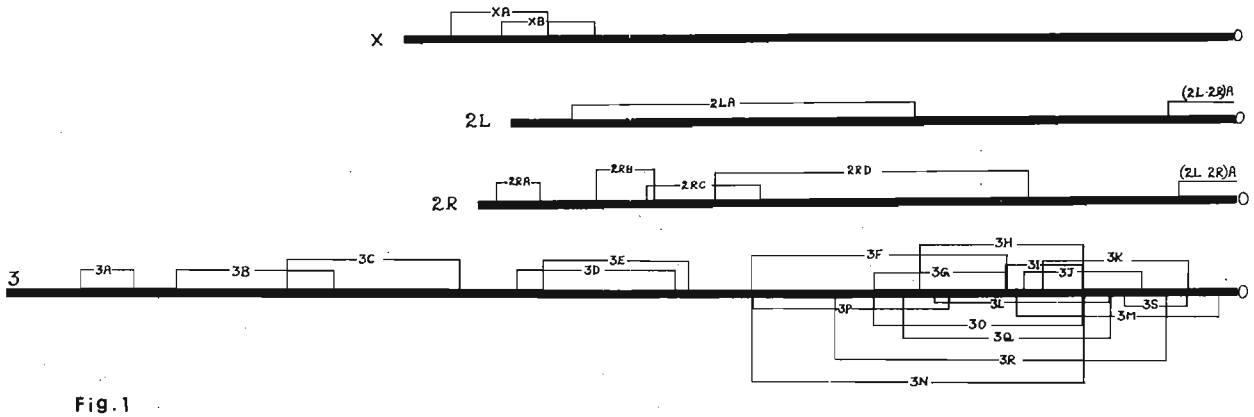


Fig.1

Fig. 1. Distribution of inversion break-points relative to the standard sequence. The open circles at the right ends represent the basal ends of the chromosomes.

The inversions and their distribution vary in space indicating that the chromosomal polymorphism in this species is of flexible type. Interestingly enough D. nasuta which is abundant and common species in all the localities studied, exhibits higher degree of polymorphism than the less abundant closely related form - D. neonasuta.

Another interesting feature in the present findings is that one of the 6 inversions in the second chromosome is a pericentric inversion - (2L-2R)A. It is so called because it is found between left and right arms of the second chromosome.

Acknowledgements: The writers are deeply indebted to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology, University of Mysore, for many constructive suggestions and criticisms. We are also thankful to Miss A. Shashikala for preparing illustrations.

References: Sajjan, S.N. and N.B. Krishnamurthy 1970, DIS #47:121.

Hayman, D.L. and R.H. Maddern. University of Adelaide, S.A., Australia. A more precise cytological location of M(1) and su(s).

Ten deficiencies induced by X-rays and one induced by ethyl methane sulphonate selected because they exposed su(s) were all found to also expose M(1)Bld. From previous cytological studies on Df(1) svr and M(1)Bld, su(s) could be localized to the region from 1B11 to 1C2-3 and more probably from 1B13 to 1C2-3 (Bridges and Brehme 1944 "The Mutants of Drosophilia melanogaster"). The smallest M deficiency recovered after X-ray treatment was found to have its proximal break point distal to 1C1 so confining both M(1)Bld and su(s) to the region 1B11 to 1B14. The proximity of su(s) and M(1)Bld are of interest in relation to the findings of Jacobson (1971, Nature New Biology 231:17) and Atwood's earlier analogy of Minutes with tRNA genes (Ritossa et al. 1966 Genetics 54:663).

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resulting from a "bradygenetic" selection were obviously superior in learning ability to the "tachygenetic" ones (1966).

References: Benzer, S. 1967, Proc. Nat. Acad. Sc. 58:1112; Elens, A., A.N. Mouravieff and M.J. Heuts 1966, Experientia 22:186; Wattiaux, J.M. 1968, Evolution 22:406.

Diamantopoulou, E. Agricultural College of Athens, Votanikos, Greece. Frequency and allelism of lethals in two natural populations of D. subobscura from Greece.

In an attempt to estimate the effective population size the frequency of lethals and the frequency of lethal allelism have been determined in two natural populations of D. subobscura from Greece: the Mt. Parnes (Attica) and the one from Alikianou (Crete). Samples of these two populations have been taken in summer and spring of 1971 respectively. Chromosome 0 (equivalent to 3R of D. melanogaster) has been analyzed by the Va ch cu/Ba balanced strain kindly provided by Dr. D. Sperlich. 145 chromosomes from Parnes and 150 chromosomes from Crete were studied. Also 52 control crosses from Parnes and 66 from Crete were made. The frequency of lethals plus semilethals was 0.310 ± 0.038 in Parnes sample and 0.227 ± 0.034 in the Cretan one. Nearly complete allelism tests of lethals (210 for Parnes and 370 for Crete) show an allelism frequency of 0.0095 ± 0.0067 for Parnes and 0.0054 ± 0.0038 for Crete. The differences of lethal frequencies (0.083 ± 0.051) and of lethal allelism (0.0041 ± .0077) are not statistically different from zero at the 5% probability level. Also the array of viabilities for homozygote chromosomes in the two populations is not statistically different. The correlation between viabilities conferred by chromosomes in homozygous and heterozygous condition has also been computed: it is +0.0569 ± 0.14 for Parnes and +0.0676 ± 0.12 for Crete. These correlation coefficients are not different from zero. On the contrary significant negative correlation coefficients (-0.421 ± 0.083 for Parnes and -0.322 ± 0.076 for Crete) are found between viabilities of quasinormal chromosomes and their coefficient of variation (from replicate culture viabilities) suggesting that chromosomes conferring higher viabilities in homozygous condition display greater homeostasis. This is not the case for the heterozygous condition where coefficients of correlation computed do not differ from zero (-0.079 ± 0.137 for Parnes and +0.031 ± 0.123 for Crete).

Pinsker, W. and E. Doschek. Institut für allgemeine Biologie, Vienna, Austria. Mating success and visual acuity in Drosophila subobscura.

The reproductive ability of D. subobscura was tested at different wave lengths of light. Groups of 3 virgin females and 3 males were set up in 15 ccm plastic vials and exposed to green light of 520 nm and to orange light of 570 nm. After a desired time the females were dissected and examined for the presence of sperm. A wild type strain and the mutant strains "ma" and "cn" were used for these experiments.

The results show that in orange light the mating success is remarkably reduced in "ma" and completely obstructed in "cn", while in wild type the insemination frequency is only lowered at an exposure time of 24 h.

Mating frequency in green and orange light
(% inseminated females)

| Color | Wave length | Exposure time | Strains + | ma | cn |
|---|---|---|---|---|---|
| green | 520 nm | 24 h | 87 | 68 | 97 |
| orange | 570 nm | 24 h | 36 | -- | -- |
| | | 72 h | 88 | 3 | 0 |

The lack of eye pigment, which causes a reduction of visual acuity, may be responsible for the impeded mating of the mutant strains in orange light. To prove this assumption, other experiments were made with respect to the optomotor response to moving striped patterns in green and orange light. The instruments for these investigations were provided by the kindness of Dr. Götz in the Max-Planck-Institute für biologische Kybernetik at Tubingen.

The figures can be considered as a measure of the average positive turning reactions.

Optomotor Response
(Rotations of the flies in the direction of the striped patterns within 10 minutes)

| Color | Wave length | Strains + | ma | cn |
|---|---|---|---|---|
| green | 520 nm | 2.93 | 0.75 | 1.35 |
| orange | 570 nm | 1.40 | 0.40 | 0.08 |

In orange light all 3 strains show a slighter optomotor response than in green light. In "cn", however, the reaction to the stripes seems to be reduced to nothing.

This finding can be compared with the results of the mating tests, in which "cn" also failed to mate in orange light. Therefore it may be concluded that mating success in D. subobscura depends on its visual acuity.

Bicudo, H.E.M. de C. São José do Rio Preto, São Paulo, Brazil. One case of partial superpolytenization in Drosophila.

In several papers, C. Pavan, A.B. da Cunha, R. Basile, and other authors, especially from the São Paulo University (Brazil), using Rhynchosciara and Sciara, have shown that polytene chromosomes may undergo an additional enlargement, in all its extent, induced by infections.

In a cytological study of salivary gland chromosomes of Drosophila septentriosaltans (saltans group) from Sevilla (Colombia), we also found one case of additional chromosomal enlargement, but limited to one part of a single chromosome: the distal end of the II L chromosome arm. The enlarged part exhibited a width approximately 2.5 times bigger than that of the same chromosome arm or any other, and was only found in 2 neighboring cells of the same salivary gland (Figs. 1 and 2). In both cells the enlargement was starting at the same band, suggesting a specific actuation over a well delimited chromosomal part. A kind of bubble under the band where the enlargement started might also be seen (Fig. 1), reminding the "large globular inclusions" described by da Cunha et al (1967) in Drosophila willistoni, which, together with other chromosomal changes, were attributed to "chemicals released by infective organisms". It is possible that some kind of infection is also involved in the present case. Unfortunate-

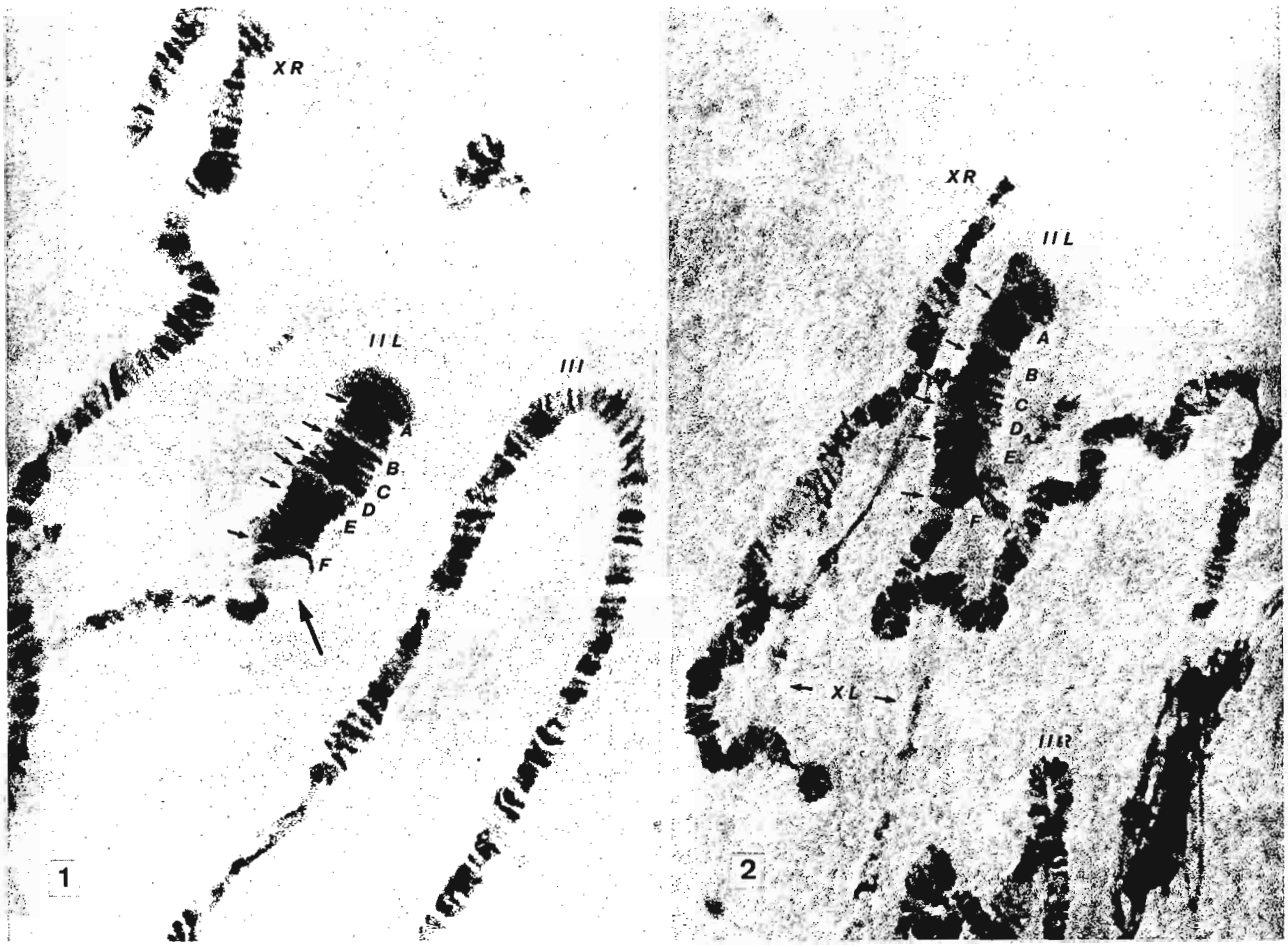


Fig. 1 and 2. Partial superpolytenization of the II L chromosome arm of D. septentriosaltans, in 2 cells of the same salivary gland. Small arrows and letters indicate that the same bands are enlarged in both cells. For a comparison of width, parts of the II L and other chromosome arms (this species has 2 pairs of metacentric and 1 of acrocentric chromosomes) may also be seen in both figures. Large arrow (Fig. 1) points to a "bubble" at the base of the enlarged part of the chromosome.

(Continues at bottom of next page)

Clark, A.M. State University of Leiden, The Netherlands. Influence of the nutritional state of females on the frequency of X/O males recovered after matings with irradiated males.

Würgler and Maier (Mutation Res. 1972, 15:41-53) have reported a maternal influence on the frequency of X/O males obtained after irradiation of mature spermatozoa. The nutritional condition of the females might be an important factor in affecting the yield of X/O males, either by way of a quantitative effect on post-fertilization repair systems in the egg, or by way of qualitative differences between eggs produced by well-fed and under-fed females respectively.

2-3 day old males of the genotype y $B/B^S$ Y $y^+$ (the X-chromosome being derived from R(1)2 that opened out) were irradiated with 3000 R and then mated overnight to (a) females aged 6 days on standard, well-yeasted food medium, or (b) females aged 6 days on unyeasted honey-agar or sucrose-agar medium. After removal of the males, the females were allowed to lay eggs on standard food medium for an initial brood A (24 hours), followed by a further brood B (48 hours). The percent of X/O males obtained in experiments with three different kinds of females, together with the total progeny counts, are given in the table.

| Female genotype | Brood | Frequencies of X/O males from: well-fed females | | starved females | |
|---|---|---|---|---|---|
| X.Y. | A | 2.91% | (1,066) | 1.61% | (124) |
| | B | 1.40% | (358) | 0.78% | (1,032) |
| $ysn^3$ | A | 1.72% | (1,394) | 1.43% | (841) |
| | B | 0.83% | (1,330) | 0.44% | (1,113) |
| Oster | A | 0.83% | (3,721) | 0.21% | (1,404) |
| | B | 0.79% | (2,393) | 0.55% | (1,995) |

The trend is consistent in each case. A lower frequency of X/O males is recorded when the females are poorly nourished during the few days prior to egg-laying. This indicates that female nutritional condition is yet another factor which must be taken into account when comparing the frequencies of X/O males recorded from different experiments.

The finding that in 5 out of 6 experiments somewhat higher loss frequencies were recorded in brood A than in brood B is consistent with a recent observation of Würgler, Bürki, and Büchi (Abstr., 3rd Europ. Dros. Res. Conf., Sept. 1972, Milan, ed. Barigozzi) on the modification of a maternal effect by storage. Herskowitz (Genetics, 1963, 48;703-710) noted an increase of the II-III translocation frequencies in sperm that had been irradiated in undernourished females in comparison to that having been irradiated in well-fed females. The contrasting effect of undernourishment of the females on the rate of recovery of X-chromosome loss and translocations from irradiated spermatozoa, may be more than a mere coincidence. Both Proust, Sankaranarayanan, and Sobels (Mutation Res. 1972 16:65-76), Maier, Graf, Ulrich, and Würgler (Abstr. 3rd European Dros. Res. Conf., Sept. 1972, Milan, ed. Barigozzi) and Mendelson (unpublished) recorded such contrasting effects for loss of chromosomes and translocation, when applying various different modifications of the material environment.

This work received support from the J.A. Cohen Institute for Radiopathology and Radiation Protection and the Health Research Organization TNO. It was carried out in part under the Association of Euratom with the University of Leiden, Contract Nr. 052-65-I BIAN.

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(Continued from preceding page)
ly we could not find again such a phenomenon in several other observations performed with the same strain; thus, we could not make a better study of the problem. In spite of this, we think that the importance of the present finding lies in suggesting that the superpolytenization, normally occurring over the entire genome, in some circumstances may partially affect a single chromosome; and, what is more relevant, it may be restricted to a well delimited part of the chromosome. Such a possibility can open good perspectives for the study of chromosomal physiology.

References: da Cunha, A.B., Z.M. França, A.M.A. Gonçalves, A. Hitelman and M. Garrido 1967, Rev. Brasil. Biol. 27:113-124.

Beck, H. University of Geneva, Switzerland. The phenotype of $X^{bb^+}/O$ males in Drosophila hydei.

In D. hydei X/O types have been reported to show a bobbed phenotype (Hess and Meyer, 1963; van Breugel, 1970, 1971) whereas such ♂♂ are wild type in D. melanogaster. Since the chromosomal situation is similar in both species, with a bb locus on the X and one on the Y chromosome, dosage regulation (Kiefer, 1968) might also occur in X/O types of D. hydei when the X carries a complete rDNA segment.

To re-examine the phenotype of $X^{bb^+}/O$, such ♂♂ were produced by crossing wild type ♂♂ (stock Madeira) to irradiated ♀♀, homozygous for ch or m. Exceptional patroclinous wild type ♂♂, produced by non-disjunction in the ♀♀, were mated singly to wild type ♀♀ to check for fertility. Of 25 ♂♂ tested, none was fertile. Only one of them showed mature sperm in the dissected testes and the receptacula of all the ♀♀ kept with these ♂♂ contained no sperm. From these data it is concluded that no Y chromosomes were present. Relative bristle length of these ♂♂ and of 25 additional ones which had not been tested for fertility is shown in Figure 1a. The average length of the two posterior scutellar bristles was measured at 50 fold magnification under a Wild M5 dissecting microscope. Relative bristle length was calculated as percentage of the distance between the two posterior dorsocentrals in each fly (Fig. 1). For comparison the distribution of bristle length indices of Madeira $X/Y^{bb}$ ♂♂ (Fig. 1b) and of $X^{bb}8/O$ types (Fig. 1c) is also given. Almost 90% of the Madeira X/O types in Figure 1a fall in the range of wild type with an index between 111 and 150, while a few are in the range of $X^{bb}8/O$ types. These latter cases are easily explained by the presence of a variety of bb alleles in the Madeira stock (Beck, unpubl.). From these observations we may conclude that $X^{bb^+}/O$ types in D. hydei show no reduction in bristle size and therefore that dosage regulation acts also in this species.

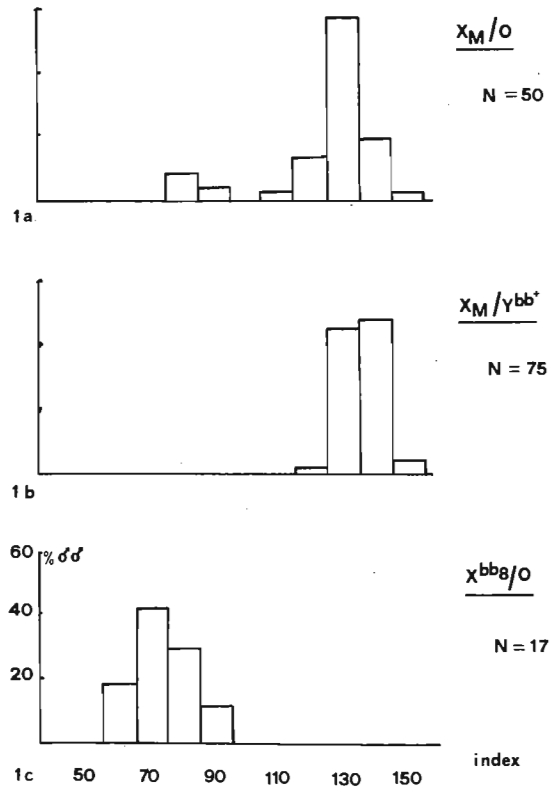



Fig. 1. Frequency distribution of relative bristle length in $X_M/O$ (1a), $X_M/Y^{bb+}$ (1b), and $X^{bb}8/O$ (1c) ♂♂. M stands for X chromosomes from wild type stock Madeira. Relative bristle length is average length of posterior scutellar bristles/distance between posterior dorsocentrals.

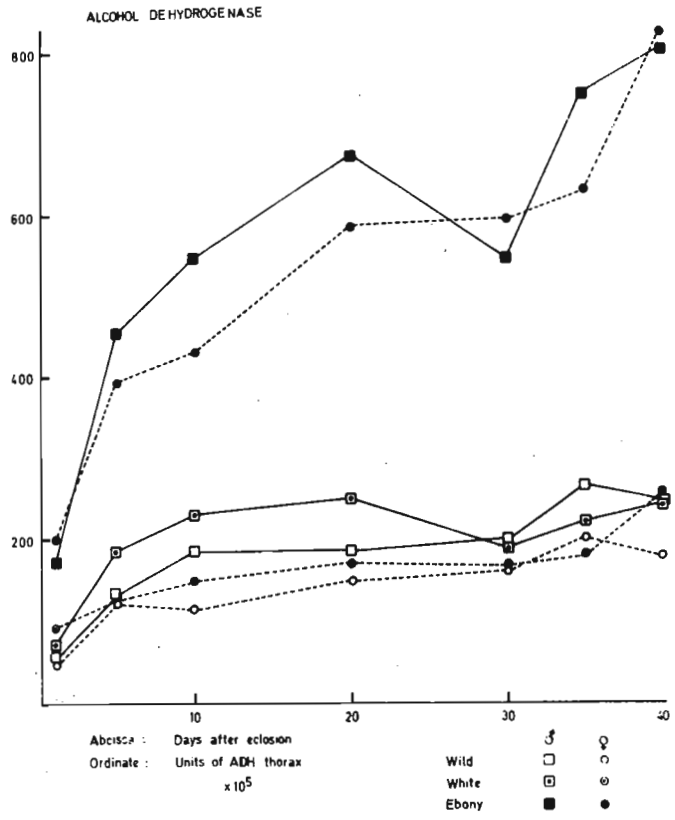
References cited: Breugel, F.M.A. van 1970, Genetica 41:589-625; _____ 1971, Genetica 42:1-12; Hess, O. and G.F. Meyer 1963, J. Cell. Biol. 16:527-539; Kiefer, B.I. 1968, PNAS 61 (1):85-89.

Ehrlich, E. University of Oregon, Eugene, Oregon. A suppressor of a suppressor.

For some years we have maintained a stock of C(2L)dp C(2R)px. An X-Y chromosome carrying $y^2$ su-$w^a$ $w^a$ was introduced into this line, along with other compounds of the arms of the second. In combination with C(2L)dp, flies with the X-Y chromosome are barely distinguishable from $w^a$; all other combinations with C(2L)+ are suppressed $w^a$ as expected. This suggests that there is a suppressor of su-$w^a$ on the C(2L) dp chromosome.

Libion-Mannaert, M. and A. Elens. Facultés Universitaires N.D. de la Paix, Namur, Belgium. Ageing in D. melanogaster ebony, white and wild: alcohol dehydrogenase and other enzyme activity changes.

The total protein content, and the activities of alcohol dehydrogenase, acid para-nitrophenyl phosphatase, and catalase, have been measured on adult flies varying from 1 to 40 days after eclosion. The tests were made according to Wattiaux et al. (1971) for total protein content Sofer and Ursprung (1968) for alcohol dehydrogenase, Neil and Horner (1964) for acid para-nitrophenyl phosphatase (but at 35°C instead of 37° and at pH 5.5 instead of 5.0) and Baudhuin et al. (1964) for catalase (at pH 7.5 instead of 7.0). A pooling of 30 thoraces was used for each measure and 10 repetitions were made for each strain, sex, and age.




A variance analysis reveals striking influences of the age, sex, and strain on the variations of the total protein content, and the alcohol dehydrogenase and acid para-nitrophenyl phosphatase activities. The influence of age and strain is marked on the catalase activity, but the differences between sexes are not significant.

The activity of catalase and acid para-nitrophenyl phosphatase falls from the 1st to the 5th day after eclosion, after which it increases. A second minimum is observed at 30 or 35 days for the catalase activity, followed by a second increase.

The most interesting fact is the much greater activity of alcohol dedydrogenase in the strain ebony ($e^{11}$). Further experiments will have to show if it depends on the gene ebony itself or on other genes present in the strain $e^{11}$, and if different isozymes of alcohol dehydrogenase are at work in the strains white and wild (Canton S).

References: Baudhuin, P., H. Beaufay, Y. Rahman-Li, O.L. Sellinger, R. Wattiaux, P. Jacques and C. de Duve 1964, Biochem. J. 92:179; Neil, M.W. and M.W. Horner 1864, Biochem. J. 92: 217; Sofer, W. and H. Urspring 1968, J. Biol. Che. 243:3110; Wattiaux, J. M., M. Libion-Mannaert and J. Delcour 1971, Gerontologia 17:289.

Nettleton, R.* University of Nebraska, Lincoln, Nebraska. A failure to demonstrate an influence of temperature on the "sex ratio" phenomenon in D. athabasca.

An attempt was made to determine whether the influence of temperature on the X chromosome caused "sex ratio" phenomenon of D. athabasca is like that found in D. pseudoobscura by Darlington and Dobzhansky (Proc. Nat. Acad. Sci. 28:45-48, 1942), who observed less extreme effects of the "sex ratio" X at high than at low temperatures. The source of the athabasca "sex ratio" X was a wild strain from Englewood, New Jersey. Since no sex-linked mutant genes were available to mark either normal or "sex ratio" X, it was necessary to use males taken at random from the progeny of females that must have been either homo- or heterozygous for the "sex ratio" X since they were daughters of males that had nearly all female progeny. The males employed were expected to be a mixture of ones with a normal X and ones with a "sex ratio" X, with the proportion of the latter at least 50%. Such males were divided at random

and mated individually with females in two separate groups, one kept at 22° and the other at 17°C (it was found that athabasca did not breed well at temperatures higher than 22°). Two test series were observed. Although at least half of the tested males should have carried the "sex ratio" X, it was found that the progenies produced by these males presented a nearly continuous variation in proportions of male offspring from 0% to 60% (all progenies including at

| | Total Flies | Number of Tested Males | Males per Progeny Mean | Males per Progeny Standard Deviation |
|---|---|---|---|---|
| Series 1 | | | | |
| "sex ratio" | | | | |
| 17°C | 507 | 9 | 3.84 | 5.3 |
| 22°C | 885 | 14 | 4.47 | 5.7 |
| "normal" | | | | |
| 17°C | 420 | 7 | 43.42 | 10.76 |
| 22°C | 161 | 3 | 46.93 | 1.4 |
| Series 2 | | | | |
| "sex ratio" | | | | |
| 17°C | 733 | 13 | 8.22 | 5.6 |
| 22°C | 1104 | 19 | 7.50 | 7.05 |
| "normal" | | | | |
| 17°C | 1825 | 30 | 44.14 | 8.9 |
| 22°C | 1131 | 21 | 47.85 | 6.3 |

least 50 individuals), a phenomenon like that reported by Sturtevant (1940) for the related species D. affinis. For purposes of making comparisons, "sex ratio" progenies were arbitrarily defined as those having a male percentage less than 25, since, for the minimum size of the progenies, less than 25% males was a significant deviation from 50% males at the 0.001 level. The results are given in the table. No significant difference between the results at the different temperatures was found.

* Presently at the University of Pennsylvania, Philadelphia. Complete address available from Dr. D.D. Miller, Dept. of Zoology, University of Nebraska, Lincoln, Neb. 68508.

Singh, A. Panjab University, Chandigarh, India. The Drosophilidae of Assam, India. As many as 853 Drosophila flies were collected from Assam, India. The collections were made over fermenting substances, mainly the fruits, by the bait-trap and sweep methods. The study of this collection revealed six species which are all reported for the first time from this area. The frequency distribution of the species at five sites is given in Table 1.

Table 1. Frequency distribution of the various species of the Drosophilidae in Assam.

| Species | Asom Hotel ♂ | Asom Hotel ♀ | Gari Gaon ♂ | Gari Gaon ♀ | Veg. Market ♂ | Veg. Market ♀ | Univ. campus ♂ | Univ. campus ♀ | Burnihat ♂ | Burnihat ♀ | Total ♂ | Total ♀ | Total ♂ & ♀ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Genus Drosophila | | | | | | | | | | | | | |
| Subgenus Sophophora | | | | | | | | | | | | | |
| 1. D. bipectinata | 21 | 2 | 3 | - | 2 | - | 28 | - | 24 | - | 78 | 2 | 80 |
| 2. D. ananassae | 41 | 31 | 17 | 20 | 30 | 37 | 108 | 125 | 111 | 146 | 307 | 359 | 666 |
| 3. D. malerkotliana | - | - | - | - | - | - | 1 | - | 1 | - | 2 | - | 2 |
| 4. D. kikkawai | 9 | - | 3 | 6 | 3 | - | 18 | 17 | 17 | 23 | 50 | 46 | 96 |
| 5. D. punjabiensis | 1 | - | 2 | - | - | - | - | - | 2 | 1 | 5 | 1 | 6 |
| Subgenus Drosophila | | | | | | | | | | | | | |
| 6. D. immigrans | 1 | - | - | - | - | - | 1 | - | - | 1 | 2 | 1 | 3 |
| Grand total | 73 | 33 | 25 | 26 | 35 | 37 | 156 | 142 | 155 | 171 | 444 | 409 | 853 |
| Period of collection | 17/4-2.5 1969 | | 30/5 1969 | | 25/4 1969 | | 19/22&30/7 & 1/8/69 | | 2/17&24/6 & 7/7/69 | | | | |

Yannopoulos, G. University of Patras, Greece. Seasonal differences of spontaneous autosomal recessive lethal mutation frequencies in a wild Greek Drosophila melanogaster population.

The present communication constitutes a preliminary report of a much wider investigation on the polymorphism of spontaneous autosomal recessive lethal mutations in a wild Drosophila population of S.W. Greece.

Behnia, A. and G. Koliantz in a recent communication (1972) reported that they have not been able to observe seasonal changes of lethal gene frequencies in a natural population of Tehran.

The data presented here come from a place called Koleika which lies at a distance of about 8 km from Patras. (The town of Patras is situated in the N.W. of Peloponnesus.) Flies were collected during three seasons, namely summer, spring and autumn. Captured males were individually mated with virgin Cy $L^4$/Pm females in order to detect in the $F_3$ the frequencies of second chromosomes bearing lethals. Table 1 shows the significant differences found between the frequencies detected from summer and autumn and spring and autumn collections, respectively. No statistically significant differences were found between the summer and spring collections. It is also worthwhile mentioning that rates of spontaneous autosomal recessive lethal mutations found in northern Greece by other research workers (Pelecanos, M. and A. Pentzos-Daponte 1970) in autumn collections, is much lower as compared with ours. Further investigation and analysis of the data presented here are in progress.

Table 1. Seasonal differences in spontaneous autosomal recessive lethal mutation frequencies.

| Season of capture | No. of parents captured | No. of chromosomes tested in the $F_3$ | No. of lethals | Lethals (percent) | No. of parents which yielded lethals | No. of parents which yielded more than one lethal | $\chi^2$ 1 d. f. |
|---|---|---|---|---|---|---|---|
| Summer (June) | 236 | 1127 | 374 | 33.19 | 114 | 100 | $\chi^2 = 2.55$, $P > 0.05$ |
| Spring (April) | 231 | 933 | 341 | 36.55 | 89 | 72 | $\chi^2 = 4.7$, $P < 0.05$ |
| Autumn (November) | 152 | 735 | 307 | 41.77 | 55 | 53 | $\chi^2 = 11.7$, $P < 0.01$ |

References: Behnia, A. and G. Koliantz 1972, DIS 48:80; Pelecanos, M. and A. Pentzos-Daponte 1970, DIS 45:79.

Miller, D.D. and J. Jaenike. University of Nebraska, Lincoln, Nebraska; Princeton University, Princeton, New Jersey. Recent extensions of the known geographical ranges of "eastern" and "western-northern" D. athabasca based on salivary gland chromosome sequences.

As speculated by Miller and Voelker (Journ. Hered. 63:3-10, 1972), the zone of overlap of "eastern" and "western" (better "western-northern") D. athabasca probably extends eastward from Minnesota to New England (suggested by earlier studies on Y chromosome types, copulation durations, crossability). However, salivary gland chromosome material with which to determine the presence of the two kinds of athabasca together had come only from Minnesota. During the summer of 1972 one of us (J.J.) collected D. athabasca in Maine (mainland and islands just east of Penobscot Bay), and these col-

lections have provided material showing, by salivary gland chromosome sequences, the presence of both "eastern" and "western-northern" athabasca in that part of New England.

As pointed out by Miller and Voelker, sequences of certain chromosome arms have been found in only one of the two kinds of athabasca and not the other (disregarding one ambiguous Minnesota strain classified only by Y type). Some of these, particularly ones involving the distal portions of arms, should be useful for recognition of "eastern" or "western-northern" athabasca even in preparations of "average" or "inferior" quality - specifically "eastern" XL MI-MII vs. "western-northern" I-distal; "eastern" XS MI vs. "western-northern" I-distal (or derived sequences); "eastern" BL MII or MIII vs. I=MI (though the latter occurs in both kinds of athabasca); "eastern" C MII and derived sequences (e.g. MIV, MV, but not III) vs. "western-northern" I-distal and derived sequences. Though complete analysis of salivary gland sequences in recent stocks from Maine has not been accomplished, observations of the above sequences (plus a few others) have shown that the cultures do contain both "eastern" and "western-northern" sequences and that each strain is consistent in having sequences of one kind only. The following numbers of strains have been so identified: Sunset (Deer Isle), 3 "eastern" and 7 "western-northern"; Bar Harbor (Mount Desert Island), 4 "eastern"; Dedham (mainland, ca. 10 miles s.e. of Bangor), 5 "eastern" and 2 "western-northern"; relatively small islands just south of Deer Isle (Dave's, Farrel, George Head, Potato, St. Helena, and Wreck), all "western-northern", 4 from St. Helena and one from each of the others.

Although Miller (Amer. Midl. Nat. 60:52070, 1958) expressed doubt that D. athabasca exists at certain points where it had been reported in the Midwest, specifically St. Louis and Lincoln, recently the presence of athabasca in these localities has been confirmed. In 1970 several apparently athabasca males collected at the St. Louis suburb of Webster Groves by Dr. H.D. Stalker came to one of us (D.D.M.), by way of Dr. Robert Voelker. Some of these males were crossed successfully to "eastern" females of a Bass Lake, Minnesota, strain, yielding offspring with strictly "eastern" athabasca salivary gland chromosomes. There is no reason now to doubt that athabasca does exist in the St. Louis area, as had been reported by Carson and Stalker (e.g. 1951). In July of 1972 one of us (D.D.M.) collected Drosophilas in southeastern Nebraska (Lincoln and Sprague, Lancaster Co., and Crete, Saline Co.) and got a few females and males that seemed athabasca-like. From five of the females (one from Lincoln, two from each of the other places) strains of athabasca have been established in the laboratory; examination of salivary gland chromosomes has shown that each has "eastern" sequences.

Angus, D.S. Salisbury College of Advanced Education, Adelaide, S.A., Australia.
Drosophila fauna of Humbug Scrub and Adelaide, South Australia.

Flies were collected over fermenting banana baits and by sweeping over vegetation between December 1971 and August 1972 at Humbug Scrub, Para Wirra National Park, a dry sclerophyll scrub 25 miles N.E. of Adelaide, and at Toorak Gardens, an Adelaide suburb.

