DROSOPHILA
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TECHNICAL NOTES

ANNOUNCEMENTS

Drosophila as a Model Organism for Ageing Studies, edited by F.A. Lints and M. Hani Soliman. The text presents a critical review, by researchers worldwide, of the genetics and gerontology of Drosophila and the extent to which available knowledge leads us to an understanding of the biological phenomena of ageing, longevity, senescena and death in higher organisms, including Man. Of special interest to geneticists, entomologists, ecologists, behavourists and cell biologists: 49 pounds hdbk, 320 pp, fully illus., details from Blackie & Son Ltd, Acad. & Professional Div., Bishopbriggs, Glasgow G64 2NZ, UK.

Aging in Drosophila: A Selectively Annotated Bibliography: by Leslie P. Gartner, published by Jen House Publ. Co., Baltimore MD (1986); 247 pages. This volume presents a listing of bibliographic references subdivided into 34 categories. Over 140 of these references have been annotated by the author.

A Laboratory Handbook of the Genetics and Biology of Drosophila: A Drosophilist's Vade-Mecum: by Michael Ashburner, Dept. of Genetics, University of Cambridge. I am attempting to write a comprehensive laboratory handbook for fly workers -- includes developmental and molecular biology, as well as classical genetics and taxonomy. In fact a draft is reasonably complete and I have a Table of Contents if anyone is interested. If anyone has methods or protocols (in any field of Drosophila biology) that they would like to share with a wider public, then I would be glad to hear from them.

UCLA Symposium: "Stress-Induced Proteins", April 10-16, 1988, at Keystone, Coloradc. Organizers: Mary Lou Pardue, James Feramisco and Susan Lindqusit; Sponsored by Hoffmann-La Roche Inc. This meeting is designed to promote future multidisciplinary approaches to studying transcriptional control mechanisms, gene cloning, translational control analyses, protein function studies, and clinical therapy as they are related to stress-induced proteins. For further information, please write or call: UCLA Symposia, 103 Molecular Biology Inst., Univ. of California, Los Angeles, CA 90024; tel. 213-206-6292.

28th ANNUAL DROSOPHILA RESEARCH CONFERENCE

Held May 20-24, 1987, at the Bismarck Hotel, Chicago, Illinois.

Plenary Session -   Wednesday, May 20
    Speakers: Minx Fuller, Cathy Laurie-Ahlberg, Hugh Robertson

Concurrent Sessions: Thursday morning, May 21
    Neurobiology I (Chair - Kalpana White)
    Techniques, Chromosomes, Natural History (Chair - William Engels)
    Heat Shock, Second Site Suppression (Chair - Elizabeth Craig)

Concurrent Sessions: Thursday afternoon, May 21
    Homeotics (Chair - Tom Kaufman)
    Neurobiology II (Chair - Barry Ganetzky)
    Transposable Elements (Chair - Michael Simmons)

Reception and Poster Sessions: Thursday evening, May 21

Plenary Session -   Friday morning, May 22
    Speakers: Vincenzo Pirrotta, Thomas Cline, Terry Orr-Weaver, Jeff Hall

Concurrent Sessions: Friday afternoon, May 22
    Development I (Chair - Michael Hoffmann)
    Gene Expression I (Chair - Gail Waring)
    Population Genetics and Evolution (Chair - Rollin Richmond)

Concurrent Session and Workshops: Saturday morning, May 23
    Segmentation (Chair - Tim Karr)
    Workshop on Aging (Chair - Robert Arking)
    Workshop on Position Effect Variegation (Chair - Janice Spofford)

Concurrent Sessions: Saturday afternoon, May 23
    Gene Expression II (Chair - David Sullivan)
    Development II (Chair - Marc Muskavitch)
    Sex Determination and Gametogenesis (Chair - Bruce Baker)

Plenary Session -   Sunday morning, May 24
    Speakers: Mark Biggin, Ian Duncan, Anthony Mahowald, Eric Wieschaus

Sociedad Venezolana de Ecologia: recently founded by a group of researchers in ecology in Venezuela. The general aim of our Society is to promote the study of tropical ecosystems and to contribute to the flow of information on ecological research in those areas. A journal, Ecotropics, will contain quality papers on tropical ecology. Write for details to Managing Editor, Sociedad Venezolana de Ecologia, Apartado 47543, Caracas 1041-A, Venezuela.

Stock Center Closed: On May 1, 1987, the NSF Drosophila Stock Center at Caltech was discontinued. The entire collection of stocks has been transferred to Indiana Univesity where the Center is under the direction of Dr. Tom Kaufman. Requests for stocks from that center should be addressed to the Curator of Drosophila stocks, Dept. of Biology, Indiana University, Bloomington IN 47405.

Journal Change: In 1987 the Journal of Embryology and Experimental Morphology to Development, reflecting the wider range of papers that are now being published. Whilst remaining the premier journal of morphogenesis, there will be greater emphasis on molecular and cellular development with a revised panel of Editors: Editor in Chief - Chris Wylie (London); UK Editors - Richard Gardner (Oxford), Peter Lawrence (Cambridge), Mike Gaze (Edinburg), Hugh Woodland (Warwick); US Editors - Richard Hynes (MIT) and Doug Melton (Harvard). Papers can be submitted direct to USA Editors, British Editors, or to Editorial Office, Development, Dept. of Zoology, Downing St., Cambridge CB2 3EJ, UK. The advantages are numerous -- fast publication time (13 weeks from acceptance to appearance, 3-4 weeks from receipt to editorial decision), published monthly, no page charges, 200 free reprints and excellent quality of production, esp. photographs. Subscriptions are subsidised by the Educational Charity that owns and runs the journal and in 1987, personal subscriptions are only $75 for 12 issues. Write to: The Biochemical Society Book Depot, PO Box 32, Commerce Way, Colchester CO2 8HP, UK.

Adell, J.C. and L.M. Botella. Universidad de Valencia, Spain. Selection for rate of pupation in crowded cultures of **Drosophila melanogaster.**

The selection for developmental time in Drosophila is mentioned in the literature as a typically assymetrical process and practically undirected, in which the selection lines for faster development do not give response (Sang & Clayton 1957; Clarke et al. 1961; Sang 1962; Bakker & Nelissen 1963). The absence of response in the fast lines has been explained as due to the optimization of the mean developmental time (MDT) due to natural selection in species that depend on transitory resources.

Our experiment consisted of 8 vials of 0.8 x 5 cm with 0.5 ml of Lewis medium with more than 150 larvae (chaos zone) from a wild strain. From these vials the first 8 individuals that pupated at each of them were collected, and the experiment was repeated with their progeny. The cycle was repeated during 3 generations.

Table 1. Mean time of pupation for each generation of selection.

Generation of selection	Mean time of pupation
0	6.73 ± 0.10
1	7.44 ± 0.12
2	6.80 ± 0.06
3	7.34 ± 0.11

Table 1 shows the values of the mean time of pupation for each one of the generations of selection. As can be seen the values of the mean time of pupation adopt a "zig-zag" aspect. This pattern is similar to the one found by Botella & Mensua working with the MDT in crowded conditions.

The observed pattern can be due to the alternative advantage of two factors: (1) individuals with faster development will be favoured since they can escape from the competition situation, and then their progeny would have a pupation time less than their parents; (2) at this moment the best adapted individuals to the competition situation will be favoured and then the mean time of pupation would increase. The progeny of these individuals will be adapted to the competition situation, and then the individuals that have a certain facility to accelerate their development will pupate, and then the cycle will start again.

References: Bakker, K. & F.X. Nelissen 1963, Ent. Exp. & Appl. 6:37-52; Botella, L.M. & J.L. Mensua 1986, DIS 63:35; Clarke, J.M., M.J. Smith & K.C. Sondhi 1961, Genet. Res. 2:70-81; Sang, J.H. 1962, Genet. Res. Camb. 3:90-109; Sang, J.H. & G.A. Clayton 1957, Jrl. Hered. 48:265-270.

Aimanova, K.G., L.M. Pereligina and N.N. Kolesnikov. Institute of Cytology & Genetics, USSR Acad. of Sciences, Novosibirsk, USSR. A biochemical analysis of the salivary gland secretory glycoproteins of **Drosophila virilis.**

There are detailed investigations of the genetic system controlling the secretory glycoprotein synthesis of the salivary gland of **D.melanogaster** (Akam et al. 1978; Korge 1981; Velissariou & Ashburner 1981; Kokoza et al. 1982; Meyerowitz & Hogness 1982). The processes of the secretion proteins synthesis have been less studied in **D.virilis** (Kress 1980, 1981; Kress & Enghofer 1975). Herein we report biochemical data on the larval salivary gland secretory proteins of **Drosophila virilis.**

The secretion isolated from the salivary gland of the third instar larvae of 35 laboratory stocks was studied in 7.5% polyacrylamide gels containing urea (Panyim & Chalkly 1969).

Two intensity staining groups of proteins are seen in the secretion patterns of the different stocks of **D.virilis.** One group (1g) consists of 3-4 fractions with the slow electrophoretic mobilities. The other of 4-5 major fractions are fast and designated sp1-sp5 (secretion protein).

Three of 5 major secretion proteins varied in electrophoretic mobility (Figure 1). It was found that protein sp2 is represented by 5 (2a-e), sp4 by 4 (4a-d) and sp5 by 2 electrophoretic variants (5a-b). Protein sp4 was not detected in secretion patterns of 12 **D.virilis** stocks (Figure 1), and it is presumably a 4"0" variant like protein sgs-4 in **D.melanogaster** (Korge 1977).

The secretion patterns differed not only qualitatively, but also quantitatively with respect to the secretory protein variants. Thus, sp5a content is relatively low in stocks 139 and 142 compared to stocks 160, 127 and others (Figure 1).

The secretion proteins were also fractionated in SDS-polyacrylamide gradient gel (5-20%) (Laemmly 1970). Four major fractions were identified: 2 were high (220 kd, 160-180 kd) and 2 were low molecular weight (14 kd, 16 kd) proteins (Figure 2).

All the secretion proteins detected are glycoproteins, i.e., they give a PAS-positive reaction. These fractions are tissue-specific because there are no proteins with the same electrophoretic mobilities in the patterns of other larval tissues such as the fat body, haemolymph, the Malpigian tubules, midgut, neural ganglia.

Figure 1. A schematic representation of the salivary gland secretion patterns of 35 **D.virilis** laboratory stocks in 7.5% polyacrylamide gel containing urea [sp = secretion protein; (a-e) = electrophoretic variants].
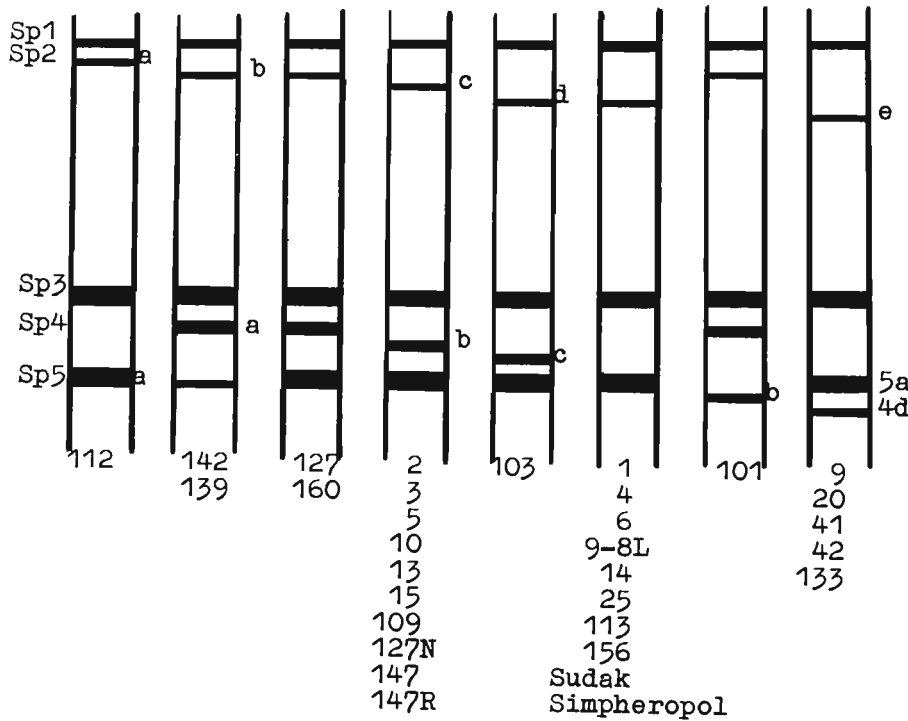
During larval development proteins sp3, sp4 and sp5 appear at 32 hr after second molt, at the time when the first PAS-positive granules are observed in the salivary gland cell. Proteins sp1, sp2 appear at 48-54 hr (Figure 3). The staining intensity of sp1 - sp5 increases during larval development, reaching maximum at the time of puparium formation. At the prepupal stage, 2 hr after secretion extrusion, only minor amounts of the major secretion proteins are observed in the proteins patterns.

A genetic analysis of the major secretion proteins with the variations in the electrophoretic mobilities was carried out. The results of reciprocal crosses within the different stocks of **D.virilis** demonstrated that the genes for the synthesis of the main secretory proteins sp2, sp4 and sp5 are located on the X chromosome. The genes controlling the synthesis of the 1g secretory proteins are located on the autosomes.

Thus, we characterized electrophoretically the secretion protein of the salivary gland of **D.virilis.** The genetic system controlling the secretory glycoprotein synthesis of the salivary glands of **D.virilis** consists of at least 3 genes mapped on the X chromosome. Other genes are located on the autosomes. All the secretion proteins detected are tissue-specific and the genes controlling their synthesis are presumably activated at different stages during larval development.
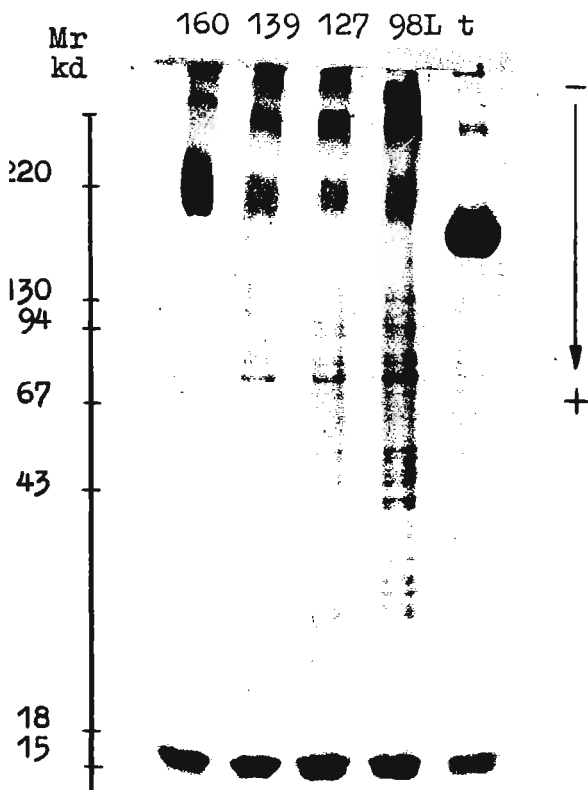
Figure 2. Electrophoregrams of the larval salivary gland secretion proteins in SDS-polyacrylamide gradient gel (5 - 20%). [160,139,127,9-8L = **D.virilis** stocks; t = **D.texana**; 160,t = secretion; 139,127,9-8L = salivary glands]
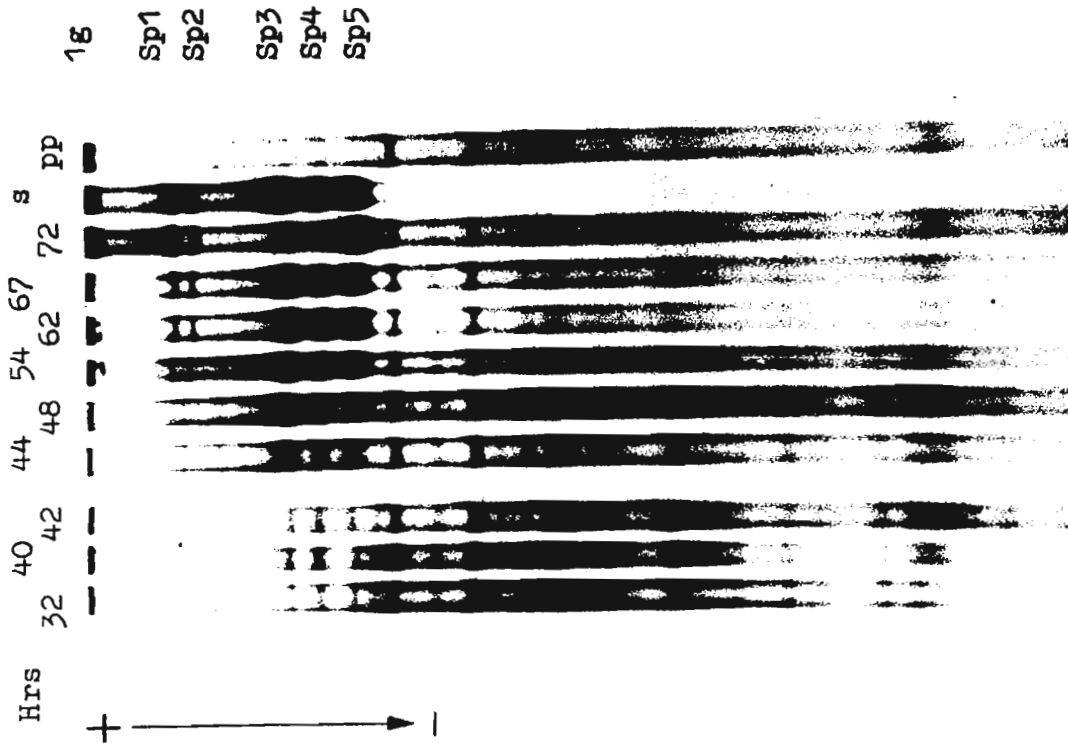
Figure 3. Electrophoregrams of the salivary gland of **D.virilis** during third larval instar [sp = secretion protein; s = secretion; pp = prepupae].

References: Akam, M.E., D.E. Roberts, G. Richards & M. Ashburner 1978, Cell 13:215-225; Kokoza, V.A., S.G. Kazakova & E.I. Karakin 1982, DIS 58:94-95; Korge, G. 1977, Devel. Biol. 58:339-355; _____ 1981, Chromosoma 84:373-390; Kress, H. 1980, Chromosoma 72:53-66; _____ 1981, Naturwissenschaften 68:28-33; _____ & E. Enghofer 1975, Insect. Biochem. 5:171-181; Laeemli, U. 1970, Nature 227:680-685; Meyerowitz, E. & D. Hogness 1982, Cell 28:165-176; Panyim, S. & R. Chalkly 1969, Arch. Biochem. Biophys. 130:337-346; Velissariou, V. & M. Ashburner 1981, Chromosoma 84:173-185.

Aimanova, K.G., L.M. Pereligina and N.N. Kolesnikov. Institute of Cytology & Genetics, USSR Acad. of Sciences, Novosibirsk, USSR. A genetic analysis of the tissue-specific salivary gland secretion proteins of **D.virilis.**

Comparative electrophoretic analysis of the salivary gland secretion glycoproteins of the third instar larvae in 35 **D.virilis** stocks and species Drosophila of the **virilis** group demonstrated qualitative and quantitative differences in their patterns.

Based on the variations in the electrophoretic mobilities, a genetic analysis of the major secretion proteins was carried out. The results of reciprocal crosses within and between the stocks and species demonstrated that the genes for the synthesis of the main secretory proteins sp2, sp4 and sp5 (secretion protein) are located on the X chromosome.

To map the gene VSP-2 (**virilis** secretion protein) in the first experiment, we used stock 127N carrying recessive mutations (cv, w), a dominant mutation (Bx) and variant 2c of protein sp2 and stock 41 with secretion protein sp2e (Table 1). The gene VSP-2 was mapped approximately 39.5 m.u. to the right of w or at about position 144.5 m.u. on the X chromosome. The genetic distance at high recombination frequencies was estimated according to Kosambi (1944).

In the second experiment, we used stock 139 with two recessive mutations (y, ap) and variant 2b of protein sp2, and 41 stock with secretion protein sp2e (Table 2). The gene VSP-2 was mapped about 8.4 m.u. to the right of ap, i.e., it was located at about position 144.4 m.u. on the X chromosome. Thus, in two independent experiments the position of VSP-2 on the X chromosome was found to be the same.

There was not a single case of recombination between VSP-2 and VSP-4 (the total number of analysed larvae was 108), thereby indicating their close linkage. Support for close linkage was derived from the genetic localization of VSP-4 in two independent experiments with two stocks, one carrying the 4a variant of sp4 and the other lacking it. The conclusion was that VSP-4 also is located in the region of 144.4 m.u. on the X chromosome (Figure 1).