The following species were found:

| Species | Humbug Scrub number | % | Toorak Gardens number | % |
|---|---|---|---|---|
| D. bryani | 1 | + | 0 | |
| D. buskii | 0 | | 3 | + |
| D. enigma | 0 | | 12 | + |
| D. fumida | 67 | 12 | 21 | 1 |
| D. immigrans | 37 | 7 | 262 | 11 |
| D. melanogaster | 31 | 5 | 66 | 3 |
| D. novopaca | 9 | 2 | 3 | + |
| D. repleta | 1 | + | 20 | 1 |
| D. simulans | 415 | 74 | 298 | 84 |
| Pholadoris sp. nov. | 2 | + | 0 | |
| Total | 563 | | 2491 | |

The cooperation of the National Parks Commission, South Australia is acknowledged. Supported by S.T.C. R.G. 25A/1.

Trippa, G., A. Lovere, A. Micheli and I. Miola. Istituto di Genetica, Università di Roma, Italia. Frequencies of SD chromosomes in natural populations of Drosophila melanogaster.

Samples of wild flies of Drosophila melanogaster from eight natural populations were collected in Puglia (Castellaneta, Otranto and Corato), Calabria (Sambiase) and Sicily (Ranna, Pedalino, Vittoria and Archi) during September-October 1971. Males, with $+_I/+_{II}$ constitution for the second chromosomes, were individually mated with virgin bw;st females from a line selected for its high degree of sensitivity to $SD^{R-1}$ chromosome (Nicoletti et al., Atti A.G.I. 14:29, 1969).

From the $F_1$ progeny of each cross, three males were collected and mated individually to bw;st females; from the $F_2$ progeny of only one cross, three males of the same constitution $+_I$/bw;st/st or $+_{II}$/bw;st/st were collected and mated to bw;st females. The k values (number of +/bw;st/st individuals/total progeny) were calculated in the $F_3$ generation.

The k values varied between 0.5 and 1.0, depending on whether the tested second chromosome behaved as a normal or as an SD chromosome. The results are presented in Table 1.

Table 1. The distribution and the properties of the SD-carrying chromosomes found in eight natural populations.

| Populations | Number of tested chromosomes | $\bar{k}$* | Absolute frequency of SD chromosomes with k values between .65-.75 | .75-.85 | .85-1.00 | TOTAL | Percent frequency of the SD chromosomes |
|---|---|---|---|---|---|---|---|
| Castellaneta | 235 | .52 ± .003 | 1 | 1 | | 2 | 0.85 |
| Otranto | 221 | .53 ± .003 | 3 | 1 | 5 | 9 | 4.07 |
| Corato | 162 | .52 ± .004 | 6 | 3 | | 9 | 5.56 |
| Vittoria | 10 | .55 ± .017 | | | 1 | 1 | 10.00 |
| Ranna | 279 | .52 ± .003 | 4 | 5 | 2 | 11 | 3.94 |
| Archi | 268 | .51 ± .003 | 12 | 1 | | 13 | 4.85 |
| Pedalino | 121 | .52 ± .005 | 1 | 1 | | 2 | 1.65 |
| Sambiase | 284 | .52 ± .003 | 8 | 9 | 5 | 22 | 7.75 |

* the k values referring to the following progenies: 1) with less than 40 individuals; 2) with a k lower than 0.35; 3) with a k higher than 0.65 have not been computed while estimating the normal mean k.

As it can be seen, SD-carrying chromosomes have been found in all the eight populations examined. Though a SD chromosome would be expected to attain high frequencies in few generations, it is a common observation that this is not the case. There can be many ways by which natural selection might balance the effects of the behaviour of SD, such as a reduction of the reproductive fitness of SD heterozygous males (Nicoletti et al., Accad. Naz. Lincei XLII: 383, 1967) or the association of SD to lethal genes, or the spontaneous appearance of insensitive $SD^+$ alleles.

SD chromosomes with inversions (Sandler et al., Genetics 44:233, 1959' Hiraizumi, and Nakazima, DIS 40:72, 1965) as well as without inversions (Nicoletti and Trippa, Atti AGI 12: 361, 1967) have been identified by means of salivary gland chromosomes examination. The present material has not yet been examined from this point of view.

Counce, S.J. Duke University, Durham, North Carolina. Variation in germ cell number and distribution in primitive dipterans.

In dipterans, germ cells are derived from cells which bud off at the posterior pole during late cleavage or early blastoderm stages. The number of primary pole cells may be constant, e.g. one in Miastor, two in Sciara, or vary, as in Drosophila, where three or more primary cells form (see Sonnenblick in BIOLOGY OF DROSOPHILA). These pole cells continue to divide, and in the first group, the number of these divisions is usually constant also, producing a predictable number of definitive pole cells. Later, during gonad differentiation, the germ cells may also divide a set number of times. For example, in Miastor, there are three divisions of the pole cells (8 definitive pole cells) which, in the gonads, divide three more times, producing 64 oogonia.

In Miastor, however, although only a single pole cell is initially produced, the number of definitive pole cells does vary. In around 20% of the more than 5000 embryos I have studied, a departure from the expected 8 pole cells has been observed as follows (see table):

| no. P.C.: | >8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 |
|---|---|---|---|---|---|---|---|---|---|
| n | 29 | 329 | 189 | 80 | 127 | 27 | 54 | 29 | 57 |
| % | 3.1 | 35.7 | 20.5 | 8.7 | 13.8 | 2.9 | 5.9 | 3.1 | 6.2 |

Five embryos with 24-32 pole cells were observed, but are not included in the above data. Obviously, one or more pole cells quite frequently undergo a supernumerary division; rarely, fewer than the normal number of divisions may occur. It is not known whether supernumerary divisions also occur during oogonial divisions in the gonad.

During gastrulation, the pole cells are passively transported to the interior as the germ band extends along the dorsal surface of the egg. During this time, they become segregated into two groups of primary germ cells (in Miastor normally 4 and 4) surrounded by lateral mesoderm which will give rise during gonad differentiation not only to supporting elements and follicle cells but to nurse cells as well. In about 20% of all older embryos (103/529) asymmetrical distribution of germ cells to the gonads was found. There was no difference between embryos with normal germ cell numbers (8) and those with aberrant numbers in the frequency with which abnormal gonadal distribution occurred. (Of course, in embryos with odd numbers of germ cells, in "normal" distribution, one of the two gonads would be expected to have an additional cell; this was taken into consideration.)

In the related genus, Heteropeza, Reitberger (Chromosoma 1) also noted a variation in the number of definitive pole cells formed. In some 600 embryos of this species I looked at, some 20% had more than the expected number of definitive pole cells (4). Asymmetrical distribution to the gonads was observed in about 20% of the older embryos, and as in Miastor, was not related to discrepancies in the number of definitive pole cells.

There is some disagreement as to whether all presumptive gonia give rise to embryos, or whether some of them become nurse cells (cf. Camenzind, Chromosoma 18; Counce, Nature 218; and Panelius, Chromosoma 23). The extent to which the number of pole cells, and hence the number of definitive germ cells, may vary in these species must be kept in mind in arguments based upon the number of offspring a single mother larva may be expected to produce.

Colaianne*, J.J. and A.E. Bell. Purdue University, Lafayette, Indiana. On the action of transformer (tra) in males of D. melanogaster.

The third chromosome mutant transformer (tra) was initially described as transforming homozygous female zygotes, tra/tra, into male-like phenotypes (Sturtevant, 1945). It has generally been thought that the gene has no effect on tra/tra male zygotes. Brown and King (1961) have hypothesized that tra is an amorphic mutant whose wild-type allele either suppresses the masculinizing action of other autosomal genes or enhances the feminizing activity of sex-linked loci. However, a recent study (Colaianne and Bell 1972) on the influence of sex of progeny on the lethal expression of the sonless gene (snl) yielded results which indicate a masculinizing effect for transformer in tra/tra males.

In this study we found that the degree of expression of sonless is directly influenced by the sex of the progeny. Given the prerequisites that both mothers and progeny are homozygous for snl, the more male-like an offspring on the male-female continuum the more susceptible it is to snl lethality. Specifically relating to the action of transformer were twenty single-pair matings (snl +/snl + Ubx/tra x + B/Y, Ubx/tra) which produced 1834 daughters and 51 sons. The sons consisted of 49 Ubx/tra and 2 tra/tra, a highly significant deviation from the expected 2:1 ratio, while the daughters were present in the ratio of 1231 Ubx/tra: 603 tra/tra.

Thus tra/tra males do differ from normal males at least in viability. Our results suggest that the tra/tra sons are, in some sense, "super males" in that they are more susceptible than normal males to the sex influenced, lethal action of snl.

References: Brown, E.H. and R.C. King 1961, Genetics 46:143-156; Colaianne, J.J. and A.E. Bell 1972, Genetics 72 (in press); Sturtevant, A.H. 1945, Genetics 30:297-299.

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Ranganath, H.A. and N.B. Krishnamurthy. University of Mysore, Manasagangotri, India. Seasonal studies on Drosophila fauna of Biligirirangana Hills, Mysore.

Biligirirangana Hills, located between 11°5'N and 77° and 78°E in the southeast corner of Mysore State are a somewhat discontinuous, north to south running hill ranges varying from 600 to 1800 meters in altitude. The chain of hills are isolated from both western and eastern ghats. These hill ranges receive both the northeast and southwest monsoon rains. The rainfall during the year varies from 150 to 200 cms and is distributed throughout except the first 3 to 4 months of the year. The first 3 to 4 months of the year is the hottest period, when the forest fire is a usual feature. Wettest period is between July to October. The area is remarkably rich in orchids, epiphytic plants, numerous ferns and club mosses also occur profusely. These variable and interesting features prompted the authors to make a seasonal study of Drosophila fauna in this region. Flies were collected during the first week of every month. The collection record for the year 1971-72 is given in Table 1.

| Year | Months | | nasuta | brindavani | immigrans | albomicans | malerkotliana | mysorensis | anomelani | jambulina | takahashii | rajasekari | pseudoananassae | busckii | nigra | varietas | unidentified | TOTALS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1971 | Nov. | M | 9 | - | 27 | - | 17 | 8 | 5 | 1 | - | - | - | - | - | - | - | 126 |
| | | F | 8 | - | 21 | - | 7 | 18 | 4 | - | - | - | - | 1 | - | - | - | |
| | Dec. | M | 18 | - | 42 | - | 3 | 25 | 21 | 6 | 4 | - | - | - | - | - | 1 | 273 |
| | | F | 14 | 3 | 96 | - | 2 | 12 | 15 | 2 | 6 | - | 1 | 1 | - | - | 1 | |
| 1972 | Jan. | M | 12 | 2 | 39 | - | 11 | 2 | 4 | 6 | 4 | 1 | 2 | - | 1 | - | 5 | 205 |
| | | F | 18 | 7 | 73 | - | 2 | 4 | - | 5 | 5 | - | - | - | 1 | - | 1 | |
| | Feb. | M | 1 | - | 4 | - | - | - | - | - | - | - | - | - | - | - | - | 15 |
| | | F | 1 | - | 7 | - | - | 1 | - | - | - | - | - | - | - | - | 1 | |
| | Mar. | M | - | - | - | - | 4 | 1 | - | 1 | 1 | - | - | - | - | - | - | 13 |
| | | F | - | - | - | - | 6 | - | - | - | - | - | - | - | - | - | - | |
| | Apr. | M | - | - | - | - | - | - | - | - | - | - | 2 | - | - | - | - | 9 |
| | | F | - | - | - | - | 5 | - | - | - | - | - | 2 | - | - | - | - | |
| | May | M | - | - | - | - | 133 | - | - | 2 | 2 | - | 9 | - | - | - | 2 | 297 |
| | | F | 2 | - | - | - | 138 | - | - | 2 | 4 | - | 1 | - | - | - | 2 | |
| | Jun. | M | 6 | - | - | - | 231 | 6 | - | - | - | 1 | 3 | - | - | - | - | 729 |
| | | F | 12 | - | - | - | 455 | 10 | - | 2 | - | 2 | 1 | - | - | - | - | |
| | Jul. | M | 5 | 1 | - | - | 51 | 1 | 3 | 1 | 4 | - | - | - | - | - | - | 243 |
| | | F | 38 | - | 3 | 1 | 126 | 3 | - | 1 | 3 | - | - | - | - | 1 | 1 | |
| | Aug. | M | 56 | 1 | 2 | - | 167 | 8 | 5 | - | 14 | - | - | - | - | - | 1 | 838 |
| | | F | 153 | - | 14 | - | 369 | 16 | 6 | - | 26 | - | - | - | - | - | - | |
| | Sep. | M | 49 | - | 4 | - | 79 | 31 | 4 | - | - | - | - | - | - | - | 2 | 314 |
| | | F | 36 | - | 43 | - | 31 | 24 | 11 | - | - | - | - | - | - | - | - | |
| | Oct. | M | 25 | - | - | - | 4 | 18 | 7 | - | 8 | - | - | - | - | - | - | 150 |
| | | F | 20 | - | 10 | - | 11 | 16 | 7 | - | 24 | - | - | - | - | - | - | |

Table 1. Drosophila fauna of Biligirirangana Hills (1971-72).

A total of 15 species have been recorded. D. malerkotliana is a seasonally versatile species occurring throughout the year while others are seasonally restricted, found only during some months of the year. From this the authors opine that D. malerkotliana is a eurythermal species while other species are stenothermal. D. nasuta and D. mysorensis

(Continued at bottom of next page)

Bock, I.R. University of Western Australia, Nedlands, W.A., Australia.
Hybridization between D. lutea and D. paralutea.

The melanogaster species group is now known to contain about 80 species classified into 11 subgroups, in several of which pairs or groups of morphologically extremely similar species occur (Bock and Wheeler, 1972). All of the 7 members of the takahashii subgroup in particular are very similar in coloration and in morphology of sex-combs and male genitalia; within this subgroup D. lutea Kikkawa and Peng 1938 (distribution: Japan and Korea) and D. paralutea Bock and Wheeler 1972 (distribution: Thailand) are a pair of siblings distinguished in the male sex by the greater number of transverse rows of bristles in the sex-comb of the latter species.

Attempts to hybridize D. lutea and D. paralutea by mass matings have now proved successful in both directions; in either case a relatively small number of $F_1$ offspring was obtained and examination of the testes of the hybrid males revealed that all were considerably reduced in size and contained no motile spermatozoa. Pairing between homologous chromosome arms in the salivary glands of hybrid larvae is consistently poor (Fig. 1); it nevertheless appears that the two species are distinguished by several chromosomal rearrangements, and it is anticipated that further work will admit of a more precise polytene chromosomal comparison between the species.

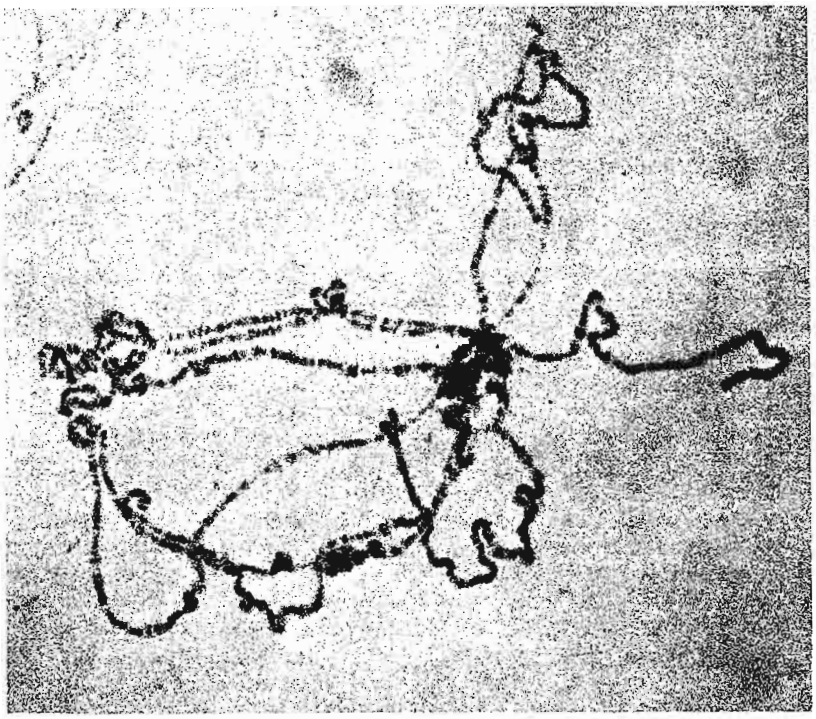

Figure 1. D. lutea x D. paralutea

Grateful acknowledgement is made to the Genetics Foundation, University of Texas, for supplying the stocks.

Reference: Bock, I.R. and M.R. Wheeler 1972, Univ. Tex. Publ. 7213:1-102.

(Continued from preceding page)

were obtained in all months except during February through May. The other species have scattered distribution over the period studied. Drosophila fauna had become virtually thinned out during the months of February, March and April, which may be due to the adverse conditions in nature. Only during the wet months of the year considerable size of Drosophila fauna were encountered.

The species D. malerkotliana and D. immigrans are characteristic in having more females than males during many months. This may be underlined by the property of greater homeostatic abilities of females. D. albomicans (?) of nasuta complex is for the first time reported from India.

Acknowledgements: We wish to express our deepest gratitude to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology, University of Mysore, for his continued help. We are especially appreciative of the hospitality and friendly courtesies of the officials of B.D.O. Department, Chamarajanagar during collections.

Matveeva, N.M., L.I. Korochkin, Institute of Cytology and Genetics, Novosibirsk, USSR. The comparison of esterase spectra of the 3rd instar larvae, pupae and imago in Drosophila virilis.

Esterases in several strains of D. virilis and their hybrids, from the collection of Prof. N. Sokoloff (Moscow, Institute of Developmental Biology), have been investigated using starch gel electrophoresis of individual flies and larvae. Differences in esterase spectra were shown to be present in different stages of the flies ontogenesis. Although esterase-1 is not usually changed, the activity of esterase-2 is higher in imago than in 3rd instar larvae and pupae. Both parental and one hybrid esterase-2 bands are expressed in 3rd instar larvae. Esterase-3 appears rather late and varies in its phenotypical expression in imago. There are some

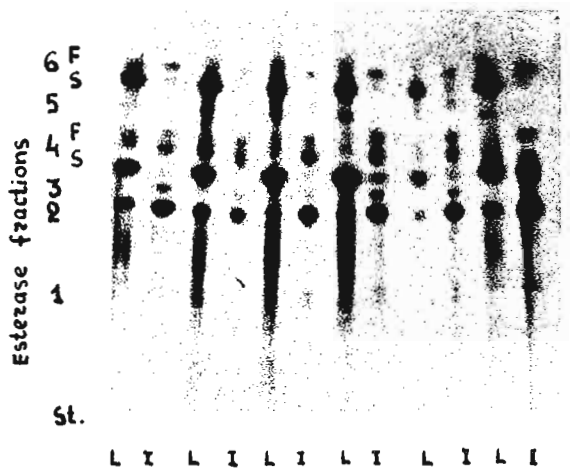


Fig. 1. Electropherograms of esterases of 3rd instar larvae and imago in D. virilis. Individual flies and larvae were used for electrophoresis in each case. I-4=103 B2 strain, 5-10=103 3a strain, II-12=147 strain, L-3rd instar larvae, I-imago. F=fast subband and S=slow subband of the corresponding esterase.

similar regularities in the phenotypical expression of esterases-4 and -6 in larvae and imago (Fig. 1, Table 1). These esterases are represented by two subfractions fast(F) and slow(S), one of which usually predominates in imago in different D. virilis strains. If the F-form of esterase-4 or esterase-6 predominates in

Table 1.
The comparison of relative activities of F- and S-subbands of esterase-6 and esterase-4 during ontogenesis of D. virilis. Activities are expressed in parts of total activity, determined from areas under the curves using a "Joyce and Loebl" microdensitometer.

| Designation of strains of D. virilis in our collection | | Esterase-6 Subfractions Fast (F) | Esterase-6 Subfractions Slow (S) | Esterase-4 Subfractions Fast (F) | Esterase-4 Subfractions Slow (S) |
|---|---|---|---|---|---|
| 103 B2 (R,gl) | Imago | 0.62 | 0.38 | 0.86 | 0.14 |
| | | 0.70 | 0.30 | 0.90 | 0.10 |
| | 3rd instar larva | 0.06 | 0.94 | 0.23 | 0.77 |
| | | 0.08 | 0.92 | 0.10 | 0.90 |
| 103 3a (R,gl) | Imago | 0.61 | 0.39 | 0.76 | 0.24 |
| | | 0.80 | 0.20 | 0.90 | 0.10 |
| | | 0.67 | 0.33 | 0.90 | 0.10 |
| | | 0.70 | 0.30 | 0.90 | 0.10 |
| | | 0.72 | 0.28 | 0.92 | 0.08 |
| | 3rd instar larva | 0.05 | 0.92 | 0.05 | 0.95 |
| | | 0.16 | 0.84 | 0.10 | 0.90 |
| | | 0.16 | 0.84 | 0.10 | 0.90 |
| | | 0.10 | 0.90 | 0.20 | 0.80 |
| | | 0.05 | 0.95 | 0.10 | 0.90 |
| | | 0.10 | 0.90 | 0.11 | 0.89 |
| 147 1B2 (b bk dt) | Imago | 0.87 | 0.13 | | |
| | 3rd instar larva | 0.13 | 1.87 | | |

(Continued at bottom of next page)

Clark, A.M. State University of Leiden, The Netherlands. Differential viabilities of X/0 males.

Würgler and Maier (Mutation Research, 1972 15: 41-53) have reported that after irradiation of mature sperm carrying a ring-X chromosome, the rate of chromosome loss, as recorded by the frequency of X/0 males, depends on the type of female used. They concluded that viability differences cannot account for their data, and suggested that a maternal influence on the repair of X-ray induced lesions might provide an explanation. The following data, obtained by methods similar to those used by Würgler and colleagues, indicate that substantial viability differences between various kinds of X/0 males may be density dependent and may in fact account for the different frequencies of X/0 males recorded in irradiation experiments.

| Egg-laying females per bottle | Mean progeny per bottle | Ratio $\frac{y\ sn^3/0}{Oster/0}$ |
|---|---|---|
| 2 | 148 | 2.22 |
| 6 | 394 | 2.12 ) * |
| 15 | 562 | 3.26 ) |

* Difference significant at the 1% level

Method 1. Females of the genotype $\frac{Oster}{y\ sn^3}$ (Oster stands for y $sc^{S1}$ In 49 $sc^8$) were mated to X·Y males which lacked a free Y chromosome, and the proportions of Oster/0 and y $sn^3$/0 males in the progeny recorded. A range in population densities was achieved by using 2, 6 or 15 egg-laying females per bottle. Egg laying was restricted to 48 hours.

Method 2. Oster females, or y $sn^3$ females, were crossed to X·Y males and the percent of X/0 progeny recorded in each case. Again, varying population densities were achieved by placing 2, 6 or 15 females in each bottle.

| Egg-laying females per bottle | Mean progeny per bottle | Percent of X/0 progeny obtained from: y $sn^3$ females | Oster females |
|---|---|---|---|
| 2 | 112 | 69.1% | 65.9% |
| 6 | 229 | 70.1% | 58.7% |
| 15 | 385 | 70.4% | 49.2% |

In method 1, developing Oster/0 and y $sn^3$/0 males are in competition in the same culture, while in method 2, the particular X/0 males are in competition with Oster/X·Y females as the case may be. Both methods lead to the same conclusion, that as population density increases, Oster/0 males are at increasing disadvantage; this effect could well be related to the Y-suppressed viability factor of chromosomes of the $sc^{S1}$ $sc^8$ type. (See, for example: Hess, Zool. Anz. Suppl. 1963, 26:87-92; Traut, Scheid and Wind, Mutation Res. 1970, 9:489-499.) On the other hand, y $sn^3$/0 males seem to be relatively insensitive to population density over the range covered in these experiments. There is no single measure of the relative viabilities of Oster/0 males and y $sn^3$/0 males. In view of the need to control population density very carefully, it seems that viability difference should still be considered as a possible explanation for the results obtained by Würgler and Maier.

This work received support from the J.A. Cohen Institute for Radiopathology and Radiation Protection, and the Health Research Organization TNO. It was carried out in part under the Association of Euratom with the University of Leiden, Contract Nr. 052-65-I BIAN.

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imago, the larvae are characterized by higher levels of the S-band activity. The changes from the "larval" type of isozymes to the "adult" type take place at the stage of middle pupae. If the S-form predominates in imago, or both the F- and S-subfractions have identical activities, a picture similar to that in imago can be seen in electropherograms of 3rd instar larvae. Equal activity of F- and S-subfractions was observed in the 3rd instar larvae and imago of hybrids between flies of "S" and "F" strains. This work on the esterases of D. virilis yielded results similar to those of Doane (1969), who studied changes in the activities of amylase subfractions, controlled by duplicating genes in D. melanogaster.

References: Doane, W. 1969, Drosophila amylases and problems in cellular differentiation. In: Problems in Biology: RNA in Development (Univ. of Utah Press) 73-109.

Faizullin, L.Z. and V.A. Gvozdev.
Kurchatov's Institute of Atomic Energy, Moscow, U.S.S.R. Genetic balance and dosage compensation in D. melanogaster.

According to Muller's views, the dosage compensation of X-linked structural genes in Drosophila is maintained by compensatory genes in the X-chromosome acting negatively and proportionally to their dose (1). This mechanism implies that the increase of a compensatory gene dosage by duplications of various regions of the X-chromosome would result in the decrease of the activity of some X-linked structural genes. We determined the activities of Pgd (1-0.65) and Zw (1-63.0) structural genes for 6-phosphogluconate dehydrogenase (6PGD) and glucose-6-phosphate dehydrogenase (G6PD), respectively, measuring the specific activities (SA) of the two enzymes in crude extracts of flies carrying various duplications. The results are presented in Table 1. Three different duplications for the distal part of the X-chromosome, containing

Table 1. Relative SA of 6PGD and G6PD of hyperploid females and metafemales. (SA of diploid females = 100. Number in parenthesis indicates the number of experiments).

| Genotype | Dose | | Relative activity %% | |
|---|---|---|---|---|
| | Pgd | Zw | 6PGD | G6PD |
| 2X (normal) | 2 | 2 | 100 | 100 |
| 2X+1-9A | 3 | | 138 ± 7 (7) | |
| 2X+1-9B | 3 | 2 | 141 ± 6 (13) | 98 ± 7 (8) |
| 2X+1-14A | 3 | | 137 ± 3 (4) | |
| 2X+16A-20 | 2 | | 107 ± 3 (12) | |
| 2X+14A-20 | 2 | | 96 ± 4 (11) | |
| 2X+9B-20 | 2 | 3 | 106 ± 5 (12) | 150 ± 7 (11) |
| 3X (metafemale) | 3 | 3 | 149 ± 5 (10) | 118 ± 3 (4) |
| 2X+Df(1)Pgd-kz(2D3-6; 2F3-4) | 2 | 3 | 99 ± 4 (12) | 125 ± 11 (6) |

Pgd locus (1-9A, 1-9B and 1-14A regions) result in the 1.4 fold increase of SA of the 6PGD irrespective of the duplication length. In fact, this increase is proportional to the enhancement of the gene dose. On the other hand, duplications for the right part of the X-chromosome (regions 16A-20, 14A-20 and 9B-20) not covering Pgd locus exert no influence on the Pgd activity. Therefore in contrast to Muller's results (2) we failed to detect any negative (or positive) regulatory genes for Pgd locus within either the right or the left parts of the X-chromosome. The similar results were obtained for Zw locus: the addition of the right or the left parts of the X-chromosome resulted in the increase of the G6PD activity proportionally to the Zw dose (9B-20 region) or showed no effect (1-9B).

Muller suggested that dosage compensation results from the simultaneous action of the group of compensatory genes scattered through the X-chromosome. Therefore the investigation of the influence of the additional entire X-chromosome in females on the SA of the two dehydrogenase was undertaken. Table 1 shows the 1.5 fold increase of 6PGD activity in metafemales (3X2A) as compared to normal females (2X2A) proportionally to the gene dosage. The 1.2 fold increase was observed for G6PD activity. The addition of the third X-chromosome with small Pgd$^-$ deficiency (2D3-6;2F3-4) had no effect on 6PGD activity although G6PD increases to 25% as compared to normal females. These results may be considered as showing the equal activity of genes in each X-chromosome in females and metafemales. The obtained data are in contradiction to Muller's idea of the existance of X-linked compensatory genes able to depress the activity of the other genes of X-chromosome proportionally to the number of X-chromosomes.

The two levels of the activity of the X-chromosomes of males and females could be deter-

Table 2. 6PGD and G6PD activities of triploids (3X3A) and intersexes (2X3A). (SA is expressed as nmoles of NADP/mg protein/min. Numbers in parenthesis indicate the number of experiments.)

| Genotype | 6 P G D | | G 6 P D | |
|---|---|---|---|---|
| | SA | SA:X-chromosome number | SA | SA:X-chromosome number |
| 3X3A | 184 ± 8 (5) | 61 | 263 ± 10 (5) | 88 |
| 2X3A | 302 ± 35 (4) | 151 | 384 ± 47 (4) | 192 |

(Continued at bottom of next page)

Ouweneel, W.J. and J.M. van der Meer.
Hubrecht Laboratory, Utrecht, Netherlands. Determination and regulation in the haltere disc.

To study the organ map of the haltere disc, mature discs were fragmented and transplanted into mature larvae. The analysis of the metamorphosed implants showed that the prospective metathorax is located in the anterior part of the disc, including many "adventitious bristles" (appearing only in implants) and two newly discovered metathoracic papillae. The knob in the posterior part of the disc is the anlage of the capitellum; this is concentrically surrounded by the anlagen of the pedicel and the scabellum, both carrying characteristic groups of sensilla campaniformia, which were also localized. The haltere anlagen are surrounded by those parts of the prospective metathorax which will be located at the base of the haltere in the imago, including the "metathoracic bristle group" and the metathoracic spiracle. This organ map is homologous with that of the wing disc (Murphy[1]).

To investigate regulative effects in disc fragments (cf. Schubiger[2] and Bryant[3] for the leg disc), anterior and posterior fragments of mature discs were cultured in the abdomina of adult hosts, where they usually underwent cell proliferation from the wound surfaces. After the culturing period (8 to 23 days) they were transplanted back into mature larvae. Analysis of the metamorphosed implants showed that 3/4 anterior (proximal) fragments formed blastemas from which posterior (distal) structures arose by regeneration. Some structures were regenerated in duplicate. It seems that the metathorax anlage is able to regenerate a complete haltere; this has to be confirmed, however, by transection at more proximal levels. (Transplantation experiments with different anterior-posterior transection levels and with medial and lateral fragments are in progress.)

On the other hand, 1/4 posterior disc fragments formed blastemas in which the distal structures normally produced by these fragments were duplicated in mirror image. This led to the formation of bilaterally symmetrical transplants. The posteriormost anlagen were duplicated most frequently, i.e., those of the metathoracic bristle group, the capitellum, and the ventral pedicellar and scabellar sensillae groups. The strictly symmetrical structure of the transplants suggests two important, preliminary conclusions which have already been anticipated theoretically[4]: (1) if proliferation is limited, a small blastema is formed; nevertheless the fragment and blastema together seem to "split up" symmetrically ("homonomous arealization") and to produce only the most posterior anlagen in duplicate, while the more anterior anlagen originally present in the fragment drop out altogether; this would imply a partial "repatterning" within the original disc fragment. (2) If proliferation is very extensive, a blastema is formed that is larger than the original fragment; here also the fragment and blastema together seem to "split up" symmetrically, and now produce not only all the structures typical for the original fragment in duplicate, but also more proximal structures for which no anlagen were present in the original fragment. This leads us to a concept of "duplicative regeneration" which is at variance with the hypothesis of Bryant[3].

References: 1. Murphy, C. 1972, J. exp. Zool. 179:51-62; 2. Schubiger, G. 1971, Devel. Biol. 26:277-295; 3. Bryant, P.J. 1971, Devel. Biol. 26:606-615; 4. Ouweneel, W.J. 1972, Acta Biotheor. 21:115-131.

(Continued from preceding page)
mined by different ratios of the number of X-chromosomes to autosome sets. Therefore the comparison of SA of the 6PGD and G6PD in triploids (3X3A) and intersexes (2X3A) was undertaken. Table 2 shows that the SA of the two dehydrogenases are higher in intersexes than in triploids. The calculation of the level of enzymatic activity per X-chromosome showed the two-fold increase of the activity of these genes in intersexes as compared to the diploid females. Thus the similar (low) levels of the activity of X-chromosome are characteristic for the normal, hyperploid and triplo-X females. In contrast, males and intersexes (the latter in our experiments had male phenotype) have a two-times higher level of activity of X-chromosome. It is possible to suggest that the low level of X-chromosome activity is determined by the X:A ratio more or equal to 1.0 while the high level of the X-chromosome activity is expressed when X:A is less than 1.0.

References: 1. Muller, H.J. 1950, Harvey Lect. 43:165-229; 2. Muller, H.J. and W.D. Kaplan 1966, Genet. Res. 8:41-59.

Ogah, F. and R. MacIntyre. Cornell University, Ithaca, New York. The persistence of Acid phosphatase-1 null mutants of Drosophila melanogaster in experimental population cages.

Four Bennett box type population cages were set up to study the fate of Acph-1 null alleles in laboratory populations. Two null allele stocks Acph-$1^{n-5}$ and Acph-$1^{n-11}$ (Bell and MacIntyre, 1972) obtained by means of EMS (Bell and MacIntyre, 1972) were used along with wild type stocks containing the fast allele, Acph-$1^{B}$, to set up the cage populations as shown in Table 1.

The initial frequencies of Acph-$1^{B}$ are 0.80 in cages 1 and 2, and 0.20 in 3 and 4. $F_1$ heterozygotes as founders were used to minimize the initial effect of "luxuriant" heterosis

Table 1. Founders of the cages.

| Cages 1 and 2 | | Cages 3 and 4 | |
|---|---|---|---|
| 120 ♂♂<br>120 ♀♀ virgins | BB stock (wild type) | 60 ♂♂<br>60 ♀♀ virgins | null stock (n-5) |
| 40 ♂♂ $F_1$<br>40 ♀♀ virgins $F_1$ | null stock (n-5) x BB (wild type) | 60 ♂♂<br>60 ♀♀ virgins | null stock (n-11) |
| 40 ♂♂ $F_1$<br>40 ♀♀ virgins $F_1$ | null stock (n-11) x BB (wild type) | 40 ♂♂ $F_1$<br>40 ♀♀ virgins $F_1$ | null stock (n-5) x BB (wild type) |
| | | 40 ♂♂ $F_1$<br>40 ♀♀ virgins $F_1$ | null stock (n-11) x BB (wild type) |

in cages founded by flies from different strains. (See MacIntyre and Wright, 1966).

For the first eleven months egg samples were taken monthly from each cage. A thin layer of food was spread on the handle of plastic teaspoons, and one of these was put in a vial and placed into the cage in the normal position of a food vial. This was allowed to stay for thirty six hours. Then the thin layer of food was transferred to a half pint bottle where the eggs were cultured. The adult flies were collected and acid phosphatase activity was assayed for by means of spot test (Bell, et al., 1972). In this assay the null homozygotes are completely lacking the bright red color indicative of Acph-$1^{B}$ homozygotes and Acph-$1^{B}$/Acph-$1^{n}$ heterozygotes. Consequently, only null homozygotes could be used to determine the Acph-$1^{B}$ frequencies in the cages.

Table 2 shows the trend in the change of the frequencies of null alleles from one gener-

Table 2. Frequency of Acph-$1^{n}$

| Months | Date | CAGES 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|
| 1 | 7/26/70 | 0.36 ± 0.03 | --- | 0.74 ± 0.03 | --- |
| 2 | 8/26/70 | 0.46 ± 0.05 | 0.36 ± 0.03 | 0.67 ± 0.03 | 0.78 ± 0.02 |
| 3 | 9/26/70 | 0.14 ± 0.03 | 0.12 ± 0.02 | 0.58 ± 0.05 | 0.75 ± 0.04 |
| 4 | 10/26/70 | 0.39 ± 0.03 | 0.18 ± 0.03 | 0.29 ± 0.03 | 0.73 ± 0.02 |
| 5 | 11/27/70 | 0.65 ± 0.04 | 0.41 ± 0.04 | 0.69 ± 0.04 | 0.66 ± 0.03 |
| 6 | 12/26/70 | 0.49 ± 0.04 | 0.55 ± 0.04 | 0.66 ± 0.04 | 0.68 ± 0.04 |
| 7 | 1/26/71 | 0.41 ± 0.04 | 0.60 ± 0.06 | 0.76 ± 0.03 | 0.57 ± 0.03 |
| 8 | 2/26/71 | 0.37 ± 0.04 | 0.48 ± 0.04 | 0.59 ± 0.03 | 0.62 ± 0.04 |
| 9 | 3/26/71 | 0.42 ± 0.04 | 0.57 ± 0.04 | 0.69 ± 0.03 | 0.61 ± 0.04 |
| 10 | 4/26/71 | 0.50 ± 0.04 | 0.45 ± 0.04 | 0.50 ± 0.05 | 0.62 ± 0.04 |
| 11 | 5/26/71 | 0.44 ± 0.04 | 0.35 ± 0.04 | 0.41 ± 0.04 | 0.52 ± 0.04 |
| 24 | 6/12/72 | 0.46 ± 0.05 | 0.54 ± 0.05 | 0.49 ± 0.05 | 0.35 ± 0.04 |

ation to the next for the four population cages. Initially in cages 1 and 2 the frequencies of null mutant rose slightly, and those for 3 and 4 decreased slightly. Then there was an apparent stabilization at frequencies of about 0.6 for null alleles in all the cages.

The apparent lack of selection against Acph-$1^{n}$ homozygotes and heterozygotes in experi-

Berger, E.M. and R. Canter. State University of New York, Albany, N.Y.

"Moulting" esterases of Drosophila.

During the ontogeny of Drosophila pseudoobscura, a series of 10 β-naphthyl esterase isozymes can be observed by electrophoresis in acrylamide gels. As seen in Figure 1, esterases 1 through 5 appear throughout development, while esterases 6 through 10 are stage specific. Esterases 8, 9 and 10 are present in first instar larvae, but gradually diminish in activity, so that by the third instar they are absent. At the onset of pupation these isozymes reappear, and continually increase in activity through metamorphosis. Esterase 6 and 7 first appear during adult development, increasing in activity until eclosion. At eclosion esterases 6, 7, 8, 9 and 10 simultaneously disappear from the adult. The loss of these isozymic forms is not due to some rapid inactivation, for all five isozymes can be observed in homogenates of the discarded pupal case. Esterases 1 through 5 are absent in these homogenates. Cohomogenization of newly eclosed adults and their empty pupal case restores the 10 banded pre-eclosion pattern.

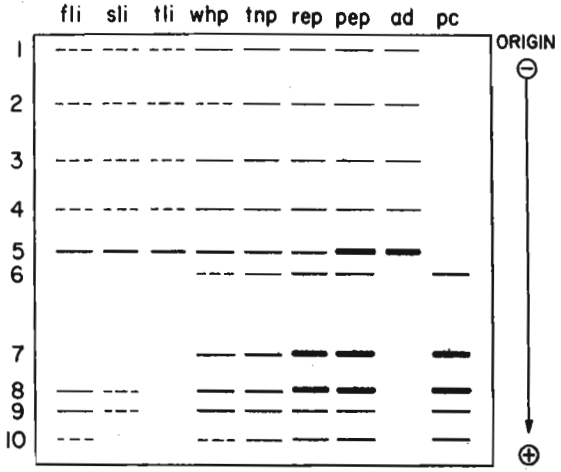



Fig. 1. Ontogeny of esterase isozymes in D. pseudoobscura. Stages are fli - first larval instar; sli - second larval instar; tli - third larval instar; whp - white pupa; tnp - tan pupa, no eye color; rep - pupa, eye colored red, no other pigmentation; pep - pre-emergent pupa, fully pigmented; ad - 9 day old adult; pc - discarded pupal case. D. pseudoobscura strain Est $5^{1.12}$ used in this study was obtained from Dr. R.C. Lewontin, Dept. of Biology, University of Chicago.

While the function of these stage specific esterases is unknown, their developmental timing and post-eclosion localization, suggest a possible role in the moulting process: we therefore designate these enzymes as "moulting esterases". Analagous esterase systems have now been observed in D. melanogaster, D. robusta, D. pattersoni, D. buskii and D. funebris. In D. pseudoobscura we now have allelic variants for several of these systems and are proceeding in genetic analyses.

This work has been supported by a grant from the NIH (GM 18910).

(Continued from preceding page)

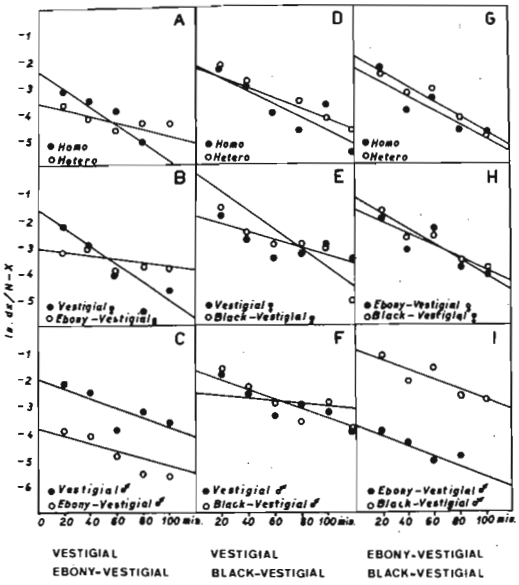
mental populations is rather surprising. In addition, other tests indicate that the viability and developmental rate of null mutants do not differ from those of wild type flies. The following question, then, might be asked: Is acid phosphatase-1 a relict enzyme? No naturally occurring null alleles have been found in populations of D. melanogaster and, in fact, in this species, the gene-enzyme system is very monomorphic (O'Brien and MacIntyre, 1969; MacIntyre, 1972 and unpublished). The observation that approximately 90% of the soluble acid phosphatase activity detected by our assay methods is due to acid phosphatase-1 also argues against a trivial role for this enzyme in D. melanogaster. (Bell and MacIntyre, 1973.)

The apparent dilemma may be due, in part, to the undoubted real differences between the environments of the laboratory and the fly's natural ecological niche. Certain factors which make this enzyme activity very importnat may be absent under laboratory conditions. We plan to test this possibility as soon as the physiological substrate(s) of the enzyme are determined.

The results described here have another important ramification. If the environment of the laboratory is so different that selection pressure against individuals deficient for a particular enzyme is absent or even reduced, experiments designed to detect selective differences between individuals carrying different allozymic forms of that enzyme, are almost certainly going to give negative results. (For example, see Yamazaki, 1971.) In other words, a demonstration of selective neutrality of certain alleles specifying allozymes is, in fact, a meaningless result if the organism doesn't even need the enzyme under the conditions of the experiment.

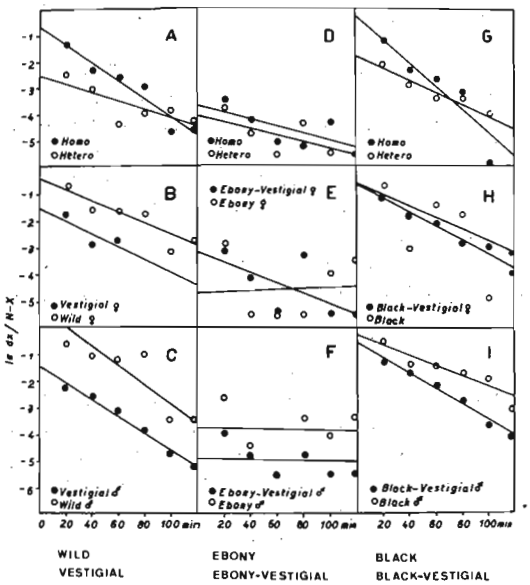
Van den Haute, J.P. and A. Elens.
Facultés Universitaires N.D. de la Paix, Namur, Belgium. Vigor factors in sexual selection.

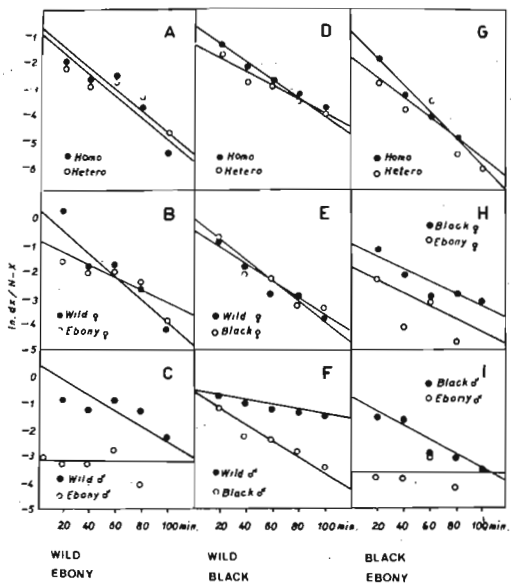
Sexual selection is a complex phenomenon. Various factors, some of which control the choice of a partner, and others control sexual activity as such, are at work at the same time. According to Faugères et al. (1971), in the beginning of the experiments of sexual selection ethological factors are primordial, but when the most receptive females are inseminated, only the males vigorous enough to court a long time have some chance to succeed with the less receptive females; the metabolic factors, controlling the sexual activity, are predominant.




To reach a deeper understanding of the phenomenon of sexual selection, it is therefore necessary to use a method enabling us to distinguish between both types of factors and to follow their action in the course of time. It seems that a method described in 1964 by Wattiaux can help in this way; the purpose of the present paper is to show its application to some cases of sexual competition between various strains of D. melanogaster.

In the multiple choice direct observation method fully described elsewhere (1958, 1964) and which has been successfully used by many authors, the graphic expression of the mating successes are curves beginning exponentially but ending as sigmoids approaching plateaux, the levels of which differ. The results of the comparisons between the various genotypes in competition can be biased by the time at which the observation is ended. Consequently it seems better to turn into straight lines the curves of mating successes, by logarithmic transformation; the value of the measure becomes independent from time.




In the formula of Wattiaux

$$\frac{dx_A}{(n_A - x_A)dt} = bk\, e^{-kt}$$

$n_A$ is the total number of individuals of one sex and genotype, $dx_A$ the number of these flies mating in the interval of time dt, and $x_A$ the number of these flies having mated. After logarithmic transformation

$$\ln \frac{dx_A}{(n_A - x_A)dt} = \ln bk - kt = a - kt \qquad \text{if} \qquad b = \frac{e^a}{k}$$

The two constants a and k can easily be estimated from the regression line

$$\hat{Y} = A - k(X - \bar{X}) \quad \text{where} \quad a = A + k\bar{X}.$$

The superiority of such an expression of the sexual activity or sexual isolation on other indices or coefficients generally used is grounded on the biological signification of the constants A and k . They give information not only on the level of sexual activity but on its variations with time as well. A steep slope of the regression line means that the sexual vigor falls rapidly after the first minutes initiating the competition. In this case the ethological factors are predominant. On the other hand, if the slope is not marked, the sexual vigor remains constant and the metabolic factors prevail.

The figures here presented concern 9 pairs of strains in competition. They give the regression lines allowing a comparison between homogamic and heterogamic matings, between the copulations of the females of both strains in competition, and also between copulations of the males. Table 1 shows the results of a covariance analysis.

Table 1. Probability of no significant differences.

| Strain A | Strain B | Homogamic versus Heterogamic Matings | | Males A versus Males B Activity | | Females A versus Females B Activity | |
|---|---|---|---|---|---|---|---|
| | | Difference | | Difference | | Difference | |
| | | Slope | Elev. | Slope | Elev. | Slope | Elev. |
| vestigial | x ebony vestigial | - | - | - | c | - | - |
| vestigial | x black-vestigial | - | - | - | - | - | - |
| black-vestigial | x ebony-vestigial | - | - | - | b | - | - |
| wild | x vestigial | - | - | - | c | - | - |
| ebony | x ebony-vestigial | - | - | - | a | - | - |
| black | x black-vestigial | - | - | - | c | - | - |
| wild | x ebony | - | - | b | - | - | - |
| wild | x black | - | - | - | c | - | - |
| black | x ebony | - | - | a | b | - | b |

- = non-significant a = $P < 0.100$ b = $P < 0.025$ c = $P < 0.005$

This indicates that a significant isolation is never found, for any of the pairs of strains in competition. Of course, the "isolation estimates" calculated according to Merrel (1950), should give the appearance of an isolation between wild and vestigial ($I = 0.45$; $X^2 = 28.57$) or black and black-vestigial ($I = 0.56$; $X^2 = 16.13$). Yet, the "isolation indices" calculated according to Petit and Ehrman (1968) for the same pairs of strains are not high enough (respectively $2.02 \pm 0.15$ and $1.72 \pm 0.10$) to confirm this opinion. But the comparison of the regression lines gives more information: the figures presented visualize quite well how the ratio of heterogamic on homogamic copulations increases with time: similar fluctuations are not uncommon (see also the case of vestigial with ebony-vestigial) and have yet been previously described (1970).

If one excepts the case of black and ebony, the females genotypes have not influence. But the most striking differences appear in comparing the male sexual activities (see Table 1)

Particularly demonstrative is the behaviour of the males ebony: the elevation of the regression line is always relatively low, but the slope is never a steep one, and such characteristics appear when in competition with wild, black, or ebony-vestigial. The sexual activity of the males ebony, although never high, remains however remarkably constant with time (a confirmation of which is easily obtained by extending the duration of observation to 4 or 6 hours); such a constance of the vigor may help (with other factors as e.g. heterosis) in protecting ebony from elimination when in competition with his wild type allele. On the other hand, the steeper slope of their regression line shows that the sexual activity of the wild males, high in the beginning of the experiment, falls rapidly; the black males behave similarly, but with a lower level of activity. For both the vigor factors seem less important than the ethological ones. The presence of the vestigial gene is in every case responsible for a lowered male sexual activity, but is does not change otherwise the behavioural characteristics of the wild, ebony or black males, since the slopes of the regression lines are nearly the same.

Generally speaking, the results of the covariance analysis are, for the male activities,

in good agreement with the calculated values of the Petit and Ehrman indices and even of the Merrel coefficients; but the comparison of the regression lines seems more interesting, in enabling us to make obvious the part played by the vigor factors.

References: Elens, A. 1958, Experientia 15:274; Elens, A. and J.M. Wattiaux 1964, DIS 39:118; Faugères, A., C. Petit and E. Thibout 1971, Evolution 25:265; Merrel, D.J. 1950, Evolution 4:326; Petit, C. and L. Ehrman 1968, Bull. Biol. 102:433; Wattiaux, J.M. 1964, Z. Vererbungsl. 95:10; Wattiaux, J.M. and A. Elens 1964, DIS 30:118.

Mahajani, S. and V.L. Chopra, Indian Agricultural Research Institute, New Delhi, India. Nitrosoguanidine mutagenesis in Drosophila melanogaster.

Nitrosoguanidine (NG) is extensively used as a mutagen in a wide variety of organisms. In Drosophila melanogaster, NG has been shown to be effective in inducing mutations in mature sperm (Khan, 1968 DIS 43:162) by feeding adult males with solution of NG in glucose, and in all stages of spermatogenesis (Browning, 1969 Mutat. Res. 8:157) following injection of saturated solution of NG into adult males. We have been studying NG mutagenesis in D. melanogaster with a view to reinvestigating the finding that the time of action of NG, at least in part, corresponds to the time of meiotic DNA synthesis in the testis. The results of some of our experiments are briefly outlined below.

Both the adult feeding method (0.05% NG in 5% glucose) and injection of NG (0.01%, in distilled water) were used. Treated males were mated to a succession of Muller-5 virgins so as to determine the mutagenic effectiveness of NG for different germ cell stages. Mature sperm were found to give the highest response to the mutagenic action of NG (SLRL frequency in the first brood was 9.1% and 6.5% for feeding and injection methods respectively). Further the two methods were found to give very similar results at concentrations which produced comparable sterility. However, premiotic germ cell stages of the treated adults remained, largely refractory to NG action. The percentage mutation (SLRL) frequencies in a representative experiment for 6 three-day broods were: 8.98, 5.61, 2.42, 0.48, 0.58 and 1.13. The considerable mutagenic effect observed for the earlier broods thus disappears by the 4th brood onwards (i.e. 10 days after treatment) when our brooding technique will sample treated spermatocytes and spermatogonia.

Treatments to larvae were next undertaken to ensure effective treatment of the premeiotic germ cell stages. Two treatment methods were used: (1) larvae were reared on basal medium containing 0.06% NG and (2) larvae were fed on 0.03% solution of NG in distilled water for 12 hours and then allowed to complete development on basal medium. Smaller, but significant, frequencies of SLRL (3.3% and 3.6% respectively) were induced in 24 hour larvae (egg laying time) by both these methods.

Table 1

| Age of treated larvae (hours) | % SLRL in broods (3 days, 2♀♀/♂) 1 | 2 | 3 |
|---|---|---|---|
| 24 | 3.85 | 3.31 | 2.67 |
| 36 | 2.73 | 3.22 | 1.33 |
| 48 | 2.16 | 1.76 | 0.00 |
| 60 | 3.16 | 0.00 | 0.00 |
| 72 | 2.05 | 2.43 | 0.20 |
| 84 | 1.07 | 0.00 | 0.00 |

(frequencies based on a minimum of about 500 tested chromosomes from 50-60, treated males)

When feeding of 0.05% NG in medium was extended to larvae of different ages and SLRL frequencies were determined in successive broods from emerging males, the results shown in Table 1 were obtained. The following conclusions emerge from the data in Table 1.

(1) For a particular age of treated larvae, the mutation frequencies decline in successive broods. This is true even for successive batches of treated spermatogonia which will be sampled, for example, in treatments of 24 and 36 hr larvae.

(2) Where analysis of successive broods permits comparison between sensitivities of spermatogonia and spermatocytes (e.g. 48 hr and older), the latter give higher mutation frequencies.

(3) The conclusion in (2) above, however, has to be viewed in the context of age of the treated larvae and the mutation frequencies obtained therefrom. For, as is obvious, the older the treated larvae, the smaller is the mutagenic effect of NG treatment.

Ives, P.T. Amherst College, Amherst, Massachusetts. Residual lethality in induced rearrangements and the chromosomal distribution of ♂ crossovers.

Two-day old virgin ♂♂ (Oregon-R/T(2;3)bw;h) after exposure to 1 or 2 kr of X-rays were mated individually and exhaustively (each to 6 new ♀♀ daily) to sets of vg bw;se or Cy bw $sp^2$;TM3 Sb Ser/T(2;3)bw;gl ♀♀, the latter to set up a test by which I could eliminate any ♂ that carried a pre-treatment lethal in either + chromosome, the former to lead to a T(2;3) test of treated +;+ pairs of chromosomes in sperm from proven lethal-free ♂♂ by the test mating of +;+/vg bw;se ♂ x T(2;3)vg $sp^2$;se ♀♀. T's involving the Y chromosome were discarded. Each T(2;3)+;+ and several of its sibling non-T +;+ pairs were tested for lethals starting with their ♂♂ x Cy bw $sp^2$;TM3 Sb Ser/T(2;3)bw;gl ♀♀. A lethal in either + chromosome or from the (rare) interaction between the +;+ members of a non-T pair, was sufficient to score a pair of non-T +;+ chromosomes as a lethal in just the same way that such a lethal must score a pair of T-linked +;+ chromosomes. The difference in frequency of lethality in those two kinds of pairs of chromosomes is a direct measure of the residual lethality (due to the rearrangement itself) in T's. Similar groups of sibling non-T lines (sometimes the same ♂♂ as used in the above lethal tests) were also tested for inversions (In's) using al cl b c $sp^2$ and scoring their $F_2$ for reduced crossing over (< 1/2 normal) in one or more regions. Residual lethality for In's was then calculated using the chromosome 2 lethal frequencies from the tests of non-T's or from appropriate SM5 tests in series which had no parallel T and non-T lethal tests. In a separate experiment, after a similarly built-in lethal screening test for both starting chromosomes, crossover (CO) chromosomes from the days 7-10 sperm broods of 1kr treated Oregon-R/al cl b c $sp^2$ ♂ x al cl b c $sp^2$ stock ♀♀ were tested for lethality (using b Bl/SM5 al $lt^v$ $sp^2$) along with samples of both kinds of sibling non-crossover (non-CO) chromosomes. The data from these experiments are given in the accompanying table. T's in the sperm of day 1 after treatment, and in days 5 and 6 sperm (which showed similar characteristics), and in both the 1 and 2 kr series, show a very similar rate of residual lethality, ranging from 40.4 to 41.9%. In's from the different days and treatments, although smaller in number, have the same order of residual lethality, 44.9 to 48.9%. Days 7 and 8 sperm show equivalent CO and lethal characteristics and their data are pooled here. Their residual lethality was 39.4%, but only 2 of the 4 broken ends of the homologous pair of chromosomes is recovered in each CO chromosome. All 4 broken ends are recovered in T's and In's. Thus, per broken end, residual lethality is

RESIDUAL LETHALITY IN X-RAY INDUCED REARRANGEMENTS

A. In Reciprocal 2;3 Translocations

| Day | Dose | T's | le | % | Non-T's | le | % | Resid. % |
|---|---|---|---|---|---|---|---|---|
| 1 | 1 kr | 74 | 41 | 55.4 | 1016 | 137 | 13.5 | 41.9 |
| 1 | 2 kr | 65 | 42 | 64.6 | 600 | 134 | 22.3 | 42.3 |
| 5+6 | 1 kr | 314 | 198 | 63.1 | 2249 | 512 | 22.7 | 40.4 |

B. In Chromosome 2 Inversions

| Day | Dose | In's | le | % | Non-In | le | % | Resid. % |
|---|---|---|---|---|---|---|---|---|
| 1 | 2 kr | 17 | 10 | 58.8 | 323 | 45 | 13.9 | 44.9 |
| 5+6 | 1 kr | 89 | 52 | 58.4 | 1544 | 194 | 12.6 | 46.8 |
| 5+6 | 2 kr | 26 | 19 | 73.1 | 351 | 85 | 24.2 | 48.9 |

C. In Male Crossovers, after 1 kr

| Day | Flies | CO | % | CO | le | % | + | le | % |
|---|---|---|---|---|---|---|---|---|---|
| 7+8 | 68,028 | 869 | 1.28 | 212 | 110 | 51.9 | 1367 | 171 | 12.5 |
| 9 | 37,716 | 373 | .99 | 142 | 23 | 16.2 | 309 | 10 | 3.2 |
| 10 | 80,821 | 524 | .65 | 199 | 17 | 8.5 | 1454 | 43 | 3.0 |

The + indicates non-crossovers. Resid. % in Day 7+8 is 39.4

much higher in CO's than in T's or In's at the time when it is at a maximum. In days 9 and 10, especially in day 10 sperm (whose clusters of crossovers were rare and small, in contrast to the situation in days 11 and 12 sperm) there was a sharp decrease in lethality in both CO and non-CO chromosomes (as well as a modest but clear decrease in frequency of recovered CO's) and residual lethality decreased to 13.0% in day 9 and to 5.5% in day 10 CO's. Residual lethality in T's and In's is presumably a position effect of the changed gene order. In ♂ CO's (homologous T's?) is it a result of possibly unequal crossovers producing chromosomes with small deficiencies or duplications, especially in days 7 and 8 where the chromosomes did not replicate before entering meiosis? Was the decrease in residual lethality in days 9 and 10 due to a repair of both lethals and rearrangements in cells whose chromosomes did replicate at least once more before meiosis, or to a loss of many days 9 and 10 cells with lethals and lethal-bearing crossovers during the subsequent replication? These questions are not answered in these data. (Minutes were relatively frequent in days 7 and 8 CO, and half the days 7 and 8 CO ♂♂ were sterile. Less than 10% of our T's and In's did not produce enough descendants for an adequate homozygous lethal test, which started, however, with several ♂♂ in each case.) Tests of a random sample of days 7-10 CO chromosomes showed no T associated with any one of them. Induced crossing over in ♂♂ appears first and very consistently in day 7 sperm (at 25° C) after radiation if the ♂ mates exhaustively (daily) during days 1-6. In another experiment 101 crossovers induced by 1 kr in al lt $stw^3$ $sp^2$/net b cn bw ♂♂ distributed themselves 17 in the net-b region, 31 in b-lt, 21 in lt-stw, 2 in stw-cn, 28 in cn-bw, and 2 in bw-sp. (The matings were treated ♂ x al lt $stw^3$ $sp^2$ ♀♀, and each $F_1$ CO x net b cn bw.) Compared to the linkage map distances those numbers are significantly high in both euchromatic b-lt and heterochromatic lt-stw and are low in euchromatic net-b and cn-bw.

This work, done in 1963-1970, was supported in turn by AEC Contract AT (30-1) 2467, NIH Grant T01-GM306, and NSF Grant GB-5680, and I am indebted to Mrs. Lucy Casey, Mrs. Virginia White, Miss Elinor Ives, and Miss Hildreth Spooner for assistance in it. Related studies from other laboratories, especially those of Bateman, Hannah-Alava, G. and A. Olivieri, and Puro, have been reported in Mutation Research during this time. Sobel's note in DIS 48:117 is perhaps the latest reference.

Široký, J. and J.K. Benedík. J.E. Purkyně University, Brno, Czechoslovakia. The changes of viability in a cage population.

The changes of the viability in the natural population during one year affected by the second chromosome were studied. The population was established on November 1st and all the time it was kept in the population cage at 25°C temperature, without light, and under a constant food condition.

The viability was tested by the Cy-method. Three tests for viability were done during the one-year period. The first in November 1970, the second in May 1971, and the third in November 1971. The modification of the Cy-method was used making possible the study of the viability of both chromosomes in each male. The results of the tests are compiled in the Table.

| | % of detrimentals | % of supervitals |
|---|---|---|
| November 1970 | 34.00 | 12.04 |
| May 1971 | 28.95 | 9.87 |
| November 1971 | 2.01 | 1.34 |

The results suggested that the frequencies of both the detrimentals and the supervitals decreased continually. The difference between this finding and the results of some other authors (Cetl, unpubl., Mukai, 1969) occurred, but these authors studied only single second chromosome lines. In their experiments the percentage of all detrimentals, especially lethals, significantly raised to the 20th or 75th generations, respectively. The situation in our experiment was rather different. The whole population, and not only the single chromosomal lines, was studied and so the natural selection connected with the competition may overlap the mutation rate in this case.

References: Mukai, T. 1969, Genetics 61:479-495.

Schwinck, I. University of Connecticut, Storrs, Conn. Imaginal eyes implanted in the imaginal abdomen.

In the study of genetic and epigenetic control of the drosopterin pathways it became desirable to study transplanted eyes in vivo during the time of drosopterin formation. It is long known that the quantity of drosopterin still increases considerably in the first days after eclosion of the fly. Experimental decrease or increase of the amount of drosopterins in post-eclosion flies by innibitor feeding respectively phenylalanin crystal implantation were recently demonstrated for several mutants (Schwinck, I. 1970, DIS 45:92; 1971, Genetics 68:s59). Here I would like to report an eye transplantation method which places the eye of a newly eclosed fly into an incision in the abdomen of a newly eclosed female host fly. The donor eye is implanted with the chitin surface sticking out of the host abdomen; the wound rims close very quickly followed by little melanin formation. A technical pilot experiment concerned with the survival rate of this post-eclosion eye transplantation method was finally terminated after more than three weeks during which the host flies had matured and produced viable offspring. The accompanying photograph shows 12 of these surface eye transplants in their 3 week old hosts; the various eye colors of the different genotypes are recognizable. In contrast to the classical larval eye disc implantation technique which results in inverted eyes deep within the abdomen, these adult surface eye transplants now allow the study of (1) pigment formation in a morphologically normal, developed eye structure, (2) the continued observation of the transplant eye, and (3) the use of the host abdomen as a tissue culture medium with genetically and/or biochemically variable contents.

Three week old host flies with surface eye implants.

Supported by a grant of the University of Connecticut Research Foundation.

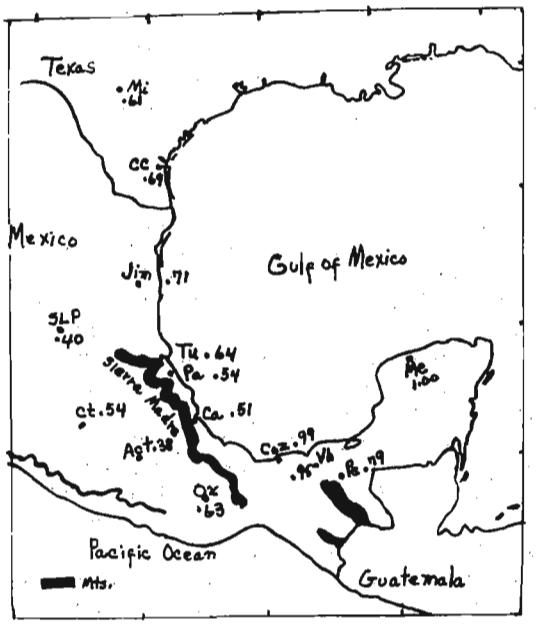
Averhoff, W.W. and M.R. Wheeler. University of Texas at Austin, Texas. A new distribution record for D. latifasciaeformis.

D. latifasciaeformis was described in 1940 by Dr. O. Duda from Uganda, Africa. It was found in Brazil in 1943 (as D. mirim) and in Mexico in 1947 (as D. baeomyia). Extensive collections in Florida in 1941 did not include the species but we found it in 1948 in South Carolina and in 1950 at 3 localities in Florida. By 1953 it was taken at 10 places in Florida and also in southeastern Georgia. By the late 1950's it was found to be widespread in Central and South America and in the West Indies.

We can now report a considerable extension of its range; it was collected at banana bait in the northeastern part of Austin, Texas in October 1972. It appears to be a very successful species, actively extending its range in the world.

Pipkin, S.B. and C. Rhodes. Howard University, Washington, D.C. Frequency of the D. melanogaster allele, $Adh^{II}$, in natural populations of Mexico.

Drosophila collections were made at 13 stations in Mexico and two in South Texas, using fruit-baited traps during the period June 15 - July 13, 1972. Species other than D. melanogaster and D. simulans, females of which are indistinguishable, were separated from one another immediately. Upon return from the collection trip, single fly assays of alcohol dehydrogenase were made using electrophoresis at 250 V for 25 min. on noble agar gels (method of Ursprung and Leone, 1965), followed by formazan staining with 2-butanol as substrate. In cultures where both D. melanogaster and D. simulans were present, only D. melanogaster males were assayed. An attempt was made to score at least 50 flies for each station in the first 3 generations after collection. However, enzyme level was so low in certain cultures that 10 cultures consisting of 3 females each were prepared and assays made of the pooled progeny. The $Adh^{II}$ allele specifies subunits with from 1/2 to 1/4 or less the specific activity of those coded by $Adh^{I}$ (Pipkin and Hewitt, 1971). For the most part the frequency of the $Adh^{II}$, allele in the populations studied can be related to temperature. Thus Fig. 1 shows that along the gulf coast region $Adh^{II}$ increases from .61 (Mico, Texas) to .69 (Corpus Christi, Texas) to .71 at Jiminez (near Cuidad Victoria) and .99 at Coatzocoalcos. Similarly in the interior high plateau region of central Mexico, $Adh^{II}$ increases from .40 at San Luis Potosi to .54 at Cuatla (near Mexico City) to .63 at Oaxaca City in the mountains. A small collection at Acatlán (southern tip of state of Puebla) gave a lower frequency of .38 for $Adh^{II}$. The frequencies of $Adh^{II}$ at 3 stations located beside the Sierra Madre Oriental (mountains) approximated corresponding frequencies in stations of about the same latitude in central Mexico, possibly because of gene flow from the cooler regions. (Tuxpan, .64; Paplantla, .54; and Cardel, near Vera Cruz, .51). $Adh^{II}$ frequencies varied little in the coastal region from Coatzococalcos (.99) to Villa Hermosa (.95) to Merida where $Adh^{II}$ reached fixation in the population sampled. However at Palenque, located in the edge of the mountains of Chiapas, the frequency of $Adh^{II}$ dropped to .79, reflecting apparently gene flow from mountain populations.




References: Pipkin, S.B. and N.E. Hewitt 1971, DIS 46:66-67; Ursprung, H. and J. Leone 1965, J. Exp. Zool. 160:147-154. (Laboratory assays were supported by NIH grant 18409-02; collection trip, courtesy of Alan C. Pipkin, Sr.)

Minamori, S. Hiroshima University, Japan. Extrachromosomal element delta correlates with "segregation distortion" phenomenon.

It has been known that Segregation-distorter gene (SD, II-55.0) in D. melanogaster is recovered in functional sperm much more often than the expected 50% when heterozygous for an $SD^{+}$ gene (Sandler, Hiraizumi and Sandler 1959). On the other hand, an SD-bearing second chromosome SD-5 was observed to retain an extrachromosomal element denoted by delta r which was assumed to be a copy of a gene locating on that chromosome ($Da^{r}$, 24.9; Minamori 1971 ; Minamori and Sugimoto, in press). The correlation of delta r to the distortion phenomenon was examined with SD-5 chromosomes. Second chromosomes recovered from SD-5/I-521($SD\ Da^{r}/SD^{+}\ Da^{+}$) females were individually tested for their sensitivity to the killing action of delta r (sensitive chromosome retains delta r; the original SD-5 is sensitive) and for their recovery from heterozygous males for the chromosome and a cn bw chromosome. The distribution of k values (the ratio of the chromosome among the total chromosomes recovered) of sensitive and insensitive chromosomes are separately shown in the follow-

ing table. It is evident that a sensitive chromosome showing no distortion (k < 0.60; presum-

| Chromosome | k value 0.46 - 0.60 | 0.61 - 0.90 | 0.91 - 100 | Total |
|---|---|---|---|---|
| Sensitive | 85 | 13 | 111 | 209 |
| Insensitive | 190 | 20 | 3* | 213 |

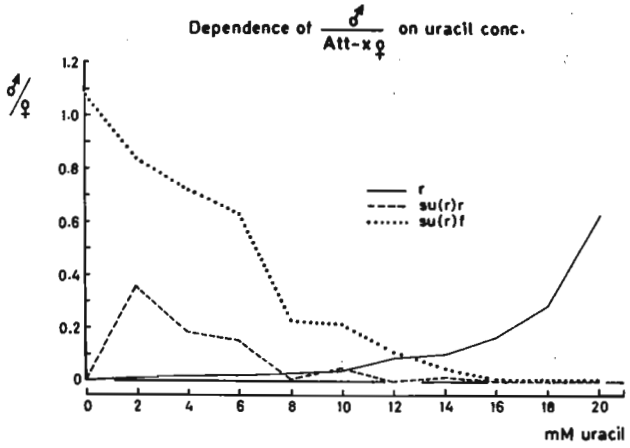
* showed no distortion in tests made in the subsequent generation

ably $SD^+ Da^r$) was produced by recombination from SD-5. The percentage was 40.7%. This finding suggests that the distortion cannot be induced by delta r alone. The percentage of insensitives which showed distorted segregation (k > 0.61) was 10.8%. Since this percentage is far lower for the expected 30%, the action of SD gene appears to be not or not fully expressed in the absence of delta r (SD $Da^+$ constitution). It is interpreted that the distortion phenomenon may be induced by a complemental interaction between SD gene and delta r.