Table 1. Mapping data for the gene VSP-2 of **Drosophila virilis.** Stocks 127N (cv, v, Bx, w, 2c) and 41 (+ + + 2e).

Genotype ♂♂ F$_2$				No. of larvae		%
+	+	+	2e	7	13	9.4
cv	Bx	w	2c	6		
+	Bx	w	2c	16	35	25.4
cv	+	+	2e	19		
+	+	w	2c	19	34	24.6
cv	Bx	+	2e	15		
+	+	+	2c	5	8	5.8
cv	Bx	w	2e	3		
+	Bx	+	2e	5	10	7.2
cv	+	w	2c	5		
+	+	w	2e	10	19	13.8
cv	Bx	+	2c	9		
+	Bx	w	2e	9	16	11.6
cv	+	+	2c	7		
+	Bx	+	2c	1	3	2.2
cv	+	w	2e	2		
	Total				138	100

Table 2. Mapping data for the gene VSP-2 of **Drosophila virilis.** Stocks 139 (y, ap, 2b) and 41 (+ + 2e).

Genotype ♂♂ F$_2$			No. of larvae		%
+	+	2e	28	35	45.45
y	ap	2b	7		
+	ap	2b	25	35	45.45
y	+	2e	10		
+	+	2b	5	5	6.5
y	ap	2e	--		
+	ap	2e	2	2	2.6
y	+	2b	--		
	Total			77	100

Based on the genetic analysis, we established recombinant classes between VSP-2 and VSP-5. The data from a total number of 108 progeny indicated that 26 were recombinants. Of 36 males, 12 were found to be recombinants. According to preliminary estimates, VSP-5 is 26.1 m.u. apart from VSP-2 (Fig. 1).

Thus, the genetic data obtained indicate close linkage of VSP-2 and VSP-4. These genes were mapped to 144.4 m.u. on the X chromosome, and VSP-2 was found to be located at a distance of approximately 26.1 m.u. from VSP-2 and VSP-4.

In crosses of stock 127N (cv, v, Bx, w) and wild **D.virilis** stock 41, the recombination frequency between mutation Bx and w was determined as 18.9%, and between stocks 109 (Bx, w) and 41 as 30.1%, although it should be about 10%. No decisive explanation can be offered for this observation. However, independent experiments with different stocks have demonstrated a recombination frequency of 20-30% in a total number of about 5000 individuals. The increase in recombination frequency in this part of the genetic map may be possibly related to an insertion of a mobile element. This suggestion needs experimental verification.

The results of the interspecific crosses **D.virilis** x **D.texana** are noteworthy. Two new fractions, which were absent in the parental secretion, appeared in the electrophoretic patterns yielded only by hybrid females of the F$_1$ generation from the **D.virilis** x **D.texana** crosses (Figure 2). Analysis of the F$_2$ generation demonstrated segregation for secretory proteins, however; only in larvae with two X chromosomes (each derived from different species), two additional fractions were consistently detected and they not under the influence of the auto-
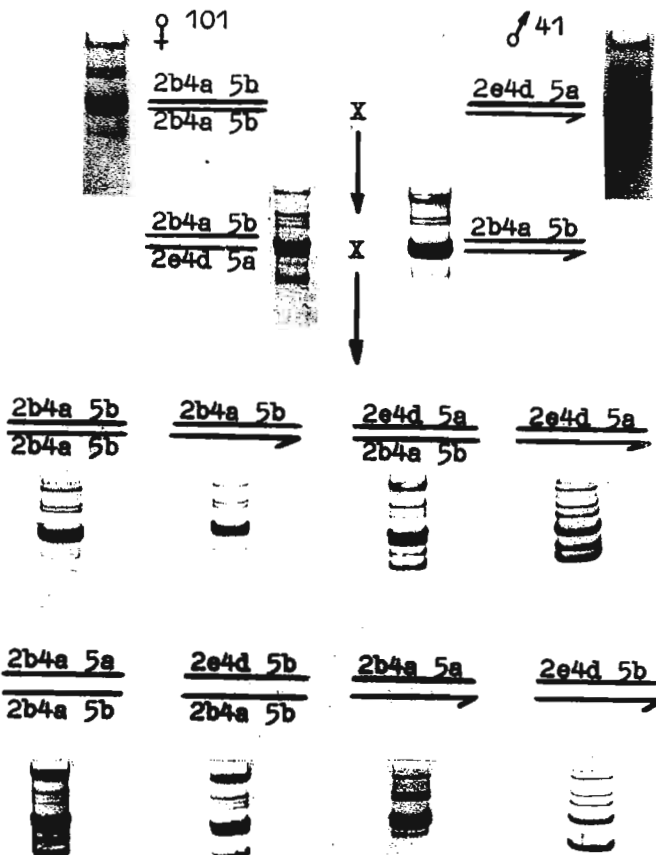
Figure 1. A scheme for the genetic mapping of VSP-2, VSP-4 and VSP-5.
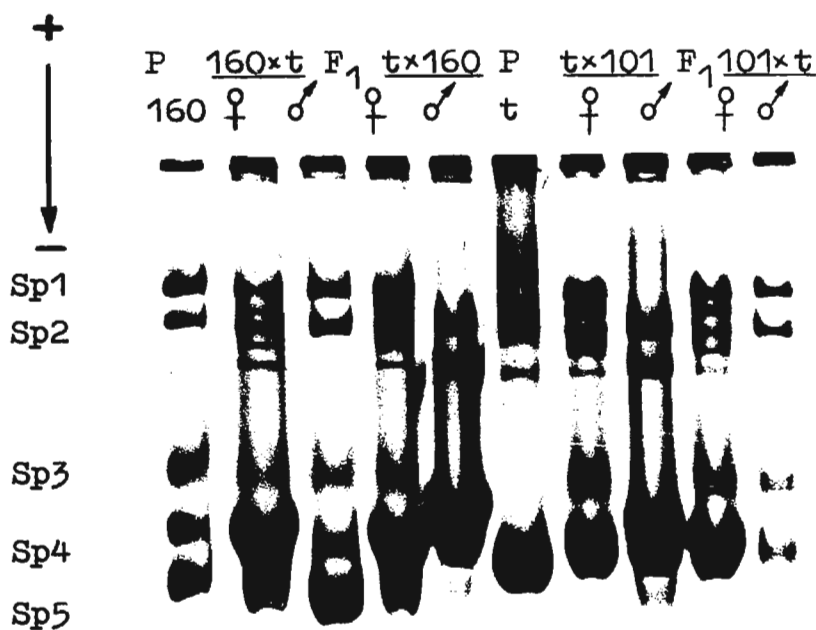
Figure 2. Electrophoregrams of the salivary gland secretion proteins of **D.texana**, **D.virilis** stocks 101, 160 and the F₁ generation from interspecific **D.virilis** x **D.texana** crosses (· = designate the new secretory proteins).

somes. The two new fractions were PAS-positive. They are possibly the final product of genes -- proteins that have undergone post-transcriptional modification. There possibly occurred an activation of new structural genes under the effect of interspecific hybridization, and analysis of the new proteins may help to elucidate the molecular mechanisms of the interaction of the genomes of various species.

Thus, in **D.virilis**, at least, three genes controlling the synthesis of secretion proteins in the salivary gland are located on the X chromosome. In contrast, in **D.melanogaster** only one of the seven genes of the secretory proteins, Sgs4, is located on this chromosome (Korge 1977, 1981; Akam et al. 1978; Velissariou et al. 1981; Kokoza et al. 1982).

Based on the results of genetic analysis of the main secretion proteins of the salivary gland of **D.virilis**, it may be suggested that the structural organization of the genes for the tissue-specific function is somewhat different in **D.virilis** and **D.melanogaster**, although they have features in common.

References: Akam, M.E., D.B. Roberts, G. Richards & M. Ashburner 1978, Cell 13:215-225; Kokoza, V.A, S.G. Kazakova & E.I. Karakin 1982, DIS 58:94-95; Korge, G. 1977, Chromosoma 62:155-174; ——— 1981, Chromosoma 84:373-390; Kosambid, D.D. 1944, Ann. Eugenics 12:172-174; Velissariou, V. & M. Ashburner 1981, Chromosoma 84:173-185.

Alcorta, E., A. Dominguez and J. Albornoz. Universidad de Oviedo, Spain. Egg retention in **Drosophila melanogaster** females.

Egg retention behavior should be very important in the ecology and population biology of Drosophila since useless waste is avoided. It is known that virgin females of **D.melanogaster** will lay few eggs (Cook 1970; Bouletreau-Merle 1982), while inseminated females can retain their eggs if no suitable substrate is available (David & Bouletreau-Merle 1971). Nevertheless, only just relating the two behavioural patterns has been attempted, discovering it to be independent (Bouletreau-Merle & Terrier 1986). The experiments described here suggest that both patterns may be related to some extent.

Five inbred strains of **D.melanogaster** from different geographic origins were used: the first, Teverga-5 (Spain), came from our own laboratory, and the other four were Crkwenica (Czechoslovakia), Israel (Israel), Kreta-75 (Greece), and Hampton Hill (Great Britain) lines which came from the Umea Drosophila Stock Center. These five lines were tested for egg laying under three different conditions: inseminated females on a suitable oviposition surface (the whole culture medium), inseminated females on a non-suitable oviposition surface (either agar or paper), and virgin females on the suitable medium.

The culture medium and the procedure to determine egg laying for virgin and inseminated females in a suitable medium were described elsewhere (Dominguez & Rubio 1986), with the exceptions that females were tested individually (one virgin female, or one female and two males for insemination, per vial), and the measurement period was extended from the second to the 20th day of the females' age. Egg laying of inseminated females on a non-suitable oviposition surface was measured as the egg laying, on the 5th day of age, of females kept on the whole medium for the earlier four days. The four non-suitable oviposition surfaces considered were either an 0.8% agar gel containing 2% ethanol and 1% acetic acid, or this same medium covered with a piece of paper. Twenty females from each strain were tested for each

of the four different conditions. Another set of virgin and inseminated females in a suitable medium were kept under the same conditions in order to be dissected later.

Females tested for egg laying in a non-suitable medium were dissected at the end of the egg laying period (i.e., on the 6th day of age), and the number of mature eggs in the ovaries was recorded. Inseminated females laying on a suitable medium were also dissected on the 6th day of age, and virgin females were dissected on the first day of laying.

Egg laying curves of virgin and inseminated females in a good egg laying medium (Figure 1) showed the typical features expected (Cook 1970). Inseminated females started egg laying on the second day following emergence; oviposition reached a maximum during days four to six and then declined. The maximum laying was delayed in virgin females, which initially laid few eggs and then reached a stable phase.

There were strong differences between strains for virgin fecundity relating to the first day of laying (Table 1b). Strains able to largely defer the deposition of their first egg also had a more reduced production. This fact has been also noted by Bouletreau-Merle & Terrier (1986) among individual females. The smaller rate of egg deposition of virgin females was not only due to a reduction of the vitellogenic activity, but also to an actual retention of eggs as shown by the large number of mature eggs that were found in the ovaries of virgin females on the first day of laying (Table 1b).

Inseminated females laid fewer eggs on a non-suitable medium than on a suitable one (Table 1a), the egg output being smaller on a paper surface than on an agar surface. Egg laying differences between strains were significant both on agar ($F = 8.76$; $p < 0.001$) and on paper mediums ($F = 5.07$; $p < 0.01$). These differences did not seem to be related to the egg output on a good surface; nor were significant within strain correlation between the egg laying of individual females on the non-suitable medium and the previous fecundity on a normal medium, as previously reported (Bouletreau-Merle & Terrier 1986).
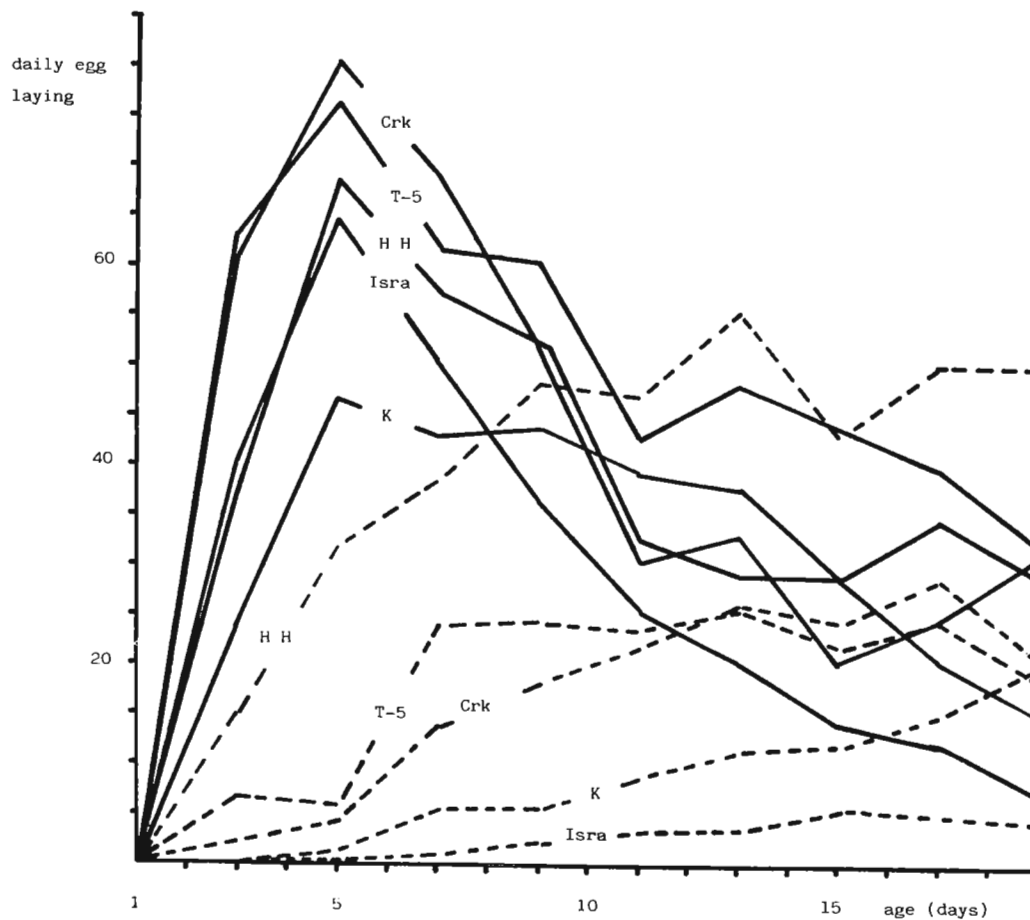
Figure 1. Daily egg-laying of virgin (- - - -) and inseminated females (———) on normal medium.

Table 1.

	Teverga 5	Hampton Hill	Crkwenica	Israel	Kreta
(a) Number of eggs laid and stored per inseminated female at the fifth day of age:					
Normal medium:					
eggs laid	64.72 ± 2.86	65.05 ± 6.13	80.00 ± 4.21	63.40 ± 4.21	48.25 ± 4.85
eggs stored	8.55 ± 1.38	27.37 ± 2.78	26.50 ± 3.60	14.22 ± 2.89	16.11 ± 2.56
Agar:					
eggs laid	17.15 ± 3.30	10.37 ± 3.17	3.64 ± 1.36	16.16 ± 2.73	1.00 ± 0.51
eggs stored	35.70 ± 3.23	67.74 ± 4.66	93.86 ± 6.04	66.21 ± 5.22	56.55 ± 2.30
Paper:					
eggs laid	1.47 ± 0.52	0.60 ± 0.26	1.53 ± 0.40	3.74 ± 0.85	0.95 ± 0.40
eggs stored	50.77 ± 2.28	71.60 ± 3.93	83.06 ± 6.35	72.79 ± 2.69	56.83 ± 3.34
(b) First day of laying and number of eggs stored by virgin females:					
1st day	3.80 ± 0.26	3.83 ± 0.38	5.40 ± 0.27	11.11 ± 0.87	7.43 ± 0.61
eggs stored	58.40 ± 5.12	72.26 ± 5.80	111.95 ± 4.78	91.44 ± 4.63	46.20 ± 2.76

Females from all the strains kept one day on a non-suitable substrate had more mature eggs in the ovaries than their respective control females (Table 1a). Within strain correlation between the number of eggs laid on the agar medium and the number of eggs retained (in those strains showing no strong retention; T5, HH and Israel) was negative and significant (r = -0.76; p < 0.001), also noted by Bouletreau-Merle & Terrier (1986) for individual females. Nevertheless, it seems that there was no such relation between strains. Nor was there any relation between the number of eggs stored in the ovaries and egg laying by virgin females.

Although no solid conclusions can be drawn from only five strains, the data show little relation between the egg retention of virgin and inseminated females. Nevertheless, there was a relation between the number of eggs stored in the ovaries by mated females kept on a non-suitable medium and virgin females of the different strainis ($r_{paper-virgins}$ = 0.907, p < 0.05; $r_{agar-virgins}$ = 0.824, p < 0.10; $r_{agar-paper}$ = 0.9534, p < 0.02). This fact suggests that females of different strains could have a different capability for egg storage that, together with other physiological, nutritional and behavioral factors, might be implicated in the two patterns of egg retention.

References: Bouletreau-Merle, J. 1982, DIS 58:28-29; _____ & O. Terrier 1986, Int. J. Invert. Reprod. 9:113; Cook, R.M. 1970, DIS 45:128; David, J. & J. Bouletreau-Merle 1971, DIS 46:83; Dominguez, A. & J. Rubio 1986, Heredity 57:305.

Alexandrov, I.D. Research Inst. of Medical Radiology, Obninsk, USSR. Enigmas of radiation mutagenesis in c(3)G mutant of **Drosophila melanogaster.**

Mechanisms of meiotic as well as radiation-induced mitotic chromatid exchanges in Drosophila known to have some intermediate steps just as their genetic control in common (see, e.g., Baker et al. 1980). It is thus not surprising that the yield of X-ray-induced chromatid exchanges in neuroblasts of mei-41 or c(3)G larvae is more low than that in the irradiated wild-type cells (Baker et al., ibid.; Pimpinelli et al. 1975). It is important too that the c(3)G genome appear to be deficient for "the fast breakage-fusion" mechanism in X-ray-induced mitotic recombination (Haendle 1974). In other words, one may well be defective for the fast recombination-dependent DNA repair which may be clearly defined as error-prone (mutagenic) repair since it leads to chromatid interchanges in wild-type genome. These early studies and the finding that the processing (i.e., repair or misrepair) of the premutational lesions in the male pronucleus is extremely restricted in time (according to Wurgler [see Racine et al. 1979], a maximum life-time of such lesions is of about 10 min) make it possible to expect that less mutations should be produced in the c(3)G genome irradiated than in wild-type ones. However, Watson's results (1972) show that this does not appear to be quite so: the c(3)G sperm gives rise to higher frequencies of both recessive lethals and translocations compared to wild-type. Since these studies have not yet been repeated, the different radiosensitivity of the somatic and germ cell chromosomes in the c(3)G mutant remains enigmatic phenomenon. Admittedly, it may well be that this difference is an artifact due to different conditions in experiments depicted (doses, radiation quality, etc.), the more so that in Watson's experiments one and enough high dose (30 Gy) was used only.