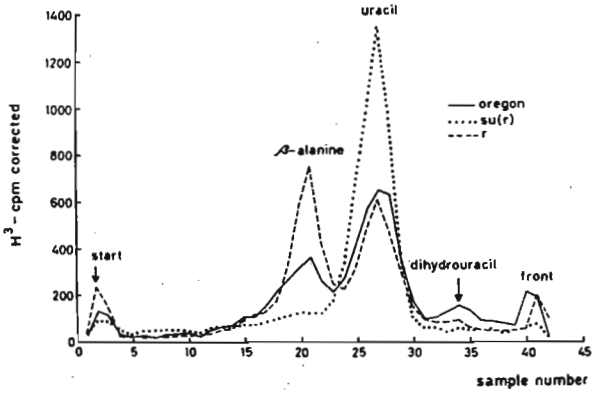
References: Minamori, S. 1971, Japan. J. Genetics 46:169-180; Sandler, L., Y. Hiraizumi and I. Sandler 1959, Genetics 44:233-250.

Bahn, E. University of Copenhagen, Denmark. A suppressor locus for the pyrimidine requiring mutant: rudimentary.

Studies have shown that an EMS induced recessive mutation situated at 1-27.7 is a suppressor of all rudimentary alleles. In contrast to the pyrimidine requiring rudimentary alleles (Nørby, 1970 Hereditas 66:205-214) this mutant is very sensitive to an exogenous supply of pyrimidines. The curves in Figure 1 show that in the cross su(r) f♂ x Att-X♀ on the minimal medium Eledon with increasing concentrations of uracil the suppressor mutant will not survive at concentrations higher than 16 mMolar of uracil. The rudimentary mutant on the contrary will hardly appear at concentrations lower than 12 mM uracil whereas the double mutant being rudimentary as well as suppressor of rudimentary has an optimum of survival at a concentration of 2 mM uracil. Thus, the r and the su(r) mutants clearly act as antagonists with respect to development of the flies on media with different concentrations of uracil. Together with the fact that dihydrouracil and dihydrothymine has no poisonous effect on the suppressor mutant the results suggested that the suppressing effect of the mutant was due to a block in the first step of pyrimidine catabolism.




Chromatography of extract of $H^3$-uracil fed whole larvae.




In an experiment using paper chromatography of the extract of $H^3$-uracil fed larvae the results depicted in Figure 2 showed no degradation of uracil in the su(r) larvae whereas the wild type and r larvae had been able to degrade uracil into dihydrouracil and further down to β-alanine. Other experiments based on in vitro degradation of $H^3$-uracil have given similar results. It is concluded that the suppressor of rudimentary mutant is a metabolic suppressor and that it causes a block in the first step of pyrimidine catabolism by greatly reducing dihydrouracil dehydrogenase activity and that the suppressing effect is due to a consequent sparesome degradation of special importance to a pyrimidine requiring mutant as rudimentary.

Tobler, H.* and S. Huber. Zoological Institute, University of Zürich, Switzerland. Effect of nitrogen mustard (HN-2) on the development of the male foreleg of Drosophila melanogaster.

Injection of nitrogen mustard into the body cavity of late third instar larvae or treatment of foreleg disks with this drug in vitro and subsequent implantation into larval hosts results in the following effects: differentiation of bristles without sockets and bracts, alteration of normal bristle patterns, reduction in size of leg parts, and formation of fewer bristles and hairs (Tobler and Maier, 1970). From these observations the question arose, as to whether or not administration of nitrogen mustard to the food would have the same effects.

10 mg of 2.2'-Dichlor-N-methyldiaethylamine (HN-2, Schuchardt, Munich) were dissolved in 1 ml of distilled water, diluted, and mixed with yeast to give appropriate concentrations of 0.05, 0.2, 0.5, 1.0 and 2.0 mg HN-2 per gr yeast. This mixture was poured in excess over standard Drosophila food (corn, sugar, agar, yeast) in a culture vial. 72 hour old larvae were either left on the drug until emergence as adults; or removed after 24 hours, rinsed with fresh water, and transferred to media without HN-2. Control flies were raised on food and yeast alone.

A concentration of 1 mg HN-2 per gr of yeast had the most significant effect on the

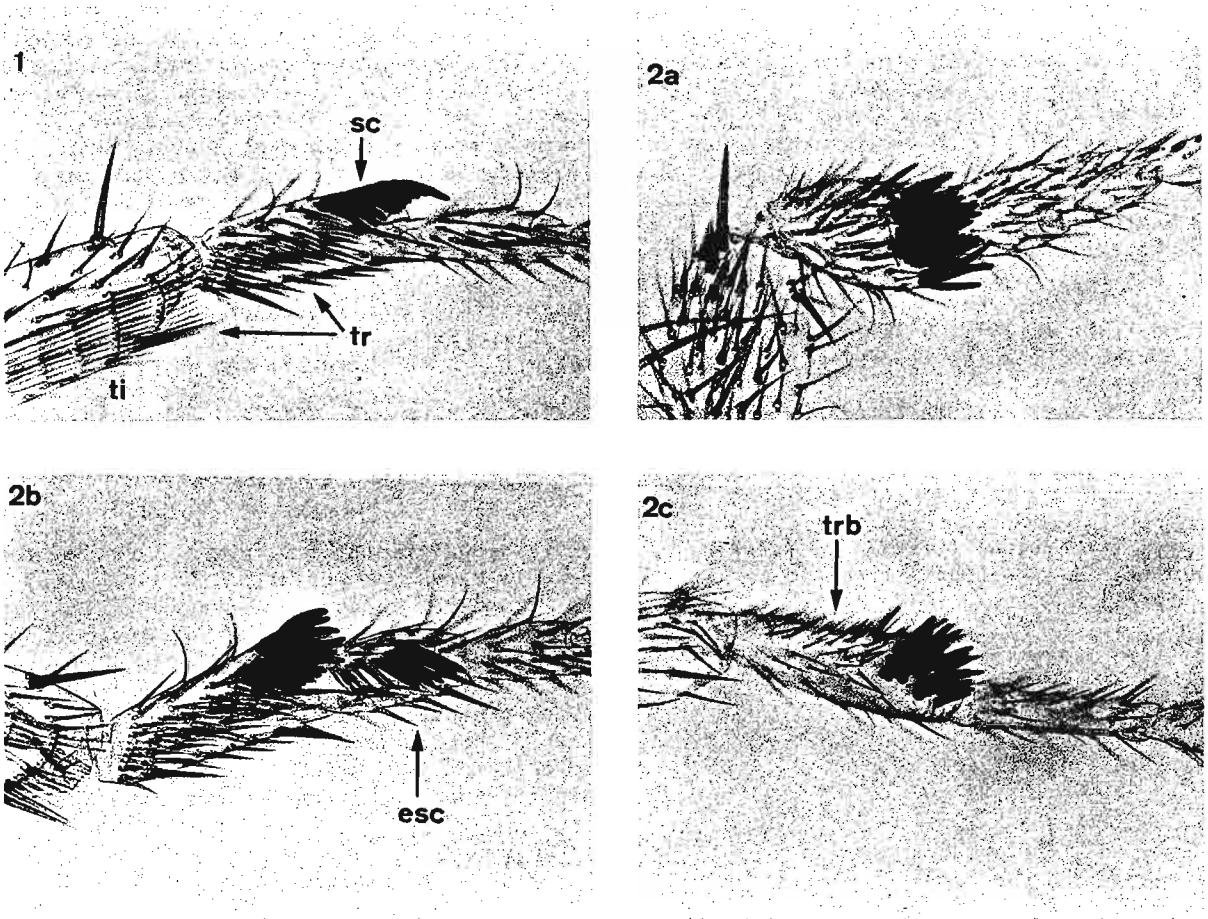



Fig. 1. Distal part of the left male foreleg of wildtype Drosophila melanogaster flies (ti = tibia, tr = transversal rows, sc = sex comb).

Fig. 2. Effects of HN-2 treatment on the bristle pattern of the male foreleg. a) Increase in the number of sex comb teeth on the first tarsal segment. b) Formation of an extra sex comb on the second tarsal segment (esc = extra sex comb). c) Alteration of the sex comb and transversal row pattern on the first tarsal segment (trb = transversal row bristles).

development of the adult foreleg. Higher concentrations proved lethal. Out of 90 treated larvae, only 2 flies were able to hatch. 31 larvae differentiated into adult flies but were unable to emerge from the pupal case and had to be dissected free. The remaining 57 larvae died during pupation. 62% of the examined flies showed typical alterations of patterns; these included the following types: 1. The number of sex comb teeth was considerably increased. Instead of the 8-13 teeth normally formed in our "Sevelen" control stock (Fig. 1), up to 35 teeth were found in the HN-2 treated animals (Fig. 2a). 2. Concomitant with the increase in the number of sex comb teeth, a striking change in the pattern was observed; the teeth are no longer arranged in an orderly row, but rather are spread over a large area of the distal part of the first tarsal segment. 3. Additional sex comb teeth may be found on the second tarsal segment (Fig. 2b). 4. The pattern of the transversal rows may also be disturbed (Fig. 2c). This is often accompanied by a reduction in the number of bristle organs.

The formation of bristles without sockets and bracts has never been observed during the present studies. The sensitive phase for the formation of socket-less bristles after injection of Mitomycin C falls between early third larval instar and 16 hours after puparium formation (Tobler and Burckhardt, 1971). It appears likely that the sensitive phase for the HN-2 and Mitomycin C effects are the same. It is not known whether or not HN-2 is metabolized to an inactive form. Alternatively it is possible that the drug does not reach the socket-forming cells ever or during the sensitive phase.

Bodenstein and Abdel-Malek (1949) treated Drosophila virilis larvae with nitrogen mustard and found that up to 50% of the exposed animals formed tarsal segments instead of aristae, thus phenocopying the mutant aristapedia. The formation of a large number of extra sex comb teeth on the distal part of the first tarsal segment may well be regarded as a phenocopy of the mutant eyeless-Dominant ($ey^D$, 4 - 2.0, Lindsley and Grell, 1968), which shows, as a pleiotropic effect, an enormous increase in the number of formed sex comb teeth. Extra sex comb teeth on the second tarsal segment of the foreleg, as is shown in Fig. 2b, are also found occasionally in the mutant combgap (cg, 2 - 71.1), which has recently been described by Datta and Mukherjee (1971).

References: Bodenstein, D. and A. Abdel-Malek 1949, J. Exp. Zool. 111:95-115; Datta, R.K. and A.S. Mukherjee 1971, Genetics 68:269-286; Lindsley, D.L. and E.H. Grell 1968, Carn. Inst. Wash. Publ. 627; Tobler, H. and H. Burckhardt 1971, Experientia 27:189-191; Tobler, H. and V. Maier 1970, Wilhelm Roux' Archiv 164:303-312.

We would like to thank Drs. E. Hadorn, H. Lebherz, and R. Nöthiger for critically reading this manuscript.

* New address: Institute of Zoology, University of Fribourg, 1700 Fribourg, Switzerland.

Baimai, V. Mahidol University, Bangkok, Thailand. A spontaneous triploid in D. birchii.

Natural populations of D. birchii exhibit remarkable karyotype variation (Baimai, 1969). The "Standard" metaphase karyotype of D. birchii consists of two pairs of V-shaped, one pair of dot-shaped, and one pair of J-shaped chromosomes (the latter the sex-chromosomes). During the course of cytogenetic investigations on this species from New Guinea, a triploid was detected in a third instar larva from a cultured stock. Figure 1 shows two X-chromosomes, one Y-chromosome, six V-shaped chromosomes, and the three dots. Although not unusual in plants, polyploidy is rare in animals; this is an example of a spontaneous triploidy in Drosophila.

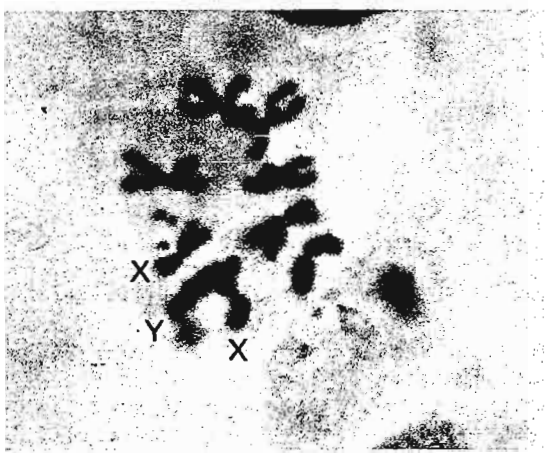



Fig. 1. A triploid in D. birchii.

Reference: Baimai, V. 1969, Chromosoma 27: 381-394.

Korochkin, L.I. and N.M. Matveeva.
Institute of Cytology and Genetics.
Novosibirsk, USSR. Esterase isozymes of Drosophila virilis group.

Some workers have demonstrated the existence of multiple forms of esterases in Drosophila virilis (Sims, 1965; McReynolds, 1967; Ohba & Sasaki, 1968). Esterase patterns have been studied in a number of D. virilis and D. texana stocks obtained from the collection of Prof. N. Sokoloff (Institute of Developmental Biology, Moscow). The starch and polyacrylamide gel procedures were used to analyse about 1000 adult individuals of D. virilis 101 (wild type), 147 (b bk dt), 140 (va), 103 (R,gl), 142 (w) and of D. texana 123 (wild type), 119 (dt) (the numbers correspond to the stocks designations used in the collection). The esterases were detected using α-naphthylpropionate as substrate (yielding best results) or a mixture of α- and β-naphthylacetate as substrates. Two types of electropherograms are distinguished: the "virilis" type and the "texana" type (Fig. 1). The zymograms of different D. virilis stocks show 6 esterase groups all of which display interstock polymorphism and some intrastock polymorphism. In adult flies the slowest esterase 1 exhibits weak activity and is represented by three bands (a,b,c). Esterase-2 in 140 and 142 stocks is stained very intensely, in stock 147 the staining is somewhat weaker and in stock 103 the staining is faint in most cases. In the incubation mixture of α- and β-naphthylacetate this esterase preferentially acts on naphthylacetate and unlike all the other D. virilis esterases is stained red. Stocks 140, 101, 142 are characterized by a slow variant of esterase-2 (B), and stocks 103 and 147 by a fast variant (A). Esterase-3 stains more intensely in males; however, sex polymorphism was clear-cut only on days 4-5 after outflow. Esterase-4 (consisting usually of two bands one deep staining and the other minor) is the second "main" esterase in the electropherogram with very high activity in D. virilis. Stocks 140 and 101 show a typical "fast" form of esterase-4 (A), stock 147 a "slow" form (B), whereas in stock 103 and particularly 142, both forms are present as homozygotes as well as heterozygotes. Esterases-5 and 6 stain less intensely in most stocks excluding 103 which differs from all the other stocks in that its activity is more evenly distributed among all the different esterase fractions.

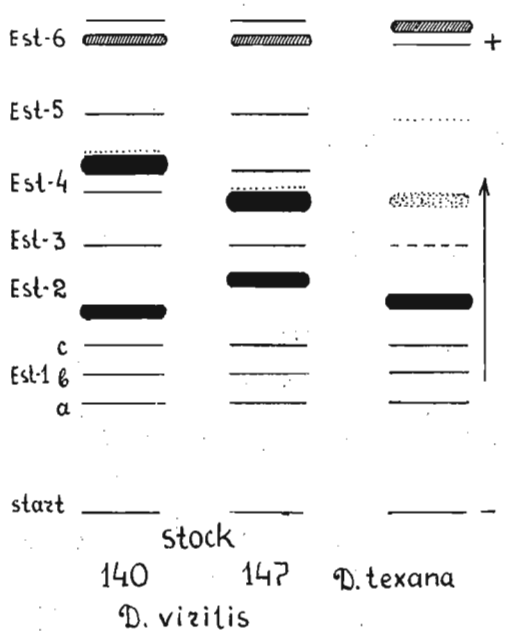



Fig. 1. Electropherograms ( starch gel )

Table 1

| Treatment | Esterases 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|
| Specificity to substrate | α | β | α | α | α | α |
| $10^{-3}$M $CuSo_4$ | ± | - | ± | - | - | - |
| $10^{-5}$M E-600 | + | ± | ± | ± | ++ | - |
| $10^{-4}$M physostigmine | + | + | ++ | + | + | + |
| Heating 50° 10 min | ± | + | ± | ± | ± | ± |
| Heating 60° 10 min | ± | ++ | ± | + | + | ++ |
| Heating 70° 10 min | ++ | ++ | ++ | + | + | ++ |

(++) - complete inhibition
(+) - weaker reaction
(±) - indefinite or variable results
(-) - no effect

D. texana stocks 123 and 119 have identical esterase patterns which differ from the pattern of D. virilis in that, first, esterases 4 and 5 are, as a rule, very indistinct, with esterase-5 usually being unidentifiable and second, in addition to esterase-2, esterase-b and esterase-6 also display an affinity for β-naphthylacetate. Table 1 shows the affinity of esterases of D. virilis stock 101 for inhibitors. D. virilis ♀ 140 x ♂ 147 and ♀ 147 x ♂ 140 esterase 4 hybrids are represented by two fractions, a paternal and maternal fraction, and esterase-2 is composed of three fractions - two parental and hybrid. Experiments with dissociation-reassociation have shown that esterase-2 is a dimer.

Acknowledgement: The authors are very grateful to Prof. N. Sokoloff and Dr. M. Evgenieff

(Continued at bottom of next page)

Clark, J.M. Flinders University of South Australia, Bedford Park, S.A., Australia.

Mutagenic effect of azathioprine in Drosophila melanogaster.

Azathioprine is a drug extensively used for immuno-suppression and the treatment of neoplasias. Since many of the patients treated with azathioprine are of reproductive age, it is important to evaluate the potential genetic risks associated with the use of the drug. Preliminary mutagenicity studies were carried out with D. melanogaster.

Canton-S males were treated for 24 hours by feeding on 10mg. azathioprine spread on the surface of the usual treacle/semolina medium (10 flies/vial). Treated males were mated to 3 virgin Canton-S females every 3 days, to give a total of 5 broods. The percentage of unhatched eggs was used as an indicator of dominant lethality. The results obtained are shown in Table 1.

Table 1.

| | Brood 1 | | Brood 2 | | Brood 3 | | Brood 4 | | Brood 5 | |
|---|---|---|---|---|---|---|---|---|---|---|
| | % u.h. | No. | % u.h. | No. | % u.h. | No. | % u.h. | No. | % u.h. | No. |
| Control | 13.8 | 3675 | 13.8 | 3824 | 15.1 | 3438 | 15.0 | 2980 | 13.2 | 2173 |
| Treated | 15.3 | 3473 | 19.1 | 3627 | 19.2 | 3350 | 19.1 | 2993 | 15.8 | 2305 |
| $\chi^2$ | 3.1 | | 24.3 | | 20.1 | | 17.8 | | 6.0 | |

u.h. - unhatched eggs

The ability of azathioprine to cause loss or breakage of chromosomes was tested by treating C(1)RM, y/R(1)2, vf/$B^S$ Y $y^+$ males as above, and mating to virgin y $w^a$ females with 3 day brood intervals for a total of 4 broods. The progeny were scored for sex, loss of $Y^L$, loss of $Y^S$, loss of X or Y and non-disjunction. The results are presented in Table II. The effect of azathioprine on the sex-ratio in rod-X flies was tested by treating C(1)RM, y/y v/$B^S$ Y $y^+$ males and mating as above.

Table 2.

| Series | Total progeny | Male/Female | Complete loss X or Y, % | Loss $Y^L$, % | Loss $Y^S$, % | N.D.J. % |
|---|---|---|---|---|---|---|
| Control | 14,212 | 0.863 | 0.197 | 0.030 | 0.060 | 0.118 |
| Brood 1 | 8,264 | 0.908 | 0.096 | 0.076 | 0.076 | 0.162 |
| 2 | 7,080 | 0.999 | 0.141 | 0.085 | 0.113 | 0.198 |
| 3 | 4,847 | 1.005 | 0.289 | 0.247 | 0.206 | 0.165 |
| 4 | 2,904 | 0.971 | 0.482 | 0.140 | 0.070 | 0.475 |

Azathioprine induced dominant lethals in Canton-S males in post-meiotic and meiotic broods. In the treated ring-X stock, chromosome breakage, as indicated by partial chromosome loss, is highest in brood 3 ($\chi^2$ = 7.10 for loss of $Y^L$). In brood 4, the maximum frequency of complete X or Y chromosome loss occurs ($\chi^2$ = 6.89), and this coincides with the highest frequency of non-disjunction ($\chi^2$ = 3.38), indicating that meiotic germ cells are being utilized in this brood. However, it is noted that although peak chromosome loss and non-disjunction coincide in brood 4, the non-disjunction does not account for all the chromosome loss. There was no evidence that preferential loss of the $Y^L$ or $Y^S$ occurred.

Only 4 mosaics were observed amongst 95 exceptional progeny in the treated series, and were subsequently found to be fertile. Only one exchange event was found. Of the 27 progeny with partial chromosome losses in the treated series, 5 were fertile, 2 of which were mosaics. Marker loss in 3 flies was not accompanied by loss of fertility factors.

In brood 3, the ring-X sex-ratio differed significantly from that of its control ($\chi^2$ = 21.12). The rod-X, male/female, sex-ratio was greatest at brood 3, and did not differ significantly from its control ($\chi^2$ = 0.23). From this it can be inferred that much of the sex-ratio shift observed in the progeny of treated ring-X flies can be attributed to breakage of the ring chromosome.

This study demonstrates the genetic activity of azathioprine in D. melanogaster. Tests in mammalian systems are required to evaluate further the mutagenicity of this drug.

(Continued from preceding page)
(Moscow) for providing them with D. virilis and D. texana stocks.

References: McReynolds, M. 1967, Genetics 56:527-540; Ohba, S. and F. Sasaki 1968, Proc. Twelfth Internat. Cong. Genetics, Tokyo:156-157; Sims, M. 1965, Nature 207:757-758.

Korochkin, L.I., M.B. Evgeniev and N.M. Matveeva, Institute of Cytology and Genetics, Novosibirsk, USSR; Institute of Developmental Biology, Moscow, USSR. The influence of the genotype on the phenotypical expression of esterases in Drosophila of the virilis group.

Previous work has demonstrated some interstrain and interspecies differences in electropherograms from single Drosophila flies of virilis group (Korochkin, Matveeva, DIS 49). The problem remains whether these differences are determined by the 2nd chromosome alone (in which esterase genes were localized), or by other chromosomes as well. Taking into consideration these data concerning the localization of esterase genes in the 2nd chromosome, which is marked by other mutations (detached, break, broken etc.) we produced two stocks with the 2nd chromosome from both species (one from D. virilis, another from D. texana) and whose other chromosomes were derived either from D. virilis or D. texana (2nd and 3rd chromosomes are linked by their proximal ends in D. texana, therefore they always remained together).

We carried out the following crosses: D. texana stock 123 +/+ ♀ x D. virilis stock 147 dt/dt ♂.

Then males from $F_1$ were crossed with D. virilis dt/dt ♀. We selected normal males in each following generation and crossed them repeatedly with D. virilis dt/dt ♀. Thus all chromosomes of D. texana, except the 2nd and 3rd, were replaced by the chromosomes of D. virilis. Through this method we obtained the stock which was designated $P_1$. In another case we crossed D. virilis stock 142 +/+ ♀ x D. texana stock 119 dt/dt ♂, then $F_1$ dt(tex)/+ (vir) ♂ x D. texana stock 119 dt/dt ♀ repeatedly as in the first experiment. This stock, in which all chromosomes of D. virilis except the 2nd were replaced by the chromosomes of D. texana, was designated $P_2$. In both cases we selected males and females of normal phenotype for electrophoretical analysis of esterases in single flies. It was found that the esterase spectra of the $P_1$ stock correspond to the "virilis" type, which has a strongly expressed esterase-4. The $P_2$ stock is similar with "texana" type, in that esterase-4 appears weak. Therefore it seems probable that the pattern of esterases in Drosophila from the virilis group is determined not only by the second chromosome, but by other chromosomes as well.

Korochkin, L.I. and M.D. Golubovsky. Institute of Cytology and Genetics, Novosibirsk, USSR. Localization of two genes controlling esterase electrophoretic mobility in Drosophila virilis.

It has been established earlier that D. virilis from Prof. Sokoloff's stocks differs with respect to the electrophoretic variants of the two main types of esterases, Est-2 and Est-4. Strain 147 with the genotype b bk dt was found to be homozygous for the fast (A) Est-2 variant and the slow (B) Est-4 one. Strain 140 - va - is homozygous for the slow variant (B) Est-2 and the fast (A) Est-4 variant. Strain 103 bearing the dominant gene R was homozygous for the A Est-2 variant and polymorphic for variants Est-4. The crosses performed have shown that both esterase variants are under monogenic control (Table 1). The progeny from ♀♀ 147 x $F_1$(147 x 140) ♂♂ crosses or

$$♀♀ \frac{b\ bk\ dt}{b\ bk\ dt}\left(\frac{Est\text{-}2^A}{Est\text{-}2^A}\ \frac{Est\text{-}4^B}{Est\text{-}4^B}\right) \times \frac{b\ +\ bk\ dt}{+\ va\ +\ +}\left(\frac{Est\text{-}2^A}{Est\text{-}2^B}\ \frac{Est\text{-}4^B}{Est\text{-}4^A}\right) ♂♂$$

with b bk dt phenotype had only A Est-2 variant and B Est-4 variant. Thus, both esterase loci are linked with visible second chromosome markers. Ohba and Sasaki (1968) have made a report concerning the location of loci for esterases on chromosome 2, however, these authors were not able to localize precisely these loci on the chromosome. For a more definite localization of the loci Est-2 and Est-4 the following crosses were carried out:

$$♀♀ \frac{b\ +\ bk\ dt}{+\ va\ +\ +}\left(\frac{Est\text{-}2^A}{Est\text{-}2^B}\ \frac{Est\text{-}4^B}{Est\text{-}4^A}\right) \times \frac{b\ +\ bk\ dt}{b\ +\ bk\ dt}\left(\frac{Est\text{-}2^A}{Est\text{-}2^A}\ \frac{Est\text{-}4^B}{Est\text{-}4^B}\right) ♂♂$$

Then the distribution of esterase variants among recombinant phenotypes was analyzed (Table 2). According to the Chino's map (1937) three visible markers have the following localization: b-143.5, bk-203.5 and dt-210 map units. The results of table 2 shows 83% of crossovers occurred in the process of the recombination between bk-Est-2 and 17% between Est-2 - dt. The locus for Est-2 is, therefore, calculated as being at 209.3 ±. On the other hand,

Table 1. The mode of inheritance of esterase variants.

Variants Est-2

| Crosses | | | | Est-2 phenot. in the progeny | | |
|---|---|---|---|---|---|---|
| Females | | Males | | AA | AB | BB |
| 147 | AA | 103 | AA | 19 | 0 | 0 |
| 103 | AA | 147 | AA | 5 | 0 | 0 |
| 147 | AA | 140 | BB | 0 | 10 | 0 |
| 140 | BB | 147 | AA | 0 | 10 | 0 |
| $F_1$(147 x 140) | AB | 147 | AA | 26 | 26 | 0 |
| $F_1$(147 x 140) | AB | $F_1$(147 x 140) | AB | 5 | 11 | 4 |
| 103 | AA | 140 | BB | 0 | 33 | 0 |
| 140 | BB | 103 | AA | 0 | 27 | 0 |
| $F_1$(103 x 140) | AB | 103 | AA | 6 | 6 | 0 |
| $F_1$(103 x 140) | AB | 140 | BB | 0 | 7 | 5 |
| $F_1$(103 x 140) | AB | $F_1$(103 x 140) | AB | 7 | 17 | 8 |

Variants Est-4

| Crosses | | | | Est-4 phenot. in the progeny | | |
|---|---|---|---|---|---|---|
| Females | | Males | | AA | AB | BB |
| 147 | BB | | AA | 0 | 23 | 0 |
| 140 | AA | | BB | 0 | 14 | 0 |
| $F_1$(147 x 140) | AB | | BB | 0 | 12 | 11 |
| $F_1$(147 x 140) | AB | $F_1$(147 x 140) | AB | 4 | 10 | 6 |

Table 2. Localization of Est-2 and Est-4 genes

| Crosses | Phenotype and number of recomb. | Est-2 A | Est-2 AB |
|---|---|---|---|
| ♀♀ $\frac{b + bk\ dt\ A}{+\ va\ +\ +\ B}$ ♂♂ | b bk + 27 | 3 | 24 |
| | + + dt 30 | 24 | 6 |
| X $\frac{b + bk\ dt\ A}{b + bk\ dt\ A}$ ♂♂ | b + dt 17 | 15 | 2 |
| | + bk + 20 | 5 | 15 |
| | Total 94 | | |

| Crosses | Phenotype and number of recomb. | Est-4 AB | Est-4 B |
|---|---|---|---|
| ♀♀ $\frac{b + bk\ dt\ B}{+\ va\ +\ +\ A}$ | b + + 90 | 76 | 14 |
| | + bk dt 90 | 21 | 69 |
| X $\frac{b + bk\ dt\ B}{b + bk\ dt\ B}$ ♂♂ | + bk + 17 | 5 | 12 |
| | b + dt 15 | 12 | 3 |
| | Total 212 | | |

79.7% crossovers arose between b - Est-4 and 20.3% between Est-4 - bk. Hence, the locus for Est-4 is located at 192 ±.

References cited: Chino, M. 1937, Jap. Journ. Gen. XII:189-210, 257-283; Ohba, S. and F. Sasaki 1968, Proc. XII Intern. Cong. Genet. 1:156-157.

Childress, D. and W. Cantelo. USDA, Fargo, North Dakota; Beltsville, Maryland. Fitness of compound chromosome strains.

It has been suggested that compound chromosomes might be suitable for changing the genetic structure of insect populations, particularly of insect pest species (Foster et al., 1972). Because an unstable equilibrium is produced in a mixed population of compound and normal individuals (a mating between a compound and a wild type is sterile), it is possible for the compound strain to replace the native strain in a very small number of generations. The release ratio at which the compound strain will replace the normal strain depends almost entirely on the fitness of the compound strain relative to that of the native population. Theoretically, the fitness of compound strains in Drosophila is near 25%, due to the production of unbalanced

Table 1. Competition experiments between the Tokyo wild and the sepia compound strain. Samples were taken every generation (10 days).

| Generation | Original release ratio Tokyo:se compound | Progeny normal (Tokyo) | Compound (sepia) | % Tokyo ± s.e. |
|---|---|---|---|---|
| 1 | 1:1 | 323 | 49 | 87 ± 1.7 |
| 2 | (20 pr + : 20 pr se) | 136 | 32 | 81 ± 3.0 |
| 3 | | 104 | 8 | 93 ± 2.4 |
| 4 | | 223 | 14 | 94 ± 1.5 |
| 1 | 1:1 (Replicate) | 145 | 108 | 57 ± 3.1 |
| 2 | | 125 | 10 | 93 ± 2.3 |
| 3 | | 264 | 17 | 94 ± 1.4 |
| 4 | | 204 | 49 | 81 ± 2.5 |
| 1 | 1:3 | 57 | 169 | 25 ± 2.9 |
| 2 | (10 pr + : 20 pr se) | 111 | 257 | 30 ± 2.4 |
| 3 | | 107 | 104 | 51 ± 3.4 |
| 4 | | 103 | 270 | 28 ± 2.3 |
| 1 | 1:3 (Replicate) | 28 | 145 | 16 ± 2.8 |
| 2 | | 57 | 69 | 45 ± 4.4 |
| 3 | | 1 | 149 | 7 ± .7 |
| 4 | | 6 | 148 | 4 ± 1.6 |
| 1 | 1:5 | 49 | 161 | 23 ± 2.9 |
| 2 | (7 pr + : 35 pr se) | 33 | 264 | 11 ± 1.8 |
| 3 | | 6 | 122 | 5 ± 1.9 |
| 4 | | 7 | 165 | 4 ± 1.5 |
| 1 | 1:5 (Replicate) | 26 | 133 | 16 ± 2.9 |
| 2 | | 8 | 123 | 6 ± 2.1 |
| 3 | | 9 | 154 | 6 ± 1.8 |
| 4 | | 14 | 223 | 6 ± 1.5 |
| 1 | 1:9 | 3 | 217 | 1 ± .8 |
| 2 | (4 pr + : 36 pr se) | - | 154 | 0 ± |
| 3 | | 12 | 167 | 7 ± 1.9 |
| 4 | | 4 | 324 | 1 ± .6 |
| 1 | 1:9 (Replicate) | 3 | 84 | 3 ± 1.9 |
| 2 | | 16 | 170 | 9 ± 2.1 |
| 3 | | 5 | 183 | 3 ± 1.2 |
| 4 | | 6 | 219 | 3 ± 1.1 |

gametes at meiosis. At this fitness, individuals bearing compound chromosomes have to be present six times as frequently as normal individuals in order to replace them. In this instance, replacement will occur in 4 generations (Childress, 1972).

Experiments performed using the Tokyo wild-type strain and a strain with wild-type compound 2nd chromosomes marked with yellow on the X chromosome closely approximate theoretical expectations. Additional experiments (Table 1) competing the Tokyo wild-type strain with a compound 3rd chromosome strain marked with sepia gave results that were also compatible with

Table 2. Results of competition experiments between the New Jersey wild-type strain and the sepia compound chromosome strain. Egg samples were taken from the cages at intervals.

| Egg samples | Release ratio | % New Jersey wild replicates | | | |
|---|---|---|---|---|---|
| Days after release | New Jersey : se compound | 1 | 2 | 3 | 4 |
| 5 | 1:5 | 57 | 75 | 100 | 0 |
| 12 | (10 prs + : 50 prs se) | 77 | 94 | 98 | 0 |
| 19 | | 94 | 94 | 93 | 12 |
| 25 | | 90 | 98 | 100 | 20 |
| 28 | | 94 | 94 | 100 | 5 |
| 31 | | 100 | 100 | 100 | 5 |
| 35 | | 100 | 100 | 100 | 0 |
| 38 | | 100 | 100 | 100 | 13 |
| 5 | 1:15 | 28 | 3 | - | - |
| 8 | (10 prs + : 150 prs se) | - | - | 65 | 0 |
| 11 | | 0 | - | 48 | 20 |
| 15 | | 77 | 55 | 38 | 28 |
| 18 | | 63 | 61 | 22 | 4 |
| 22 | | 77 | 61 | 2 | 2 |
| 25 | | 82 | 48 | 6 | 32 |
| 29 | | 99 | 87 | - | - |
| 5 | 1:20 | 11 | 27 | 18 | 16 |
| 8 | (10 prs + : 200 prs se) | 40 | 8 | 30 | 9 |
| 11 | | 0 | 67 | 68 | 12 |
| 15 | | 0 | 45 | 32 | 49 |
| 18 | | 45 | 61 | 18 | 37 |
| 22 | | 47 | 46 | 55 | 34 |
| 25 | | 49 | 38 | - | - |
| 29 | | 67 | 75 | - | - |

the theoretical expectation. At a release ratio of 1 Tokyo : 5 sepia, the compounds were very close to replacing the wild-type strain after 4 generations. However, when the same sepia compound strain was put in competition with a wild-type strain collected in New Jersey (Table 2), a ratio of 1 New Jersey wild : 5 sepia was not sufficient to fix the sepia compound strain. In fact, even a ratio of 1 : 20 did not result in replacement of the wild-type strain by the compound strain.

These contradictory results are undoubtedly due to the difference in fitness between the Tokyo wild-type strain and the New Jersey wild-type strain. Tokyo wild has been maintained in the laboratory for many years and has doubtless accumulated many genes that are detrimental to overall viability. The New Jersey strain was collected approximately 2 years ago and is evidently much more vigorous. Some preliminary field observations on the fitness of the sepia compound strain relative to the New Jersey strain verify the laboratory observations. It is clear that accurate estimations of the performance of a compound strain relative to a wild-type strain can be made only if the genetic background of the two strains is similar.

References: Childress, D. 1972, Genetics 72:183-186; Foster, G., M. Whitten, T. Prout, R. Gill 1972, Science 176:875-880.

Butterworth, F.M., H. Dene and D. Wachnicki, Oakland University, Rochester, Michigan. Acid phosphatase in the protein granules of the larval fat body.

Cells and tissues programmed to die during development undergo a preparative and lytic phase. The preparative phase is generally marked by an increase in the number of lysosomes and during the lytic phase the acid hydrolases contained in these bodies are released presumably causing the breakdown of the cells. Although there are many kinds of hydrolases in lysosomes (Barrett, 1969), the usual criterion for identifying lysosomes cytochemically is by the presence of acid phosphatase activity. In the following discussion it will be demonstrated that granules containing acid phosphatase activity are present in the larval fat body, and that the presence of these granules is correlated with the cell death of normal and mutant tissue.

Acid phosphatase was demonstrated cytologically by both the lead nitrate method of Gomori (1950) and the azo dye technique of Seligman and Mannheimer (1949). The lead nitrate method was modified from Gomori (1950, as prescribed by Pearse, 1968, P. 728) which utilized sodium glycerophosphate (Sigma) as the substrate, lead nitrate and acetate buffer at pH 5. The azo dye method of Seligman and Mannheimer (1949) used was modified (Seligman, personal communication). The reaction medium consisted of 50 mg sodium $\alpha$-naphthol phosphate (Dajac Laboratories), 30 mg fast blue salt B (Matheson, Coleman and Bell), 90 ml of 4.0M sodium chloride, and 10 ml of 0.07M acetate buffer at pH 5.0. Both media were mixed just prior to each experiment, and the control media were identical in every way except the respective substrate was omitted. Although similar results were obtained from both procedures, the azo dye method was used predominantly.

Both methods were used on whole tissues or sections of paraffin embedded tissues. The whole tissues were either unfixed or fixed in acetone for 24 hours at 4°C. Material to be sectioned was fixed in acetone (as above), dehydration completed in several changes of acetone, cleared first in cedarwood oil followed by xylene, infiltrated in paraffin at 50-53°C for not more than 1 hour, embedded in paraffin, and sections were placed on slides without fixative. Incubation in the reaction media was at 37°C for 15-24 hours. Paraffin was removed from the sections just prior to mounting in balsam. Whole tissues following the reaction procedure were rinsed in distilled water and either mounted directly in glycerine jelly or dehydrated and embedded in paraffin for sectioning. Tissues were also prepared for general cytological examination according to Butterworth and Bodenstein (1967). The larvae, pupae and adults studied were reared under standard conditions (see Butterworth 1972).

In the normal development of the larval fat body cell, during the third instar, the cells achieve a 5-fold increase in cross-sectional area. Midway in the instar proteinaceous granules begin to form, increasing in size and number such that by the end of the instar they comprise 20% of the cytoplasm. During metamorphosis the cells separate and begin to histolyze. Most of the cells survive metamorphosis and have the same percentage of protein inclusions. However the cells have become smaller due to a loss of background cytoplasm. During the first two days of adult life the cells rapidly histolyze until, by the third day of adult life, none remain (Butterworth et al., 1965; Butterworth, 1972). In mutant, $ap^4$, the larval fat body which has protein granules in normal amounts histolyzes in the adult state at a much lower rate than normal (Butterworth and King, 1965; Butterworth, 1972).

Acid phosphatase activity was found in the protein granules of the fat body of wild type animals from the time that the granules were first detected in the mid-third instar larvae and throughout development as long as the cells were present. Acid phosphatase could not be demonstrated in cytoplasm at earlier stages although activity in small granules might not be resolvable in the light microscope. Individuals homozygous for $ap^4$ which could be detected as early as the second day of pupation also had acid phosphatase in the protein granules of the fat body for the remainder of metamorphosis and through the fifth day of adult life. Flies of this genotype rarely survive longer than five days. Nevertheless large numbers of their larval fat cells are present during that period; and the intensity of the acid phosphatase stain in the granules is similar throughout this period. Furthermore, the level of staining of enzyme in the granules of both $ap^4$ and wild-type flies is similar.

It is of interest to note that the fat body of Acph-1 null-activity mutants (Bell, et al. 1972), generously supplied by Dr. R.J. MacIntyre, possesses qualitatively normal numbers of protein granules; and that the cells of this tissue break down at the normal rate in the adult stage. Preliminary experiments with several alleles generally indicated no acid phosphatase activity in the granules. Occasionally activity was detected particularly in whole tissues. Presumably other genetically distinct acid phosphatases present are being inactivated by the

cytological processing. Clearly cell death in the fat body does not require acid phosphatase-1.

Acid phosphatase has been demonstrated in the protein granules of the larval fat body in other insect species (beetles: DeLoof, 1972; moths: Locke and Collins, 1968; muscid flies: Stay, 1959) but the function of acid phosphatase in the protein granules is not yet clear. Since the protein granules of the larval fat body of Drosophila contain acid phosphatase, and since Gaudecker (1963) finds the granules to be autophagic, it is reasonable to think of these granules as being lysosomal. Attempts to identify other acid hydrolases in the granules are underway.

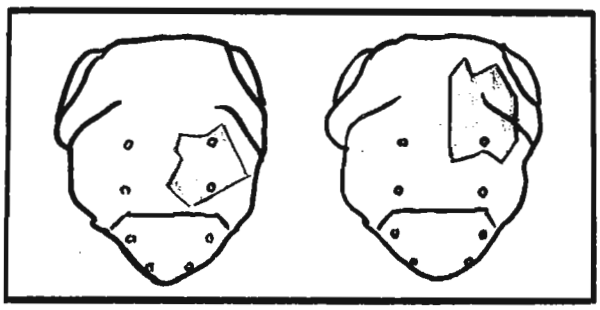
References: Barret, A.J. 1969 In: Lysosomes in Biology and Pathology, Vol. 2 (Ed. by Dingle and Fell) Chapt. 6, American Elsevier Co., N.Y.; Bell, J.B., R.J. MacIntyre and A.P. Olivieri 1972, Biochem. Gen. 6:205-216; Butterworth, F.M. 1972, Devel. Biol. 28:311-325; Butterworth, F.M. and D. Bodenstein 1967, J. Exp. Zool. 164:251-266; Butterworth, F.M. and R.C. King 1965, Genetics 52:1153-1174; Butterworth, F.M., D. Bodenstein and R.C. King 1965, J. Exp. Zool. 158:141-154; DeLoof, A. 1972, J. Insect Physiol. 18:1039-1047; Gaudecker, B.V. 1963, Z.F. Zellforsch. 61:56-95; Gomori, G. 1950, Stain Tech. 25:81; Locke, M. and J.V. Collins 1968, J. Cell Biol. 36:453-483; Pearse, A.G.E. 1961, Histochemistry, Theoretical and Applied, 2nd ed. Little, Brown & Co., Boston; Seligman, A.M. and L.H. Mannheimer 1949, J. Nat. Cancer Inst. 9:427-434; Stay, B. 1959, J. Morph. 105:457-494.

Behnia, A. and G. Koliantz. Teachers Training College, Tehran, Iran. The effect of uric acid on a wild-type strain of D. melanogaster.

In the summer of 1970, from a natural population of Azarbaijan province in Iran, a wild type strain, called Gayaneh, was isolated which showed a high viability, fecundity and also a longevity more than Java wild type (Mostashfi, P. and G. Koliantz, 1970, 1971). The strain was divided into three lines each of which was exposed to different genetical experiments.

Two-day old males of the second line, were let feed for 24 hours on Whatman filter paper which had been saturated with uric acid in distilled water. Then each male was made to mate with two virgin females in fresh Mostashfi medium. After 24 hours the males were discarded a and females remained to lay eggs for 48 hours. To obtain $F_2$, a mass mating method was used (Spencer 1947a, Tsuno 1970). In this experiment the evidence was saccharose solution with the same concentration as uric acid.

The effect of uric acid in 1, 3, 5 and 10 percent concentrations are summarized in Table 1.



| % Concent. | Gener. | l | sl | qn | malform. | mosai. |
|---|---|---|---|---|---|---|
| 1 | $F_1$ | 1 | 2 | 7 | - | - |
| | $F_2$ | - | 1 | 6 | 3 | - |
| 3 | $F_1$ | - | - | 10 | 1 | - |
| | $F_2$ | - | 2 | 8 | 3 | - |
| 5 | $F_1$ | 2 | - | 8 | 8 | - |
| | $F_2$ | - | 1 | 7 | 3 | 9 |
| 10 | $F_1$ | - | 1 | 9 | 9 | - |
| | $F_2$ | 5 | 3 | 2 | 13 | - |

The number of malformations in the $F_1$ increased with uric acid concentrations and reached its highest point in the $F_2$ of 10% concentration.

In 5% concentration, two gynandromorphs and nine cases of mosaism were observed, two of which are drawn diagramatically. Highest lethality in $F_1$ and $F_2$ was observed in 5% and 10% concentrations, respectively.

The results obtained by double bound (with saccharose solution), showed relatively lower malformations and lethal effects.

References: Godbole, N.N., R.M. Kothari and V.G. Vaidya 1971, DIS 46:116; Hochman, B. 1971, Genetics 67; Mostashfi, P. and G. Koliantz 1971, 3rd Iran. Cong. of Genet.

Matthews, D.L. and C.P. Wright. Western Carolina University, Cullowhee, North Carolina. Development of Gluful-4, 1(1)EN5, a lethal mutant of D. melanogaster.

Gluful-4, 1(1)EN5, is a sex-linked lethal mutant of Drosophila melanogaster which was X-ray induced by Novitski (1963). Death in this mutant usually occurs in the larval stage. The objective of this study was to determine general developmental characteristics of the life history of this lethal mutant. As a control, y, w, spl, sn larvae, which came from the stock in which Novitski (1963) induced the lethal mutant, were used.

To observe development of control larvae, sixty 48-hour larvae were placed in food containers at 25°C and observed until puparium formation. The control larvae exhibited normal development. All 60 control larvae underwent puparium formation at 110 ± 17 hours after oviposition. To observe development of 1(1)EN5 larvae, 130 48-hour larvae were placed in food containers and observed until death. The results of these observations are summarized in Table 1.

Table 1. Subsequent development of 130 1(1)EN5 larvae separated from non-lethal sibs and placed in food containers 48 hours after oviposition.

| Age in hours after oviposition | Total live larvae | Total dead larvae | Total puparia formed |
|---|---|---|---|
| 48 | 130 | 0 | 0 |
| 72 | 78 | 52 | 0 |
| 96 | 28 | 102 | 0 |
| 120 | 9 | 121 | 0 |
| 144 | 5 | 125 | 0 |
| 168 | 5 | 125 | 0 |
| 192 | 5 | 125 | 0 |
| 216 | 5 | 125 | 0 |
| 240 | 2 | 126 | 2 |
| 264 | 1 | 126 | 3 |
| 288 | 1 | 126 | 3 |
| 312 | 1 | 126 | 3 |
| 336 | 0 | 126 | 4 |

It is evident that most 1(1)EN5 individuals died before the stage of puparium formation. About 3% of the 1(1)EN5 larvae did undergo puparium formation. The rare instances of puparium formation in 1(1)EN5 larvae occurred much later than puparium formation in control larvae. Whereas control larvae formed puparia at about 110 hours after oviposition, the lethals formed puparia from 240 to 336 hours after oviposition. After puparium formation no further development was evident in the 1(1)EN5 individuals. All apparently died within 5 days after puparium formation.

Reference: Novitski, E. 1963, List of biochemical mutants DIS 37:51-53.

This work was supported by a grant from the North Carolina Academy of Science.

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

In August 1971, one hundred and thirty one isolines were established from Taiwan. In 128 of these the males had a white face. These are designated as belonging to Taxon A. In 1 line the males had white orbits. This is designated as belonging to Taxon B. The remaining 2 lines had males without white on the face. These are designated as belonging to Taxon C.

In Taxon A three simple heterozygous inversions were detected. Of these E and L had previously been detected at Cebu (Mather & Thongmeearkom 1972) but the third designated B' is new (figure). The Taiwan population appears to be far less variable than the Cebu population. In the Taiwan population a total of 7 inversions were recorded and 37/131 flies were free of heterozygous inversions. Of the three inversions recorded E and B' occur in high frequency and L in low frequency (table).

The one line of Taxon B had inversion C, already detected at Cebu, in heterozygous condition.

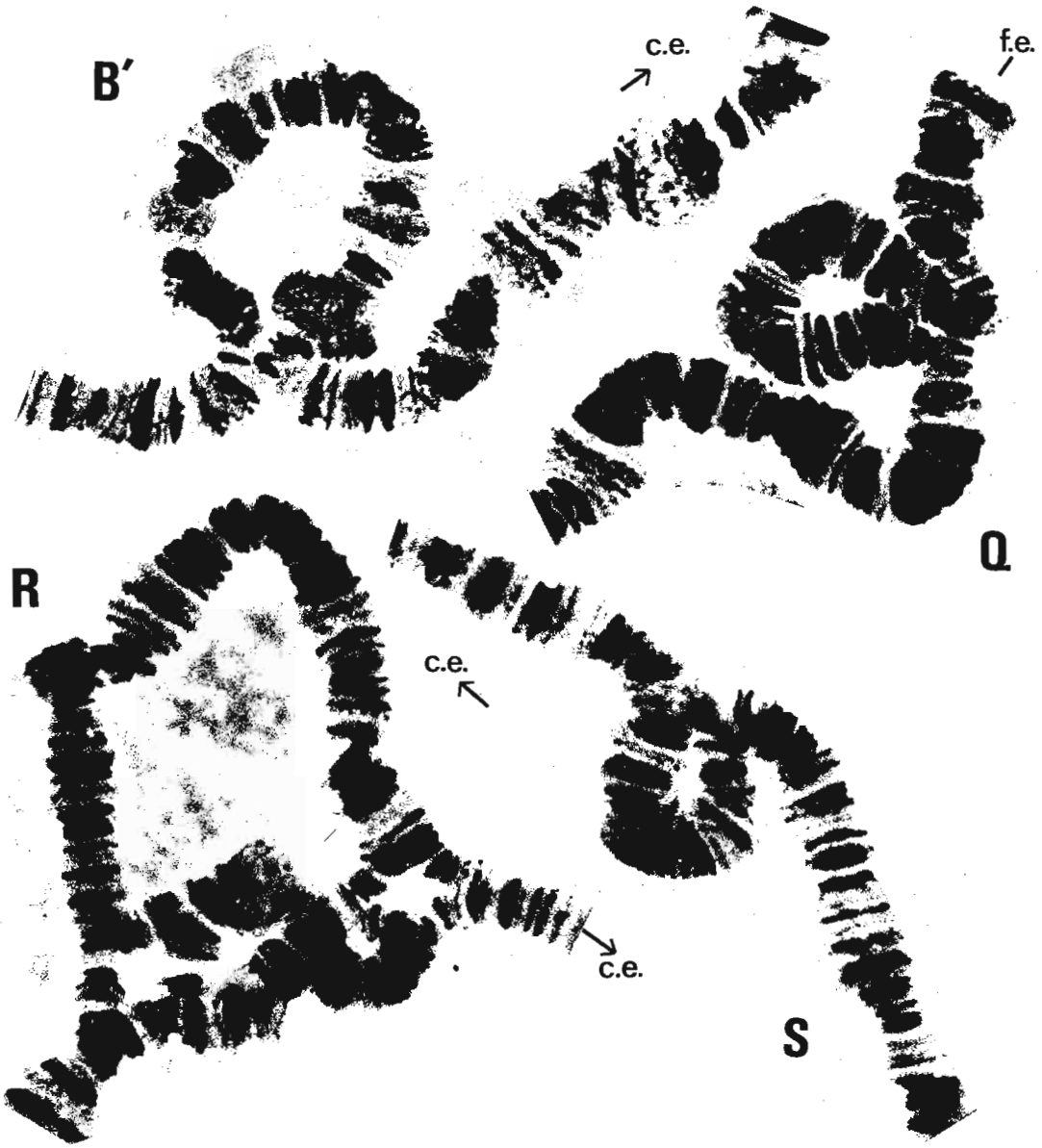



Figure. Inversions. c.e., centromere end; f.e., free end. Arrows point toward centromere ends.

| Inversion | Type | Position | Heterozygosity Frequency % |
|---|---|---|---|
| E | Sim | II LC | 39.1 |
| B' | Sim | III P | 47.7 |
| L | Sim | III P | 1.6 |

Sim=simple, C=Central, P=proximal to centromere

One line of Taxon C had three new inversions A, R and S and the other the Q inversion (figure).

The hybridisation relationships between the Cebu flies and the Taiwan taxons, which will throw light on their taxonomic status will be reported on in a later communication.

References: Mather, W.B. and P. Thongmeearkom 1972, Inversion polymorphism in D. sulfurigaster albostrigata, DIS 48:40.

(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.)

Sidhu, N.S. Indian Veterinary Research Institute, Izatnagar, U.P., India. A note on floating muscle fibres in seminal vesicles of D. melanogaster (Meig).

During October 1961, the author investigated the genetics of spermatozoa in Drosophila species using various types of microscopes like one with phase contrast, Ultraphot-II, electron microscope etc. in addition to the microscope-projector. During the course of the studies with Ultraphot-II of living spermatozoa, structures like polytene chromosomes were noticed which had too regular bands at equal intervals to be polytene chromosomes. These were discovered to be the floating muscle fibres in the seminal vesicles of Drosophila melanogaster. The pictures were taken at a magnification of X 3000. Fig. 1 shows the muscle fibres at X 6000 magnification.

The muscle fibres are clearly identifiable single bodies overlapping or floating freely in the seminal vesicles. Each muscle fibre consists of large numbers (hundreds) of sarcomeres. Each sarcomere in D. melanogaster muscle has been found to be 1 μ in length. Thickness at Z bands ranges from 1 to 1.17 μ while it is only 0.833 μ at the A type bands (myosin region). One can clearly identify Z and A types of bands. The Z-bands are dark striations 0.333 μ in width while the A bands are 0.162 μ in width with a gap of about 0.200 μ between the two A type bands of a sarcomere.

Fig. 1. Floating muscles taken from seminal vesicles of D. melanogaster seen at X 6000 (under an Ultraphot-II under ordinary light). A = A-type bands (Myosin); M = mitochondrial body (sarcosome); MEP = motor-end-plate of muscle fibre; N = nucleus; S = sarcomere; ST = spermatozoan tail; Z = Z-type band of sarcomere.

It is difficult to differentiate the sarcolemma from the sarcoplasm. The nucleus of the muscle fibre is seen to be 2 μ in diameter floating besides one of the sarcomeres but attached to it by a thin fibril. The motor-end-plate of the muscle fibre, a dark knob-like structure at the end of the muscle fibre is 1.3 μ in length and 1 to 1.5 μ in breadth. Mitochondria or sarcosomes are seen attached as dark beads 1.17 to 1.66 μ in length, either round or elongated structures appearing dark like the Z bands of the muscle fibre. The myofilaments (primary = myosin or secondary = actin) cannot be differentiated under the Ultraphot-II nor any other details like M or N band lines.

The occurrence of the floating muscles in seminal vesicles of D. melanogaster is still a strange phenomenon. Their exact location of occurrence too is not worked out, though one comes across such bodies only in some preparations.

(Continues at bottom of next page)

Golubovsky, M.D. and I.K. Zaharov.
Institute of Cytology and Genetics, Novosibirsk, USSR. The new autosomal mutation "almond eye" and its interaction with certain dominant mutants in Drosophila melanogaster.

In the first generation derived from crossing a male from a natural wild population (Uman) with females of strain D In(3LR)/Sb approximately half of the flies with the marker Dichaete had almond eyes and all the Stubble flies had normal eyes. A line was obtained from Dichaete offspring with abnormal eyes. In crosses with the normal alternative mutation named "almond eye" (symbol "ale") behaved as a usual monogenically inherited recessive. Thus ale becomes dominant in the presence of the gene Dichaete. Then we found that ale is located in the third chromosome. The position of the gene ale at the third chromosome was determined by using dominant markers Sb and Dl. The results enabled us to establish the position of locus ale in

The phenotype of ale/ale

The phenotype of Mc ale/Mc ale

the region of 47.5 ±. In the same region is located the gene Deformed. The dominant allele Dfd is lethal in the homozygote and in the heterozygote produces two main visible effects: reduced eye and tufted vibrissae. The phenotype of Dfd/ale heterozygotes show no deviants from normal size and shape of eyes and only about 20% of flies have the vibrissae near the eyes tufted. It remains unclear whether ale is closely linked with Dfd new gene producing in heterozygous condition the phenotypic supression of Dfd or ale is a new recessive allele of Dfd locus which in Dfd/ale restores the normal phenotype. In a preliminary recombination test Dfd/ale females were crossed to D/Sb males. In the $F_1$ 436 flies D/ale and 133 D/Dfd occurred and no flies with marker D and normal eye (recombinant). In heterozygotes Dl/ale, Mc/ale, Dr/ale and L/+; D/ale the mutant phenotype is more pronounced with respect to eye size. Mc ale homozygotes are completely eyeless, the facets are replaced by occasional bristles, head size is considerably reduced. Crosses between Mc ale homozygotes are sterile, whereas crosses involving females or males of other strains are fertile. Mutation Mc is a dominant supressor of the phenotypic effect of genes Dfd and ale exerted on eye shape but not its size. It is concluded that though the ontogenetic events determining normal eye shape and size are interrelated at certain early stages, subsequently they follow distinct courses of development since they are independently affected by genetic factors.

(Continued from preceding page)

Anything seen somewhat resembling these floating muscle fibres are flight muscle fibres of Drosophila which have been described to be fibrillar, striated and having giant round mitochondria associated with them as described by Watanabe and Williams (1953). Such giant mitochondria are not found associated with these floating muscles described in the present research note.

Reference: Watanabe, M.I. and C.M. Williams 1953, as quoted by Herbert H. Ross (1956) in "A text book of Entomology", John Wiley & Sons, Inc. (New York) pp 519.

Hardy, R.W. University of California, San Diego; La Jolla, California.

Aberrant cytodifferentiation of aneuploid spermatids in D. melanogaster.

Herskowitz and Muller (Genetics 39:836-850, 1954) reported results which are consistent with the hypothesis that there is a morphological difference between hypo- and hyperploid sperm. They used a camera lucida to measure the lengths of heads of sperm from the seminal vesicles of flies that had either attached XY or free X and Y chromosomes. The attached XY stock is expected to produce in each bundle 32 spermatids that contain both the X and Y and 32 that have no sex chromosome. The ratio of the amount of chromatin in the two types of nuclei, based on the size of ganglion metaphase chromosomes, is between 6:4 and 7:4. Their results showed a unimodal distribution of head lengths similar to that found in Oregon-R males except that the dispersion was greater in the attached XY stock. They interpreted the data as indicating that all the sperm heads probably have the same shape but they are distributed around two modes of lengths related as $\sqrt[3]{3} : \sqrt[3]{2}$. The width of the mature sperm head is less than 0.4μ and tapers to less than 0.1μ toward the acrosome (Bairati and Perotti, Comparative Spermatology, Academic Press, pp 333-346, 1970). Hence, an exact measurement of nuclear length as well as other nuclear dimensions is unlikely as these distances are below the resolution of usual light microscopy. The failure of Herskowitz and Muller to find a bimodal distribution of sperm lengths may therefore have been a consequence of the method used.

Recently, testes from flies that produce aneuploid sperm were prepared for electron microscopy by the method of Tokuyasu, Peacock and Hardy (Z. Zellforsch. 124:479-506, 1972). Electron micrographs of cross sections through the testes show that there are differences in spermatid dimensions. In $Y^S X.Y^L$, In(1)EN, y B/O flies there are approximately equal numbers of nuclei of two different sizes (Fig. 1, B and L) in all bundles of early (uncondensed chromatin) pre-individualization (Tokuyasu, Peacock and Hardy, Z. Zellforsch. 124:479-506, 1972) spermatids observed. Sequential sectioning of two bundles in one testis confirmed that the difference in size is continuous along the lengths of the nuclei. The lengths of the

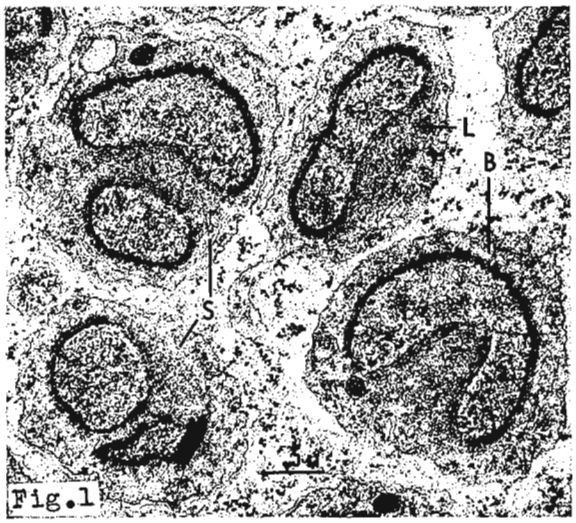


Fig.1

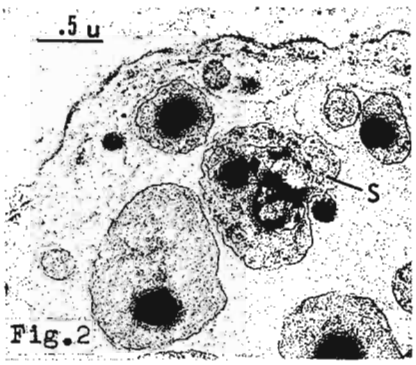


Fig.2

nuclei at this stage are comparable. Longitudinal sections of such nuclei have not been obtained, hence the exact measurement of nuclear lengths remains a question. These data do, however, suggest that dimensions of the spermatid nucleus other than length are affected by the addition of removal of chromosomes. The time during spermiogenesis at which the size difference first becomes apparent is not yet known.

In addition to the size bimodality, other deviations from normal morphology are seen at various levels along the bundles. Some nuclei are divided longitudinally into two unequal parts (Fig. 1, S). The division may be complete or incomplete. By this I mean that some nuclei are split in two from the acrosome end to the axoneme end whereas other nuclei are split only at the acrosome end giving a "forked" morphology. One nucleus was found which appears to be folded back on itself forming a hairpin configuration. By following a particular nucleus at all levels, it can be shown that this phenomenon occurs among the larger nuclei. A preliminary investigation of the occurrence of nuclei of this type in late (condensing chromatin) pre-individualization spermatids suggests that they may not complete spermio-

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Ranganath, H.A. and N.B. Krishnamurthy.
University of Mysore, Manasagangotri, India. Rate of development and fecundity in Drosophila nasuta.

Natural selection is the result of at least three component processes - differential survival, differential mating success and differential fecundity (Ayala 1970). Differential survival at different densities during preadult stages has been shown in D. nasuta (Ranganath and Krishnamurthy 1972). The experiment was conducted at 4 different densities - 200, 100, 50 and 25 eggs per vial (A, B, C and D groups respectively). To estimate the fecundity, the flies that emerged on the first day (Fast) and those that emerged on the last day (Slow) were selected. Virgins were isolated, aged for 5 days and then pair matings were made. The number of eggs laid were noted for the following 10 days. The fecundity test (Table 1) has shown

Table 1. Summary of the $\chi^2$ homogenity test for the fecundity of Fast and Slow developing flies.

| Group | No. of Pairs | | Observed | Expected | $\chi^2$ | P value |
|---|---|---|---|---|---|---|
| A | Fast | 4 | 390 | 318.28 | 36.9 | 0.0001 |
| | Slow | 3 | 167 | 238.72 | | |
| B | Fast | 6 | 697 | 480.60 | 243.5 | 0.0001 |
| | Slow | 4 | 104 | 320.40 | | |
| C | Fast | 4 | 441 | 336.50 | 65.4 | 0.0001 |
| | Slow | 4 | 232 | 336.50 | | |
| D | Fast | 6 | 798 | 478.80 | 432.0 | 0.0001 |
| | Slow | 4 | - | 219.20 | | |

that the fast emerging flies are more fecund than the slow ones. The average number of eggs laid by fast individuals in the A, B, C and D groups are 97, 116, 110 and 133 respectively, while the corresponding slow emerging flies have 55, 26, 58 and none respectively.

The characters of organisms are an outcome of the combined effects of its genetic constitution and the process of development. The egg laying capacity varies significantly between the fast flies of the 4 groups and also those of slow flies. This is indicative of the effect of different crowdings during development on the fecundity of adult flies.

Fast developing individuals having shorter generation time with a greater average reproductive output are the positive contributors to the process of adaptation at the population level.

Acknowledgements: 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. 1970, Essays in Evolution and Genetics in Honor of Th. Dobzhansky. Ed. M.K. Hecht & W.C. Steere, 121-158; Ranganath, H.A. and N.B. Krishnamurthy 1972, DIS 48:132.

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genesis (Fig. 2, S). If these spermatids do not mature and participate in fertilization the number of $F_1$ females would be less than the number of $F_1$ males. Such a deficiency of progeny from XY-bearing sperm relative to nullo-XY sperm is commonly observed in crosses of XY/O males. Males from the attached XY stocks studied with the electron microscope also yield this altered sex ratio. In a cross of the $Y^SX.Y^L$, In(1)EN, y B/O males to Canton-S females the fraction of Bar $F_1$ females was .409 (790/1929). The relation between abnormal nuclei and sperm function is being investigated.

The phenomena have also been seen in flies of the following genotypes: (1) C(2L)RM, dp; C(2R)RM, px (2) C(3L)RM, se h $rs^2$;C(3R)RM, sbd gl $e^s$ (3) $y^2/B^SY$;C(2L)RM, dp;C(2R)RM, px, C(3L)RM, $h^2$;C(3R)RM, + (4) mei-S332.

The nuclear profiles observed in these stocks have more variation than the attached XY stock. This is explicable if the kinds of aneuploidy that can occur in each stock are taken into consideration. For instance in stock (1) there are four kinds of nuclei that could occur: 1/4 no 2, 1/2 either 2L or 2R and 1/4 both 2L and 2R.

These preliminary results suggest that head size during spermiogenesis depends on chromatin content and that there are constraints on the physical amount of genetic material that a spermatid nucleus can contain and undergo normal cytodifferentiation. An analysis of nuclei in various stages of spermiogenesis is now in progress.

Diebolt, J.R. Queens College, Charlotte, North Carolina. The effect of inhomogeneous electrostatic fields on D. melanogaster egg hatch; negative results.

Avio and Tarozzi (1956, 1958) have reported that an electrostatic field of 125 V/m had no effect on oviposition or egg hatch of D. melanogaster. Similarly, Steen and Oftedal (1967) found that D. melanogaster egg hatch time was unnaffected by homogeneous magnetostatic fields of 0.16 and 0.5 tesla.

In this study D. melanogaster eggs were subjected to a 0.3 kV/cm or a 0.6kV/cm, inhomogeneous, (-) or (+) polarity, electrostatic field to determine the effect on egg hatch time. The treatment apparatus consisted of two aluminum plates measuring 0.3 thick x 39.7 cm long x 18.4 cm high, arranged parallel and separated by a distance of 10 cm. The plates were connected to one another by two porcelain spacers at each end. Culture vials, plugged with a one hole, size 4 rubber stopper, through which a piece of 5mm (o.d.) flint glass tubing about 8-9 cm long was inserted, were arranged in a single, uniform row between the plates. A piece of 12 guage copper wire about 18-19 cm long was inserted through the glass tubing and immersed into the corn meal, molasses, Brewer's yeast medium in the vials. Each wire emerging from each vial was connected at right angles to a long single piece of 12 gauge copper wire which was connected by one end to the ground wire of the D.C. voltage output source and the capacitor plates were connected to the positive terminal wire. To reverse the polarity of the field the capacitor plates were connected to the ground wire from the D.C. voltage output source and the single copper wire was connected to the positive

Table 1. Accumulated mean % egg hatch. Eggs subjected to inhomogeneous electrostatic fields.

| Treatment | Hours after egg count 20 | 21 | 22 | 23 | # eggs |
|---|---|---|---|---|---|
| Control | 8.5 | 44.0 | 85.6 | 90.5 | 327 |
| 0.3 kV/cm (-) | 5.9 | 40.9 | 84.1 | 89.8 | 405 |
| 0.3 kV/cm (+) | 6.5 | 44.7 | 86.6 | 91.5 | 344 |
| Control | 8.1 | 41.3 | 84.6 | 91.1 | 738 |
| 0.6 kV/cm (-) | 6.3 | 42.7 | 87.3 | 92.3 | 761 |
| 0.6 kV/cm (+) | 5.3 | 42.1 | 86.7 | 92.2 | 519 |

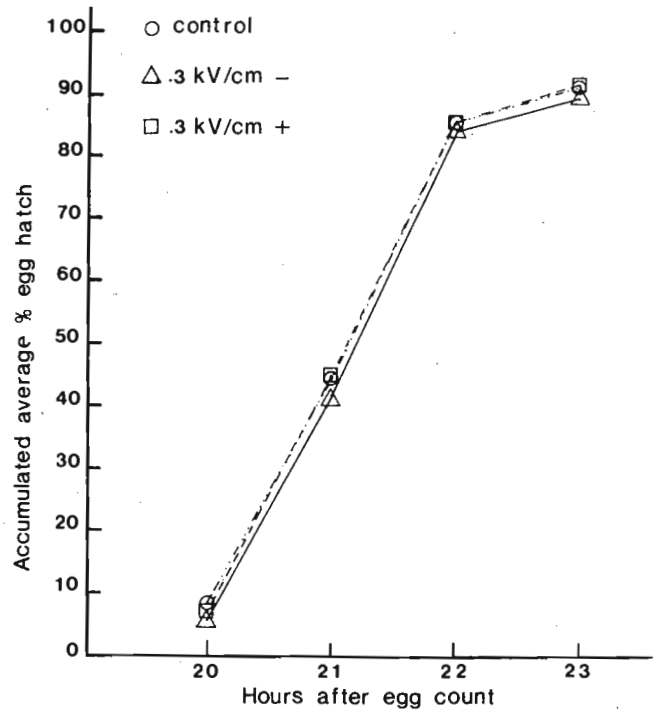


Figure 1. The relationship of eggs subjected to a 0.3 kV/cm inhomogeneous, (+) or (-) polarity, electrostatic field for 23 hours to percentage hatch.

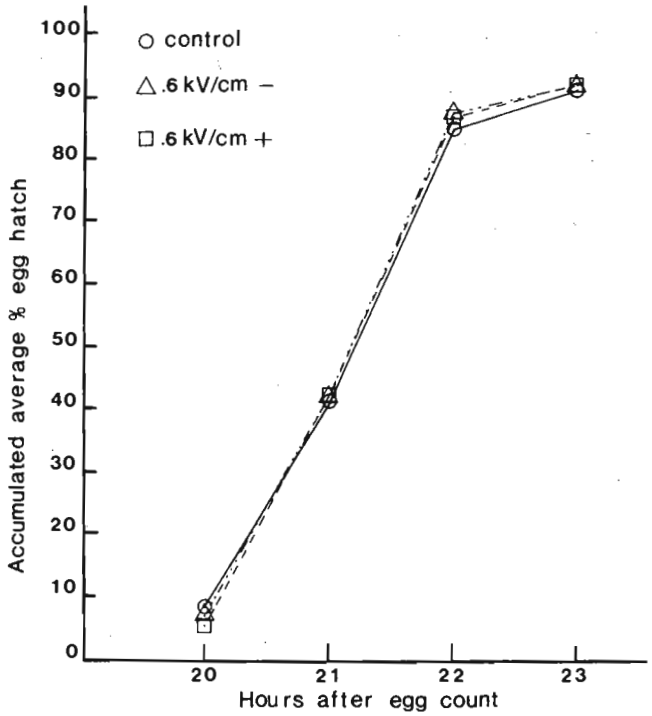


Figure 2. The relationship of eggs subjected to a 0.6 kV/cm inhomogeneous, (+) or (-) polarity, electrostatic field for 23 hours to percentage hatch.