To inquire more carefully into the processing concerned in c(3)G pronucleus as compared with wild-type, the relationship between dose and effect was studied for different genetic end-points [dominant and recessive sex-linked lethals (RSLL), macrodeletions of X chromosome occurring as hyperploid w^a males, visible mutations as the y, w, b, cn and vg loci which have been classified into a true gene (VVg) and chromosome (SV+LV+ different VV^{ch}) mutations; see, e.g., Alexandrov (1984) and DIS 61, 63 for details] induced by gamma-rays of ^{60}Co or fission neutrons ($\bar{E}$ = 0.85 MeV) in mature sperms of 2-3 day old c(3)G or wild-type males (treated ones were mated individually to 5 virgin females of genetic constitution y sc^{S1} In49 sc^8 w^a; b cn vg for one day only). The same st c(3)G^{17} ca chromosome as described earlier (Alexandrov 1980) was used throughout. Homozygous c(3)G females are almost fully sterile and c(3)G/c(3)G males are far less fertile than the wild-type ones from the stock D-32 used. In particular, the progeny of the one D-32 male under mating conditions (note that inseminated females were twice subcultured at three-day intervals) depicted totals on an average about 264 sibs, whereas the offspring of the c(3)G male averages only about 48 under the same mating conditions. Thus, the untreated c(3)G males unlike wild-type
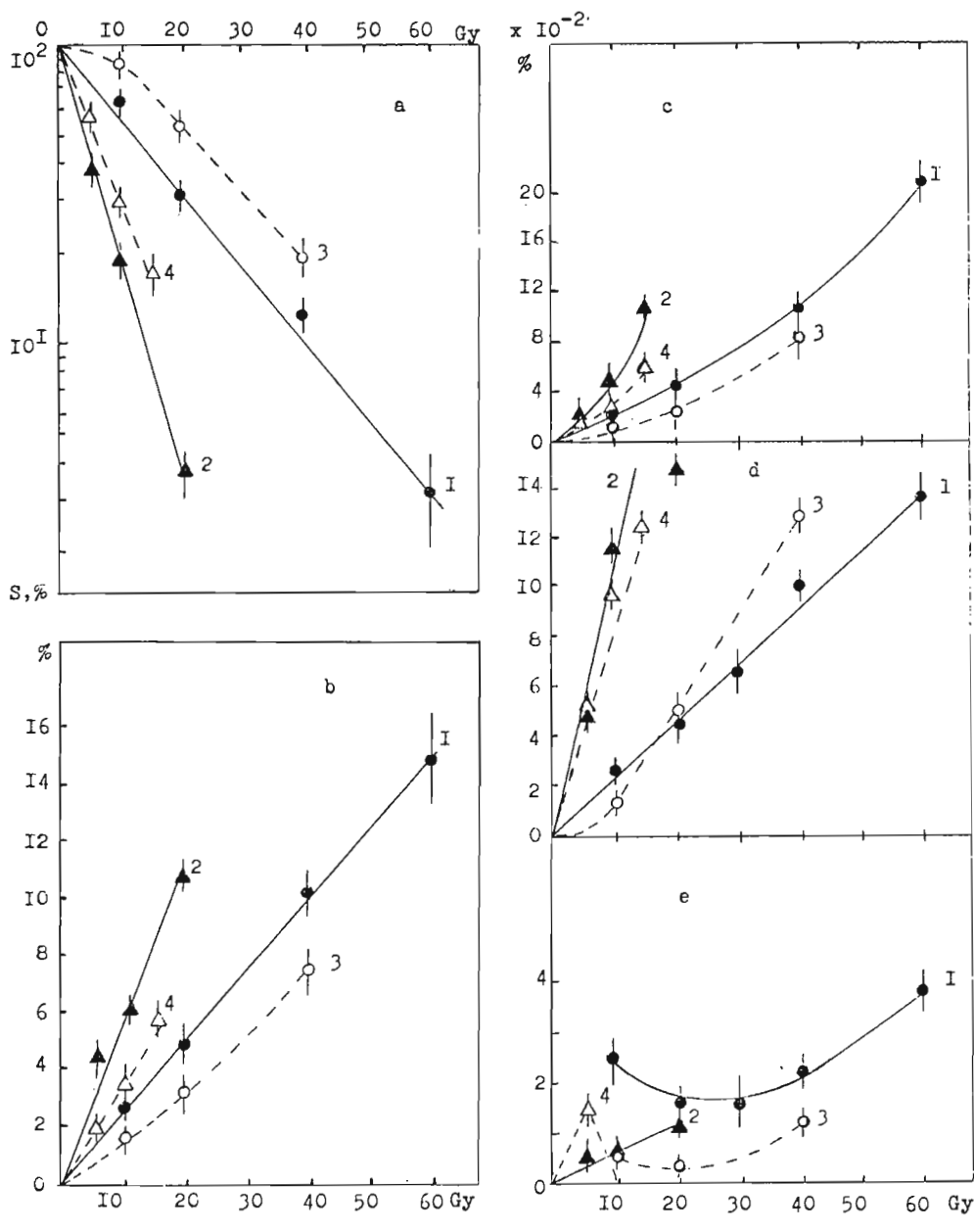
Figure 1. Dose-response curves for the relative survival (a), RSLL (b), macrodeletions of X chromosome (c), visible chromosome (d), and gene (e) mutations after irradiation of the wild-type (1,2) or chromosome (3,4) sperm of **Drosophila melanogaster** by gamma-rays of ^{60}Co (1,3) or fission neutrons (2,4) (details for end-points, see text).

show a high spontaneous dominant lethality. In so doing, however, the frequencies of the spontaneous RSLL in mature c(3)G and wild-type sperms are practically the same (0.061 and 0.075%, respectively, under about 4000 X chromosomes tested for each genome studied).

Analysis of the dose-response curves obtained (Figure 1) shows that the c(3)G genome exhibits a number of intriguing features in response to gamma-rays as well as to neutrons as compared with wild-type. Among unusual effects detected first of all one notices that the relative survival (the ratio of the treated survival to the control) of the mutant is much higher than that of wild-type at all dose levels (Fig. 1a). Moreover, in contrast to the latter where survival curve is exponential for both radiations studied, the mutant-survival curve shows the characteristic shoulder after gamma-ray-irradiation but does not after treatment by neutrons. These findings show that, on the one hand, radiation-induced dominant lethals were formed more rarely in the c(3)G genome than in the wild-type one, and, on the other hand, there is very strong evidence (a shoulder in the mutant-survival curve for gamma-rays, but not neutrons) for the existence of the efficiently acting repair system in the mutant, but not wild-type, zygote. Further, data for other end-points studied (Fig. 1 b-e) show that those arise more rarely in the mutant genome than the same ones in the wild-type as well. It is important to note (and this is another enigmatic finding) that a linear curve for the visible chromosome mutations (as a whole for all scored) induced by gamma-rays in the wild-type sperms (Fig. 1d) is transformed into linear-quadratic one in the mutant. As a result, the mutation frequencies for c(3)G genome is fewer at the low dose levels and more at the high dose levels than those in wild-type at the same doses absorbed. As the dose-response curve for RSLL shows (Fig. 1b), this tendency appears to be observed for the end-point named also, but the further investigations with more higher doses are required to verify this suggestion.

Returning to the first unusual effect, namely, more higher survival of c(3)G bearing zygotes in comparison to wild-type, one can note that the results showing more low yield of the chromosome changes (Fig. 1 b-d) in the mutant as compared with wild-type may explain this enigmatic phenomenon if one bears in mind that dominant lethality in Drosophila sperm has been interpreted as being due to the chromosome rearrangements.

Another new and exclusively enigmatic finding is a non-linear but rather asymmetrical "U"-shaped curve in the low-dose-region for the true gene mutations (summary data for five loci studied as a whole) induced by gamma-rays in the wild-type as well as c(3)G sperm (Fig. 1e). It is important to note that qualitatively the same dose-response curve nearly in the same dose interval has been obtained for gamma-ray-induced mutations to streptomycin nondependence in **E.coli** (Hussain & Ehrenberg 1979). Therefore, the deviation from linearity at low doses may well be a general phenomenon for radiation mutagenesis in pro- and eukaryotes mechanisms which are still unknown.

Thus, in addition to the enigmatic effects in Drosophila radiation genetics described earlier and considered by Novitski elsewhere (1976), the recent studies depicted have added some new ones among which the role of the locus c(3)G$^+$ in the zygotic processing of the radiation-induced premutational lesions is particularly intriguing. Taking account of more low yield of almost all radiation-induced genetic effects studied in the c(3)G genome than that in the wild-type one, it had been proposed that the c(3)G$^+$ locus is one of genetic elements controlling the recombination-dependent misrepair [so-called haploid-specific and mutagenic form of recombinational repair (Alexandrov 1986; Alexandrova & Alexandrov 1986)] non-efficiency of which [e.g., effect of c(3)G] must result in a noticeable reduction of the mutations recorded. It is this effect that has been found depicted in our researches. The premutational lesions which have not undergone the misrepair at the pronucleus stage are recognized and efficiently removed by diploid-specific error-free recombination repair just after the union of the maternal and paternal genomes occurs at the end of the first cleavage division (Alexandrova & Alexandrov 1986). The hypothesis about haploid-specific error-prone and diploid-specific error-free recombinational repair systems (among others occurring on the pronucleus-early cleavage stages and competing with each other for the same lesions) apparently may explain most, if not all, enigmas in Drosophila germ cell radiation mutagenesis.

References: Alexandrov, I.D. 1980, Int. J. Radiat. Biol. 37:183-188; _____ 1984, Mutat. Res. 127:123-127; _____ 1986, in: Recombinogenesis: Its Role in Evolution and Selection, Shtiinza, Kishinev; Alexandrova, M.V. & I.D. Alexandrov 1986, in: Recombinogenesis: Its Role in Evolution and Selection, Shtiinza, Kishinev; Baker, B.S., M. Gatti, A.T.C. Carpenter, S. Pimpinelli & D.A. Smith 1980, in: DNA Repair and Mutagenesis in Eukaryotes (Generoso, Shelby & de Serres, eds.), Plenum Press, NY pp189-208; Hanedle, J. 1974, Mol. Gen. Genet. 128:233-239; Hussain, S. & L. Ehrenberg 1979, Hereditas 91:111-116; Novitski, E. 1976, Science 194:1387-1390; Pimpinelli, S. et al. 1975, Atti Assoc. genet. ital. 20:26-28; Racine, R.R., A. Beck & F.E. Wuergler 1979, Mutat. Res. 63:87-100; Watson, W.A.F. 1972, Mutat. Res. 14:299-307.

Alexandrov, I.D. Research Inst. of Medical Radiology, Obninsk, USSR. Modification of radiation-induced rates of intra- and intergenic changes at the vestigial locus of **Drosophila melanogaster** by genotype, caffeeine, actino-mycin-D, sodium fluoride and radiation quality.

In the following, the spectrum and frequencies of the vestigial mutations recovered simultaneously with the black ones in the experiments described earlier (Alexandrov 1986) are presented. Each of the four hundred and sixty-seven vesitigal mutants scored was at first mated, as with the black ones, to b Pm/In(2LR)Cy, net dp^{tx1} Cy b pr Bl lt^3 cn^2 L^4 sp^2 tester flies of the appropriate sex to obtain in the end the vestigial homozygotes. At this step of the genetical analysis, all the vestigial mutations, as with the black ones, have been classified into 3 main and regularly occurring mutant types: (1) sterile F$_1$ visibles (SV) (301 out of 467 putative vestigial mutations scored), (2) transmissible visibles with recessive lethality (LV) (82 out of 166 fertile F$_1$ mutants), and (3) transmissible and viable in homozygote visibles (VV) (84 out of 166).

TABLE 1. LV^F = true LV^F without the "twin" vestigial mutants the number of which are given in the parentheses; micro-deletions = sterile and non-complementing (with the nw83b27, see Alexandrov & Alexandrova, this issue: Research Note) vestigial mutants without visible rearrangements; a.m.f. = average mutation frequency, locus/r x 10^{-8}.

Conditions of experiment #	VV^g	SV	LV^S	LV^F	VV^{ch} Micro de-letions	VV^{ch} Visible re-arrangements	Lost before analysis	Total a.m.f.
[# = radiation, dose, modifier used, genotype of male treated, No. of F$_1$ progeny scored]								
1. gamma-rays, 40 Gy, D-32, No. = 192939								
	6	55	6	13(7)	15	3In,lt	1	100
/a.m.f./=	/0.8/			/12.1/				/12.9/
2. gamma-rays, 40 Gy, D-18, No. = 156127								
	5	76		11(2)	11	2In,I T	1	107
/a.m.f./=	/0.8/			/16.2/				/17.1/
3. caffeine (0.2%) + gamma-rays, 40 Gy, D-32, No. = 91729								
	2	41		4	5	1 In,I T	1	55
/a.m.f./=	/0.6/			/14.2/				/15.0/
4. actinomycin-D (100 mg/ml) + gamma-rays, 40 Gy, D-32, No. = 69046								
	2	35		6(2)	7			50
/a.m.f/=	/0.7/			/17.4/				/18.1/
5. sodium fluoride (0.2%) + gamma-rays, 40 Gy, D-32, No. = 30861								
	3	12		3	2	1 In,I T		22
/a.m.f./=	/2.4/			/15.4/				/17.8/
6. 0.1, 0.35 and 0.85 MeV fission neutrons, 8-10 Gy, D-32, No. = 79839								
	1	25	2	6	1	1 In,I T		36
/a.m.f./=	/1.3/			/43.8/				/45.1/
7. ^{252}Cf, 14 Gy, D-32, No. = 24072								
	1	8		2(1)		1 In	1	13
/a.m.f./=	/0.3/			/3.6/				/3.9/
8. 0.85 MeV fission neutrons, 10 Gy + gamma-rays, 10 Gy, D-32, No. = 13377								
	1	7				1 In		9
/a.m.f./=	/3.7/			/29.9/				/33.6/
9. X- or gamma-rays, 40 Gy, c(3)G, No. = 151780								
	5	34	1	11(4)	3	8 In,I T,I Tp		64
/a.m.f./=	/0.8/			/9.7/				/10.5.
10. 0.35 and 0.85 MeV fission neutrons, c(3)G, 5 Gy, No. = 20605								
	1	5						6
/a.m.f./=	/9.7			/48.5/				/58.2/
............................, ..., 10 Gy, No. = 10357								
	0	4		1				5
/a.m.f./=	/0.0/			/48.3/				/48.3/

According to date of the further analysis of LV, about 11% of them (9 out of 82) were LV^S (see Alexandrov 1986), about 22% (16 out of 73) proved to be so-called "twins" vestigical mutants (i.e., those having an independent recessive lethal separated from the vestigial mutation crossing over in the vg^X/Bl female heterozygotes) and the rest of the mutants considered were a true LV^F due to all kinds of the chromosome rearrangements revealed by means of genetical (Alexandrov 1984) and cytogenetical (Alexandrov & Alexandrova, this issue: New Mutants) studies. Further, polytene chromosome analysis of the 69 VV preserved was also carried out, and the number of the VV associated (VV^{ch}) with the visible chromosome rearrangements was detected (Alexandrov & Alexandrova, this issue: New Mutants). In addition, the pattern of the complementation for the remaining VV was determined. This test was made possible by the finding that gamma-ray induced mutant nw83b27 had complementation properties when compounded to the pseudo- (vg, np) or to some of the homo-alleles (nw) producing a phenotype more normal than that of either allele (Alexandrov & Alexandrova, this issue: Research Note). On the other hand, no nw83b27 was found to complement with deficiency the part or whole locus in question, i.e., heterozygotes nw83b27/vg deficiency has the nw83b27 phenotype. Using this approach, the remaining VV were further classified into associated (VV^{ch}) and unassociated (VV^g) with micro-deletions vg visibles.

Analysis of the relative proportion of a true gene mutation (VV^g) versus all chromosome (SV, LV^S, LV^F and VV^{ch}) alterations at the locus of interest after action of the variable studied (Table 1) shows that VV^g arise most rarely in the wild-type (D-32, D-18) and c(3)G male germ cells (the post-meiotic stages as a whole) after gamma- or neutron-irradiation, and the chromosome rearrangements of all kinds found to be predominant in the spectrum of the vg mutations induced. Pretreatment of the D-32 males with caffeine or actinomycin-D do not appear to modify this picture. On the other hand, sodium fluoride increases significantly the yield of VV^g, but no chromosome, vestigial mutations (of the black ones, Alexandrov 1986). It is important that the mutagenic effect of the consecutive irradiation by neutrons and gamma-rays found to be nearly twice as high as one should expect for the additive action of the radiations studied. This finding for the vestigial VV^g is analogous to that for VV^g at the black locus (Alexandrov 1986). Like the latter, vestigial VV^g induced by fission neutrons in the c(3)G sperm arise more often than that induced by gamma-rays in the same germ cells under otherwise equal conditions. Thus, neutrons appear to be more efficient than gamma-rays for producing VV^g mutations in the c(3)G germ cells and this picture is opposite to that found for the wild-type germ cells. The researches to verify these first observations are in progress now.

References: Alexandrov, I.D. 1984, DIS 60:45-47; ———— 1986, DIS 63:19-21.

Alexandrov, I.D. and M.V. Alexandrova.
Research Inst. of Medical Radiology, Obninsk, USSR. A new nw allele and interallelic complementation at the vg locus of **Drosophila melanogaster.**

Among viable pseudoalleles at the vg locus of **Drosophila melanogaster** the nw known to be the most extreme mutant occupying the farthest distal site on the map of the gene in question (Carlson, Ferriola & Schuchman 1980) and bringing about the most marked phenotype in which the four major effects are all recessive and result in completely lacking of wings and halteres, erected postscutellars and sterility of the females (Lindsley & Grell 1968). The complex phenotype of the nw shows that the product of the vg$^+$ gene seems to display at least the four different metabolic functions which are simultaneously affected by the lesions at the distal domain of the gene.

The nw alleles with two or three functions affected only appear to be unknown as yet (Lindsley & Grell 1968). However, five exactly the same nw alleles (3 of them have been lost unfortunately for some reasons) among forty-five radiation-induced mutations of interest have been recovered in different experiments described elsewhere (Alexandrov & Alexandrova, this issue: New Mutants). All five nw alleles in homozygote are fertile and one of them (nw83b27) have a normal postscutellar as well. Thus, affecting the wings and halteres, the nw83b27 does not disrupt the other two functions. The further crosses revealed that heteroallele np/nw83b27 had fully wild-type phenotype whereas vg/nw83b27 show normal halteres and far less mutant wings varying from s2 in the females to np in males. Recurrence of the wings is, however, accompanied by their new defects, namely, the wings found to be often crumpled and/or blistered. The complementation pattern for the radiation-induced fertile vg mutations, when compounded to the nw83b27, proved to be the same (Alexandrov & Alexandrova, this issue: New Mutants). On the other hand, only some of the radiation-induced nw homoalleles unassociated with visible chromosome rearrangements are found to complement with nw83b27 whereas almost all radiation-induced pseudoalleles (s, np), here also unassociated with rearrangements, are complementary ones. It is important to note that none of 15 apparent vg deficiencies studied are complementing with the nw83b27. Hence it follows that, first, the

nw83b27 appear to complement only with the true gene mutations at the locus in question and, second, test on the complementation with nw83b27 seems to make it possible to tell the gene mutations from micro-deficiencies within the locus under study. These findings are, in principle, analogous to those for the white locus described earlier (Green 1959). Thus, the discovery of the second genetic system which shows the distinctive complementation pattern for gene mutations and microdeletions is essentially offering the possibility of studying the mutagenic action of the harmful environs simultaneously on the gene and chromosome levels in Drosophila.

References: Carlson, E., P. Ferriola & E. Schuchman 1980, DIS 55:23-24; Green, M.M. 1959, Z. indukt. Abstamm. -u. Vererb. 90:375-384; Lindsley, D.L. & E.H. Grell 1968, Carn. Inst. Wash. Publ. 627.

Aparisi, M.L. and C. Nájera. Universidad de Valencia, Spain. Location of eye colour mutants from natural populations of **D.melanogaster.**

A great quantity of genetic variability exists in natural populations. Nájera (1985) studied the genetic variability with respect to mutations affecting both eye colour and eye morphology in six natural populations of **D.melanogaster.** The flies were captured in three different places: wine cellar, vineyard and pinewood; at two different seasons of the year: Spring and Autumn.

By means of allelism tests inside and between populations, 87 different mutants were detected. Some of these mutations (43) had been localized in their linkage group and mapping after making the allelism crosses with all the eye mutations located in the same chromosome.

Table 1. Frequencies of the eye mutants in the different natural populations (number of mutations/number of chromosomes analyzed).

	CELLAR		VINEYARD		PINEWOOD	
	Autumn	Spring	Autumn	Spring	Autumn	Spring
Described mutants:						
bw(brown,2:104.5)	---	0.0063(2/320)	---	0.0036(1/280)	---	---
ca(claret,3:100.7)	---	---	---	0.0036(1/280)	---	---
cd(cardinal,3:75.7)	0.0037(1/272)	0.0125(4/320)	---	0.0036(1/280)	0.0111(2/180)	0.0075(2/268)
cn(cinnabar,2:57.5)	---	0.0031(1/320)	0.0049(1/204)	---	---	---
dke(dark eye,2:73)	0.0037(1/272)	0.0094(3/320)	---	---	0.0056(1/180)	---
g(garnet,1:44.4)	---	0.0031(1/320)	0.0049(1/204)	---	---	---
Hn^{r3}(Henna recessive3,3:23)	0.0073(2/272)	0.0031(1/320)	---	0.0036(1/280)	---	0.0075(2/268)
mah(mahogany,3:88)	---	0.0094(3/320)	---	---	---	0.0037(1/268)
p(pink,3:48)	---	---	---	---	---	0.0037(1/268)
pn(prune,1:0.8)	0.0037(1/272)	0.0063(2/320)	---	0.0036(1/280)	---	0.0037(1/268)
rb(ruby,1:7.5)	---	---	---	0.0036(1/280)	---	---
rs^2(rase2,3:35)	---	---	---	0.0036(1/280)	---	---
se(sepia,3:26)	0.0073(2/272)	0.0063(2/320)	0.0049(1/204)	---	0.0056(1/180)	---
sf(safranin,2:71.5)	0.0257(7/272)	0.0313(10/320)	0.0098(2/204)	0.0286(8/280)	0.0222(4/180)	0.0149(4/268)
swb(strawberry,1:2.2)	0.0037(1/272)	---	---	---	---	---
v(vermilion,1:33)	---	0.0063(2/320)	0.0049(1/204)	0.0036(1/280)	---	0.0037(1/268)
vi(vin,3:36.3)	0.0037(1/272)	0.0031(1/320)	---	---	---	---
w(white,1:1.5)	0.0037(1/272)	0.0063(2/320)	---	---	---	---
New mutants:						
alm(almond,3:25.6)	0.0037(1/272)	---	---	---	---	---
ches(chestnut,2:101.8)	0.0037(1/272)	---	---	---	---	---
dch(dark chestnut,3:25.7)	---	---	---	0.0036(1/280)	0.0056(1/180)	---
dr(dark-red,2:11.1)	---	0.0031(1/320)	---	---	---	---
gra(grape,3:34.7)	0.0037(1/272)	---	---	---	---	---
ifa(irregular facet,3:23.4)	---	---	0.0049(1/204)	---	---	---
jew(jewel,2:107.5)	0.0037(1/272)	---	---	0.0036(1/280)	---	---
man(mandarin,2:54.3)	---	---	---	---	---	0.0075(2/268)
pur(purplish ruby,3:39.5)	0.0037(1/272)	---	---	---	---	---
sb(spoony-brown,3:38.3)	0.0037(1/272)	---	---	---	0.0056(1/180)	---

To localize each mutation in its respective chromosome, one double recessive mutant was employed: vg.e[11] (vestigial, 2:67.0; ebony, 3:70.7). Standard eye colour mutants from laboratory stocks were used for the allelism tests and two double recessive mutants were employed for mapping: b.vg (black, 2:48.5; vestigial, 2:67.0) and e.tx (ebony, 3:70.7; taxi, 3:91.0).

Table 1 shows the distribution of alleles of described mutations found with the allelism tests and the new mutants found in these natural populations.

The great number of alleles of eye mutations and the existence of mutations not described previously and found in these populations confirm the hypothesis of Gassparian & Fadjami (1974) about the higher mutability of eye colour loci.

References: Gassparian, S. & S. Fadjami 1974, Genetics 77:524; Nájera, C. 1985, Ph.D. Thesis, Valencia.

Aparisi, M.L. & C. Nájera. Universidad de Valencia, Spain. Quantitative estimations of the red and brown pigments of eye colour mutant from natural populations of Drosophila melanogaster.

Quantitative estimation of the red and brown eye pigments of D.melanogaster in eye colour mutants has normally been carried out following Clancy's "double extraction" procedure (1942). Ephrussi & Herold (1944) systematically studied Clancy's procedure and found it inappropriate for analysis of the brown pigment in mutants having also drosopterins. Real et al. (1985) revised the previous procedures for selective extraction of the eye pigments and developed one method for quantitative estimation of the red pigments, and modified the procedure of Butenandt et al. (1960) to quantify the brown pigment.

We have used this method to quantify both red and brown pigments of eye colour mutants coming from six natural populations of D.melanogaster, captured in three different places: wine cellar, vineyard and pinewood; in two different seasons of the year: Spring and Autumn (Nájera 1985).

In total, 43 strains were analyzed. Ten of them are new mutants and the rest (33) are alleles of known mutations. Using this method, we have also calculated the amount of red pigment of all the standard mutants of which we have found some allele in the natural populations cited above.

Table 1. Quantitative estimation of the red pigment of eye colour mutants from natural populations of **D.melanogaster.**

Stock mutants	Absorbance at 480nm	Relative % with respect to Oregon strain	Stock mutants	Absorbance at 480nm	Relative % with respect to Oregon strain
bw	0.010±0.001	1.44±0.13	dke^{ca}	0.276±0.012	41.82±1.85
ca	0.069±0.003	10.38±0.48	g^{cs}	0.210±0.016	31.82±2.44
cd	0.396±0.007	60.00±0.94	g^{va}	0.046±0.003	6.93±0.43
cn	0.574±0.008	86.97±1.26	Hn^{r3} vs	0.273±0.014	41.29±2.18
dke	0.481±0.006	72.92±0.91	Hn^{r3} ps	0.204±0.003	30.83±0.45
g	0.107±0.004	16.18±0.68	Hn^{r3} cs	0.156±0.007	23.60±1.14
Hn^{r3}	0.177±0.003	26.82±0.41	Hn^{r3} ca	0.163±0.004	24.73±0.60
mah	0.436±0.005	66.06±0.70	mah^{cs}	0.580±0.013	87.88±2.00
p	0.211±0.004	31.93±0.54	p^{ps}	0.122±0.005	18.41±0.72
pn	0.109±0.004	16.40±0.68	pn^{ca}	0.090±0.004	13.64±0.53
rb	0.135±0.002	20.45±0.28	pn^{cs}	0.090±0.004	13.64±0.53
rs^2	0.284±0.007	43.07±1.11	rb^{vs}	0.066±0.001	9.96±0.12
se	0.065±0.001	9.78±0.17	rs^2 vs	0.129±0.009	19.58±1.43
sf	0.117±0.013	17.69±1.92	se^{va}	0.051±0.001	7.69±0.12
v	0.523±0.017	79.17±2.71	se^{cs}	0.053±0.002	7.96±0.34
vi	0.112±0.007	16.97±1.06	se^{pa}	0.049±0.004	7.35±0.54
Natural mutants			sf^{vs}	0.288±0.020	43.56±3.01
bw^{vs}	0.008±0.001	1.25±0.12	sf^{va}	0.323±0.010	48.94±1.49
bw^{cs}	0.008±0.001	1.21±0.19	sf^{ca}	0.502±0.025	75.98±3.82
ca^{vs}	0.105±0.006	15.91±0.91	sf^{pa}	0.575±0.011	87.05±1.65
cd^{ps}	0.535±0.008	81.10±1.25	sf^{cs}	0.607±0.017	91.93±2.51
cd^{ca}	0.622±0.014	94.19±1.82	swb^{ca}	0.281±0.005	42.58±0.59
cd^{pa}	0.764±0.017	115.76±2.23	v^{va}	0.736±0.020	111.48±3.06
cn^{cs}	0.479±0.018	72.61±2.72	v^{ps}	0.661±0.014	100.15±1.87
dke^{cs}	0.540±0.027	81.74±4.08	vi^{cs}	0.220±0.006	33.29±0.87

Table 2. Quantitative estimation of the brown pigment of eye colour mutants from natural populations of **D.melanogaster.**

Natural mutants	Absorbance at 492nm	Relative % with respect to Oregon strain
bw^{vs}	0.267±0.004	82.92±1.24
bw^{cs}	0.267±0.010	82.76±2.95
ca^{vs}	0.078±0.011	24.23±3.42
cd^{ps}	0.054±0.003	16.62±0.78
cd^{ca}	0.057±0.003	17.70±0.93
cd^{pa}	0.054±0.003	16.77±0.93
cn^{cs}	0.027±0.002	8.39±0.63
dke^{cs}	0.296±0.018	91.77±5.00
dke^{ca}	0.327±0.006	101.40±1.71
g^{cs}	0.167±0.006	51.87±1.87
g^{va}	0.073±0.001	22.67±0.31
Hn$^{r3\ vs}$	0.363±0.005	112.58±1.40
Hn$^{r3\ ps}$	0.344±0.005	106.84±1.56
Hn$^{r3\ ca}$	0.279±0.003	86.49±0.78
Hn$^{r3\ cs}$	0.346±0.008	107.46±2.49
mah^{cs}	0.497±0.020	154.20±6.06
p^{ps}	0.095±0.005	29.35±1.40
pn^{ca}	0.205±0.001	63.67±0.32
pn^{cs}	0.107±0.003	33.08±0.78
rb^{vs}	0.088±0.004	27.33±1.24
rs$^{2\ vs}$	0.169±0.001	52.33±0.16
se^{va}	0.364±0.003	112.89±0.79
se^{cs}	0.190±0.003	58.85±0.78
se^{pa}	0.277±0.001	86.03±0.32
sf^{vs}	0.305±0.018	94.72±5.59
sf^{va}	0.357±0.011	110.72±3.27
sf^{ca}	0.339±0.006	105.28±1.86
sf^{pa}	0.404±0.004	125.47±1.25
sf^{cs}	0.439±0.003	136.34±0.94
swb^{ca}	0.272±0.006	84.32±1.71
v^{va}	0.034±0.004	10.41±1.09
v^{ps}	0.023±0.003	6.99±0.78
vi^{cs}	0.208±0.018	64.60±5.59

Table 3. Quantitative estimation of the red and brown pigments of the new mutants from natural populations of **D.melanogaster.**

Strain	Absorbance at 480nm	Rel. % with respect to Oregon strain
Red pigments:		
ches^{ca} (2:101.8±3.0)	0.598±0.020	90.56±2.63
gra^{ca} (3:34.7±1.3)	0.378±0.010	57.24±1.48
sb^{ca} (3:38.3±0.6)	0.398±0.009	60.35±1.12
pur^{ca} (3:39.5±0.2)	0.512±0.007	77.54±1.10
jew^{ca} (2:107.5±1.0)	0.314±0.005	47.54±0.72
alm^{ca} (3:25.6±1.7)	0.399±0.015	60.46±2.20
ifa^{va} (3:23.4±0.4)	0.354±0.019	53.60±2.90
dr^{cs} (2:11.1±1.6)	0.444±0.008	67.20±1.24
dch^{vs} (3:25.7±1.4)	0.617±0.026	93.45±3.46
man^{ps} (2:54.3±2.0)	0.435±0.006	65.91±0.88

Strain	Absorbance at 492nm	Rel. % with respect to Oregon strain
Brown pigments:		
ches^{ca}	0.263±0.003	81.53±0.78
gra^{ca}	0.298±0.005	92.39±1.40
sb^{ca}	0.137±0.007	42.39±2.02
pur^{ca}	0.393±0.015	121.90±4.51
jew^{ca}	0.125±0.003	38.67±0.78
alm^{ca}	0.369±0.004	141.44±1.09
ifa^{va}	0.227±0.001	70.50±0.31
dr^{cs}	0.313±0.004	97.05±1.09
dch^{vs}	0.476±0.015	147.67±4.50
man^{ps}	0.058±0.004	18.01±1.24

Table 1 shows the quantitative estimation of red pigment (absorbance at 480 nm) and the relative percentage with respect to Oregon strain (wild strain) and Table 2 shows the quantitative estimation of the brown pigment (absorbance at 492 nm) and the relative percentage with respect to Oregon strain, for the known mutations.

In Table 3, the quantitative estimation of both pigments for the new mutants is reflected.

These analyses indicate that the amount of both red and brown pigments can vary between the different alleles of the same mutation; this is a new demonstration of the great variability of the eye colour loci.

References: Butenandt, A., E. Biekert, H. Kubler & B. Linzen 1960, Hoppe seyler's Z. Physiol. Chem. 319:238-256; Clancy, C.W. 1942, Genetics 27:417-440; Ephrussi, B. & J.L. Herold 1944, Genetics 29:148-175; Nájera, C. 1985, Ph.D. Thesis, Valencia; Real, M.D., J. Ferre & J.L. Mensua 1985, DIS 61:198-199.
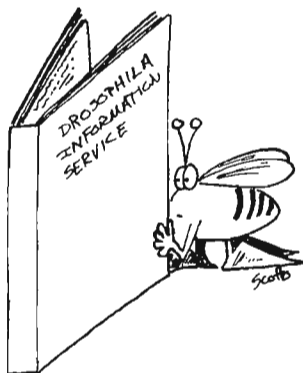
Original Graphics
by
Scott P. McRobert

Arbona, M. and R. de Frutos. Universidad de Valencia, Spain. Induction of new puffs during recovery from anoxia in **Drosophila subobscura.**

We exposed individuals at Oh prepupa (just at the beginning of prepupation) to different periods of anoxia, 4 and 24 hr, before analysing the effects of recovery from anoxia on the gene expression in polytene chromosomes of **Drosophila subobscura.** In both cases we observed: (1) at a morphological level the larva has interrupted its development, i.e., has not attained the pupal stage; (2) at a level of gene expression a stop is observed, i.e., the same puffs appear as at Oh prepupa though 4 and 24, respectively, have passed.

Secondly, the effects of recovery from anoxia on gene expression were analysed. To attain this purpose, after exposing individuals at Oh prepupa to 4 and 24 hr of anoxia, they are allowed to recover by keeping them in air in short periods of time. The appearance of 7 new puffs is observed: 2C, Chromosome A; 18C and 27A, Chromosome J; 54CD, Chromosome E; 85AB, 89A, and 94A, Chromosome O. These puffs are the same which appear after exposing these individuals at Oh prepupa to heat shock at 31°C.

Armstrong, E., L. Bass and K. Staker. University of Maryland, College Park. Transovarial transmission and longevity of **Drosophila ananassae** and **Drosophila simulans** infected with **Nosema kingi.**

Eleven species of Drosophila have been reported to be susceptible to the microsporidium, **Nosema kingi** (Armstrong 1976; Armstrong et al. 1986). Of these species, **Drosophila ananassae** and **Drosophila simulans** are presently maintained in our laboratory. The objectives of this report were to determine if the parasite could be transovarially transmitted and determine its effect on the longevity of these flies.

Eggs from heavily infected **D.ananassae** and **D.simulans** were retrieved, separated into lots of 20-25 and placed directly on noninfected regular stock diet as described previously (Armstrong 1976). Offspring from the eggs were killed upon emergence and examined for the presence of the parasite.

Two percent (n=328) of the offspring from eggs laid by infected **D.ananassae** were parasitized while 4% (n=350) of the offspring from **D.simulans** were infected upon emergence. In both species, the abdomens of infected flies were swollen.

Table 1. Effects of **Nosema kingi** on the longevity of **Drosophila ananassae** and **D.simulans** at 25±1°C. Number in parentheses represents flies used in calculations.

Observation X̄ longevity(days) ± SE	D.ananassae		D.simulans	
	Control	Infected	Control	Infected
♀♀	23.3±0.8 (72)	17.6±0.8* (50)	24.3±1.1 (58)	14.1±0.5* (66)
♂♂	33.3±1.7 (40)	14.4±1.0* (38)	36.2±2.1 (51)	15.2±1.1* (46)
♂♂ + ♀♀	28.8±2.1 (112)	16.2±0.6* (88)	29.9±1.3 (109)	15.7±1.3* (112)

* = significant at 1% level using Student's t-test.

N.kingi, in each species, also significantly reduces the longevity of the flies (Table 1). Males tended to live longer than females although in **D.ananassae** the infected females lived longer than the males.

References: Armstrong, E. 1976, Z. Parasitenk 50:161-165; _____, L. Bass, K. Staker & L. Harrell 1986, J. Invertebr. Pathol. 48:124-126.

Band, H.T. Michigan State University, East Lansing. Attraction of **chymomyzids** to a tree wound.

Attraction of **chymomyzids** to cut wood is documented (Steyskal 1952; Wheeler 1952; Watabe 1985; Band 1986). In summer 1986 multiple **chymomyzid** species were attracted to a single wound, 5 x 15 in, on a wild cherry tree along the trail to Bald Knob approximately 2 mi from the Mt. Lake Biological Station in Virginia's Allegheny Mountains. This was the only wound ringed by sap exudate in a stand of trees, some of which also had wounds. At this more shaded location flies could be collected and/or observed throughout the day in contrast to morning and evening displays on the cut firewood in an open area the year before (Band 1986) or in timberyards in Japan (Watabe 1985).

Species collected during the period of observations, July 15-25, are shown in Table 1. Males outnumber females (Watabe 1985; Band 1986). No eggs or larvae were found in the sap exudate, suggesting it was primarily a male aggregation site (Thornhill & Alcock 1983). Matings were observed July 16-25, including attempted mate stealing. Mating pairs captured included **C.aldrichii** and **C.procnemoides.**

Table 1. Chymomyzid species collected from 15 through 25 July 1986 on a wound on a wild cherry tree near Mt. Lake Biological Station in Virginia.

dates	Species	no. males	no. females
15-17	C.caudatula	4	1
15-25	C.procnemoides	12	7
17-25	C.aldrichii	10	1
15	C.amoena	1	0
17	sp. #2	1	0
		28	9

The identified species, **C.caudatula, C.aldrichii, C.procnemoides** and **C.amoena**, represent 3 species lineages (Okada 1976). Both unidentified species collected at MLBS in 1985 fall into a separate lineage since both have a blackish frons and light tan mesoscutum; they differ in tarsal traits. Therefore, both are different from **chymomyzid** sp. A and sp. B of Wheeler (1952).

The two captured mating pairs, **C.aldrichii** and **C.procnemoides**, are in the same lineage. This indicates females can discriminate between simple courtship patterns and calls into question species' selection (Gould 1982). Interspecific fights occurred, but neither combatant left the site. Species strongly attracted to the site, **C.caudatula, C.procnemoides,** and **C.aldrichii,** were captured repeatedly. Species rarely attracted, **C.amoena** and sp. #2, were captured only once or seldom seen. Again, **C.amoena** was conspicuous by its banded wing pattern.

The fact that dark and light forms of both **C.aldrichii** and **C.procnemoides** were captured points to the need for a key in which color is not a primary taxonomic discriminant. It also questions species' stability.

Male aggregation for the purpose of attracting mates has been described among **tephritids** (McDonald 1987; Thornhill & Alcock 1983). The latter also include mate stealing as a cost of male aggregation. The lek behavior of Hawaiian Drosophila males has also been described as male aggregations (Thornhill & Alcock, ibid.).

Acknowledgements: Thanks are extended to Marshall Wheeler for identifying the dark forms of **C.aldrichii** and **C.procnemoides**. Charles Werth and Rob Simpson identified the lek tree as a wild cherry. A fellowship from the University of Virginia for research in summer 1986 is gratefully acknowledged. Thanks are also extended to Blaine Cole, Director, and J.J. Murray, Biology Dept. Chairman, for providing research space.

References: Band, H.T. 1986, DIS 63:26-27; Gould, S.J. 1982, in: Perspectives on Evolution, Milkman (ed.), Sinauer, Sunderland, MA, pp. 83-104; McDonald, P.T. 1987, Ann. Entomol. Soc. Amer. 80:17-20; Okada, T. 1976, Kontyu, Tokyo 44:496-511; Steyskal, G. 1952, Letter to Marshall Wheeler; Thornhill, R. & J. Alcock 1983, Insect Mating Systems, Harvard Univ. Press, Cambridge; Watabe, T. 1985, DIS 61:183-184; Wheeler, M. 1952, Univ. Texas Publ. 5204:162-218.

Band, H.T. Michigan State University, East Lansing. Pgm in Virginia **Chymomyza amoena** populations.

The frequencies of Pgm alleles were surveyed in four **C.amoena** populations in summer 1986. These were populations emerging from apples collected at Pamplin (near Lynchburg), in a Danville neighborhood, from a Blacksburg orchard and at a site along Rt. 700 to Mt. Lake Biological Station. The first two sites represent Southside Virginia localities, the latter two Western Virginia localities. Times of collections varied. The Pamplin collection was made in early June, the Danville collections in mid and late June, the Rt. 700 collections in late June and early July, the Blacksburg collection in early July. Due to low egg hatchability in later collections from Danville, Blacksburg and Rt. 700 sites, F_1's from initial emergees, also grown on apples, were included to boost sample sizes. Table 1 compares the frequencies of Pgm alleles in the four populations. No significant differences emerge (d.f. = 6, G = 6.04). The average frequency of F is 74±3% and of M is 25.5±3%.

However, if genotypic frequencies are compared between populations, significant differences emerge (d.f. = 9; G = 17.41; p<0.05). Genotypes among the sampled populations are shown in Table 2. When pair-wise comparisons are made, the Pamplin population is found to differ significantly from both the Danville and Rt. 700 populations. Comparisons among Danville, Blacksburg and Rt. 700 populations are not significant. The distance between Pamplin and Danville is about 75 mi, between Danville and Blacksburg 130 mi, Blacksburg and the Rt.700 site at least 11 mi. The M allele appears responsible for the genotypic differences. In Pamplin and Blacksburg populations, the frequency of MM homozygotes is reduced; the heterozygote FM is the major source of the M allele. In the Danville and Rt.700 populations, the major source of the M allele is from the homozygotes. Whether or not these results are due to small sample sizes remains unknown. Whether or not frequencies of F and M alleles shift between summer and winter, as in Michigan, also remains unknown (Band & Band 1987).

Table 1. Frequencies of Pgm alleles in four Virginia **C.amoena** populations, summer 1986.

Location	n	2n	F+	F	M
Pamplin	33	66	1	52	13
Danville	22	44		30	14
Blacksburg	25	50		39	11
Rt. 700	24	48	_	32	16
	104	208	1	153	54

Table 2. Genotypes for PGM allozymes in the Virginia **C.amoena** populations.

Location	n	F+F	FF	FM	MM
Pamplin	33	1	19	13	0
Danville	22		13	4	5
Blacksburg	25		15	9	1
Rt. 700	24	_	13	6	5
	104	1	60	32	11

References: Band, H.T. & R.N. Band 1987, Experientia (in press).

Banerjee, Snigdha, S.K. Banerjee and A.S. Mukherjee. University of Calcutta, India. Separation of two populations of nascent replicons in polytene chromosomes in **Drosophila hydei** by agarose gel electrophoresis.