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Hollis, R.J. College of William and Mary, Williamsburg, Virginia. Allozymic variation in D. melanogaster from Virginia.

Drosophila melanogaster populations were collected using baited traps at four locations (Toano, Short Pump, Afton Mt., and Monterey Mt.) in Virginia. Individual flies were then assayed via starch gel electrophoresis for Esterase 6 and Alcohol dehydrogenase using the methods of Johnson (p.c.). These allozymes are described in Wright (1963) and Johnson and Denniston (1964).

Table 1. Data for Esterase 6

| Site | Miles* | n | Genotypes S/S | F/S | F/F | Frequency of S |
|---|---|---|---|---|---|---|
| Toano | 0 | 50 | 12 | 27 | 11 | 0.51 |
| Short Pump | 56 | 56 | 20 | 23 | 13 | 0.56 |
| Afton Mt. | 130 | 35 | 13 | 21 | 1 | 0.67 |
| Monterey Mt. | 182 | 36 | 16 | 19 | 1 | 0.71 |

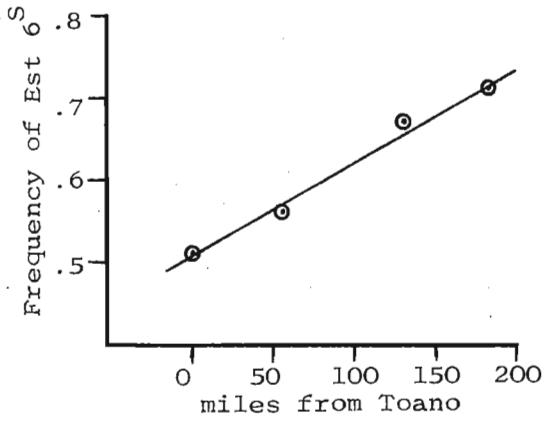
* approximate air males west of Toano

Table 2. Alcohol dehydrogenase data

| Site | Miles* | n | Genotypes S/S | F/S | F/F | Frequency of S |
|---|---|---|---|---|---|---|
| Toano | 0 | 96 | 36 | 39 | 21 | 0.58 |
| Short Pump | 56 | 68 | 37 | 28 | 3 | 0.75 |
| Afton Mt. | 130 | 41 | 16 | 16 | 9 | 0.59 |
| Monterey Mt. | 182 | 35 | 14 | 12 | 9 | 0.57 |

* approximate air miles west of Toano

Figure 1.




Tables 1 and 2 summarize the results of this study. Both of the loci were found to segregate in all populations sampled. As the sample sites lie on an approximate east-west line which passes through several topographic regions (coastal, piedmont, and montains), linear regressions were performed by least squares analysis using miles from the most easterly site as the abscissa and allele frequency as the ordinate. No apparent pattern was observed for Adh. For Est 6, a cline is suggested, with $r^2 = 0.94$. A graph of the Est 6 data, including the regression line is presented in Figure 1. I fully recognize that both the sample sizes and numbers were extremely limited in this preliminary study; however, this cline, if it does indeed exist, would indicate that selection is operative.

References: Johnson, F.M., personal communication; Johnson, F.M. and C. Denniston 1964 Nature 204:906-907; Wright, T.R.F. 1963, Genetics 48:787-801.

Supported in part by NSF GZ 2153.

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terminal wire.

From 80-100 isogenic Canton-S wild type females were collected and placed in each of several 1/2 pint bottles along with several males, on fresh, yeasted cornmeal medium for 24-48 hours. The flies were then very lightly etherized and 20 females were placed in each of several vials for 1 hour to lay eggs after which time the deposited eggs were counted. The vials containing the eggs were then subjected to the treatment field or were used for the control study. The control vials were placed between capacitor plates but were not subjected to the treatment field. The temperature in the treatment field or in the control field was $24 \pm 0.5^{\circ}C$.

Egg hatch counts were begun 20 hours after the egg counts and continued for 3 hours. Egg hatch was scored by counting the number of empty egg cases per vial.

The accumulated average number of eggs hatching at each counting time was calculated. Table 1 shows the percent egg hatch for each experimental treatment. Figures 1 and 2 show the relationship of the accumulated average percent egg hatch to time of count for eggs subjected to a 0.3 kV/cm and 0.6 kV/cm electrostatic field respectively.

The electrostatic fields used have no effect on egg hatch time or frequency of egg hatch.

References: Avio, C.M. and G. Tarozzi 1956, Riv. Biol. 48:49-74; _____ 1958, Riv. Biol. 48:145-152; Steen, H.B. and P. Oftedal 1967, Experentia 23:814.

Golubovsky, M.D. and Y.N. Ivanov. Institute of Cytology and Genetics, Novosibirsk, USSR. Autosomal mutation in Drosophila melanogaster killing the males and connected with female sterility.

In 1967 we performed an experiment on the frequency of occurrence of the second chromosome mutations in the offspring of flies isolated from natural population of Magarach (Crimea). The wild males were crossed individually to Cy/$L^2$ females. In the $F_3$ of one of such cross where parental males and females had Cy/+ genotype we obtained the following results: 548 Cy/$+^M$; 44 $+^M/+^M$. The "$+^M$" indicates the individual chromosome. The Cy offspring consisted of both females and males. But all the 44 flies homozygous for second chromosome were females. These females had reduced viability and were absolutely sterile. We could not explain these results by suggesting the occurrence of any chromosomal rearrangement between the sex chromosomes and the autosomes. The stock was maintained in balanced condition for some years. Then we examined the segregation again (see Table 1). Thus, again, the second chromosome homozygotes were only females. Their survival increased for some years of culturing but yet remained much lower than that of Cy/+ females. As earlier, all the second chromosome homozygous females $+^M/+^M$ were absolutely sterile. We had suggested that the second chromosome $+^M$ had a recessive factor we named "male killer" (symbol: mak) which in homozygous condition kills the non-Cy males and also causes female sterility. The gonads of the non-Cy females were often rudimentary. Assuming a monogenic nature of the "mak" factor, we mapped it by means of dominant genes Bl and $L^2$.

Table 1. The results of crosses of ♀ Cy/$+^M$ x ♂ Cy/$+^M$

| phenotype of offspring | | | |
|---|---|---|---|
| ♀♀ Cy | ♂♂ Cy | ♀♀ + | ♂♂ + |
| 535 | 543 | 216 | - |

Table 2. The results of crosses of ♀ Bl $L^2$/mak x Cy/mak ♂

| Phenotype and sex | Cy ♀♀ and ♂♂ | | | | non-Cy ♀♀ | | | | non-Cy ♂♂ | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Bl L | + | Bl | L | Bl L | + | Bl | L | Bl L | + | Bl | L |
| Frequency | 460 | 636 | 36 | 64 | 319 | 210 | 18 | 29 | 296 | 1 | 18 | - |
| Total | | | | | 576 | | | | 315 | | | |

The data of table 2, especially the sex ratio among the non-Cy flies, confirm the suggestion about the monogenic nature of the mak factor. Then the data show that the mak factor is to the left of the Bl locus and closely linked with Bl. Its position is no more than one m.u. from Bl, at approximately 54 ±. The second chromosome containing mak had an obviously reduced crossing over frequency, as shown by the reduction of the recombinant zygotes Bl and L among Cy progeny and non-Cy females. Unexpected results were observed in testing the sterility of different non-Cy females and Bl males. Of 15 tested males, 14 proved to be sterile. On the other hand, more than half of the females in the progeny from the above mentioned crosses with expected genotypes of mak/mak and $L^2$ mak/+ mak were fertile. It is interesting to note here that in the region 54 of standard second chromosome map, a cluster of mutations responsible for male and female sterility is known.

Smith, J.W. Purdue University, Lafayette, Indiana. Mating behavior of Drosophila nigromaculata.

This description of the mating behavior of Drosophila nigromaculata, a member of the quinaria species group, is based on 66 observations using one male with three females. All observations were made at 25° C in a moistened filter paper lined shell vial. The terminology used in describing mating behavior follows that of Spieth (1952).

The male taps at the side or occasionally at the front; moves to the rear of the female, assumes a head under the wings position and using the tarsal portion of his forelegs begins to stroke the dorsal and lateral surfaces of the female's abdomen. The forelegs of the male move alternately. Less frequently, the male will stroke the dorsal surface of the female's wings. Initial stroking is slow with well defined pauses but as courtship continues, stroking becomes

faster with fewer breaks between bursts of stroking.

As the speed and intensity of stroking increases, the male pauses in stroking and flicks one wing rapidly forward 90°. The extended wing vane is parallel to the substrate. As courtship continues, both wings are simultaneously flicked forward 90°. Wing flicking reciprocates rapidly with stroking so that just prior to mounting, it is difficult to temporally separate these two courtship elements.

A receptive female spreads her wings 90°. Infrequently, (27% of all observations) after this acceptance signal is given, the male quickly places his head close to the terminalia of the female, extends his proboscis and rapidly licks the genitalia once or twice prior to mounting. In 9.1% of the observed courtships, males mounted females which had failed to give an acceptance signal.

The male mounts the female by crawling forward up onto her dorsum. The hindlegs of the male remain on the substrate. The midlegs are curved around the lateral surfaces of the abdomen with the tarsi resting on the midlegs of the female. The forelegs of the male rest on tergites one and two with the tarsi curved over the dorsal surface of the wing halteres of the female. While mounting, the male curls his abdomen under and thrusts forward and up until genitalic contact is make. The now coupled flies turn in a circle for several seconds and stop.

Prior to completion of copulation, the female, using her hindlegs, kicks back at the area of genitalic contact. Copulation is terminated however, by the male straightening his abdomen and breaking contact. The male then backs off the dorsum without turning, after which both flies begin cleaning movements beginning at the terminalia and ending at the head.

Non-receptive females kick back with their hindlegs, flutter their wings, depress their abdomen or decamp. Males court one another and countersignal by fluttering.

Of the 66 observed courtships, 48 resulted in copulation; of these 48 pairs, 43 of the females were inseminated as determined by microscopic examination of the seminal receptacles and/or spermathecae for the presence of motile sperm. Both males and females used in these observations were virgin, six day flies. Mean courtship time was 39.8 seconds with a range from 4 to 177 seconds. Mean copulation time was 5 minutes 11 seconds with a range from 3 minutes 29 seconds to 14 minutes 22 seconds.

Reference: Spieth, H.T. 1952 Bull. Amer. Mus. Nat. Hist. 99:395-474.

Palabost, L. Université Paris, France.

The influence of density on the larval viability of Drosophila melanogaster.

The influence of density on the larval viability was studied in two strains of Drosophila melanogaster, either in pure culture or in competition. The two strains were a wild one, M68 CA, and the mutant rosy scarlet (ry st). They were grown on a rich nutrient medium and on a medium deficient in both proteins and vitamins, at 20 and 25°.

The following results were obtained on a rich medium, where there is no competition for nutrients; the larval viability of the wild strain is density independent, for all tested densities from 25 to 1000 larvae. The mean hatching percentage is 81.5 ± 0.6% at 25° and 72.8 ± 0.6% at 20°. The larval viability of ry st is density-dependent, with a threshold at 400-500 larvae. This sudden fall of viability, in the absence of larval competition, depends on the excretion of auto-toxic products by the ry st larvae. The ry st larvae have no influence on the viability of the wild ones; neither have the wild ones on those of ry st.

On a medium deficient in proteins and vitamins which involves larval competition, I observed the following results; the larval viability of the wild strain is density independent, but lower than on the rich medium at 25°, while it is density-dependent at 20°. The larval viability of ry st is density-dependent both at 25° and at 20° and lower than on the rich medium. The ry st larvae have no influence on the viability of the wild ones, but living wild larvae reduce the viability of ry st larvae. On the contrary, when the ry st larvae are grown on a medium conditioned by the excretory products of wild larvae, their viability is higher; in other words there is facilitation.

These results prove that when two strains are in competition, three situations are possible: 1) no interaction, 2) inhibition, 3) facilitation.

Sobels, F.H. University of Leiden, The Netherlands. The induction of non-disjunction of compound second chromosomes in stage-14 oocytes by X-rays.

The findings recorded for the induction by X-rays in stage-14 oocytes are controversial. After exposure to 500 R, Day and Grell (Mutation Res. 3:503, 1966) obtained values similar to those expected from stage-7 oocytes, provided linear extrapolation from 4000 R data for stage-7 oocytes is correct. For both the X and the fourth chromosomes, Kiriazis and Abrahamson (Genetics 60:193, 1968) did not observe an increase over the control values. The latter result was confirmed by Traut (Mutation Res. 10:156, 1970), who categorically concludes that a dose of 400 R does not induce non-disjunction in either mature or immature (Mutation Res. 10:125, 1970) oocytes. More recently, however, Clark and Sobels (Mutation Res. in press) and Sobels and Clark (Abstr. 3rd Europ. Dros. Res. Conf. Milan, Sept. 1972, ed. Barigozzi), by making use of compound second chromosomes, were able to show that, in contrast to Traut's findings, exposures of 500, and even 250 R do significantly raise the non-disjunction frequency in stage-7 oocytes over that in the controls. A re-investigation of the induction of non-disjunction in stage-14 oocytes, using this method, seemed therefore indicated. Advantage is taken of the fact that compound reverse metacentrics (or isochromosomes) in females usually disjoin regularly, whereas in males they show disjunctional and non-disjunctional segregation with equal frequencies. Since disomic and nullosomic female gametes will result in viable zygotes after fertilization with nullosomic and disomic sperm, respectively, this system makes the quantitative recovery of non-disjunctional progeny from various states of oocyte development possible. It should be noted that disomics can only result from non-disjunction, whereas nullosomics, in part, also originate from chromosome loss following breakage.

Four-day old females of the genetic constitution C(2L)RM, $j^{63}$; C(2R)RM, px were exposed to 400 R and mated to C(2L)RM, bpr; C(2R)RM, vg males. To record stage-dependent sensitivity differences, egg samples were collected from 9:00 a.m. until 5:00 p.m., and from 5:00 p.m. until 9:00 a.m., over a period of 72 hours in total; in this context only the data for the first two broods, sampled during 24 hours after exposure to irradiation, are presented.

Pooled data of 7 replicate experiments.

| Brood | Hours after irradiation | No. of progeny | Disomic eggs no. | Disomic eggs % | Nullosomic eggs no. | Nullosomic eggs % |
|---|---|---|---|---|---|---|
| A | 0 - 8 | 682 | 6 | 0.88 | 11 | 1.6 |
| B | 8 - 24 | 2,734 | 26 | 0.95 | 24 | 0.88 |
| | Controls | 12,142 | 50 | 0.41 | 42 | 0.35 |
| | Stage-7 oocytes (500 R) | 7,393 | 70 | 0.94 | 33 | 0.44 |

Considering first the data for disomics, it may be noted that their frequency does not significantly differ between broods A and B, and hence the data of these two sampling periods were pooled. The frequency of 0.93% thus obtained, is significantly higher (at the 0.01% level, Kastenbaum and Bowman, Mutation Res. 9:527, 1970) than in the controls, and does not at all differ significantly from that found after exposing stage-7 oocytes to 500 R. The present data thus confirm earlier findings that there is no difference in radiosensitivity with regard to the induction of non-disjunction between stage-14 and stage-7 oocytes. Moreover, they are in line with the findings of Day and Grell for X-chromosome non-disjunction in stage-14 and slightly earlier oocyte stages, but different from those recorded by Kiriazis and Abrahamson, and Traut.

With regard to nullosomics, it can be seen that these appear to be induced at higher frequency in brood A, than in brood B, though the difference is not significant. The frequency obtained in brood A is significantly higher ($P < 0.01$) than in the controls, and that induced by 500 R in stage-7 oocytes. A similar result has been recorded by Traut, and the different findings for disomics and nullosomics clearly originate from the fact that chromosome breakage phenomena contribute to the induction of nullosomics, but not to that of disomics.

This work received support from the J.A. Cohen Institute for Radiopathology and Radiation Protection and the Health Research Organization TNO. It was carried out in part under the Association of Euratom with the University of Leiden, Contract Nr. 052065-1 BIAN.

Koliantz, G. Teachers Training College, Tehran, Iran. Recombination changes in D. melanogaster.

| Generation | Low line | High line |
|---|---|---|
| 1 | 16.6 | 18.1 |
| 2 | 16.2 | 18.4 |
| 3 | 15.9 | 18.6 |
| 4 | 16.1 | 18.2 |
| 5 | 16.1 | 18.2 |
| 6 | 15.9 | 18.6 |
| 7 | 15.9 | 18.5 |
| 8 | 16.1 | 18.6 |
| 9 | 16.0 | 18.7 |
| 10 | 15.9 | 18.7 |

The recombination value between Bl-$L^2$, derived from Bl $L^2/al^2$ Cy $lt^v$ sp, was studied under artificial selection. Virgin Bl $L^2$/+ + females were crossed in two lines with Gayaneh wild type males. In the $F_1$ four recombination classes (Bl-$L^2$, +, Bl and $L^2$) were obtained. In each selected line, Bl $L^2$/+ + females were made to cross with their wild-type brothers in over ten generations. All flies were raised in Mostashfi medium and reared at 23 ± 1°C.

Results and conclusion: The table shows the recombination values within ten generations.

For each selected line a plateau was formed, which shows slight variability in the recombination classes.

No changes in fecundity of Bl $L^2$/+ + females were observed in either line.

In the 12th generation, a reciprocal cross was made between the two selected lines. The average 16.9 for Bl $L^2$/+ +♀L x + +/+ +♂H and 17.1 for Bl $L^2$/+ +♀H x + +/ + +♂L was observed, which does not show a significant dominance of one over the other.

Reference: Kidwell, M.G. 1972, Genetics 70:419-443.

Evans, W.H. Western Washington State College, Bellingham, Washington. Non-random segregation in Drosophila females.

In the course of experiments designed to show the extent of major autosome nondisjunction in inversion heterozygous females a case of non-random segregation was found.

XX;Sm1/+;CxD/+ females 2 to 4 days of age were mass-mated to attached 2 males (C(2)L, dp;C(2)R, px). Tallies of offspring genotypes are given in Table 1.

These results show that patroclinous-2 progeny are predominantly +/+ for the third chromosomes while most matroclinous-2 progeny receive the CxD chromosome from their mothers. The non-randomness is in the tendency for the CxD chromosome to be included in diplo-2 eggs beyond expectation ($P < .05$).

Table 1. Progeny from XX;Sm1/+;CxD/+ ♀♀ x C(2)L, dp;C(2)R, px ♂♂.

| Matroclinous-2 | | Patroclinous-2 | |
|---|---|---|---|
| Sm1/+;+/+ | 4 | dp, px;+/+ | 19 |
| Sm1/+;CxD/+ | 14 | dp, px;CxD/+ | 5 |
| Total | 18 | Total | 24 |

The simplest model that could account for these results would be that the Sm1 and CxD chromosomes tend to the same pole during meioses that are nondisjunctional for the second chromosomes. If this were so one would expect a similar but reciprocal tendency for the Sm1 chromosome to be included in diplo-3 eggs. As shown in Table 2, the results of such a test do not conclusively support this model.

Table 2. Progeny from XX;Sm1/+;CxD/+ ♀♀ x C(3)L, ri;C(3)R, sr ♂♂.

| Matroclinous-3 | | Patroclinous-3 | |
|---|---|---|---|
| +/+;CxD/+ | 31 | +/+;ri, sr | 38 |
| Sm1/+;CxD/+ | 38 | Sm1/+;ri, sr | 24 |
| Total | 69 | Total | 62 |

Another model that fits the results better is this: All three unpaired major autosomes may compete for pairing sites on the arms of the non-inverted third chromosome. The CxD chromosome often pairs with its homolog, at least along one arm. However, either of the second chromosomes may pair along the other arm. The remaining second chromosome is assumed to remain univalent. During segregation this situation, as often as it occurs, leads to the formation of diplo-2 eggs that are CxD or nullo-2 eggs that are not.

The inconclusive results shown in Table 2 indicate that if the non-inverted second chromosome has similar pairing sites they are less effective than those of the third chromosome in finding pairing partners. Perhaps this could be simply because the second chromosome is shorter. A question naturally arises as to why the CxD chromosome cannot pair with more than one partner as well as its non-inverted homolog. It may be that in the CxD chromosome one of the pairing sites has been disrupted. Further work is underway to test the validity of the multiple pairing site idea.

Ehrman, L., J. Leonard and A. Lewis. State University of New York, Purchase, New York. On the mechanism of selective mating between two D. paulistorum semispecies.

Sexual isolation between the Centro-american and the Amazonian semispecies of D. paulistorum has been shown to be almost complete (Koref-Santibanez, 1972a and 1972b; Perez-Salas and Ehrman, 1971). Tests in Elens-Wattiaux (Elens and Wattiaux, 1964) direct observation chambers in these laboratories produced no intersemispecific matings when Belem, Brazil, (Amazonian) ♀♀ (hereafter = B) were courted by Lancetilla, Honduras, (Centroamerican) ♂♂ (hereafter = H) or when the reciprocal cross was attempted. But 5% intersemispecific matings occurred when ♀♀ and ♂♂ of both semispecies were present simultaneously.

Extracts of Belem ♂♂ were prepared by grinding 1000 of them in chilled dichloromethane in a tissue grinder. The crude extract was painted on H ♂♂ and crosses with B ♀♀ attempted unsuccessfully in two trials involving 25 ♂♂ and 25 ♀♀ each trial. Although the numbers of intersemispecific matings were not enhanced in this way, the rate of mating (matings/unit time) could be reduced between H ♂♂ and H ♀♀ by painting this B ♂♂ extract on the H ♂♂ (that is relative to flies painted with only $CH_2Cl_2$). It was further found that this negative assay was possible even when strips of filter paper, coated with the $CH_2Cl_2$ extract and allowed to dry, were added to the mating chambers (foam-stoppered 2 cm diameter vials containing 8 H ♀♀ and 6 H ♂♂). Subsequent chromatography of the extract on Quantum LQDF silica gel thin-layer plates indicated that the activity (measured by negative assay) is present in the fraction of $R_f$ = o.77 to 1.0, when developed with $CH_2Cl_2$, indicating a relatively nonpolar compound. Illustrative times to 4 H ♀♀ x 4 H ♂♂ matings in these tests are:

| Fraction ($R_f$): | 0-0.23 | 0.23-0.35 | 0.35-0.53 | 0.53-0.77 | 0.77-1.0 | $CH_2Cl_2$ |
|---|---|---|---|---|---|---|
| Time (min.) | 81 | 77 | 50 | 19 | 136 | 58 |

Variability of these preliminary results is high, but relative ordering appears to be consistent. Comments and suggestions on the proper treatment of the negative assay data are invited.

While determining baseline data, an interesting side observation was made. The effect of the pheromone of the common house fly (Musca domestica; Carlson, et al., 1972; the compound is 9-Z-tricosene commercially available from Hampstead Chemical Co.) on Drosophila mating behavior was measured. D. pseudoobscura, a North American species, was strongly inhibited by the presence of the pheromone, whereas the mating behavior of D. paulistorum (same two semispecies) was unaffected. Whether this difference is due to interspecific contact between D. pseudoobscura and M. domestica (and, inversely, the absence of such contact with D. paulistorum, a Neotropical species) or to some other factor (e.g., similarity of the Musca pheromone to other significant D. pseudoobscura sensory cues), has not been tested.

References: Carlson, D.A. et al. 1971, Science 174:76-78; Elens, A.A. and J.M. Wattiaux 1964, DIS 39:118-119; Koref-Santibanez, S. 1972a, Evolution 26:108; _____ 1972b, Evolution 26:in press; Perez-Salas, S. and L. Ehrman 1971, Genetics 69:63-70.

Supported by NIH Research Grant 1 R01 GM 18907-01.

Sang, J.H. University of Sussex, Brighton, England. Can ecdysones be fed to Drosophila larvae?

There are conflicting reports in the entomological literature on the effects of feeding ecdysones to insects. The following data suggest that both α- and β-ecdysone are ineffective when fed to D. larvae grown on Sang's (1956)[1] medium under germ-free conditions. The tu bw strain was used for these experiments to test if ecdysone suppressed melanotic tumor formation, and dihydrocholesterol was provided in place of the usual cholesterol since this raises tumor frequency and is itself unlikely to be converted to ecdysones[2]. The ecdysones were added at 45 hours of larval development, just prior to the

| | Control | + Tween 80 | + α-ecdysone | + β-ecdysone | |
|---|---|---|---|---|---|
| Larval period | 0.743 | 0.786 | 0.757 | 0.764 | log. days |
| Tumor frequency | 91.2 | 87.7 | 98.7 | 98.1 | per cent |

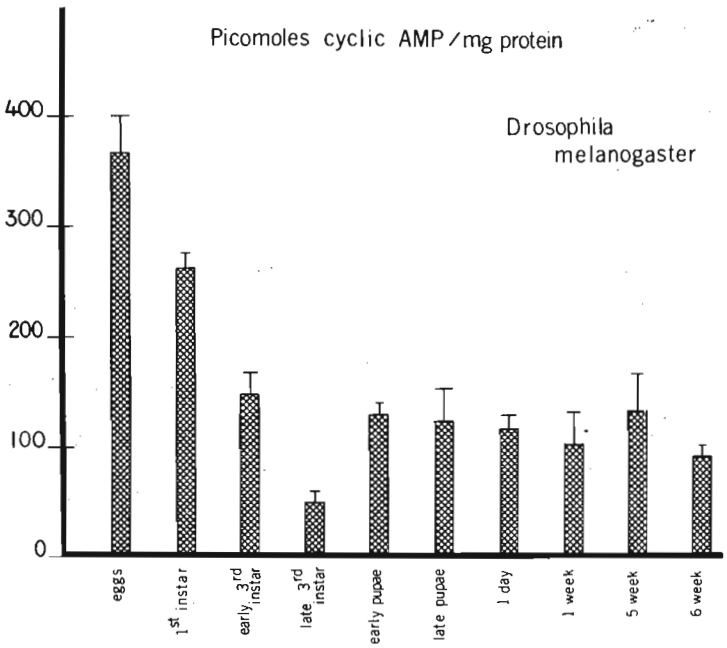
time when tumors form, and were soluabilised with Tween 80. Since none of the differences are significant the presumption is that the hormones are destroyed during absorption by the larvae

and that previous negative results are not a consequence of interactions with the flora of usual culture media.

References: 1) Sang, J.H. 1956, J. Exp. Biol. 33:45-72; 2) Cooke, J. and J.H. Sang 1972, Genetical Res. in press

Nicolosi, R.J., M.B. Baird, H.R. Massie, and H.V. Samis. Masonic Medical Research Laboratory, Utica, N.Y. Cyclic AMP levels in pre-adult and adult D. melanogaster.

Sustaining levels of Adenosine 3,'5'-cyclic monophosphate, (cyclic AMP) were measured in pre-adult and male adult Oregon-R, D. melanogaster. Pre-adult organisms at different stages of development were reared and collected as described elsewhere (1). All samples were homogenized in 0.32 M sucrose and adjusted to a final volume of 5 ml. Chitin was eliminated from the homogenates essentially as described in a previous note (2). An aliquot of the homogenate was then added to preloaded centrifuge tubes containing 1 ml of ice-cold 10% TCA. Following centrifugation at 17,000 x g for 10 minutes, at 0°C, the recovered supernatants were assayed for cyclic AMP by the method of Gilman (3).




Protein was determined on the homogenates according to the method of Lowry et al., (4).

No appreciable differences in sustaining levels of cyclic AMP were apparent in adult Drosophila which range in age from 1 day to 6 weeks, (Fig. 1). However, there appears to be considerable differences in the levels of cyclic AMP present in different pre-adult stages, (Fig. 1). It is evident that Drosophila eggs and 1st instar larvae possess levels of cyclic AMP which are 2 and 3 fold greater, respectively, than the remaining pre-adult Drosophila, as well as in those found in later stages. These preliminary results suggest that higher levels of cyclic AMP are present during those developmental stages of Drosophila in which there are high levels of mitotic activity.

References: (1) Samis, H.V.Jr., F.C. Erk and M.B. Baird 1970, Exp. Geront. V. 6:9-18; (2) Samis, H.V. and F.C. Erk 1969, DIS 44:132; (3) Gilman, A.G. 1970, Pro. Nat. Acad. Sci. 67:305-312; (4) Lowry, O.H., M.J. Rosebrough, A.L. Farr and R.J. Randall 1951, J. Biol. Chem. 193:265.

Malpica, J.M. Institute of Animal Genetics, Edinburgh, Scotland. Enzyme polymorphisms in four populations of D. melanogaster.

Four laboratory populations of D. melanogaster were characterized at seven loci on the third chromosome controlling biochemical polymorphisms. The populations - Kaduna, Pacific, Canberra and Stellenbosch - differ in origin being from Nigeria, the Pacific coast of the U.S., Australia and South Africa respectively. The four stocks have been maintained for 23, 17, 13 and 3 years respectively since their capture in large population cages in the laboratory. The foundation stocks for all of them were above 100 females except Kaduna where the number is not known. The number of individuals analyzed and the frequencies of the different alleles are given in the following Table, A standing for the fastest anodic migrating form and the others in this order within each locus. (Table on next page).

Table - Numbers of individuals analyzed and results

| | | Populations | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Kaduna | | Pacific | | Canberra | | Stellenbosch | |
| Locus | Allelic form | Flies scored | frequency | Flies scored | frequency | Flies scored | frequency | Flies scored | frequency |
| Idh | A | 86 | 1.0 | 34 | 1.0 | 30 | 1.0 | 29 | 1.0 |
| Est 6 | A | 184 | 0.31 | 100 | 0.74 | 100 | 0.76 | 104 | 0.35 |
| | B | | 0.69 | | 0.26 | | 0.24 | | 0.65 |
| Pgm | A | 84 | 0.0 | 44 | 0.0 | 44 | 0.08 | 37 | 0.03 |
| | B | | 1.0 | | 1.0 | | 0.92 | | 0.85 |
| | C | | 0.0 | | 0.0 | | 0.0 | | 0.12 |
| Est C | A | 184 | 0.0 | 100 | 0.0 | 100 | 0.0 | 104 | 0.19 |
| | B | | 1.0 | | 1.0 | | 1.0 | | 0.81 |
| Odh | A | 86 | 1.0 | 42 | 1.0 | 32 | 1.0 | 29 | 1.0 |
| Xdh | A | 84 | 1.0 | 27 | 1.0 | 27 | 1.0 | 27 | 1.0 |
| Aldox | A | 86 | 0.0 | 47 | 0.0 | 39 | 0.0 | 36 | 0.04 |
| | B | | 0.0 | | 0.0 | | 0.0 | | 0.18 |
| | C | | 1.0 | | 1.0 | | 1.0 | | 0.78 |
| No. loci polymorphic | | 1 | | 1 | | 2 | | 4 | |
| Proportion of genome heterozygous per individual | | 0.061 | | 0.055 | | 0.073 | | 0.197 | |

Bijlsma, E. and W. van Delden. University of Groningen, Haren, The Netherlands.
Polymorphism at the alcohol dehydrogenase locus in D. melanogaster.

Experiments were performed to investigate the nature of the widespread polymorphism at the alcohol dehydrogenase locus in natural populations of D. melanogaster. In addition, experiments were carried out on the probability of survival in extreme environments of populations differing in genetic composition concerning the Adh locus. To characterize the genotypes of individual flies, agar gel electrophoresis was carried out at 140 V and 28 mA for one hour, using a 0.02 M veronal buffer pH 8.5. After electrophoresis, the gels were put in a solution consisting of 97 ml 0.1 M veronal buffer pH 8.5, 3 ml aethanol 96% and 50 mg NAD. Sites of Adh activity on the gel can then be detected by fluorescence under UV (366 nm). Five homozygous S and five homozygous F lines were isolated from the Groningen population. This population was started in 1967 with several inseminated ♀♀ from a local fruit market and kept in the laboratory for three years. The frequency of the F-allele of Adh in this population was rather stable at 0.50-0.60. Cage populations were started from the homozygous lines with different initial gene frequencies. Frequency changes in these populations are given in Table 1. Extinction under more or less extreme conditions was measured in the following way.

Table 1. Frequency changes in cage populations.

| Population | Initial frequency F-allele | Frequency F-allele after 5 months | Frequency F-allele after 19 months |
|---|---|---|---|
| Base population | 0.62 | 0.53 | 0.54 |
| 1 | 0.20 | 0.37 | 0.66 |
| 2 | 0.20 | 0.55 | 0.54 |
| 3 | 0.40 | 0.55 | 0.59 |
| 4 | 0.40 | 0.54 | 0.63 |
| 5 | 0.60 | 0.55 | 0.45 |
| 6 | 0.60 | 0.65 | 0.64 |
| 7 | 0.80 | 0.60 | 0.60 |
| 8 | 0.80 | 0.64 | 0.68 |

Four kinds of vial populations, differing in genetic composition, were compared in each of the eight different environments given in table 2. These four types of populations were: monomorphic F, monomorphic S, polymorphic with an initial frequency of 0.20 F and polymorphic with an initial frequency of 0.80 F. Each type of population was represented in each environ-

Table 2. LD 50 values in different environments.

| | Environments * | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Type of population | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| monomorphic F | 13.9 | 10.9 | 3.6 | 5.6 | 1.5 | 9.2 | 21.6 | 10.1 |
| monomorphic S | 13.7 | 5.5 | 4.0 | 4.3 | 2.2 | 9.8 | 22.3 | 34.2 |
| polymorphic (0.20 F) | 21.4 | 10.1 | 7.7 | 5.6 | 2.3 | 9.9 | 16.7 | 14.4 |
| polymorphic (0.80 F) | 20.2 | 14.7 | 7.0 | 5.5 | 2.7 | 9.0 | 16.4 | 12.9 |
| number of generations | 11 | 8 | 10 | 9 | 5 | 11 | 11 | 9 |

* Environments: 1. Control: 25°C, 70% R.H., food medium consisting of 1000 ml water, 19 grams of agar, 54 grams of sucrose, 32 grams of yeast, 13 ml nipagine solution (10 grams nipagine in 100 ml aethanol 96%); 2. medium supplemented with 20% aethanol; 3. temperature 29.8°C; 4. yeast amount reduced to 5 grams; 5. medium supplemented with 10% aethanol, yeast reduced to 5 grams; 6. sucrose content reduced to 25 grams, after 6 generations further reduced to 12.5 grams; 7. 35% R.H.; 8. 95%-100% R.H.

ment with 50 vial populations at the start of the experiment. The number of founders per vial was 10 inseminated ♀♀. Surviving flies were shaken each generation into new vials without mixing between vials. Extinction was measured each generation as the fraction vials which failed to produce adults. Table 2 gives the calculated LD 50 values. In addition gene frequencies were determined in this experiment in some of the environments, these are given in Table 3.

The change in gene frequencies (Table 1) in the cage populations, leading to an increase of the F-frequency in the initial low frequency populations and to a decrease in the initial high frequency populations, leading to values comparable to the base population, strongly suggests some kind of balancing selection. The frequency changes in all but one of the environments applied to the vial populations (Table 3) confirm this finding. In aethanol supplemen-

Table 3. Gene frequency changes in polymorphic vial populations.

| | | Initial frequency 0.20F | 0.80 F |
|---|---|---|---|
| environment | generation | frequency F-allele | frequency F-allele |
| 1 | 7 | 0.42 | 0.65 |
| 2 | 5 | 0.65 | 0.87 |
| 3 | 5 | 0.42 | 0.67 |
| 4 | 4 | 0.43 | 0.70 |
| 5 | 3 | 0.42 | 0.62 |

ted medium however an increase of the F-frequency occurred both in the 0.20 F and 0.80 F populations, which may lead to a higher equilibrium value compared with regular medium or maybe even loss of the S-allele.

Table 2 shows that the probability of survival, at least in some environments, depends on the genetic composition of the population. Monomorphic S-populations e.g. possess a relatively low survival on medium, supplemented with aethanol, a relatively high survival however is found in the high R.H. environment. The probability of survival of both types of polymorphic populations in relatively high under control and high temperature conditions and relatively low under low R.H. conditions. Polymorphism at the Adh locus may thus contribute to an increased probability of population survival in alternating, extreme environments.

Steiner, W.W.M. University of Hawaii, Honolulu, Hawaii. A simple and effective desiccating system.

Because the study of genotype-environmental relationships may offer a positive approach to problems of current evolutional interest, it seems appropriate to offer for use an inexpensive system to control relative humidity (R.H.) in the laboratory. It has been specifically designed for the study of individual reaction to the stress offered by low humidity environments.

Figure 1 illustrates the overall design of the system. The arrows indicate the direction of air flow. The air pump (Hush II, Metaframe Corp., Maywood, N.J.) has a double outlet. The

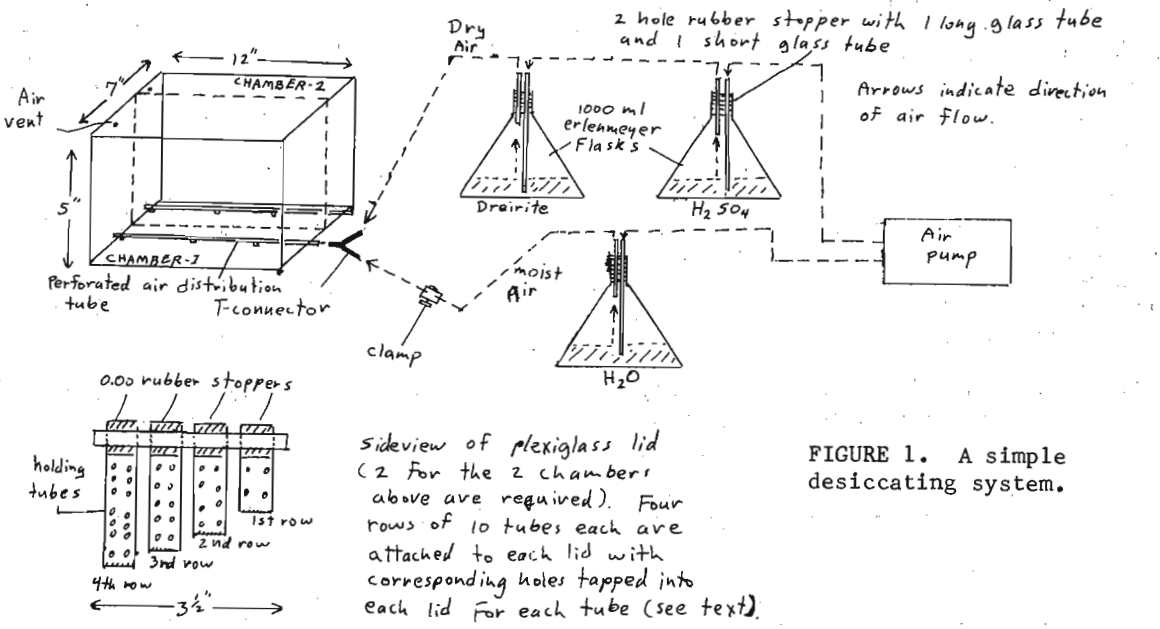



FIGURE 1. A simple desiccating system.

first outlet pumps air through two 1000 ml erlenmeyer flasks containing 300 ml of concentrated sulfuric acid ($H_2SO_4$) in the first and 300 grams of Drierite in the second. (In addition, a sodium bicarbonate sump may be attached to the line between these flasks to assure neutralization of $H_2SO_4$ byproducts.) These serve to remove all moisture from the air; the Drierite providing, in addition, a warning when the $H_2SO_4$ has reached it's saturation point (this usually occurs in 3-4 days, depending on the relative humidity of the ambient air). When this happens, another flask containing fresh $H_2SO_4$ may be quickly substituted for the first.

Where humidities above 0% R.H. are desired, the second outlet to the air pump may be used to pump air through a 1000 ml erlenmeyer flask containing 300 ml of distilled $H_2O$. This procedure enables saturation of the air with moisture. The clamp attached to the inlet line from the $H_2O$ reservoir can then be manipulated such that the correct ratio of dry to wet air can be obtained which will yield the desired relative humidity. A hygrometer-temperature indicator (Doral #118, Airguide Instr. Co., Chicago) can be placed within the chamber receiving the the conditioned air to record internal temperature and humidity. Two-hole rubber stoppers (no. 9) containing one 8 cm glass tube for the outlet hose and one 30 cm glass tube for the inlet hose are used to cap the erlenmeyer flasks. These should be carefully attached and taped down since pressure on the system can force these loose. The long glass tube should extend into the flasks contents.

The desiccation box consists of 2 narrow chambers holding approximately 2800 cc of air each. It and the cover plates for each chamber are constructed of 1/4 inch clear plexiglass. Air enters via a 1/4 inch plexiglass tube which runs the length of each chamber. This "distribution tube" has unequal numbers of perforations on the bottom with more located on the terminal end than near the entrance. It is designed to equalize air delivery throughout the chamber. Over all dimensions of the 2-chambered box with the lid on are 6.75 inches (wide) x 5.5 inches x 12.5 inches.

Attached to half of the lid (cover plate) of each chamber are four rows of 1/2 inch

plexiglass tubes (10 tubes to a row) with corresponding holes tapped through the lid for each tube. These tubes, which have an inside diameter of 3/8 inches, are designed to hold 1 to 5 flies each. These may have 1/16 inch perforations and have a fine mesh cloth screening glued to their bottom. The perforations and screening enable the tubes to equilibrate evenly with the rest of the chamber to the desired relative humidity and maximizes exposure of the organisms placed in them to the chambers internal environment.

These "holding" tubes are staggered in length. The rearmost (4th) row of tubes should be 3 1/2 inches in length, the next row of 10 tubes 3 inches in length, the next row 2.75 inches and the firstmost row 2.5 inches. This order enhances visual observation of the tubes since the flies, when they reach a state of exhaustive dehydration, will fall to the bottom. When this occurs, the rubber stopper (no. 0.00) plugging the aperture of the tube may be removed and an aspirator inserted through the corresponding hole in the cover plate to remove the organism. Stopcock grease can be used to seal the chamber lids, with a small vent located at the rear of the chambers serving to equalize air pressure on the sealed system. A second air system must be installed to operate the second chamber, which can also be used to house an undesiccated control.

I have found it takes 6-8 hours to equilibrate the chamber at 0% R.H. Somewhat longer time intervals may be required where equilibration is desired at higher humidities.

Table 1 lists the component parts necessary to build a 2-chambered desiccation box. Ethylene dichloride is used as a plexiglass cement and all line connections are made with 1/4 inch amber rubber tubing. All plexiglass components can usually be ordered precut from local distributors, but specifications must usually be in inches. Total cost of the system (Hawaii) is less than $30.00.

Table 1. Components necessary to build a 2-chambered desiccating system. A fine mesh screen of durable quality should be used to cover the bottom of each holding tube. Plexiglass components are of 1/4" thickness.

| ITEM | NO. PER SET | DIMENSIONS |
|---|---|---|
| base | 1 | 6 3/4" x 12" |
| endpieces | 2 | 6 3/4" x 5 1/4" |
| sidepieces + middlepiece | 3 | 5" x 12" |
| bars (for lid support) | 2 | 1/2" x 12" |
| blocks (for distrib. tube support) | 6 | 1/2" x 1/2" |
| lid (coverplate) | 2 | 3 1/4" x 12 1/2" |
| distribution tube | 2 | 1/4" x 13" |
| holding tubes: 1st row | 20 | 1/2" x 2 1/2" |
| 2nd row | 20 | 1/2" x 2 3/4" |
| 3rd row | 20 | 1/2" x 3 |
| 4th row | 20 | 1/2" x 3 1/2" |
| rubber stoppers | 80 | no. 0.00 |
| rubber stoppers | 6 | no. 9.0 (2-hole) |
| glass connecting-Y | 2 | 1/4" |
| amber rubber tubing | - | 1/4" x 144" |
| rubber tubing clamps | 2 | --------- |
| erlenmeyer flasks | 6 | 1000 ml capacity |
| glass tubing, short | 3 | 1/4" x 3" |
| glass tubing, long | 3 | 1/4" x 10-12" |

Acknowledgements: Dr. H.L. Carson's timely suggestion helped spark this note. Discussions with Ernest Kawasaki and Dr. Mike Kambysellis proved invaluable to the design of this system.

Research supported by NSF grant GB-23230 to the Island Ecosystems Stability and Evolution Subprogram of the International Biology Program, University of Hawaii.

Bennet-Clark, H.C. University of Edinburgh, Scotland. Microphone and pre-amplifier for recording courtship song of Drosophila.

The sound produced during wing vibration is a valuable specific character in Drosophila (1). This has been recorded by using elaborately sound insulated pressure sensitive microphones but this technique requires quiet conditions and makes observation very difficult.

In the course of an analysis of the acoustics of the song, it became apparent to me that with very small acoustic sources, the particle velocity in the sound wave was far larger than the pressure at very short ranges (2). The female fly's receptor, the antennal arista, responds to particle displacement. With this appreciation of the acoustic situation, I started using a displacement sensitive ribbon microphone to record courtship song.

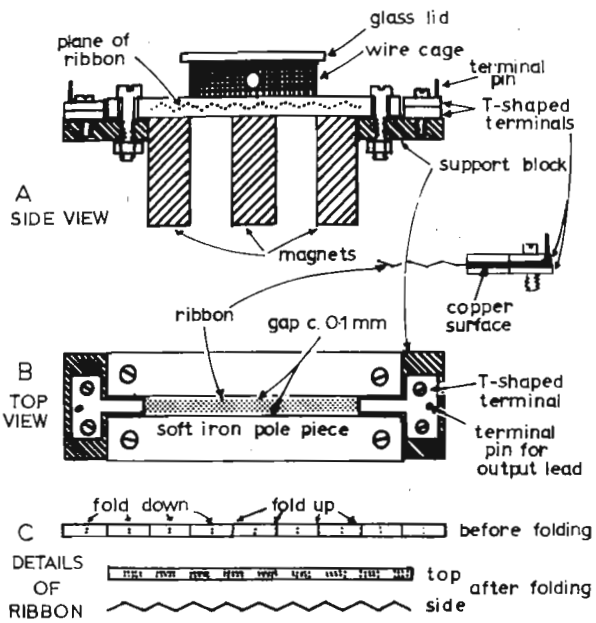



fig. 1. 50mm

RIBBON MICROPHONE CONSTRUCTION

One microphone used at Edinburgh is the Reslo ribbon, made by Reslosound, Spring Gardens, London, London Road, Romford, Essex England. This is unfortunately no longer in production though some stocks of the 30-50 ohm model remain at a price around $50.00. The outer protective grille is removed and the flies are placed in a wire cage 20 mm long, 10 mm wide and 10 mm high directly on top of the inner protective gauze about 2 mm above the ribbon diaphragm. Flies can be inserted if a hole is made in the side of the cage and observed if the top is made of glass (Fig. 1.A). Ribbon microphones are very sensitive to wind blast and care must be taken to avoid blowing out the ribbon when the flies are inserted.

If funds are low, an adequate ribbon microphone can be made for a few cents using horseshoe magnets and foil from a capacitor. The components are a pair of soft iron pole pieces about 50 mm long drilled at each end, a pair of Paxolin support blocks drilled and tapped to hold the pole pieces about 3 mm apart, four T shaped pieces of copper plated printed board held in pairs by screws to the support blocks, two or three small horseshoe magnets and a ribbon cut from the foil of a paper-foil capacitor.

The general assembly is shown in Fig. 1.A & B. The size of the pole pieces will depend on the size of the magnets but should not exceed 10 mm width. The ribbon is cut 3 mm wide from 5 µm thick aluminium foil and is concertinaed as in Fig. 1.C. After folding, the ribbon is held between the copper faces of the T shaped terminal clamps in the roughly aligned frame formed of the pole pieces and support blocks. The poles can then be adjusted to the minimum gap consistent with free movement of the ribbon. The top of the ribbon should be protected by a wire gauze. After assembly, the magnets are attached to the underside of the pole pieces with their north poles all on one and south poles all on the other pole piece.

The output is taken from the terminals either by a brass lug or a pin soldered to one of the copper foils. The shortest possible leads are used to connect to an

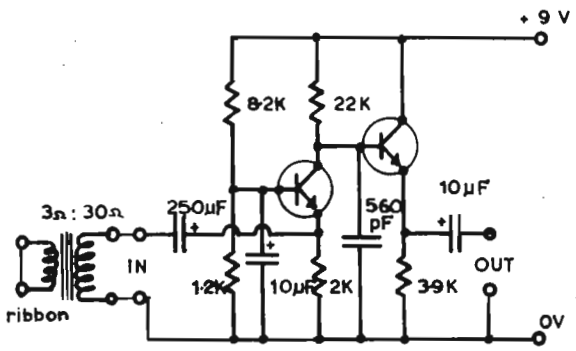



fig. 2. LOW IMPEDANCE PRE-AMPLIFIER

impedance matching transformer. The transformers used to couple the output transistors to the speaker in a personal radio are ideal.

The ratio should be from 1:3 and 1:10 step-up or to match 3 ohms and 30 to 300 ohms. The transformer is used to step down in the radio so the ribbon should be connected to the speaker or secondary winding and the primary to the pre-amplifier (Fig. 2).

The microphone can be tested by feeding from a signal generator when it will radiate sound or by feeding it into a high sensitivity oscilloscope, when it should respond to sound.

The output from a ribbon microphone is at low impedance and very low level. A suitable pre-amplifier with a gain of 200 and output impedance of 200 ohms is shown in Fig. 2 and will give an output compatible with most transistorised tape recorders. The first stage operates in the grounded base condition with low input impedance, high gain and high output impedance. This drives an emitter follower to give a low output impedance. Suitable transistors in Europe are BC 109 and in North America, types 2N 3904, 2N 3906, 2N 5087 etc. As current drain is about 1 mA, small 9 V batteries can be used.

The frequency response of the microphone is substantially flat between 50 to 70 Hz and about 5 to 7 kHz. The response of the pre-amplifier is flat within 1 dB over this band and falls at 6 dB per octave at the extremes. The background noise of microphone and pre-amplifier is equivalent to a sound of less than 40 dB so adequate signal to noise ratios can be obtained with many species of flies which produce sounds whose particle velocity is 70 to 100 dB at a range of 2 to 5 mm (2).

A detailed account of the theory and construction of ribbon microphones is given by Olson (3).

References: 1. Ewing, A.W. and H.C. Bennet-Clark 1968, Behaviour 31:288-301; 2. Bennet-Clark, H.C. 1971, Nature (London) 234:255-259; 3. Olson, H.F. 1957, Acoustical Engineering, xix+718, Van Nostrand, Princeton, N.Y.

Tartof, K.D., D. Tartof and M. Jones. Institute for Cancer Research, Philadelphia, Pennsylvania. A fly trap.

When fruit flies escape their culture bottles, they become a nuisance. Indeed, they are a danger to experiments involving sucrose gradients, cell cultures or sophisticated enzymology and can threaten the amicable relations between neighboring laboratories. Therefore, we sought to find a convenient trap for fruit flies that we could dispense to our neighboring cell culture and biochemical colleagues. It was imperative that the trap contain no yeast since yeast, like fruit flies, is anathema to those working with cell cultures. For reasons that we consider imprudent, we shall not discuss those events which led to our discovery of the ideal fly trap. Suffice is to say that a little (10-20 ml) Mogen David Concord Grape Wine in a half-pint bottle containing a Kleenex tissue and capped with a cardboard disc perforated with a 1-2 mm hole makes an extraordinarily efficient fly trap. The effectiveness of various vineyards and vintages awaits further exploration.

Novitski, E. University of Oregon, Eugene, Oregon. Instant triploids.

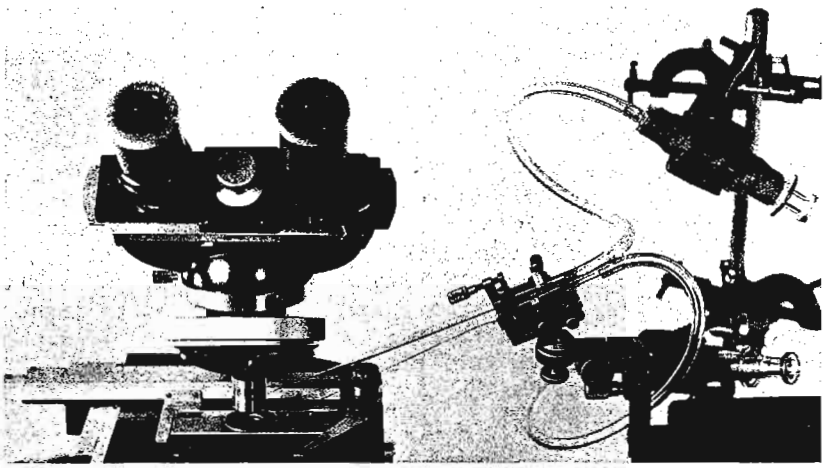
For many purposes the triploid condition is a convenient tool, as for the insertion of mutants into attached X's and other compound chromosomes. One convenient way of achieving this result quickly is to mate attached X females to males carrying compounds for both the second and third chromosomes. Since the males produce some small fraction of sperm that are essentially diploid with respect to the autosomes, along with a single X and a single fourth chromosome, a new triploid zygote may be produced when such a sperm fertilizes an egg with the attached X.

In a test of about 7000 C(1) females mated to C(2);C(3) males, 57 triploids were produced and the desired genetic change was achieved; i.e., the Bar locus (which had previously been attached to the tip of 2L, see DIS 47:91) was added to the tip of 2L on C(2L). It should be noted that 3N ♀♀ with compound autosomes are quite infertile and a stock is difficult to keep going, even for a few generations, so that this technique is useful only in a hit-and-run type of experiment, where the 3N condition is needed only ephemerally.

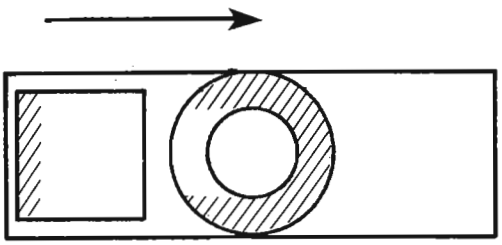
Hartmann-Goldstein, I.J. University of Sheffield, England. A simple method for isolating nuclei from individual salivary glands of Drosophila.

Drawing on the work of Boyd, Berendes and Boyd (1968), we have recently developed a quick method for isolating salivary gland nuclei from 3rd instar larvae. The procedure can be carried out in well under 5 min and permits investigation of nuclei from single individuals.

APPARATUS A microscope with a x10 phase objective is placed on the base plate of a Leitz micromanipulator equipped with a double instrument holder. Inserted in the holder are two glass needles made from tubing of I.D. 2 mm, O.D. 3mm. One of the needles is drawn to a length of about 100 mm, with a cleanly-broken, circular tip of O.D. about 150 µm. The dimensions of the other needle are not critical providing that its I.D. is not less than 300 µm. Both needles are connected by PVC tubing of I.D. 3 mm, wall thickness 0.75 mm, to 10 ml glass syringes mounted on a clamp stand. We have found standard syringes to give better results than those with screw-advance supplied as micromanipulator accessories. Our isolation chambers are made by sealing smoothly-polished brass washers of I.D. 13 mm, O.D. 25 mm and thickness 1.5 mm to clean slides with Gurr's Kronig Cement. We happen to have access to these materials; no doubt equally suitable chambers can be made in other ways. All glassware is siliconed.



METHOD Before starting the dissection, the coarser needle is filled with buffered Ringer made up of 1.1 mM $CaCl_2$, 0.11 M NaCl, 1.9 mM KCl, 2.3 mM $NaHCO_3$ and 0.08 mM $NaH_2PO_4$ at pH 7.3 and the drawn part of the finer needle is filled with detergent solution made up of 10% Tween 80, 10% sodium deoxycholate and buffered Ringer in the proportions 10:4:86. The position of the needles is adjusted so that only minor further adjustment is necessary once the gland has been placed on the microscope. Now the glands are dissected in buffered Ringer, transferred to a small drop of detergent in the center of an isolation chamber and placed on the microscope stage. The finer needle is pointed approximately at the middle of the gland and at right angles to it. Applying suction with the attached syringe, the gland is forced into the needle and the pressure is immediately reversed. In the large majority of cases this results in rupture of the gland and rapid expulsion of numerous nuclei, which settle on the slide (success depends largely on the speed with which the manipulations are carried out). With a minimum of delay the tip of the coarser needle is dipped into the solution and gradually, so as not to disturb the nuclei adhering to the slide, Ringer is added to dilute the detergent. Gland detritus can now be removed by gentle suction. More Ringer is added until a meniscus projects above the surface of the washer. The washer and the underside of the trailing edge of a 22 x 22 mm coverslip are coated with petroleum jelly as shown in the diagram (shaded areas). The coverslip is slowly slid over the washer so that the preparation is sealed, no air bubbles are trapped and the center of the coverslip remains clean. Glassware and solutions are pre-cooled to 3°C and the dissection is carried out at that temperature.



Some nuclei obtained by this method are deformed or have adhering cytoplasmic material. Almost all preparations, however, yield an abundant supply of clean, undamaged nuclei as judged by morphological criteria - we make no claims about submicroscopic or chemical purity. The preparations are suitable for study with x10 objectives; by replacing the slide at the

base of the isolation chamber with a coverslip they should also be suitable for work at higher powers, using an inverted microscope.

Reference: Boyd, J.B., H.D. Berendes and H. Boyd 1968, J. Cell Biol. 38:369-376.

Grossfield, J. New England Institute, Ridgefield, Connecticut. Gaseous anesthesia of Drosophila.

Carbon dioxide anesthetization has often been used to avoid some of the deleterious effects of etherization. While different species and mutant strains respond differently to ether, the use of $CO_2$ introduces the difficulty of $CO_2$ sensitivity with some strains. For such cases, anoxia produced by nitrogen, helium or argon is an effective narcotizing agent. Administration of pure $O_2$ does not narcotize. The major detail involved in using gaseous anesthetization is the use of a low flow rate through the regulator valve and short exposure time to avoid the dessicating effects of gas flow. If extended periods (over 5 minutes) of gas flow are required, then humidification of the gas is suggested.

Bricks, B.G. and J. Grossfield. New England Institute, Ridgefield, Connecticut. A timing control circuit for fluorescent lamps.

The spectral distribution and heat characteristics of fluorescent lamps are superior to incandescent bulbs for illuminating small chambers on a rigid light cycle in studies of circadian rhythms. Controlling the on-off cycle of small fluorescent lamps, such as those used to light a dissecting microscope stage, cannot be done by simply attaching the lamp to a timer. Such an arrangement will turn the lights off but will fail to turn them on, because a voltage greater than that of the AC line is required to initiate the gas discharge in the lamp. This increased voltage is normally provided by the induced EMF of the ballast inductor of the

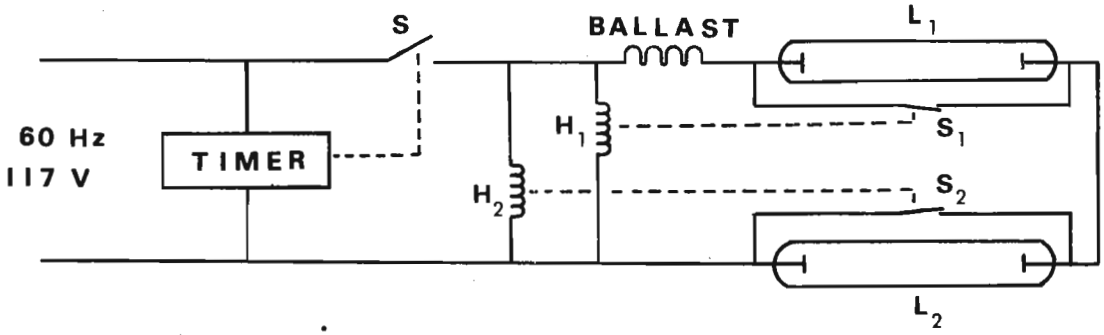



Fig. 1. $L_1$ and $L_2$ are fluorescent lamps connected in series with the usual ballast. $H_1$ and $H_2$ are heater filaments of the time delay relays, and $S_1$ and $S_2$ are the respective relay switches connected across each lamp. Switch S is internal to and activated by the timer clock mechanism. When S is closed, the lamps and heater filaments are connected to the AC line. Since $S_1$ and $S_2$ are shunting the lamps, sufficient current is drawn so that the induced EMF of the ballast will ignite the tubes. About 20 seconds later the heat generated by $H_1$ and $H_2$ causes $S_1$ and $S_2$ to open; all the current passes through $L_1$ and $L_2$ and lamp ignition is completed.

lamps. If the lamps and inductor are simply connected by a timer to the AC line, the induced EMF is insufficient owing to the small current drawn because of the high impedance of the lamps prior to breakdown of the gas. We present here a simple circuit (Fig. 1) employing thermal time-delay relays to overcome this difficulty. One relay (Amperite 115C20T) with contacts ($S_1$ and $S_2$) in the normally closed (NC) position is connected in parallel with each fluorescent lamp (GE F4T5-D). When the timer closes switch S the relays short circuit each tube, allowing sufficient current so that the induced EMF of the ballast can ignite the tubes. After ~20 seconds the relays open permitting full current through the lamps and producing maximum illumination. Relay tubes for two lamps will fit inside the case of an Intermatic model T101 24 hour timer.

Atherton, D. and J. Gall. Yale University New Haven, Connecticut. Salivary gland squashes for in situ nucleic acid hybridization studies.

In situ nucleic acid hybridization studies require chromosome preparations that can withstand air-drying, alkali denaturation, incubation at elevated temperatures, and conventional autoradiography, while still maintaining adequate morphological detail. We have found Drosophila polytene chromosomes among the most challenging objects so far tested. The following procedure provides permanently stained autoradiographs of in situ hybrids in which banding patterns are nearly as good as in the best temporary mounts.

Select a late third instar larva, which has crawled up the wall of the bottle and has stopped moving. Those with extruded spiracles and somewhat hardened cuticle often provide the best glands. Animals should be from well-fed, uncrowded bottles. D. melanogaster larvae should be raised at a low temperature (18°C).

Dissect out the salivary glands in Ringer's solution, including the duct but without the fat bodies. Transfer the glands on a needle to a small dish of freshly prepared ethanol-acetic fixative (3:1) and cover the dish. The glands will turn white immediately.

While the glands are fixing (2-5 min.) prepare a siliconized coverslip by removing it from ethanol and wiping it to remove all dust. On the coverslip place a small drop of 45% acetic acid (7-8µl). The volume is critical.

Remove the glands from the 3:1 with forceps and watch them under the dissecting microscope while the fixative evaporates. Just before the 3:1 is completely evaporated place the glands in the drop of 45% acetic acid and allow them to swell. Remove any pieces of dust from the coverslip. We prefer strong incident illumination against a black background.

Gently lower a subbed slide toward the preparation until it just touches the drop. Pick up the coverslip with the slide. Survey the preparation under low or medium powers of the phase contrast microscope (16X-40X). At this point there should be many intact cells as well as free, but intact nuclei.

Place the slide, coverslip up, between two pieces of bibulous paper. While holding one end of the slide, tap the area over the coverslip about ten times with the eraser end of a pencil. Tap very hard for melanogaster, less hard for some other species with larger nuclei, such as virilis and hydei. Do not make any special effort to prevent lateral motion of the coverslip. Under no circumstances should sustained pressure be applied to the coverslip area.

Check under the microscope. At this point cytoplasm should be flowing and the chromosomes should have broken out of the nuclear envelope. Tap again in the same manner as before, and recheck under the microscope. Continue until the chromosome arms are in the desired configuration.

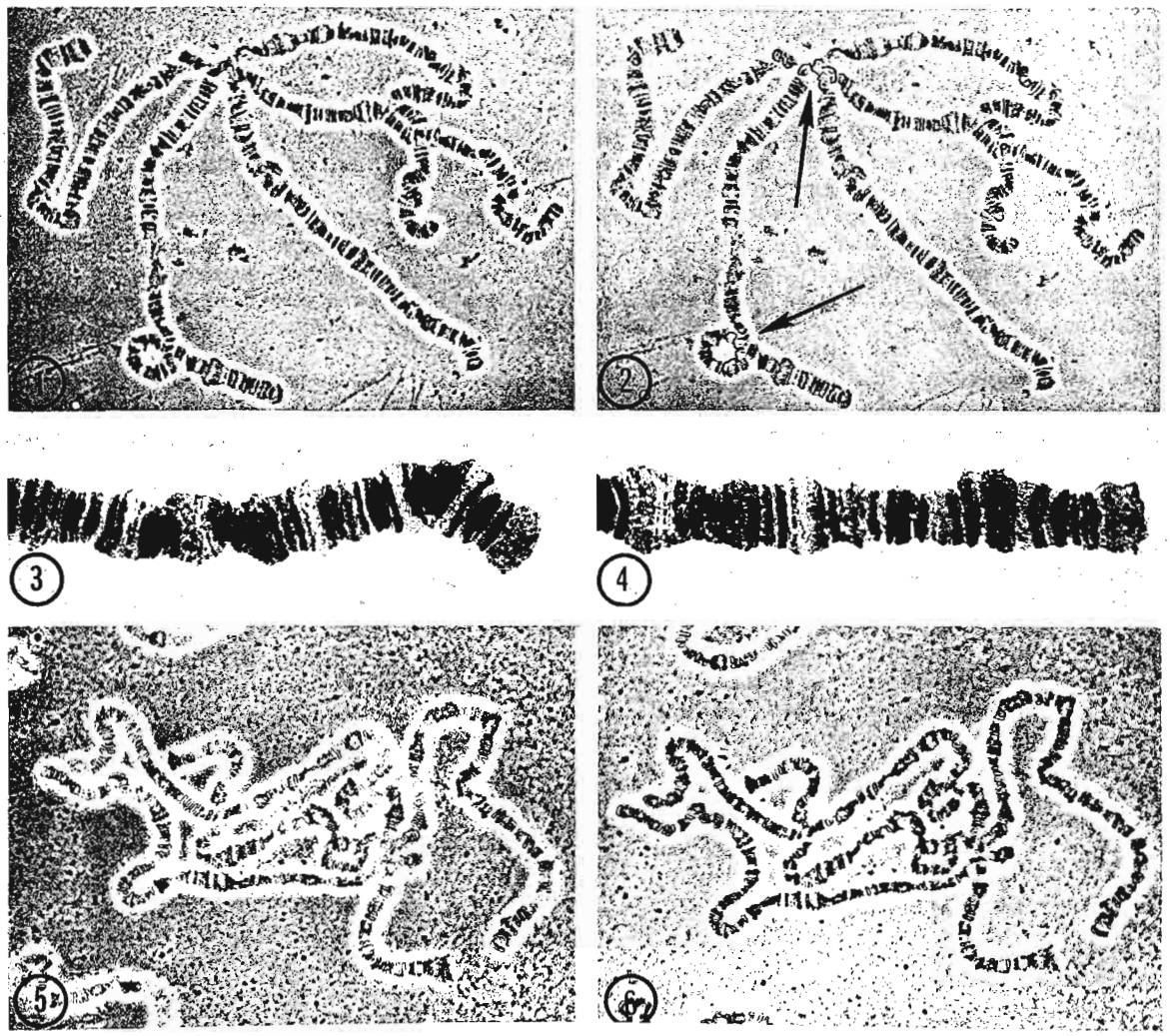
Note that the first taps should break the nuclear envelope, make the cytoplasm flow, and cause the chromosome arms to extend freely into the liquid. Subsequent taps eliminate the excess liquid and cement the chromosome arms into the desired position on the slide. Do not "squash" the preparation as is usually done for salivary gland material. Preparations which are adequately squashed for conventional cytology will not be flat enough for in situ hybridization, while those squashed flat enough by finger pressure will usually be badly distorted.

Flatten the preparation by placing the slide on a warming plate at 40-45°C. About once a minute check the preparation by observing under the 25X phase objective for a) cytoplasmic flow, b) the presence of heavy, refractile bands, and c) the condition of the chromocenter. A preparation should be left on the warming plate until the cytoplasm has stopped flowing, the heavier bands have been pressed until they are no longer refractile, and the chromocenter has not been pressed into a gray blob. These criteria apply specifically to observations made with the Zeiss 16X, 25X, or 40X Neofluar objectives. If during the monitoring stage the chromocenter begins to lose its morphology, remove the slide whether or not the cytoplasm is flowing and some bands are still refractile. Dust between the slide and coverslip will permit air to enter as the solution evaporates, thereby ruining the preparation.

Place the slide, coverslip down, on a flat block of dry ice in a covered container, and leave for 10-20 minutes. With a sharp razor blade in hand and an open Coplin jar of 100% ethanol standing by, quickly lift the slide from the dry ice, place the razor blade under the edge of the coverslip, flip off the coverslip and plunge the slide into the ethanol. Leave the slide in the ethanol for at least 12 hours in order to harden the tissue; it may be left for several days if desired. Remove the slide, stand it vertically, and allow it to dry. Check the slide under the phase contrast to see how good the preparation is. The best slides will have little cytoplasm, well spread chromosome arms, and all bands grey or black, even though viewed in air without a coverslip or mounting medium. Adequate preparations will have

some refractile bands. In these the alkali denaturation step during the in situ hybridization procedure will cause some swelling.

For in situ hybrids proceed as described in Gall and Pardue (Methods in Enzymology, XXI,




Figs. 1 and 2. Chromosomes of D. melanogaster before (1) and after (2) treatment with 0.07N NaOH for 2 minutes to denature the DNA. This preparation demonstrates the ideal degree of flattening, and shows only minimal damage after the alkali treatment (arrows in (2) indicate "ballooning" in the chromocenter and at regions of overlaps). Photographed as a dry preparation without mounting medium or coverslip. Because of the great difference in refractive index between the specimen and air, only the very flattest preparations will show gray or black bands. Zeiss 25X Neofluar phase, X310.

Figs. 3 and 4. Two examples of chromosome 2L from a preparation which had been air dried, denatured with 0.07N NaOH, stained with Giemsa, and permanently mounted in Permount. Fig. 3 is from the nucleus shown in (1) and (2). Zeiss 100X planapochromat, bright field, X1,200.

Figs. 5 and 6. Chromosomes before (5) and after (6) 0.07N NaOH to demonstrate the appearance of an adequate, but not ideal squash. Note the overall high refractility with many bands brighter than background. As in (1) and (2) the contrast is slightly lower after alkali, presumably due to extraction of material. Photography exactly as in (1) and (2). X330.

470-480, 1971). However, omit step 9 of that paper (acid treatment to remove histones). Acid treatment reduces the amount of hybrid formed and interferes with good staining.

The procedure outlined here can, of course, be used for routine stained preparations without hybridization. In this case, the flatness of the chromosomes is less critical. Giemsa stain provides excellent contrast of bands and demonstrates nucleolar DNA clearly. It may be preceded by RNase or a brief acid hydrolysis to reduce cytoplasmic staining (1N HCl, 60°C, 5 min.).