Replication of chromosomal DNA occurs exclusively during the S-phase of the cell cycle (Howard & Pele 1953) at numerous small replicating units. These replicating units are termed "replicons" (Taylor 1963). Within each replicon DNA replication starts at a fixed origin and progresses bi-directionally (Hubermann & Riggs 1968). Furthermore, the size of the replicons varies in different cell types with different growth conditions (Blumenthal et al. 1973; Callan 1972) and the size of the replicons are mostly in the range of 15-100 μm or 50-300 kbs (Hand 1978).

Two distinct types of replicons have been recovered in highly polytenized nuclei in salivary glands of Drosophila by fibre autoradiography (Lakhotia & Sinha 1983) at different stages of the S-period. However, the work of Lakhotia & Sinha differs from earlier DNA fibre autoradiographic study (Steinmann 1981).

In the present work, we re-investigated the nature of nascent replicons in unsyncrhonized salivary glands of **D.hydei**, by 1.5% agarose gel electrophoresis. Third-instar larvae were
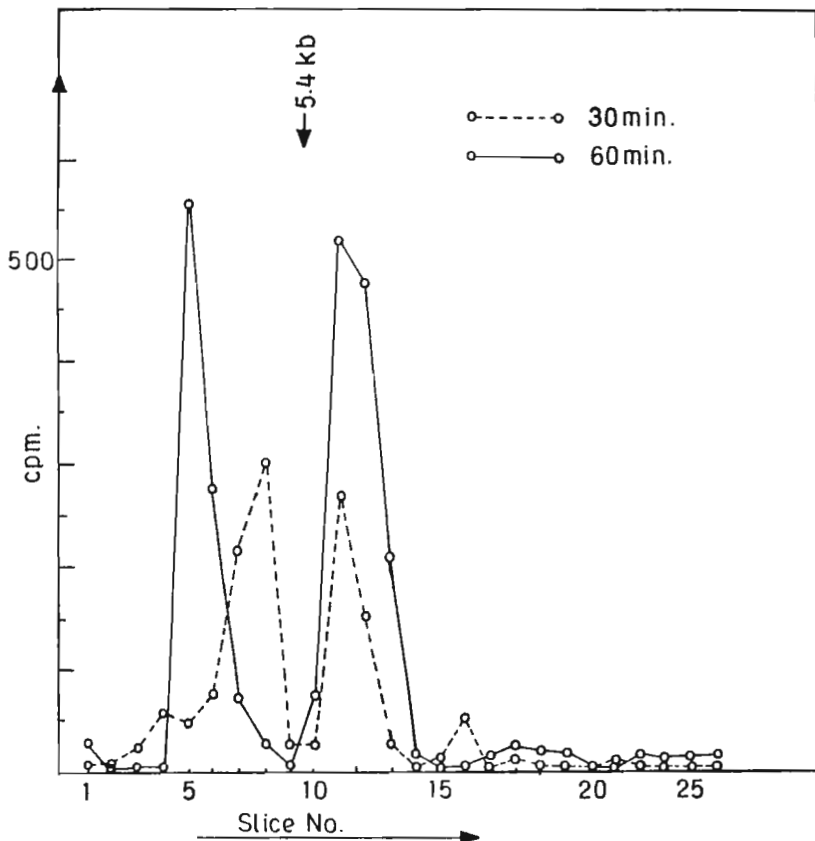
Figure 1. Electrophoresis separation of labelled salivary gland DNA was extracted at 25°C and analysis in 1.5% agarose gels. The 30 min and 60 min separation are demonstrated by two peaks. First peak located at slices 5-8 and second peak located at slices 10-15. Arrow indicates the position of marker DNA.

dissected out and salivary glands were incubated in ^{3}H-TdR for two different time periods (i.e., 30 min and 60 min). After incubation, glands were immediately transferred to lytic buffer containing 0.05M Tris-HCl (pH 7.4), 0.002M Na$_2$EDTA and 1% SDS and incubated at 25°C for 3 hrs. After incubation in the buffer, the lysate was run in 1.5% agarose flat-bed gel for electrophoresis with 150 m volts for 3 hrs. After completion of electrophoresis 3mm slices were cut from the gel and DNA was eluted and the radioactivity was counted in a Scintillation counter as described by Lonn (1980).

The results showed two distinct populations of labelled DNA molecules whether glands were incubated with ^{3}H-TdR for 30 mins or 60 min (Figure 1). The position of the first peak at 60 min changed toward high molecular weight, but the low molecular weight peak did not. However, peak heights were higher with 60 mins as expected.

The non-ligated labelled nascent DNA is double-strand DNA and migrates to slice no. 4 to 15, and it has an apparent size ranging between 10 kb to 3.2 kb.

The appearance of two peaks suggest the existence of two different categories of active replicons in polytene nuclei of **D.hydei**. The first type, which is located between slice no. 5-8, is high-molecular weight and faster replicating in nature, but the second type which is located between slice no. 10-15 is slow replicating and of low molecular weight.

References. Blumenthal, A.B., H.J. Kriegstein & D.S. Hogness 1973, Cold Spring Harbor Symp. Quant. Biol. 38:205-223; Callan, H.G. 1972, Phil. Trans. Roy. Soc. (Lond) 181:19-41; Hand, R. 1978, Cell 15:317-325; Howard, A. & S.R. Pele 1953, Heredity 6:261-273; Hubermann & Riggs 1968, J. Mol. Biol. 32:327-341; Lakhotia, S.C. & P. Sinha 1983, Chromosoma 88:265-276; Lonn, U. 1980, Chromosoma 77:29-40; Steinmann, M. 1981, Chromosoma 82:289-307; Taylor, J.H. 1963, J. Cell Comp. Physiol. 62(supp) 1:73-86.

Baricheva, E.M., V.F. Semeshin, I.F. Zhimulev and E.S. Belyaeva. Inst. of Cytology & Genetics, Novosibirsk, USSR. Electron microscopical mapping of some puffs of **Drosophila melanogaster** polytene chromosomes.

In this paper electron microscopical (E.M.) mapping of puffs, developing in the regions 3CD, 35AB, 56EF, 85F, 90B and 99EF of **Drosophila melanogaster** polytene chromosomes of Batumi stock is presented. Techniques of squashed chromosome preparations for E.M. have been described earlier (Semeshin et al. 1982).

The 3CD region. In the 3C region 4 single bands and 4 "doublets", and in the 3D region 5 bands with one to be doublet were found by Bridges (Figure 3a, see Lindsley & Grell 1968). By E.M. in the 3C region we revealed 8 bands (Figure 1b): 3C4 and 3C8 bands are not seen, 3C5-6 and 3C9-10 "doublets" (Fig. 1d and 1b, respectively) are seen as pairs of tightly adjoined bands. The 3C1, 3C2-3 and 3C11-12 bands look like single bands (Fig. 1b, c, d). Thin band 3C11-12 is seen only on some stages of development and only in stretched chromosomes (Fig. 1b). By E.M. in 3D region we revealed 5 single bands: the band 3D5-6 is not a doublet. In the 3CD region of larvae (Fig. 1d) puff develops from 3C11-12 band (marked by black triangle on Fig. 1). In 4-11 hr prepupae puff develops from bands 3D1 and 3D2 (marked by open triangle on Fig. 1). The 3C9-10 and 3D3 bands restrict the puffing zone (Fig. 1e). Thus, E.M. mapping data confirm Korge's suggestion (Korge 1977) that in larvae and prepupae puffs develop from different bands.

The 35AB region. In the 35B region we found 6 bands, 4 of them were doublets (Figure 2a). E.M. reveals all bands mapped by Bridges: the 35B1-2 "doublet" is pair of tightly adjoined bands 35B1 and 35B2 (Fig. 2d), which is clearly seen only on some preparations. Other bands look like single bands. By E.M. we revealed that in larvae puff develops from one loose band 35B3 (marked by black triangle on Fig. 2). All other bands of the region preserve their morphology.

In prepupae (Fig. 2e) puffing pattern drastically changes: band 35B3 becomes compact and puff develops from the 35B1 band. The 35B2 band does not take part in puff formation. At this stage we also saw the lengthening of space between two dense bands which correspond to the Bridges 35B1-2 doublet (marked by open triangle on Fig. 2).

The 56EF region. In the 56E region there are 4 bands with two of them to be doublets on Bridges map (Fig. 3a). By E.M. we reveal only two single bands (Fig. 3b). Puff formation in the 56E region starts with irregular decondensation of the 56E1-2 and 56E4-5, which look like grains of compact material in decondensed chromatin (Fig. 3c). When puff reaches its maximum, decondensation becomes more regular and involves bands of the 56D region (Fig. 3d). In the 56F region Bridges found 11 bands, six of them to be doublets (Fig. 3a). By E.M. in the 56F1-9 region we revealed a group of tightly adjoining bands, at least 5 (Fig. 3b), but, in some other cases, more (Fig. 3e). In the 56F10-17 region we revealed 8 single bands; this is in a good agreement with Sorsa's data (Sorsa 1983). At all stages of larval and prepupae development when polytene chromosomes are suitable for observation, we did not detect puffing activity in the 56F region.
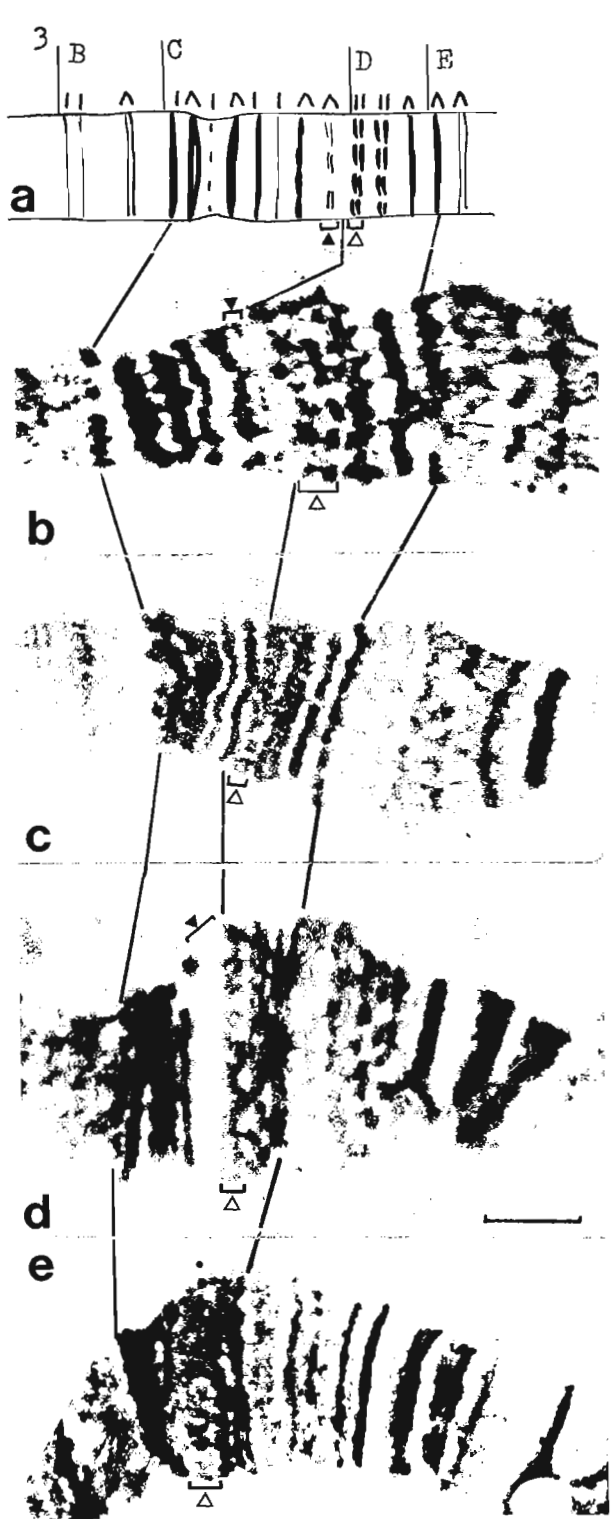
Figure 1. Puffs formation in the region 3CD. a =
Bridges map; b-e = EM map: b,c = 0-h prepupae;
d = PS1-2; e = 6-h prepupae. Δ = indicates bands 3D1
and 3D2. ▲ = indicates band 3C11-12.
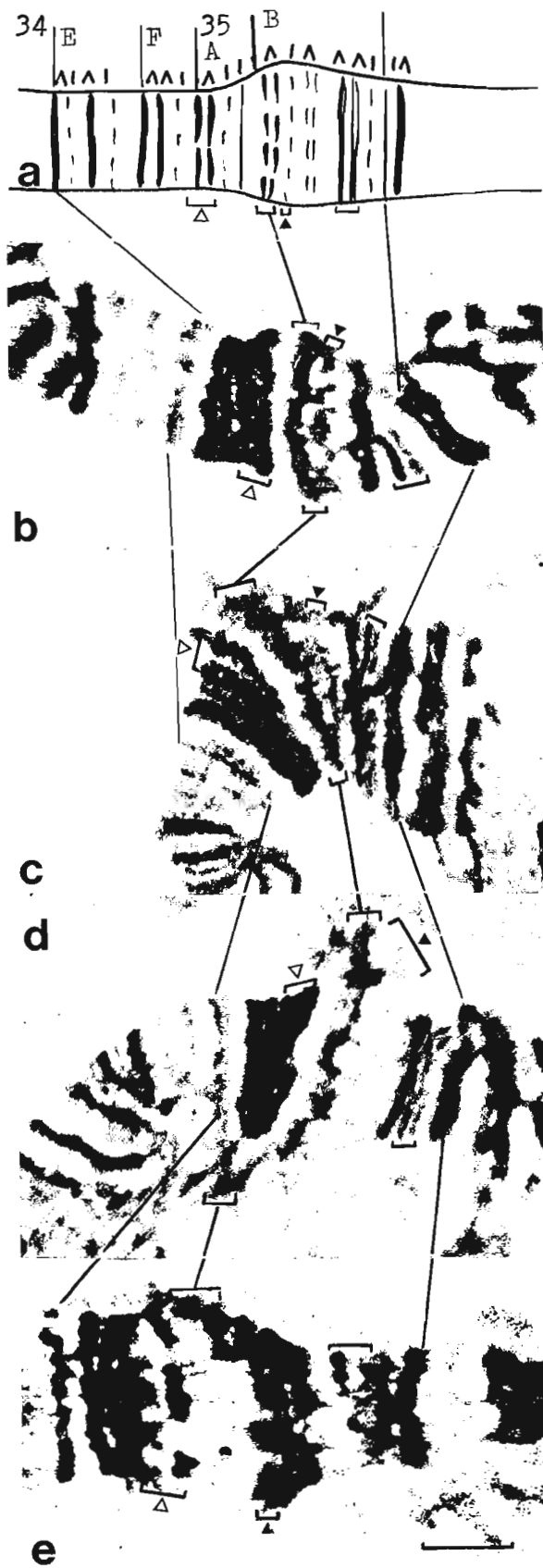
Figure 2. Puffs formation in the region 35AB. a = Bridges map; b-e = EM map: b,c = PS1-2; d = PS4-5; e = 0-h
prepupae. Δ = indicates bands 35A1-2. ▲ = indicates band 35B3.
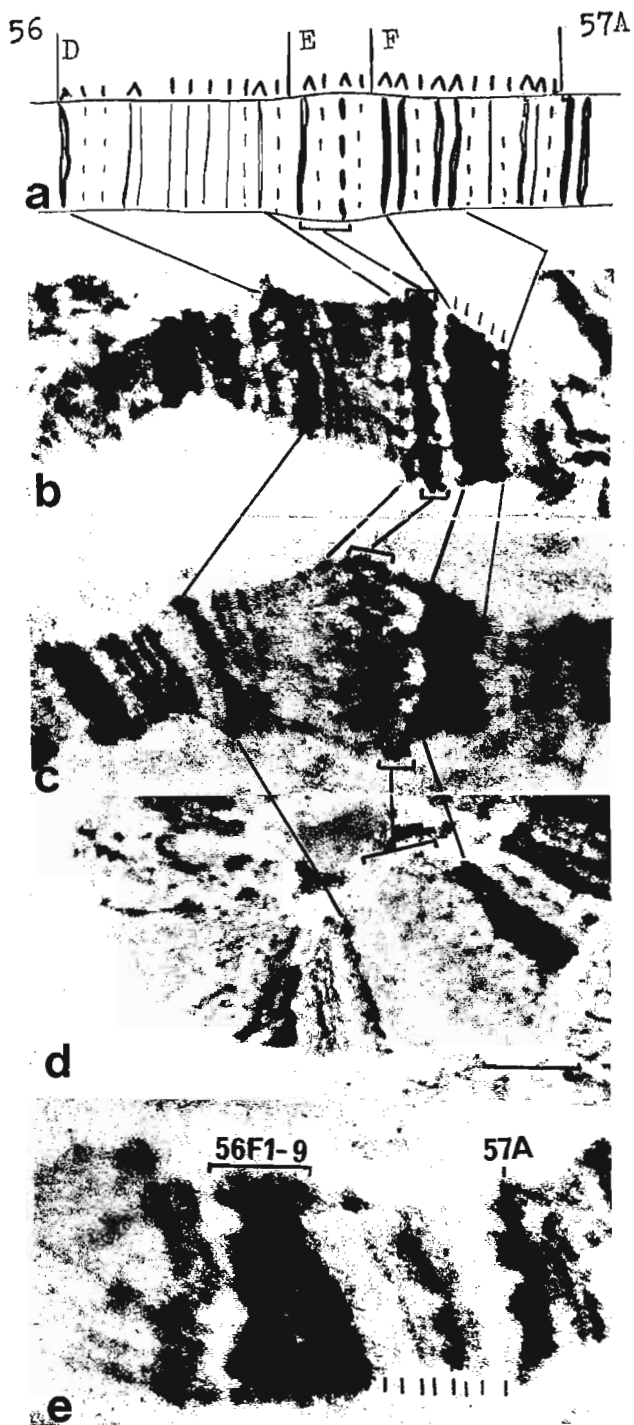
Figure 3. Puffs formation in the region 56EF.
a = Bridges map; b-3 = EM map: b = 0-h prepupae;
c = PS1-2; d = PS4-5; e = PS1-2.
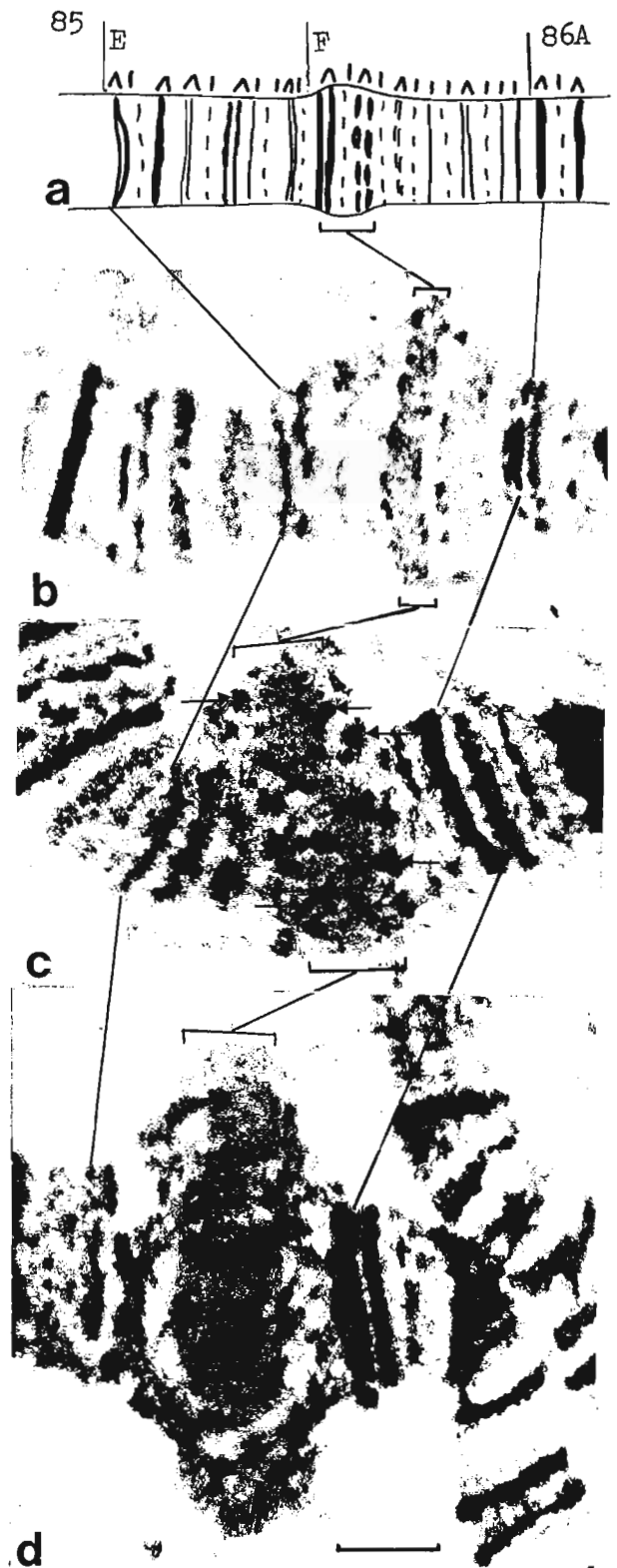
Figure 4. Puff formation in the region 85F. a = Bridges map; b-d = EM map: b = PS1; c = PS4-5; d = 0-h prepuape.
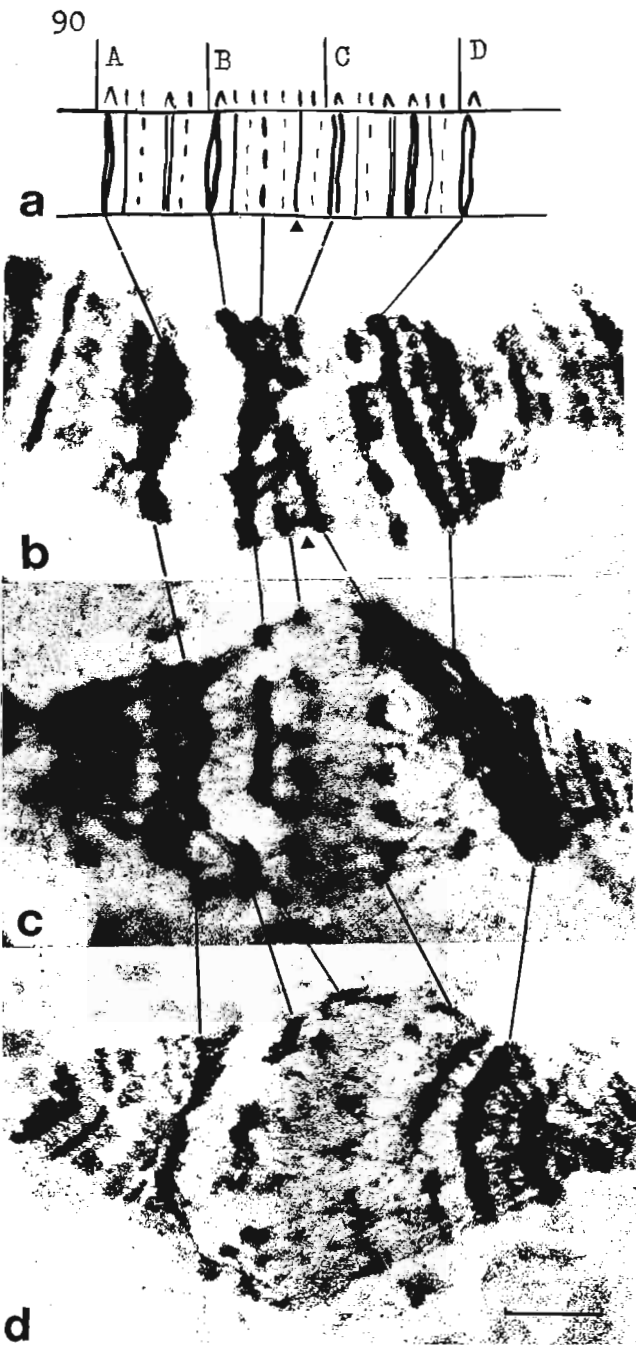
Figure 5. Puff formation in the region 90B. a = Bridges map; b-d = EM map: b = 0-h prepupae; c,d = PS1-2. △ = indicates band 90B7.
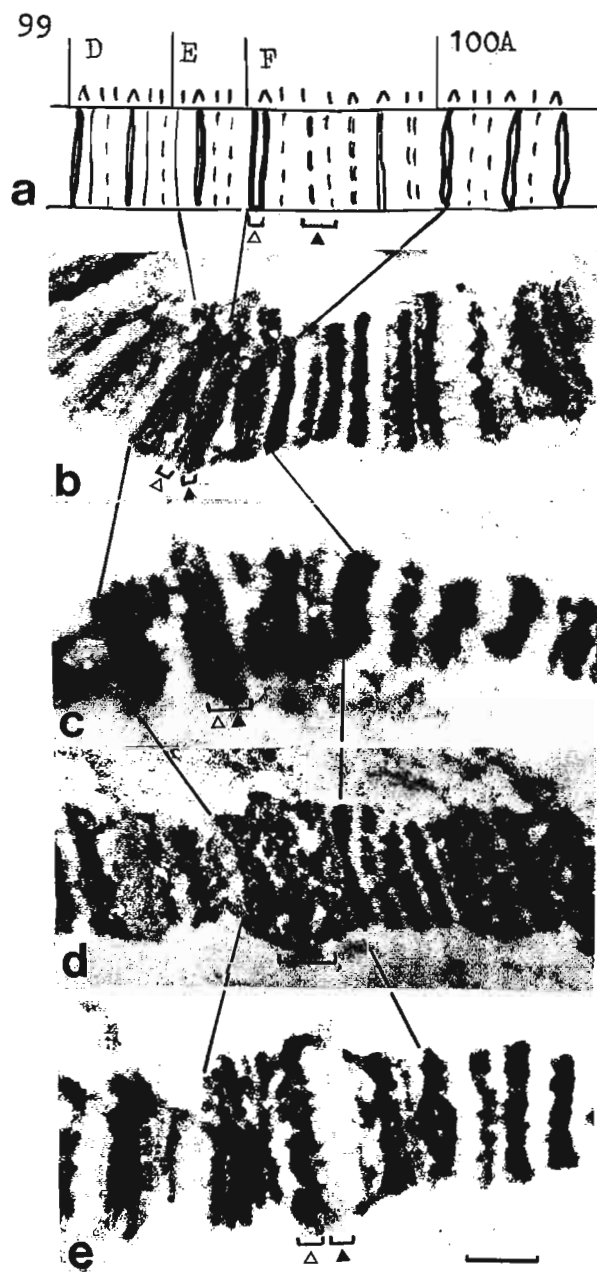
Figure 6. Puffs formation in the region 99F. a = Bridges map; b-e = EM map: b,c = PS1; d = 4h prepupae; e = 0-h prepupae. △ = indicates the 99F1-2 bands. ▲ = indicates the 99F4 and 99F5 bands.

The 85F region. In the 85F1-2 region Bridges found 5 bands, with two doublets 85F1-2 and 85F4-5 (Fig. 4a). By E.M. we reveal that the 85F1-2 and 85F4-5 bands are dense single bands. We failed to detect thin 85F3 band: between 85F1-2 and 85F4-5 bands we saw dark, granular, decondensed material (Fig. 4b). When the size of puff increases (Fig. 4c) dense bands 85F1-2, 85F4-5 and 85F7-8 become granular (marked by arrows on Fig. 4d) and when puff reaches it maximum, we reveal only material of 85F1-2 and 85F7-8 bands.

The 85F4-5 band is greatly decondensed and involved in the zone of puff formation (Fig. 4d).

The 90B region. In the 90B region there are 7 bands on standard map, first of them to be doublet. The 90C1-9 band is also seen in light microscope as a doublet (Fig. 5a). By E.M. in the 90B region only 3

bands are revealed (Fig. 5b), corresponding to bands 90B1-2, 90B5 and 90B7 of Bridges. The 90C1-2 band is a pair of bands similar in thickness. In the 90B region puff develops from thin single 90B7 band (marked by black triangle on Fig. 5). The 90B5 and 90C1-2 bands which delimit the puff, are seen on all stages, although when puff reaches its maximum, they greatly change their morphology (Fig. 5d).

The 99EF region. In the 99E region there are 4 bands on Bridges map with one to be a doublet, in the 99F region 8 bands, including 3 doublets (Fig. 6a). By E.M. in 99E region 5 single bands were found and in the 99F region 8 single bands (Fig. 6b). Bridges 99F1-2 doublet appeared to correspond to pair of band similar in thickness (marked by open triangle on Fig. 6), but thin 99F3 band is not visible. In the 99EF region puff formation starts with partial decondensation of bands in the 99F1-2 - 99F5 interval (Fig. 6c), which look like mixtures of compacted and decondensed material of these bands. When puff reaches its maximum, irregular decondensation involves bands in the 99E4 - 99F6-7 interval (Fig. 6d). In O-h prepupae pattern of puff formation is changed (Fig. 6e). All bands in the 99E and 99F regions are compact, with exception of 99F4 and 99F5 bands (marked by black triangle on Fig. 6), which are decondensed and form morphologically homogenous puff, unlike the preexisting ones (Fig. 6e).

Thus, we examined the formation of 10 puffs which can develop from one band (3C11, 35B1, 35B3 and 90B7), or from several bands (3D1-2, 56E, 85F and 99E) and in one case it looks like lengthening of space between bands 35A1 and 35A2. The puffs also differ in their morphology: with uniform decondensation of bands in the 3C11, 3D1-2, 35B1, 35B3, 90B7 and 99F4-5 regions and irregular decondensation in the 56E, 85F and 99EF regions. In our study it is difficult to reveal the reasons of such irregular decondensation of bands in course of puff formation. It may be due to some peculiarities of gene transcription there, or of mechanical nature, as for puff 2B3-5 (Belyaeva et al., in press).

Puff formation in the 3C11 and 90B7 regions correlates with the expression of genes of 4th (Korge 1977) and 5th (Guild & Shore 1984) fractions of salivary gland secret, which are mapped in these regions. In the 56EF region where are situated about 200 genes of 5S RNA, we detected only one puff, developing from two bands. Unfortunately, it is still unclear, whether the puff formation correlates with 5S RNA genes expression.

References: Belyaeva, E.S. et al. 1987, in press; Guild, G.M. & E.M. Shore 1984, J. Mol. Biol. 179:289-314; Korge, G. 1977, Chromosoma 62:155-174; Lindsley, D.L. & E.H. Grell 1968, in: Genetic Variations of **Drosophila melanogaster**; Semeshin, V.F. et al. 1982, Chromosoma 87:229-237; Sorsa, V. 1983, Cell Diff. 12:137-147; Winber, D.E. & D.M. Steffensen 1970, Science 170:639-641.

Bell, W.J. and K. Nagle. University of Kansas, Lawrence. Pattern of inheritance of adult rover/sitter traits.

Adults of rover and sitter **Drosophila melanogaster** morphs, the larvae of which were previously shown to differ in their foraging tactics (Sokolowski 1980), can be discriminated as individuals with relatively straight paths (rovers) and looping search paths (sitters) (Nagle & Bell 1987). Larval progeny of rover and sitter adult-selected lines, selected from a population of field-collected flies, had respective rover and sitter larval traits (Nagle & Bell 1987). We report here the results of 8 crosses between rover and sitter lines, and an analysis of the genetic and non-genetic influences on this behavior.

Rover and sitter phenotypes are discriminated on the basis of distance searched after consuming a drop of sucrose, which is an indirect measure of how long a fly searches after feeding. Within a period of 30 sec, rovers walk as far as 42 cm, whereas sitters seldom walk more than 2 cm before stopping or flying away. The straight-line distance walked from the sucrose drop within a period of 30 s is referred to as displacement score. Methods are detailed in Nagle & Bell (1987) and Tortorici & Bell (1987).

The parents of each of the crosses described in Table 1 were between 5 and 10 days old when mated. All strains and progeny of crosses were maintained under conditions of 24-26°C, 40-60% RH and a 12:12-h photocycle.

Contrast analysis of variance was performed by J.S. de Belle & M.B. Sokolowski, York University, Ontario, Canada, using the SAS general linear models procedure, to determine the effects of autosomes, transient maternal effects, permanent cytoplasmic factors and/or sex chromosomes on differences in search displacement (Wahlsten 1979; de Belle & Sokolowski 1987). Not all of the potential conclusions could be drawn because only 8 of the required 16 crosses were made in this study. The data were normalized using a LN(SQRT) transformation, but untransformed means and SD are provided as an index of actual displacement.

Male and female data are presented in Table 1. These results suggest a classical additive, incomplete dominance, polygenic model, with the two F_1's intermediate between rover and sitter. The backcrosses led to a progressive increase in displacement scores from low values in progeny of crosses with male sitter against females from the low F_1 (derived from male sitter) to high values in progeny of crosses with male rover against females from the high F_1 (derived from male rover). The variances of F_2's are

Table 1. Mean transformed (ln[sqrt]) displacement scores and variances of male and female progeny of crosses between rover (R) and sitter (S) **D.melanogaster** lines (untransformed means and ± SD are given in paren.).

	Crosses		Male			Female		
Mother	Father		N	$\bar{x}$	V	N	$\bar{x}$	V
1	R x R		52	1.76	0.03	36	1.68	0.08
			(35.5 ± 9.8)			(32.2 ± 12.0)		
2	S x S		50	0.51	0.06	41	0.68	0.12
			(3.2 ± 2.3)			(5.0 ± 3.9)		
3	R x S		20	0.97	0.19	20	0.94	0.14
			(9.5 ± 6.9)			(8.5 ± 6.5)		
4	S x R		20	1.16	0.07	20	1.15	0.11
			(11.9 ± 7.9)			(12.1 ± 7.3)		
5	(S x R) x R		30	1.16	0.18	30	1.41	0.10
			(13.7 ± 10.0)			(20.0 ± 11.5)		
6	(R x S) x R		29	1.21	0.24	31	1.02	0.16
			(16.3 ± 11.9)			(10.1 ± 6.9)		
7	(S x R) x S		28	0.75	0.16	29	0.97	0.14
			(6.5 ± 7.0)			(8.9 ± 6.0)		
8	(R x S) x S		30	0.70	0.17	30	0.78	0.17
			(5.9 ± 5.9)			(6.9 ± 6.8)		
9	(R x S) x (R x S)		30	1.05	0.14	30	1.12	0.25
			(10.5 ± 7.2)			(14.5 ± 12.8)		
10	(S x R) x (S x R)		30	1.02	0.17	31	1.26	0.14
			(10.5 ± 8.8)			(15.6 ± 9.4)		

larger than those of F_1's.

No significant differences were found between the sexes (F[1,597] = 2.06, p>0.10), but differences between strains were highly significant for both males (F[9,309] = 39.72, p<0.0001) and females (F[9,288] = 20.87, p<0.0001). Rover and sitter differ significantly in both males (F[1,309] = 294.5, p<0.0001) and females (F[1,288] = 130.0, p<0.0001). The following conclusions, using an alpha of 0.05, hold for both males and females. The absence of a dominance effect is evident from a comparison between parental strains (#1 and #2) and F_1 hybrids (#3 and #4). F_1 hybrids are not significantly different, indicating that non-autosomal inheritance (sex chromosomes, permanent cytoplasmic factors, transient maternal factors) is not involved. Backcrosses to sitter autosomes (#8) vs F_2 autocrosses (#9) are significantly different, whereas backcrosses to rover autosomes (#5) vs F_2 autosomes (#10) are not, suggesting the possibility of an additive genetic influence.

The number of loci involved in the adult rover/sitter trait, 2.57, was estimated according to Wright (1968, equation 15.8); this is a rough estimate, since the influence of linkage and interactions have not been determined. That more than one gene is involved is also indicated by the percentage of individuals with extreme rover phenotypes (>42 cm displacement) in the parental stock (>50%), as compared to the F_1's (0%), backcrosses (<8.3%) and F_2's (5.0%).

References: Bell, W.J. & C. tortorici 1987, J. Ins. Physiol. 33:51-54; de Belle, J.S. & M.B. Sokolowski 1987, Heredity (in press); Nagle, K. & W.J. Bell 1987, Behav. Genet. (in press); Sokolowski, M. 1980, Behav. Genet. 10:291-302; Wahlsten, D. 1979, in: Theoretical Advances in Behavioral Genetics, J.R. Royce & L. Mos (eds.), pp 426-481, Sijthoff & Nordhoff, Germantown, MD.

Belo, M. and D.A. Banzatto. UNESP, Jaboticabal, Brasil. Association between Drosophila and yeasts. V. Breeding sites preferences in **D.ananassae** strains.

Description of the flies and yeasts used can be found in Belo & Banzatto (1984). The results were reached from successive tests conducted to select two strains: one (SK-1 strain) with preference to **Saccharomyces kluyveri** (S.k.) and the other (PM-1 strain) to **Pichia membranaefaciens** (P.m.). In the imagoes attraction tests to yeasts, a special box described by Belo & Lacava (1980, 1982) was used. In each test 318 pairs of flies were released by generation in the attraction box. For each yeast species two bottles (1/4 liter) with Mittler (1952) medium were used and two without yeast, only with synthetic medium. After each attraction test, the bottles were maintained in a chamber at 25±0.5°C and after five days the number of larvae were counted, and used to determine the productivity of the two strains (Figure 1).

According to Belo & Banzatto (1984), the SK-1 strain imagoes attracted to the selective yeast during the selection tests increased, showing a frequency that follows the linear regression model; however, the PM-1 imagoes decreased, and a few flies were attracted to selective yeast; for this reason only nine tests with **P.m.** are shown in Figure 1. The line which corresponds to the regression equation for productivity of SK-1 strain in **S.k.** (F = 8.32, p<0.05) is presented, and for the PM-1 strain the polynomial regression test was not significant. In the initial test for SK-1 strain, 45% of the larvae were obtained in **S.k.**, and in the last three tests the production were 67, 72 and 70%. The larval frequency presented by PM-1 strain in **P.m.** did not indicate a clear pattern. The correlation coefficients between the frequencies and the number of tests realized are presented in Table 1.

The abundance of larvae produced by the SK-1 strain raised on **S.k.** shows for this yeast, a positive, significant correlation, while for the other ones, only negative, non-significant correlations occurred. This strain presented no descendants on **Torulopsis pinus**, nor on the yeast-free bottles containing only synthetic

Figure 1. Frequencies (in percentages) of larvae produced in **S.k.** (o) by the SK-1 strain and in **P.m.** (●) by the PM-1 strain, during the generations of selection.

Graph: % Larval frequencies vs Generations of selections. Line corresponding to the equation: $Y = 29.3382 - 4.6184X + 0.4453X^2$

Table 1. Values of correlation coefficients (r) between the percentage of larvae produced in each yeast species, and the number of tests conducted.

Yeast species	SK-1 strain (15 d.f.)	PM-1 strain (7 d.f.)
P.membranaefaciens	-0.2464^{ns}	-0.3411^{ns}
S.kluyveri	0.7266^{***}	0.0297^{ns}
S.chevalieri	-0.3868^{ns}	0.1420^{ns}
T.delbrueckii	-0.0691^{ns}	0.2385^{ns}
L-9	-0.3243^{ns}	0.4115^{ns}
S.roseus	-0.3572^{ns}	-0.4191^{ns}
T.pinus	--	-0.3922^{ns}
H.saturnus	-0.2437^{ns}	0.0565^{ns}
H.anomala	-0.4000^{ns}	-0.3025^{ns}
R.glutinis	-0.4657^{ns}	-0.5355^{ns}

ns=non-signif. (p>0.05); ***=signif. (p<0.001)

Table 2. Means and standard error values for the number of larvae produced in each species of yeast by the two strains and results of the "t" Student test.

Yeast species	SK-1 strain	PM-1 strain	"t" Test
P.membranaefaciens	15±7	46±19	1.55^{ns}
S.kluyveri	177±41	45±17	2.99^{**}
S.chevalieri	125±28	237±80	1.32^{ns}
T.delbrueckii	150±50	110±45	0.59^{ns}
L-9	17±9	50±25	1.48^{ns}
S.roseus	1±1	5±5	1.18^{ns}
T.pinus	--	2±1	0.81^{ns}
H.saturnus	19±10	27±15	0.44^{ns}
H.anomala	7±3	22±10	1.56^{ns}
R.glutinis	1±1	2±2	0.25^{ns}

ns=non-signif. (p>0.05); **=signif. (p<0.01)

medium; the same was shown for PM-1 strain. The correlation coefficients corresponding to larval frequencies of descendants of the PM-1 strain are not significant. Thus, the tests revealed differences in larval production among the yeast species and between fly strain. The preference tests with imagoes produced a convergence in SK-1 strain for **S.k.** as breeding site. This was not presented by PM-1 strain on **P.m.** Table 2 shows the average number of larvae produced in all yeast species by the two strains and the results of the "t" Student test applied. One single significant difference between means was found in **S.k.**, where the SK-1 strain presented more descendants than PM-1 strain.

The three most productive yeasts in descendants of the SK-1 strain (**S.k.**, T.delbrueckii and S.chevalieri), and of the PM-1 strain (**S.chevalieri**, T.delbrueckii and L-9), produced 88% and 73% of all larvae, respectivley. The sum of the means of larvae produced in all yeasts by the PM-1 strain results in a value which is slightly higher than that of the SK-1 strain. Application of the "chi-square" test demonstrated that these totals did not differ significantly (X^2 = 1.09, p<0.05). This datum serves to show that the PM-1 strain distribution frequency of larvae in the yeasts was more equitable than that observed for the SK-1 strain, and shows that the PM-1 strain was more versatile in its choice of breeding sites than the SK-1 strain.