Materials needed: Siliconized coverslips: Place coverslips (18 mm square, #1 thickness) in a 1% Siliclad solution for a few moments, rinse in distilled water, and allow to dry on paper toweling. Store the coverslips dry and do not worry about water marks. Just before needed, place a few coverslips in 95% ethanol. Subbed slides: Clean new slides in detergent, rinse well with tap water, and place for a few moments in a solution of gelatin and chrome alum (0.1% gelatin in 0.01% chromium potassium sulfate). Dry slides vertically in a slide carrier, and store in a dust-free box. Solutions: Immediately before use, prepare "3:1" fixative, consisting of 3 parts absolute ethanol + 1 part glacial acetic acid. Keep stock solutions of 45% acetic acid, and any convenient insect Ringer's solution.

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## PERSONAL AND LABORATORY NEWS

J.L. Mensua, formerly Assistant Professor in the Department of Genetics, University of Barcelona, has accepted a charge of Professor in the Autonomous University of Barcelona (Bellaterra), where he is head of a new Laboratory of Genetics.

Heinz Tobler has left the University of Zürich and is now Full Professor and Director of the Institute of Zoology, University of Fribourg, 1700 Fribourg, Switzerland. He heads a group working on problems of determination and differentiation of imaginal discs.

Ross MacIntyre will be on sabbatic leave at the Department of Genetics, University of California at Davis, Davis, California from February through July, 1973 (from Cornell University, Ithaca, New York).

The Drosophila Species Collection at the University of Texas at Austin still exists, and stocks are available upon request from either M.R. Wheeler or R.H. Richardson. Although support is insufficient for long-term survival, efforts are still being made to obtain additional support necessary to maintain the collection. With tightening of research funds, the financial support for the facility was no longer possible as a component of a research project. With the enthusiastic and invaluable world-wide support of numerous Drosophila workers, about 1/3 of the N.I.H. approved support level has been shifted to the N.I.H. Research Resources Branch. Until August 31, 1973, sufficient additional interim support for temporary maintenance of the facility is being supplied from local and piecemeal support. In the meantime, N.S.F. has reviewed a proposal to share support with N.I.H., but there were no funds for new museum support and N.S.F. has not been able to allocate funds to this facility. Unless additional funds become available by next August the facility will be lost, but optimism still prevails.

Ravi Dutt Narda, 32, former Associate Professor of Genetics, Department of Genetics, Punjab Agricultural University, Ludhiana, India, died on August 23, 1971. Dr. Narda did his M.Sc. in 1963 and Ph.D. in 1966 from Punjab University, Chandigarh, India, under the supervision of Dr. G.P. Sharma. He joined the Department of Genetics, Punjab Agricultural University, in 1966 as Assistant Professor and was promoted to Associate Professor in 1970. His research work included behavioural genetics, speciation and mutation studies in Drosophila.

Klug, W.S. and D. Weller. Wabash College, Crawfordsville, Indiana. A computer simulation of a Drosophila transmission genetics experiment.

Many general genetics courses utilize some form of a Drosophila transmission genetics experiment in the laboratory. Such an experiment can take on a number of forms and many provide the student with an unknown mutant stock. The student's exercise is to localize the unknown gene on its appropriate linkage group. This can be accomplished in several ways. The mutant females may be crossed with males from a multiply marked stock containing a series of dominant genes such as Cy/Pm;H/Sb (Strickberger, 1962, p. 53). The cross is then carried to an $F_2$ generation after selecting $F_1$ males and performing a back cross to mutant females. Alternatively, two simultaneous but separate crosses may be made where mutant females are mated to males marked with a recessive gene on either of the two major autosomes (King, 1967 p. 429) and carried to the $F_2$ generation.

In either case, if the crosses are properly executed, the unknown gene may be unequivocally located on its proper chromosome. Such an exercise may precede a mapping exercise, a prediction of the locus and finally a confirming allelism test.

This paper describes a computer program which simulates the localization of the linkage group of an unknown recessive mutant gene by the previously described "two cross" method. Such a program may be utilized as an effective teaching technique in several ways. It may be applied during the initial phases of the actual laboratory experiment to reinforce the students' understanding prior to their data collection or when time is the essence or no laboratory is included in the course, it can effectively replace the laboratory work.

The simulation was written in RSTS (Resource Timesharing System) BASIC-PLUS, an advanced BASIC language used by the PDP-11 family of computers. For convenience of execution, it is divided into four parts: 1) an instructor's program, 2) the $F_1$ exercise, 3) the $F_2$ exercise, and 4) a Chi square analysis.

The instructor's program is used to set up an information file for any number of unknown mutants. The file contains an identification number, the linkage group, and a phenotypic description for each unknown.

The $F_1$ program randomly selects an unknown from this file each time the program is run. The readout provides the user with the identification number, the phenotypic description, and $F_1$ data for two crosses between the unknown female and a recessive marker male from the II and III linkage group. The 300-500 offspring for each cross are number keyed for all eight possible male and female phenotypes and the data code numbers are printed in rows of varying length which finally assume the shape of a half-pint milk bottle. Since all of the unknowns are recessive, the $F_1$ results are of one of two types. If the unknown is sex-linked, only wild type females and unknown mutant males result. If the unknown is an autosomal gene, all $F_1$ offspring for both crosses are wild type. In the former case, the gene has been localized on the X-chromosome.

If the unknown mutant is determined to be autosomal, the user can then call up the $F_2$ program and input the identification number of his unknown. This program provides the $F_1 \times F_1$ data for both of the original crosses, using the same number coding system for the phenotypes and printing the data in the same bottle shape. These data, when analyzed, provide the necessary information for the determination of the linkage group in question.

Both programs are designed to simulate the normal probability distributions encountered in an actual experiment and therefore give different numbers of offspring and different random variations from the ideal phenotypic ratio each time the program is run. These deviations from the ideal may be analyzed with the separate Chi square program which can provide $\chi^2$ values for any data analysis, including actual laboratory work.

We have found this to be a valuable experience for general genetics students. The entire three programs may be completed in 15 minutes and can be run any number of times by the same student. In addition to simulating the transmission experiment it assures that all students at this educational level will have at least an introduction to the use of the computer.

If you have a sincere interest in applying such a simulation to your genetics teaching, we will be pleased to provide you with the programs as written in BASIC-PLUS for use with our PDP-11/20 timesharing system.

References: King, R.C. 1967, Genetics, Oxford, N.Y.; Strickberger, M. 1962, Experiments with Drosophila, John Wiley, N.Y.

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Eaves, L.J. Birmingham, England
Ebitani, N. Tokyo, Japan
Eggert, R. Davis, California
Ehrlich, E. Eugene, Oregon
Ehrman, L. Purchase, New York
Eiche, A. Stockholm, Sweden
Elens, A. Namur, Belgium
Elgin, S. Pasadena, California
Elmer, W.A. Atlanta, Georgia
Englert, D.C. Carbondale, Illinois
English, D.S. Flagstaff, Arizona
Engstrom, L. Muncie, Indiana
Enns, R.E. Pasadena, California
Erickson, J. Bellingham, Washington
Erk, F.C. Stony Brook, New York
Espinós, A. Valencia, Spain
Evans, W. Bellingham, Washington
Ezell, S.D. Madison, Wisconsin
Fabergé, A.C. Austin, Texas
Fahmy, M.J. Chalfont St. Giles, England
Fahmy, O.G. Chalfont St. Giles, England
Falk, R. Jerusalem, Israel
Farmer, J.L. Provo, Utah
Farnsworth, N.W. Buffalo, New York
Fattig, W.D. Birmingham, Alabama
Feijen, H.R. Limbe, Malawi
Felix, R. Mexico City, Mexico
Fenner, H. Santiago, Chile
Feuerbach-Mravlag, H. Vienna, Austria
Figueroa, J.A. Chicago, Illinois
Fischer, B. Bochum-Querenburg, Germany
Fitz-Earle, M. Vancouver, Canada
Fleuriet, A. Clermont-Ferrand, France
Fontdevila, A. Davis, California
Forbes, C. Moscow, Idaho
Forrest, H.S. Austin, Texas
Fouillet, P. Villeurbanne, France
Fowler, G.L. Düsseldorf, Germany
Fox, A.S. Madison, Wisconsin
Fox, D.J. Knoxville, Tennessee
Fox, D.P. Aberdeen, Scotland
Frankel, A. Iowa City, Iowa
Frankham, R. Sydney, Australia
Franklin, I.R. Sydney, Australia
Friedman, T.B. Bethesda, Maryland

Friedman, L.D. St. Louis, Missouri
Friedman, S. New Haven, Connecticut
Fritz-Niggli, H. Zürich, Switzerland
Frutos, R. Barcelona, Spain
Fuchs, M.S. Notre Dame, Indiana
Fujii, H.M. Fukuoka, Japan
Fukatami, A. Sakado-Machi, Japan
Fullilove, S.L. Austin, Texas
Fuscaldo, K.E. Philadelphia, Pennsylvania
Futch, D.B. San Diego, California
Fuyama, Y. Tokyo, Japan
Gabay, S.J. Urbana, Illinois
Gale, S.S. Birmingham, England
Gall, J.G. New Haven, Connecticut
García, P. Barcelona, Spain
Garcia-Bellido, A. Madrid, Spain
Garen, A. New Haven, Connecticut
Garen, S. New Haven, Connecticut
Gay, H. Ann Arbor, Michigan
Geer, B.W. Galesburg, Illinois
Gelbart, W. Storrs, Connecticut
Gerace, L. Baltimore, Maryland
Gerresheim, F. München, Germany
Gersh, E.S. Philadelphia, Pennsylvania
Gethmann, R.C. Catonsville, Maryland
Gibson, J.B. Cambridge, England
Gill, K.S. Ludhiana, India
Gill, R.W. Riverside, California
Glass, H.B. Stony Brook, New York
Glätzer, K.H. Düsseldorf, Germany
Gloor, H. Geneva, Switzerland
Godbole, N.N. Poona, India
Gonzalez, F.W. Upton, New York
González, R. Barcelona, Spain
Gottlieb, F.J. Pittsburgh, Pennsylvania
Götz, K.G. Tübingen, Germany
Gould, A.B. Newark, Delaware
Goux, J.M. Paris, France
Grabicki, E. New Haven, Connecticut
Graf, U. Zürich, Switzerland
Grant, B.S. Williamsburg, Virginia
Green, M.M. Davis, California
Grell, E.H. Oak Ridge, Tennessee
Grell, R.F. Oak Ridge, Tennessee
Grossfield, J. Purchase, New York
Grossman, A. Jerusalem, Israel
Grünberg, H. London, England
Guest, W.C. Fayetteville, Arkansas
Gupta, R. Calcutta, India
Guzmán de Jiménez, J. Mexico City, Mexico
Haapala, O. Turku, Finland
Hackman, W. Helsinki, Finland
Hadorn, E. Zürich, Switzerland
Haendle, J. München, Germany
Halfer, C. Milan, Italy
Hale, G. New York, New York
Hall, J. Pasadena, California
Hall, L. Vancouver, Canada
Hama, H. Chiba, Japan
Hanks, G.D. Gary, Indiana
Hannah-Alava, A. Turku, Finland
Hanson, T.E. Pasadena, California
Hara, K. Fukuoka, Japan
Harford, A.G. New Haven, Connecticut
Hardy, D.E. Honolulu, Hawaii
Hardy, R.W. San Diego, California
Harrison, B.J. Norwich, England
Hartman-Goldstein, I.J. Sheffield, England
Hauschteck-Jungen, E. Zürich, Switzerland
Hay, D.A. Bundoora, Australia
Heath, C.E. Knoxville, Tennessee
Hedrick, P.W. Lawrence, Kansas
Heed, W. Davis, California
Heisenberg, M. Tübingen, Germany
Hengstenberg, R. Tübingen, Germany
Hennig, W. Tübingen, Germany
Herskowitz, I. New York, New York
Hess, O. Düsseldorf, Germany
Hexter, W.M. Amherst, Massachusetts
Hildreth, P.E. Charlotte, North Carolina
Hillman, R. Philadelphia, Pennsylvania
Hinton, C.W. Wooster, Ohio
Hiraizumi, Y. Austin, Texas
Hiroyoshi, T. Osaka, Japan
Hochman, B. Knoxville, Tennessee
Hodgetts, R.B. Edmonton, Canada
Heokstra, R.F. Haren, Netherlands
Hoenigsberg, H.F. Bogotá, Colombia
Hollander, W.F. Ames, Iowa
Hollingsworth, M.J. London, England
Hollis, R.J. Williamsburg, Virginia
Holm, D. Vancouver, Canada
Holmgren, P. Umeå, Sweden
Honda, Y. Nagasaki, Japan
Hooper, G.B. Poughkeepsie, New York
Horn, E. Zürich, Switzerland
Houze, C. Duarte, California
Huang, S.L. Madison, Wisconsin
Hubby, J.L. Chicago, Illinois
Hunt, D.M. London, England
Huth, A.C. Tübingen, Germany
Ikeda, H. Tokyo, Japan
Ikeda, K. Duarte, California
Illmensee, K. München, Germany
Imberski, R.B. College Park, Maryland
Inoue, A. Nagasaki, Japan
Ives, P.T. Amherst, Massachusetts
Iyama, S. Misima, Japan
Jacobs, M.E. Goshen, Indiana
Jacobs, P.A. San Diego, California
Jacobson, A.G. Austin, Texas
Janning, W. Münster, Germany
Jeffery, D.E. Provo, Utah
Jha, A.P. Bhagalpur, India
Jinks, J.L. Birmingham, England
Johannisson, R. Düsseldorf, Germany
Johnsen, R.C. Garden City, New York
Johnson, F.M. Raleigh, North Carolina
Johnson, J.H. Chicago, Illinois
Johnston, J.S. Austin, Texas
Jones, D.A. Birmingham, England
Jones, G.H. Birmingham, England
Jong, G. de Utrecht, Netherlands
Jonker, F.H. Haren, Netherlands

Jousset, F.X. St. Christol les Alès, France
Judd, B.H. Austin, Texas
Jungen, H. Zürich, Switzerland
Kaji, S. Kobe, Japan
Kale, P.G. Varanasi, India
Kalicki, H. Garden City, New York
Kalisch, W.-E. Bochum-Querenburg, Germany
Kambysellis, M.P. New York, New York
Kamra, O.P. Halifax, Canada
Kanehisa, T. Kobe, Japan
Keneko, A. Sapporo, Japan
Kang, M.-J. Seoul, Korea
Kang, S.-J. Seoul, Korea
Kang, Y.J. Seoul, Korea
Kang, Y.S. Seoul, Korea
Kankel, D.R. Pasadena, California
Kaplan, M.L. New York, New York
Kaplan, W.D. Duarte, California
Karlik, A. Vienna, Austria
Kastritsis, C.D. Thessaloniki, Greece
Kaufman, T. Vancouver, Canada
Kaufmann, B.N. Philadelphia, Pennsylvania
Kaufmann, B.P. Ann Arbor, Michigan
Kawabe, M. Kobe, Japan
Kawaharada, M. Nagasaki, Japan
Kawanishi, M. Misima, Japan
Kearsey, M.J. Birmingham, England
Keller, E.C. Morgantown, West Virginia
Kercher, M.D. Lexington, Kentucky
Kezer, J. Eugene, Oregon
Khishin, A.F. Assuit, Egypt
Kikkawa, H. Osaka, Japan
Kim, K.W. Kwangju, Korea
Kimura, M. Misima, Japan
King, R. Evanston, Illinois
Kitagawa, O. Tokyo, Japan
Klug, W.S. Crawfordsville, Indiana
Knaap, A.G.A.C. Leiden, Netherlands
Kobayashi, Y. Hiroshima, Japan
Kobel, H.R. Geneva, Switzerland
Koch, P. Ketzerbach, Germany
Koref-Santibañez, S. Santiago, Chili
Korge, G. München, Germany
Kosuda, K. Tokyo, Japan
Kothari, R.M. Poona, India
Kramers, P.G.N. Leiden, Netherlands
Krause, E. South Orange, New Jersey
Kress, H. München, Germany
Krimbas, C. Athens, Greece
Krivshenko, J.D. Rochester, New York
Kroman, R.A. Long Beach, California
Krunić, M. Belgrade, Yugoslavia
Kubli, E. Zürich, Switzerland
Kumagai, K. Sakado-Machi, Japan
Kunz, W. Düsseldorf, Germany
Kuo, G. New Haven, Connecticut
Kuroda, Y. Misima, Japan
Kurokawa, H. Tokyo, Japan
Laird, C. Seattle, Washington
Lakhotia, S.C. Burdwan, India
Lakovaara, S. Oulu, Finland
Lamb, M.J. London, England
Lambertsson, A. Umeå, Sweden
Lankinen, P. Oulu, Finland
La Pushin, R. Houston, Texas
Latter, B.D.H. Sydney, Australia
Laughnan, J.R. Urbana, Illinois
Lawrence, M.J. Birmingham, England
Lechien, J. Namur, Belgium
Lechner, J.F. Philadelphia, Pennsylvania
Lee, C.C. Seoul, Korea
Lee, C.S. Austin, Texas
Lee, C.S. Seoul, Korea
Lee, T.J. Seoul, Korea
Lefevre, G. Jr. Northridge, California
Legay, J.M. Lyon, France
Leigh, B. Leiden, Netherlands
Leister, F. Baltimore, Maryland
Leonard, J. Purchase, New York
Levins, R. Chicago, Illinois
Lewis, A. Purchase, New York
Lewis, E.B. Pasadena, California
Lewontin, R.C. Chicago, Illinois
L'Helias, C. Gif-sur-Yvette, France
L'Héritier, Ph. Clermont-Ferrand, France
Libion, M. Namur, Belgium
Lieb, E. München, Germany
Lifschytz, E. San Diego, California
Lindsley, D.L. San Diego, California
Lints, C. Heverlee, Belgium
Lints, F.A. Heverlee, Belgium
Llorens, A. Barcelona, Spain
LoCasio, N. Buffalo, New York
Lokki, J. Helsinki, Finland
Long, T.C. Chapel Hill, North Carolina
Louis, M. Gif-sur-Yvette, France
Loukas, M. Athens, Greece
Lowy, P.H. Pasadena, California
Lucchesi, J.C. Chapel Hill, North Carolina
Luce, W.M. Urbana, Illinois
Lüers, H. Berlin, Germany
Lumme, J. Oulu, Finland
Lüning, K.G. Stockholm, Sweden
MacBean, I.T. Bundoora, Australia
Machová, H. Brno, Czechoslovakia
MacIntyre, R. Ithaca, New York
Maeda, Y. Kobe, Japan
Magalhães, L.E. São Paulo, Brazil
Magnusson, J. Stockholm, Sweden
Mainx, F. Vienna, Austria
Majumdar, S.K. Easton, Pennsylvania
Mange, A. Seattle, Washington
Manna, G.K. Kalyani, India
Manning, B. Ann Arbor, Michigan
Marien, D. New York, New York
Marinković, D. Belgrade, Yugoslavia
Maroni, G. Madison, Wisconsin
Marques, E.K. Pôrto Alegre, Brazil
Martin, A.O. Cleveland, Ohio
Martinez, M.N. Pôrto Alegre, Brazil
Maruyama, T. Misima, Japan
Massie, H.R. Utica, New York
Mather, E.P. Aberdeen, Scotland
Mather, K. Birmingham, England

Mather, W.B. Brisbane, Australia
Matsubara, T. Nagasaki, Japan
Mazar Barnett, B. Buenos Aires, Argentina
McCarron, M. Storrs, Connecticut
McCarthy, P.C. New Wilmington, Pennsylvania
McCormack, M.K. Minneapolis, Minnesota
McCrady, E. Greensboro, North Carolina
McKenzie, J.A. Bundoora, Australia
McMurtrey, M. Houston, Texas
Meer, J. van der Utrecht, Netherlands
Megna, F. Naples, Italy
Meltzer, P. Cambridge, England
Mendelson, D. Leiden, Netherlands
Mercader, J. Mexico City, Mexico
Merrell, D.J. Minneapolis, Minnesota
Merriam, J.R. Los Angeles, California
Merritt, R. Rochester, New York
Messerschmid, V. München, Germany
Metcalfe, J.A. York, England
Mettler, L.E. Raleigh, North Carolina
Meyer, G.F. Tübingen, Germany
Meyer, H.U. Madison, Wisconsin
Micheli, A. Rome, Italy
Miglani, G.S. Ludhiana, India
Milkman, R. Iowa City, Iowa
Miller, D.D. Lincoln, Nebraska
Millington-Ward, A.M. Leiden, Netherlands
Minamori, S. Hiroshima, Japan
Minato, K. Misima, Japan
Mindek, G. Zürich, Switzerland
Mitchell, H.K. Pasadena, California
Mitra Nivedita, Miss Calcutta, India
Mittler, S. DeKalb, Illinois
Mizobuchi, K. Chiba, Japan
Moffitt, S. Oxford, England
Mohler, D. Iowa City, Iowa
Moisand, R. Buffalo, New York
Momma, E. Sapporo, Japan
Monclús, M. Barcelona, Spain
Montalenti, G. Rome, Italy
Montelius, I. Stockholm, Sweden
Montell, I. Umeå, Sweden
Moree, R. Pullman, Washington
Mori, S. Nagasaki, Japan
Moriwaki, A. Misima, Japan
Morrison, W.J. Ithaca, New York
Mortensen, M. Copenhagen, Denmark
Moskwinski, T. Notre Dame, Indiana
Mosna, G. Milan, Italy
Mossige, J. Oslo, Norway
Mourad, A.M. Alexandria, Egypt
Muckenthaler, F.A. Albany, New York
Mukai, P. Raleigh, North Carolina
Mukherjee, A.S. Calcutta, India
Muñoz, E.R. Buenos Aires, Argentina
Murakami, A. Misima, Japan
Murata, M. Chiba, Japan
Murnik, M.R. Macomb, Illinois
Myszewski, M.E. Des Moines, Iowa
Nakai, S. Chiba, Japan
Nakai, S. Osaka, Japan
Nakao, Y. Hiroshima, Japan
Nakashima-Tanaka, E. Sakai, Japan
Narise, S. Sakado-Machi, Japan
Narise, T. Sakado-Machi, Japan
Nash, D. Edmonton, Canada
Nash, W.G. Washington, D.C.
Nawa, S. Misima, Japan
Neeley, J.C. Portland, Oregon
Nelson, N. Seattle, Washington
Nicoletti, B. Rome, Italy
Nicolosi, R. J. Utica, New York
Nigon, V. Lyon, France
Nilsson, B. Stockholm, Sweden
Nix, C.E. Oak Ridge, Tennessee
Nørby, S. Copenhagen, Denmark
Nöthel, H. Berlin, Germany
Nöthiger, R. Zürich, Switzerland
Nouand, D. Paris, France
Novitski, E. Eugene, Oregon
Nuez, F. Valencia, Spain
Obinata, M. New Haven, Connecticut
Oftedal, P. Oslo, Norway
Ogah, F. Ithaca, New York
Ogaki, M. Sakai, Japan
Ogita, Z. Osaka, Japan
Ohba, S. Tokyo, Japan
Ohkubo, K. Nagasaki, Japan
Ohnishi, S. Misima, Japan
Ohring, L. Umeå, Sweden
Ohta, T. Misima, Japan
Oikarinen, A. Oulu, Finland
Oishi, K. Kobe, Japan
Okada, T. Tokyo, Japan
Oksala, T.A. Turku, Finland
Olivieri, G. Rome, Italy
Olofsson, E. Umeå, Sweden
Ondrej, M. Prague, Czechoslovakia
Ortiz, E. Madrid, Spain
Oshima, C. Misima, Japan
Oster, I.I. Bowling Green, Ohio
Oster, P. Bowling Green, Ohio
Ostrowski, R.S. Charlotte, North Carolina
Ouweneel, W.J. Utrecht, Netherlands
Paik, Y.K. Honolulu, Hawaii
Pak, W.L. Lafayette, Indiana
Palabost, L. Paris, France
Paribi, G. Naples, Italy
Park, M.S. Kwangju, Korea
Parker, D.R. Riverside, California
Parry, D. Seattle, Washington
Parshad, R. Chandigarh, India
Parsons, P.A. Bundoora, Australia
Paterson, H.E. Nedlands, Australia
Pavlovsky, O. Davis, California
Paxman, G.J. Lancaster, England
Paz, C. Buenos Aires, Argentina
Peacock, W.J. Canberra, Australia
Peers, E. New York, New York
Pelecanos, M. Patras, Greece
Pentzos-Daponte, A. Thessaloniki, Greece
Perekovic, V. Edmonton, Canada
Perkins. J.M. Birmingham, England
Persson, K. Umeå, Swedenien

Petit, C. Paris, France
Petitpierre, E. Barcelona, Spain
Petri, W.H. Cambridge, Massachusetts
Petrovich, S.B. Catonsville, Maryland
Phillips, J. Austin, Texas
Picard, G. Clermont-Ferrand, France
Pierce, S. Chapel Hill, North Carolina
Pinsker, W. Vienna, Austria
Pipkin, S.B. Washington, D.C.
Plaut, W.S. Madison, Wisconsin
Plus, N. St. Christol les Ales, France
Policansky, D. Chicago, Illinois
Poodry, C. Vancouver, Canada
Portin, P. Turku, Finland
Postlethwait, J.H. Eugene, Oregon
Potter, J.H. College Park, Maryland
Poulson, D.F. New Haven, Connecticut
Powell, J.R. New Haven, Connecticut
Prakash, S. Rochester, New York
Prendergast, L. San Diego, California
Presley, J.M. Davis, California
Prevosti, A. Barcelona, Spain
Prout, T. Riverside, California
Puro, J. Turku, Finland
Pyati, J. Tempe, Arizona
Quinn, W. Pasadena, California
Rae, P.M.M. Tübingen, Germany
Rahman, S.M.Z. Bhagalpur, India
Rai-Chaudhuri, S.P. Calcutta, India
Ramel, C. Stockholm, Sweden
Rasmuson, B. Umeå, Sweden
Rasmuson, M. Umeå, Sweden
Ratnayake, W.E. Nugegoda, Ceylon
Ratty, F.J. San Diego, California
Ray, C. Atlanta, Georgia
Razzini, A. Milan, Italy
Reddi, O.S. Hyderabad, India
Relton, J.M. Sheffield, England
Remondini, D.J. Spokane, Washington
Rendel, J.M. Sydney, Australia
Ribó, G. Barcelona, Spain
Richard, J. Berkeley, California
Richardson, R.H. Austin, Texas
Rico, M. Valencia, Spain
Rinehart, R.R. San Diego, California
Rivera, M.L. Barcelona, Spain
Robbins, L.G. East Lansing, Michigan
Roberts, C.F. Leicester, England
Roberts, D.B. Oxford, England
Roberts, P. Corvallis, Oregon
Robertson, E. Edinburgh, Scotland
Robertson, F.W. Aberdeen, Scotland
Röck, B. Tübingen, Germany
Rodinò, E. Padova, Italy
Romero, I.F. Bogotá, Colombia
Rose, R.W. Glenside, Pennsylvania
Ruderer-Doschek, E. Vienna, Austria
Rudkin, G. Philadelphia, Pennsylvania
Runger, E. Geneva, Switzerland
Russell, M.R. Edmonton, Canada
Rutherford, P. Aberdeen, Scotland
Ryman, N. Stockholm, Sweden
Sakaguchi, B. Fukuoka, Japan
Sakai, K.I. Misima, Japan
Sakoyama, Y. Osaka, Japan
Salai, L. Albany, New York
Samis, H.V. Utica, New York
Sanders, T.G. Princeton, New Jersey
Sandler, L. Seattle, Washington
Sang, J.H. Brighton, England
Sanjeeva Rao, M. Hyderabad, India
Sankaranarayanan, K. Leiden, Netherlands
Sappington, J. Ann Arbor, Michigan
Sarkar, D.N. Varanasi, India
Saul, S. Chicago, Illinois
Saura, A. Helsinki, Finland
Savontaus, M.-L. Turku, Finland
Sayers, E.R. Tuscaloosa, Alabama
Schabtach, E. Eugene, Oregon
Schäfer, U. Düsseldorf, Germany
Schaffer, H.E. Raleigh, North Carolina
Schalet, A. Leiden, Netherlands
Scharloo, W. Utrecht, Netherlands
Scheid, W. Münster, Germany
Scheilds, G. Brighton, England
Schneider, I. Washington, D.C.
Schouten, S.C.M. Utrecht, Netherlands
Schweizer, P.M. Charlottesville, Virginia
Schwinck, I. Storrs, Connecticut
Schwochau, M. Düsseldorf, Germany
Seecof, R.L. Duarte, California
Seki, T. Osaka, Japan
Semeonoff, R. Leicester, England
Seybold, W.D. Zürich, Switzerland
Seyffert, W. Tübingen, Germany
Shafer, S.J. Philadelphia, Pennsylvania
Sharma, G.P. Chandigarh, India
Shearn, A. Baltimore, Maryland
Sheldon, B.L. Sydney, Australia
Shellenbarger, D. Iowa City, Iowa
Shen, M.W. Austin, Texas
Sherald, A. Charlottesville, Virginia
Shiomi, T. Nagasaki, Japan
Shorrocks, B. Leeds, England
Sick, K. Copenhagen, Denmark
Sidhu, N.S. Izatnagar, India
Singeisen, C. Fribourg, Switzerland
Singh, A. Chandigarh, India
Singh, B.N. Varanasi, India
Široký, J. Brno, Czechoslovakia
Slatis, H.M. East Lansing, Michigan
Slizynska, H. Edinburgh, Scotland
Smith, B.R. Aberdeen, Scotland
Smith, D.A. Birmingham, England
Smith, P.D. Atlanta, Georgia
Scowcroft, W.R. Canberra, Australia
Sobels, F.H. Leiden, Netherlands
Södergren, A. Umeå, Sweden
Sofer, W. Baltimore, Maryland
Sokoloff, A. San Bernardino, California
del Solar, E. Santiago, Chile
Somero, M.G. San Diego, California
Sonnenblick, B.P. Newark, New Jersey
Sorsa, M. Helsinki, Finland

Sorsa, V. Helsinki, Finland
Sparrow, J.C. Charlottesville, Virginia
Speers, L. Ottawa, Canada
Sperlich, D. Tübingen, Germany
Spiers, G. Edinburgh, Scotland
Spiess, E.B. Chicago, Illinois
Spieth, H.T. Davis, California
Sprechman, L. Austin, Texas
Springer, R. Vienna, Austria
Stafford, D.W. Chapel Hill, North Carolina
Steffensen, D.M. Urbana, Illinois
Steiner, W.W. Honolulu, Hawaii
Stern, C. Berkeley, California
Strickberger, M.W. St. Louis, Missouri
Strøman, P. Copenhagen, Denmark
Sugimoto, K. Hiroshima, Japan
Sullivan, D.T. Syracuse, New York
Sundbom, R. Northridge, California
Suomalainen, E. Helsinki, Finland
Suter, K. Zürich, Switzerland
Suzuki, D. Vancouver, Canada
Svahlin, H. Umeå, Sweden
Takaya, H. Kobe, Japan
Tallantire, A.C. Kampala, Uganda
Tantawy, A.O. Alexandria, Egypt
Tartof, K. Philadelphia, Pennsylvania
Temin, R.G. Madison, Wisconsin
Thoday, J.N. Cambridge, England
Thomasson, W.A. Chicago, Illinois
Thompson, S.R. Ithaca, New York
Thongmeearkom, P. Brisbane, Australia
Thörig, G.E.W. Utrecht, Netherlands
Throckmorton, L.H. Chicago, Illinois
Tigerstedt, P. Helsinki, Finland
Tiivola, A. Helsinki, Finland
Tobari, I. Chiba, Japan
Tobari, Y.N. Tokyo, Japan
Tobler, H. Fribourg, Switzerland
Tokunaga, C. Berkeley, California
Tomkins, J.K. Clayton, Australia
Tonomura, Y. Tokyo, Japan
Torroja, E. Madrid, Spain
Tracey, M.L. Davis, California
Traut, A. Münster, Germany
Traut, H. Münster, Germany
Triantaphyllidis, C.D. Thessaloniki, Greece
Trippa, G. Rome, Italy
Trout, W.E. Duarte, California
Tsacas, L. Gif-sur-Yvette, France
Tsakas, S. Athens, Greece
Tsuchiyama, S. Fukuoka, Japan
Tsuno, K. Sakado-Machi, Japan
Tuinstra, E.J. Utrecht, Netherlands
Tung, P.S.-C. University Park, Pennsylvania
Ulrich, H. Zürich, Switzerland
Urbischek, E.F. Swansea, Great Britain
Ursprung, H. Zürich, Switzerland
Vaidya, V.G. Poona, India
Valencia, J.I. Madison, Wisconsin
Valencia, R.M. Madison, Wisconsin
Valentin, J. Stockholm, Sweden
Van den Haute, J. Namur, Belgium
Van Valen, L. Chicago, Illinois
Vepsälälinen, K. Helsinki, Finland
Vigue, C. Baltimore, Maryland
Viinikka, Y. Turku, Finland
Voelker, R.A. Raleigh, North Carolina
Vreezen, W.J. Leiden, Netherlands
Vyse, E.R. Bozeman, Montana
Waddington, C.H. Edinburgh, Scotland
Wakahama, K.-I. Matsue, Japan
Walker, G.W.R. Edmonton, Canada
Wallace, B. Ithaca, New York
Wallace, H. Birmingham, England
Ward, C.L. Durham, North Carolina
Wargent, J.M. Sheffield, England
Warren, H. Pittsburgh, Pennsylvania
Wasserman, M. New York, New York
Watanabe, T.K. Misima, Japan
Watson, W.A.F. Aberdeen, Scotland
Wehner, R. Zürich, Switzerland
Welshons, J. Ames, Iowa
Welshons, W.J. Ames, Iowa
Westerman, J. Bundoora, Australia
Wheeler, L. Austin, Texas
Wheeler, M.R. Austin, Texas
White, K. New Haven, Connecticut
Whitney, J.B. Chapel Hill, North Carolina
Whittinghill, M. Chapel Hill, North Carolina
Whittle, J.R.S. Brighton, England
Wildeboer-du Pui, M.L.L. Haren, Netherlands
Wilkerson, R.D. Oak Ridge, Tennessee
Williamson, D.L. Stony Brook, New York
Williamson, J.H. Calgary, Canada
Wimber, D. Eugene, Oregon
Winge-Cordeiro, H. Pôrto Alegre, Brazil
Winkel, E.U. Berlin, Germany
Wöhrmann, K. Tübingen, Germany
Wolf, H. München, Germany
Wong, P.T. Duarte, California
Wood, D.D. Rochester, Michigan
Woodruff, R.C. Austin, Texas
Woolf, C.M. Tempe, Arizona
Wright, C.P. Cullawhee, North Carolina
Wright, T.R.F. Charlottesville, Virginia
Wui, I.S. Kwangju, Korea
Würgler, F.E. Zürich, Switzerland
Yakoumi, G. Athens, Greece
Yamada, M.A. Misima, Japan
Yamazaki, H.I. Tokyo, Japan
Yamazaki, T. Misima, Japan
Yannopoulos, G. Patras, Greece
Yasuda, N. Chiba, Japan
Yoon, J.S. Austin, Texas
Yoon, S.B. Madison, Wisconsin
Yoshikawa, I. Nagasaki, Japan
Yost, H.T.Jr. Amherst, Massachusetts
Younis, S.A. Assuit, Egypt
Ytterborn, K.H. Stockholm, Sweden
Zeguers, M.W.S. Haren, Netherlands
Zonneveld, B.J.M. Leiden, Netherlands
Zouros, E. Chicago, Illinois
Zubeldia, A. Valencia, Spain