Such observations show that selection preference tests to yeast with imagoes produced differences in the preference of breeding site choice by the strains of flies (work supported by CNPq-PIG IV).

References: Belo, M. & D.A. Banzatto 1984, Naturalia (UNESP) 9:7-14; Belo, M. & P.M. Lacava 1980, DIS 55:164-165; &　　　1983, Naturalia (UNESP) 7:35-45; Mittler, S. 1952, Science 155:271-272.

Belo, M. and E.B. Malheiros. UNESP, Jaboticabal, Brasil. Association between Drosophila and yeasts. VII. Crosses with **D.ananassae** strains selected for development.

Belo (1982) carried out studies concerned about successive generations for developmental rate on three yeast species, **Pichia membranaefaciens (P.m.)**, **Saccharomyces kluyveri (S.k.)** and **Torulaspora delbrueckii (T.d.)**, producing thus, three **D.ananassae** strains. Each one of the three lines of selections generally showed during the experimental period increases of the number of flies (NF), biomass (BI), viability (VI) and bias to diminish the developmental time (DT). To analyze comparatively the results of such selective process, virgin male (m) and female (f) of the F_{12} generation were placed in bottles (1/4 liter) with Mittler (1952) medium modified (Belo & Banzatto 1984) for two days to crosses and digestive tube sterilization; following, males and females stayed together more three days in bottles with Mittler (1952) medium eating the yeast specie, which would serve to test the cross. The yeast species were stored in bottles with synthetic medium six days before the females to be placed (15 per bottle), where they stayed three days to oviposit, after which they were removed. Thus were organized 12 (crosses between flies of the same strain) or 8 bottles (crosses between individuals of different strains), and the VI was measured by the formula: NF divided by sum of the NF, number of larvae and number of pupae. For DT the formula DT = [t + (t - t')/2] + T (Carvalho 1981) was used; where t is the number of days between the withdrawal of the parental flies from bottles and the first imago counting; t' is the interval between two countings (one day) and T is the maintenance time of the females in the bottles. The tests were conducted at 25±0.5°C.

Table

Table 1. Means presented by descendants from the crosses among the fly strains on different yeast species.

Crosses	NF	BI	VI	DT
mSk x fSk (S.k.)	10.96	59.78	0.63	10.16
mSk x fSk (P.m.)	8.24	41.39	0.32	12.91
mSk x fSk (T.d.)	7.48	39.50	0.31	10.10
mPm x fPm (P.m.)	12.99	81.72	0.71	12.29
mPm x fPm (S.k.)	6.45	18.55	0.35	12.02
mPm x fPm (T.d.)	9.06	33.87	0.51	10.68
mTd x fTd (T.d.)	12.64	72.90	0.68	9.49
mTd x fTd (P.m.)	8.90	43.66	0.38	12.32
mTd x fTd (S.k.)	8.29	43.38	0.60	10.18
mTd x fPm (P.m.)	11.37	95.31	0.90	14.55
mTd x fPm (T.d.)	8.55	35.51	0.38	12.27
mPm x fTd (P.m.)	9.09	36.52	0.50	14.32
mPm x fTd (T.d.)	10.21	47.78	0.45	11.50
mTd x fSk (S.k.)	9.03	51.52	0.44	11.57
mTd x fSk (T.d.)	10.33	81.75	0.56	10.88
mSk x fTd (S.k.)	11.01	61.46	0.52	10.66
mSk x fTd (T.d.)	10.02	55.26	0.47	11.55
mSk x fPm (P.m.)	7.74	55.05	0.34	14.43
mSk x fPm (S.k.)	7.93	32.95	0.33	11.76
mPm x fSk (P.m.)	9.63	44.55	0.45	12.55
mPm x fSk (S.K.)	10.01	67.18	0.54	11.79

Table 1 shows every kind of cross realized with the flies. The means analyses were done with ANOVAs and Tukey tests. The crosses that involved individuals from the same strain, which the yeast species was the same used to select such flies during the generations, showed according to the data, a general pattern of the best performance in the exploration of the selective yeasts. An example is the value to the NF, BI and VI of the cross between males and females of strain selected to develop on **P.m.**, i.e., mPm x fPm (**P.m.**); such values are significantly superior to the values produced by the same cross, where the females oviposited on the non-selective yeasts, mPm x fPm (**S.k.**) and mPm x fPm (**T.d.**). These results indicated the best performance to exploration of the selective yeasts which served to select the strains, showing an evolution of the flies on such microorganisms in relation to the non-selective ones. The descendants of the crosses mSk x fSk (**S.k.**) and mTd x fTd (**T.d.**) showed the DT equal or smaller than the values to the same crosses on the non-selective yeasts. On the other hand, the flies DT on **P.m.** were generally longer, which may be due to nutritional condition of such yeast. The means to the NF, BI and VI produced by crosses between flies of the same strain on the selective yeast generally are not different significantly from the largest values presented by the crosses between individuals from different strains; these last values were not different even from the smaller ones, obtained from crosses among the same differing strains. Thus, an example is the values showed to NF by the crosses mSk x fSk (**S.k.**), mTd x fTd (**T.d.**), mPm x fPm (**P.m.**), mTd x fPm (**P.m.**), mSk x fTd (**S.k.**) and mPm x fSk (**S.k.**) which are statistically equal; and the last two values to the crosses cited did not differ from values obtained to the crosses mSk x fPm (**P.m.**) and mSk x fPm (**S.k.**). While the value to the cross mTd x fPm (**P.m.**) differed, it is true for BI and VI also.

The results seem not to indicate any maternal effect in relation to the analysed parameters. The cross mTd x fPm (**P.m.**) produced high values to NF, BI and VI, showing a heterotic effect, while the cross mSk x fPm (**P.m.**) produced low values; the crosses mSk x fTd (**T.d.**) and mPm x fTd (**T.d.**) did not differ with regard to such parameters.

References: Belo, M. 1982, Free-Docent Thesis (UNESP); _____ & D.A. Banzatto 1984, Naturalia (UNESP) 9:7-14; Carvalho, S. 1981, Ph.D. Thesis, Fac. de Med. de Ribeirao Preto, USP; Mittler, S. 1952, Science 155:271-272.

Boedigheimer, M. and M. Simmons. University of Minnesota, St. Paul. Studies on the ability of an M′ strain to suppress P element-induced hybrid dysgenesis.

In the P-M system of hybrid dysgenesis in **D.melanogaster**, the P cytotype is the condition that regulates the activity of transposable P elements in the germ line. Females with this condition transmit it to their progeny, in whom it suppresses nearly all P element action. This maternal transmission has been shown to persist for at least two generations (Engels, 1979a); ultimately, however, the maintenance of the P cytotype depends on the presence of P elements in the chromosomes. All strains with the P cytotype possess these elements, but not all strains with chromosomal P elements possess the P cytotype.

Many laboratory strains of **D.melanogaster** lack P elements altogether: these have the M cytotype, a condition that permits P elements to move whenever they are introduced into the strain by a cross or by microinjection (Engels 1983). This movement also requires the activity of the P transposase, an enzyme encoded by the gene carried by structurally intact P elements (Karess & Rubin 1985; Laski, Rio & Rubin 1986; Rio, Laski & Rubin 1986).

Some strains of **D.melanogaster** appear to have an attenuated version of the M cytotype (Kidwello 1983, 1985). These M′ or pseudo-M strains possess P elements, but most of their elements appear to be defective in the production of the P transposase (Bingham, Kidwell & Rubin 1982; Simmons & Bucholz 1985). This attenuated M cytotype is evident when M′ females are crossed to males from a P cytotype strain that has many autonomous (i.e., transposase-producing) P elements. The progeny of such crosses exhibit significantly less hybrid dysgenesis than the progeny of crosses between pure M cytotype females and the same P males. This suggests that the M′ females possess a partial P cytotype, that this condition may be maternally transmitted, and that it arises from nonautonomous P elements.

We have studied an inbred line derived from the M′ strain Muller-5 Birmingham (Bingham, Kidwell & Rubin 1982; Simmons & Bucholz 1985). This line, called M5-B#1, was produced by ten generations of sib-mating and was subsequently maintained by mass-matings in half-pint culture bottles. Previous work had established that the parent stock possessed many P elements and that none of these was able to produce the P transposase. We therefore assumed that the P elements of M5-B#1 were all nonautonomous; this assumption has subsequenlty been validated by extensive tests with the M5-B#1 line itself (data to be reported elsewhere).

In one set of experiments, we determined the cytotype of the M5-B#1 line. This was done by crossing M5-B#1 females to males from strains possessing many autonomous P elements. The daughters of these crosses were then examined for evidence of P element activity in their germ lines. For controls, the daughters of pure M females (from a bw; st stock), which had been crossed to the same type of males, were also examined.

One test involved a classic dysgenic trait, GD sterility. The π_2 strain was used as the source of the males for the dysgenic crosses; the tested females were mated individually to these males and their progeny were reared at 29°, the restrictive temperature for GD sterility. 132 hybrid daughters from the bw; st females were examined and all proved to be sterile. In contrast, 94 hybrid daughters from the M5-B#1 females were examined, and only 7 were sterile. This very low frequency of GD sterility shows that most of the daughters of the M5-B#1 females were able to suppress P element activity in their germ lines, presumably because they had inherited some feature of the P cytotype from their mothers.

Further evidence for the P cytotype in the M5-B#1 line came from a second test that utilized the P element insertion mutation, sn^w (Engels 1979b, 1984). This mutation is unstable in germ lines with the M cytotype, so long as autonomous P elements are present; however, in the P cytotype, little or no instability is observed. For these tests, sn^w: π_2 males from a stock with many autonomous P elements were crossed to bw; st or M5-B#1 females in mass cultures at 21°; the lower temperature was to reduce GD sterility among the progeny. Then the sn^+/sn^w daughters of these crosses were mated individually to y sn^3 v car males at 25° so that any germ line instablity of sn^w could be detected by scoring the bristle phenotypes of the next generation. Under the influence of the P transposase, sn^w mutates to sn^e and sn^+ alleles. In these experiments, however, only the sn^e mutations could be detected. These mutations were much less frequent in the cultures where the tested flies had M5-B#1 mothers. The average (unweighted) mutation rate for 67 daughters of the bw; st females was 13.3 ± 1.2% (1,384 flies counted), whereas, it was only 2.8 ± 0.4% (2,970 flies counted) for 79 daughters of the M5-B#1 females. In both sets of crosses, the flies were counted until the 15th day after the cultures were established. These data clearly demonstrate that the daughters of the M5-B#1 females had considerable ability to suppress sn^w mutability, suggesting that they had inherited some aspect of the P cytotype from the M5-B#1 line. However, neither these data nor those from the sterility tests could discriminate between cytoplasmic and chromosomal determinants of M5-B#1′s cytotype.

We therefore investigated whether this ability to suppress hybrid dysgenesis could be inherited maternally through two generations. The experiment utilized a pure M strain with balancers on the second

Table 1. GD sterility and sn^W hypermutability of different genotypes derived from pure M and M' maternal lineages.

| | A Hybrids (CyO;TM6/Xa mothers) | | | | | | B Hybrids (M5-B#1 mothers) | | | | | |
| | GD Sterility | | sn^W Hypermutability | | | | GD Sterility | | sn^W Hypermutability | | | |
Genotype	No. fe-male	% ster-ile	No. cul-tures	sn^W	sn^e	Total	Mutation rate (%)±s.e.	No. fe-male	% ster-ile	No. cul-tures	sn^W	sn^e	Total	Mutation rate (%)±s.e.
1 P/M';P/M';P/M'	45	97.7	51	1181	124	1305	9.2±1.1	48	100.0	48	1533	101	1634	6.3±0.7
2 P/M';P/CyO;P/M'	40	100.0	56	1054	106	1160	9.1±1.2	58	100.0	62	1868	125	1993	7.4±1.1
3 P/M';P/M';P/TM6	30	100.0	87	740	90	830	10.6±1.3	94	100.0	90	1239	117	1356	9.1±1.2
4 P/M';P/CyO;P/TM6	8	100.0	37	118	7	125	5.9±2.0	57	98.2	67	374	34	408	8.5±1.6
5 P/+;P/M';P/M'	91	98.9	45	1339	107	1446	7.2±1.2	58	91.4	44	1615	69	1684	4.0±0.7
6 P/+;P/CyO;P/M'	94	100.0	56	1405	165	1570	10.2±1.3	84	97.6	49	1639	95	1734	5.7±0.8
7 P/+;P/M';P/TM6	172	94.2	81	1280	128	1408	8.7±0.9	184	94.0	76	1662	103	1765	6.3±0.8
8 P/+;P/CyO;P/TM6	137	98.5	62	570	39	609	6.0±1.2	114	94.7	68	875	63	938	7.6±1.3

The P chromosomes were derived from the $sn^W;\pi_2$ stock, the M' chromosomes from the M5-B#1 inbred line, and the +,CyO and TM6 chromosomes from a pure M stock. The mutation rate is the unweighted average among the cultures of a group. The standard error of this average was computed empirically.

and third chromosomes. This strain, CyO; TM6/Xa, was crossed in both ways to M5-B#1 flies (at 25°) to produce +/M5'; CyO/M' TM6/M' reciprocal hybrids (the chromosomes with the primes are the ones from the M5-B#1 line). We refer to the flies that had M5-B#1 fathers as the "A" hybrids, and to those that had M5-B#1 mothers as the "B" hybrids. These flies were then used in tests for the suppression of GD sterility and sn^W hypermutability. The procedure was to cross individual sn^W; π_2 males to each kind of hybrid female. The crosses were initially performed at 21° to produce fertile daughters that could be tested for sn^W hypermutability, but after six days, the mated females were transferred to fresh cultures, which were then incubated at 29°C to induce GD sterility in the second brood. Each culture could theoretically produce eight distinguishable types of females. Our intention was to determine the incidence of sterility among these, as well as to estimate the mutability of sn^W in their germ lines. For the latter investigation, each type of female was mated to three y sn^3 v car males (at 25°) and the frequency of sn^+ mutations in the germ line was determined by counting the sn^W and sn^e progeny until the 15th day after mating. The mutation frequencies were then averaged over all the females in a group.

Table 1 presents the results of this experiment. Although the data on GD sterility are not extensive, there is no evidence for variation in the incidence of this trait among the diverse flies that were tested. The frequency of sterility was uniformly high among all the females from both kinds of hybrids. Neither genotype nor maternal lineage seemed to have any effect on this dysgenic trait. It would seem, therefore, that the ability of M5-B#1 to suppress GD sterility had been lost in only one generation of hybridization with a pure M strain. This contrasts with the results of Kidwell (1985), who found that reciprocal hybrids between M' and pure M strains exhibited a level of suppression that was intermediate between the two parental types.

The data from the tests for the ability to suppress sn^W hypermutability were more extensive than those from the sterility tests. Nearly 20,000 flies were counted to determine the 16 mutation rates given in Table 1. There was variation among these rates, indicating the possible influence of maternal lineage and chromosomal genotype in controlling sn^W mutability. However, since the uncertainty of the estimated mutation rates was relatively large, it was difficult to identify the effects of specific genotypes. This problem was exacerbated by the viability effects of the autosomal balancers, which could have been especially pronounced in the case of the sn^e flies. Viability differences among the various genotypes might have led to distorted estimates of the mutation rate. Therefore, for these data the best approach seemed to be to compare the mutation rates within genotypes and across maternal lineages, thus controlling for any genotypic-specific effects that might be present.

In six out of the eight comparisons, the flies derived from the B hybrids had lower average mutation rates than those derived from the A hybrids. By the sign test, the probability of this and more deviant results is 0.144, which is not significant; however, the low probability suggests that the maternal lineage of the B hybrids, i.e., the one from M5-B#1, reduced the mutation rate, irrespective of the chromosomal genotype. It is worth noting that the two comparisons unfavorable to this hypothesis involved genotypes 4 and 8, for which the fewest data were available.

Although not decisive, the results of this experiment suggested that the ability of M5-B#1 to suppress sn^W mutability could be transmitted maternally through at least two generations. If this pattern is confirmed by more precise experiments, it would conform to that classically established for the P cytotype, indicating that M5-B#1 shares this feature with bona fide P cytotype strains. A repressor with a limited ability for self-replication has been postulated to explain the maternal inheritance of cytotype

(Engels 1981). What is significant in the case of M5-B#1, however, is that this line is devoid of autonomous P elements. This fact, combined with the suggestions provided by the data above, implies that some aspect of the P cytotype may exist in this line independently of any autonomous P elements.

Simmons & Bucholz (1985) showed that the chromosomes of the parent strain, Muller-5 Birmingham, reduced sn^W mutability when they were inherited paternally. Their results, which excluded any maternal effect, indicated that nonautonomous P elements on the chromosomes could titrate the P transposase. Some of the variation in sn^W mutability seen in the present experiments could also be due to a titration effect, but the generally lower mutability of the flies with the M5-B#1 cytoplasm implies that transposase titration by chromosomal P elements can only be part of the story. Simmons & Bucholz (1985) suggested a model in which the P cytotype depended on the titration of the P transposase by extrachromosomal P elements that could be inherited maternally. Although this is still a formal possibility, other evidence (H. Robertson & W. Engles, personal communication) now favors a different model. It has been suggested by Robertson & Engels that some nonautonomous P elements might produce a repressor of P element activity, and that this repressor might accumulate to bring about the P cytotype. In this view, strains with a partial P cytotype, such as M5-B#1, might produce lesser amounts of the repressor than strains with the bona fide P cytotype. Crosses of M5-B#1 females to pure M males would, therefore, be expected to dilute the repressor more than crosses of P cytotype females to pure M males. This would lead to a more pronounced loss of the ability to suppress hybrid dysgenesis. The data reported here are consistent with this view so long as we postulate that the reduction of sn^W hypermutability requires less repressor than the reduction of GD sterility. This postulate seems reasonable since the repressor need act at only one locus to quench sn^W mutability, whereas to prevent GD sterility, it might have to cover a large number of chromosomal P elements. Further studies utilizing the M5-B#1 line are currently underway to clarify these aspects of the repressor hypothesis.

References: Bingham, P.M., M.G. Kidwell & G.M. Rubin 1982, Cell 29:995-1004; Engels, W.R. 1979a, Genet. Res. 33:219-236; _____ 1979b, PNAS USA 76:4011-4015; _____ 1981, Cold Spring Harbor Symp. Quant. Biol. 45:561-565; _____ 1983, Ann. Rev. Genet. 15:315-344; _____ 1984, Science 226:1194-1196; Karess, R. & G.M. Rubin 1984, Cell 38:135-146; Kidwell, M.G. 1983, PNAS USA 80:1655-1659; _____ 1985, Genetics 111:337-350; Laski, F.A., D.C. Rio & G.M. Rubin 1986, Cell 44:7-19; Rio, D.C., F.A. Laski & G.M. Rubin 1986, Cell 44:21-32; Simmons, M.J. & L.M. Bucholz 1985, PNAS USA 82:8119-8123.

Bolshakov, V.N. and I.F. Zhimulev. Inst. of Cytology & Genetics, Novosibirsk, USSR. Distribution of rearrangement's breakpoints within the 11A6-9 intercalary heterochromatin band of **Drosophila melanogaster** X-chromosome.

It was shown that in euchromatic portions of Drosophila salivary gland chromosomes there are so-called "intercalary heterochromatin" (IH) regions (dense, dark bands or groups of bands) showing the properties of pericentric and centromeric heterochromatin (for review and more detailed description of IH regions, see Zhimulev et al. 1982). For further investigation of IH phenomena, we started analysis of the 11A6-9 band, the most prominent IH region of the X-chromosome. We mapped the breakpoints of nine translocations and three deletions within this band to find out whether the "weak point" in its central part is the "hot spot" of chromosomal rearrangements. Salivary glands of 3-d instar larva, males or females, were squashed using standard technique in 55% lactic acid after acetoorcein staining and analysed under light microscope.

It was found that the breakpoints are randonly distributed within the 11A6-9 band. According to the morphology and sizes of the broken parts of the band, breakpoints of three translocations are mapped in the distal part (Figure 1, a-d), three translocations in the central part (Fig. 1 e-h), and three translocations and three deletions in the proximal part (Fig. 1 i-m) of the band. In the cases of two deletions, KA6 and KA10, we could map the breakpoints in relation to the "weak point", because in females analysed we did not find any constrictions in the rearranged homologues, while in FM homologues it was often present (Fig. 1 k), thus meaning that the "weak point" region was deleted. Unfortunately, such analysis is impossible for the translocations and one deletion, because males were analysed, where the constrictions in the X-chromosome are absent (Zhimulev et al. 1982).

It is worth mentioning, that males bearing T(1;Y) are all viable and fertile, thus implying that the translocations do not disrupt some essential genetic loci in the 11A6-9 band, even distributed randomly. Thus, we can suppose the existence of at least three zones of "silent DNA" in the 11A6-9 band ("weak point" region and distally and proximally to it, Figure 2). Our data cofirm earlier findings of Lefevre (1981) that all breakpoints found by him in the 11A6-9 band were nonmutant and nonlethal. Therefore, the high frequency of rearrangement breakpoints in the 11A6-9 band and in some other IH regions in comparison with other chromosome regions (if the relative DNA content is taken into consideration, see Bolshakov et al. 1985) may be probably due to the "genetic inertness" of some parts of IH regions or IH regions in whole.
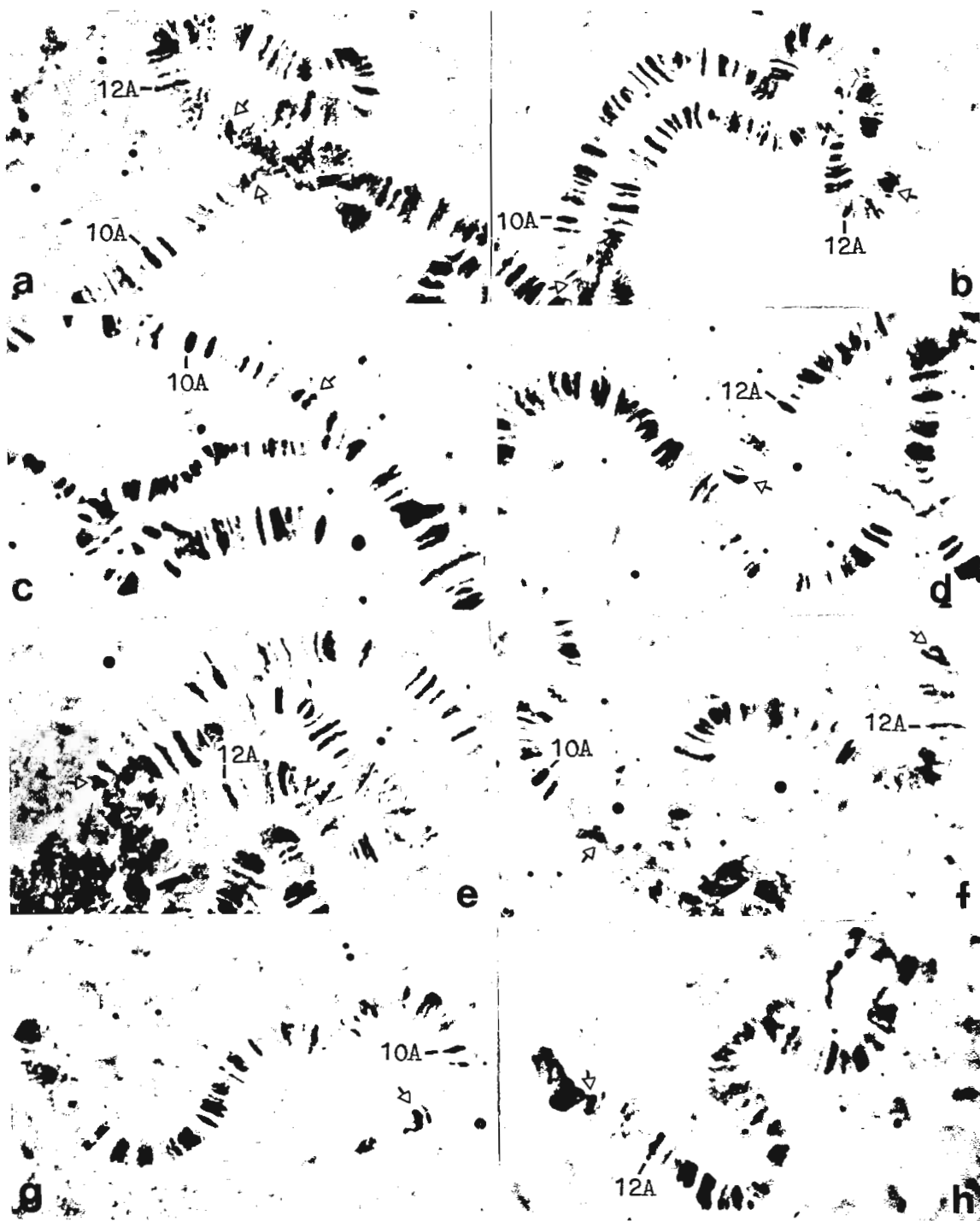
Figure 1. Rearrangements in the 11A6-9 IH band with the breakpoints in distal (a-d), central (e-h) and proximal (i-n) parts of the band. a = T(1:Y)B44; b = T(1;Y)B45; c,d = T(1;3)01249; e = T(1;Y)B87; f = T(1;Y)D8; g,h = T(1;Y)D9; i = T(1;Y)B53; j = T(1;Y)20; k = Df(1)KA6; l = Df(1)KA10; m = T(1;Y)4; n = lzDf(1)v^{65b}. a-j, m, n = males and k, l = females were analyzed. Open-headed arrows indicate the parts of broken 11A6-9 band. Black-headed arrow in k indicates "weak point" in the 11A6-9 band in FM7 homologue.

Figure 1:
(contin.)
i thru n.
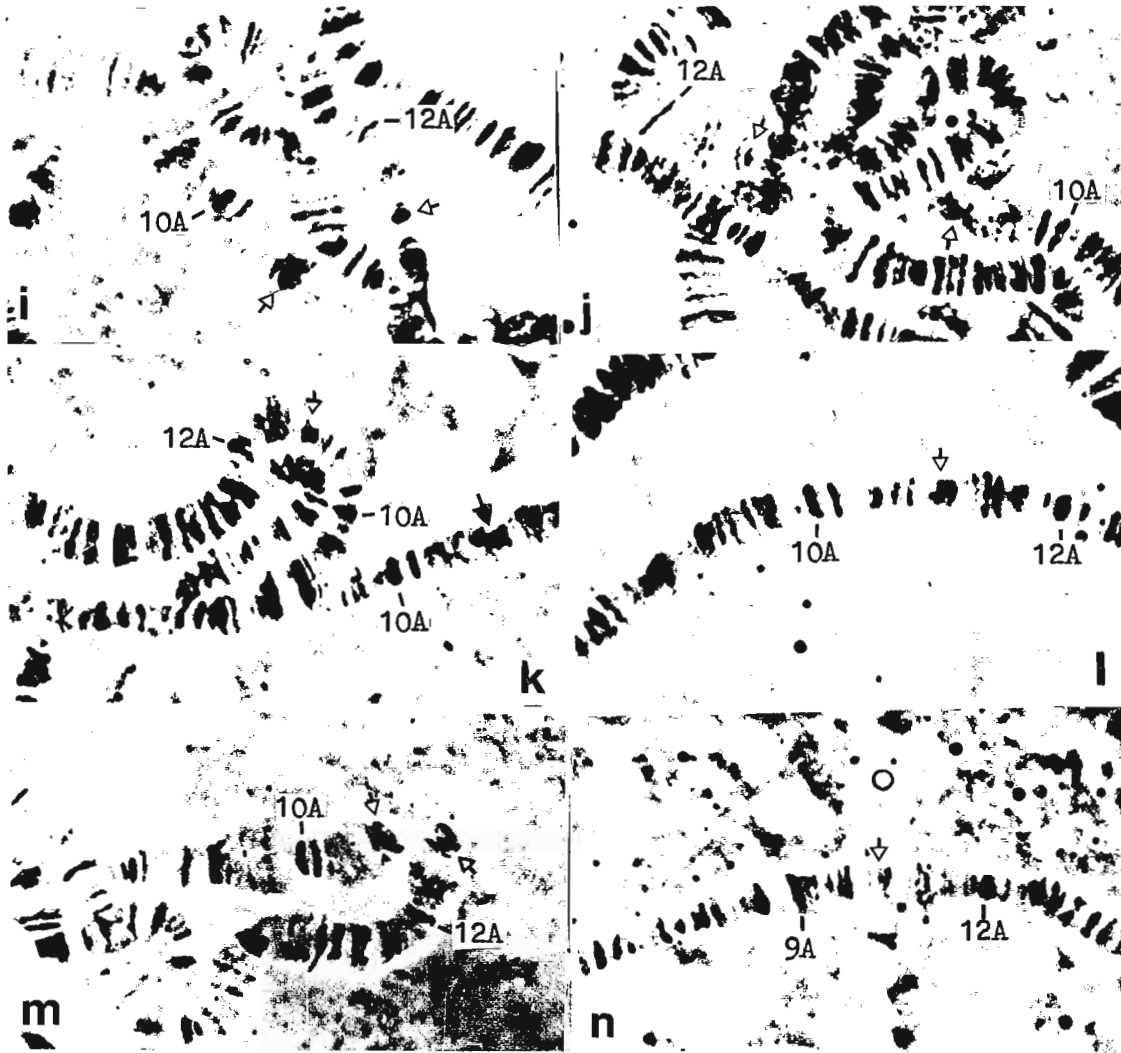
11A6-9
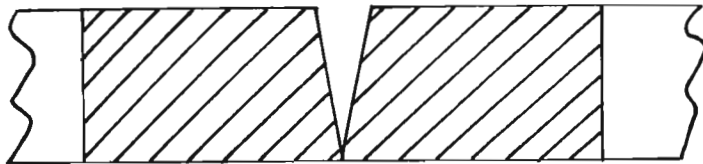
Figure 2. Schematical representa-
tion of distribution of rearrange-
ment breakpoints within the 11A6-9
IH band. "Weak point" is indica-
ted in its central part.

References: Bolshakov,
V.N. et al. 1985, Chromosoma 92:
200-208; Lefevre, G. 1981, Gene-
tics 99:461-480; Zhimulev, I.F.
et al. 1982, Chromosoma 87:197-
228.

T(1;Y)B44
T(1;Y)B45
T(1;3)01249
T(1;Y)B87
T(1;Y)D8
T(1;Y)D9
T(1;Y)B53
T(1;Y)20
Df(1)KA6 (10E1-2)
Df(1)KA10 (10F11/11A1-2)
T(1;Y)4
Df(1)v^{65b} (9F12-13)

Bove, C. Stanford University, Stanford, California.[*] Hybridization between **S.cerevisiae** CDC genes and **D.melanogaster** DNA reveals no homology.

Several aspects of the cell cycle (e.g., DNA replication, DNA repair, chromosome segregation) are common to all eukaryotic cells. It is known that some of the genes involved in these processes have been conserved during evolution and share homology between species (e.g., tubulins, histones).

In **S.cerevisiae** many cell division cycle (CDC) genes have been cloned. I have used eleven yeast CDC clones as hybridization probes to try to detect and isolate homologous sequences from **D.melanogaster**. The CDC clones used in this study are shown in Table 1. These clones were used only because of their immediate availability and not because their roles in the cell cycles made them more likely to be conserved.

The first experiment was to see if homology to the CDC clones could be detected in total fly DNA. Southern blots of total **D.melanogaster** DNA, digested with various restriction enzymes, was probed with DNA of the CDC containing fragment of yeast (excised from the vector) labeled by nick translation. In addition to the CDC sequence, these fragments contained variable lengths of yeast flanking regions, including other transcription units of unidentified function.

The hybridization conditions used in this experiment were: (1) a standard condition (50% formamide, 5X SSPE, 5X Denhardt's solution, 0.1% SDS, 100 µg/ml denatured salmon sperm DNA), and (2) a low stringency condition (identical to the standard condition but with 29% formamide). About 5×10^5 cpm/ml of hybridization mix was used in each case. Hybridizations were for 12-24 hr at 42°C. The filters were washed at 50°C for 4 hr in 0.1% SDS, 0.1x SSPE for the standard condition and 0.1% SDS, 1x SSPE for the low stringency condition. Autoradiographic exposure of the Southerns for 10-15 days at -70°C with an intensifying screen did not reveal hybridization in any case. This negative result is not itself sufficient to eliminate the possibility of homology. The amount of DNA eventually homologous might be present in too low a concentration to be detectable on a Southern of total genomic DNA.

To increase the sensitivity of the screen, I probed a lambda library of **D.melanogaster** genomic DNA (Maniatis et al. 1978). The library was plated in order to have a set of plates containing ten Drosophila genome equivalents. Filters prepared from these plates were hybridized with each CDC probe using the low stringency condition described above. After exposures ranging from one to three weeks, the autoradiograms showed no unambiguous positive signals with any of the tested probes. However, some ambiguous positive signals were present with some CDC probes. The corresponding phages were picked and rescreened. After this second screening some ambiguous signals were again present. DNA from the phage exhibiting possible hybridization was prepared, digested with Eco R1, transferred to nitrocellulose and hybridized with the CDC probe used to detect them. In no case was homology confirmed.

While these results do not exclude the presence of homology between the tested yeast CDC genes and Drosophila genes, they do say that if this homology exists it is not very high (less than roughly 85%) because it was not detectable with the hybridization conditions used.

Table 1. Yeast CDC clones used to screen for related **D.melanogaster** sequences.

CDC	plasmid designation	insert size	source	CDC necessary for
4	pBR322-CDC4(71)	1.9	Byers (unpub)	DNA syn., spindle pole body formation
8	YE24-CDC8-113-7	3.0	Hartwell (unpub)	chromosome rep., error prone repair
11	YEp24-CDC11-5#12	4.5	Pringle (unpub)	microfilament ring formation
12	YRp12-CDC12	3.5	Pringle (unpub)	microfilament ring formation
13	YEp24-CDC13-161-4	7.5	Hartwell (unpub)	medial nuclear division
17	YEp24-CDC17-112-5	12.0	Hartwell (unpub)	medial nuclear division
24	YRp10-CDC24	2.7	Pringle (unpub)	bud emergence, chitin ring form.
28	pBR322-CDC28.2		Reed 1982	start
34	YRp7-CDC34 (79)	2.8	Byers (unpub)	DNA syn., spindle pole body formation
37	pBR322-CDC37.1		Breter et al. 1982	start
39	pBR322-CDC39.1		Breter et al. 1982	start

    References: Breter et al. 1983, Molec. Cell. Biol. 3:881; Reed et al. 1982, Molec. Cell. Biol. 2:412; Maniatis et al. 1978, Cell 15:687.

Bove, C. Stanford University, Stanford, California.* pBR 322 homologous sequences found in a library of **D.melanogaster** genomic DNA.

Some of the cloned cell division cycle (CDC) genes of the yeast **S.cerevisiae** have been used as probes to screen a standard lambda library of **D.melanogaster** genomic DNA (Maniatis et al. 1978) under low stringency hybridization conditions (see previous note). The probes used in the initial screens of this library were prepared by nick translation of insert plus plasmid vector. During this screening, I isolated a Drosophila clone containing several Eco RI fragments (see Figure 1). One of these Eco RI fragments, of about 8 kb, hybridized with four different CDC containing plasmids. Since the CDC inserts are different in these four cases, the homology should reside in some other sequence present on the vector and presumably common to all of them. Three of the four CDCs used were cloned in YEp24 and one in YRp10; both are pBR322 derivatives. YEp24 contains the yeast 2µ origin of replication and the yeast URA 3 sequence. YRp10 contains the yeast Ars 1 origin of replication and URA 3. Thus, these vectors have in common both yeast and bacterial plasmid derived sequences that might be the source of the homology. A second concern was whether the phage isolated from the Drosophila library really contained Drosophila DNA or whether it might have a piece of yeast DNA accidentally inserted during the cloning procedure and, present by chance also in the two CDC vectors, YEp24 and YRp10 (Drosophila stocks are usually kept on food containing yeast!). To distinguish between these two possibilities, and to verify which sequence of the vectors was responsible for the observed hybridization with the "Drosophila" 8 kb fragment, this fragment was gel purified, nick translated and used to probe different indicative digests.

In the first experiment, total Drosophila (Canton S) DNA and total **S.cerevisiae** DNA were digested with Eco RI, run on an agarose gel, transferred to nitrocellulose, and hybridized with this probe using standard stringency conditions (see previous note). The probe hybridized only with Drosophila DNA, thus excluding the possibility of **S.cerevisiae** contamination in the library.

The next step was to identify the sequence in the vector responsible for the homology. For this purpose the two vectors were cut with appropriate enzymes. YEp24 (which was available without the CDC insert) was cut with Eco RI; this gives rise to a fragment containing the 2µ origin of replication and a fragment containing pBR322 and URA 3. YRp10 was cut with Eco RI plus Hind III; this digestion gives rise to four fragments: one containing pBR322, one containing the CDC insert, one containing the URA 3 sequence and a final fragment containing the Ars 1 origin of replication. The digests of these DNAs were separated on a gel, transferred to nitrocellulose, and hybridized with a probe made from the 8 kb Eco RI fragment under standard conditions. The result of this experiment was quite unexpected: the 8 kb fragment hybridized with the fragment containing pBR322 in both cases.

That pBR322 homologous sequences can be present in Drosophila genomic DNA preparations has been shown by other investigators (Singh & Schneider 1984). Consequently, it is possible that a piece of pBR322 homologous plasmid may have been cloned with Drosophila sequences during the construction of the library used in these experiments. This conclusion is also supported by the finding that a probe made from pure pBR322 hybridizes with a fragment of about 5 kb included in the 8 kb fragment of the Drosophila clone at one end of the insert (see Figure 1).

To see if the remaining portion of this clone contains Drosophila sequences, the whole phage was used to prepare a probe for in situ hybridization with salivary chromosomes. The probe hybridizes on the left arm of the second chromosome at about 25A. Thus the phage actually contains Drosophila DNA as well as the contaminating pBR322 "homologous" fragment. Taken together these findings support the conclusion of pBR322 contamination in this particular Drosophila clone from the Maniatis library and may be a useful warning to other investigators.

References: Breter et al. 1983, Molec. Cell. Biol. 3:881; Reed et al. 1982, Molec. Cell. Biol. 2:412; Maniatis et al. 1978, Cell 15:687.

Figure legend (map labels):

λR

B- Bam HI
S- Sal I
R- Eco RI
H- Hind III

R S H R
R B
R H R
H H R
H
R R
R
R

B S H

pBR322 homology

λL

1 kb

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Transient expression of hsp-CAT1 and
copia-CAT1 hybrid genes in **D.melanogaster**
and **D.virilis** cultured cells.

One of the unique opportunities offered by the development of DNA-mediated gene transfer techniques is the ability to study the regulated expression of a gene obtained from one species in the environment of another. Transient expression systems also provide a functional assay for defining DNA sequences flanking structural genes that are necessary for control of their expression.

The chloramphenicol acetyltransferase (CAT) assay developed by Gorman et al. (1982) offers a rapid and sensitive method to detect the functional activity of different promoter sequences. This paper reports experiments on the transient expression of the hsp-CAT1 and copia-CAT1 recombinant plasmid constructed by Di Nocera & Dawid (1983) in **D.melanogaster** and D.virilis cultured cells. Both plasmids are the gift of Dr. I. Dawid. It is well known that promoters of the heat-shock protein genes act just as universal promoters in different species since copia-like elements of **Drosophila melanogaster** are strongly specia specific sequences.

Plasmids were introduced into the 67j25D **D.melanogaster** cultured cells (Kakpakov et al. 1969) and 79f7Dv3g **D.virilis** cells (Braude-Zolotarjova et al. 1986) by the calcium phosphate precipitation method (Wiger et al. 1978). Following transfection cells were heat shocked (where indicated) and harvested by centrifugation. After a wash in phosphate buffered saline, pH 7.4, the cell pellets were recovered and resuspended in 250 µl of Tris-HCl, pH 8.0. Cell lysates, prepared by several freeze-thaw cycles were spun for 5 min in microcentrifuge. Aliquots of the clear supernatants were then assayed for CAT activity. Reaction products were visualized by ascending thin-layer chromatography and autoradiography as described (Gorman et al. 1982). First of all, we have optimized conditions for introducing plasmid DNA into cultured cells. We carried out transfection varying the pH of HeBs buffer for Ca-phosphate precipitation, transfection time and concentration of acid-phenol purified plasmid hsp-CAT1. We found that the level of CAT activity induced by heat shock was highest at HeBs pH 6.7. There was an increase in CAT induction in the range of 5-10 µg DNA per 5 ml of the medium, and then a leveling off. In subsequent studies a DNA concentration of 10 µg/plate was used. CAT activity peaked in 48 hr after transfection (Figure 1).

Figure 1. CAT synthesis assayed in Drosophila cells after transfection with hsp-CAT1 plasmid DNA. Cells were incubated at 25°C (A,B) or at 37°C (C,D) for 30 min and then allowed to recover at 25°C for 60 min. A, C = 67j25D and B, D = 79f7Dv3g cells.

After we have optimized conditions for introducing hsp-CAT1 plasmid into both cell lines used, comparative examinations of the level of CAT induction produced by copia-CAT1 plasmid DNA were carried out.

Copia-Cat1 is taken up more effectively and expressed more strongly in **D.melanogaster** cells than in **D.virilis** cells (Figure 2, A & C). This result indicated that copia promoter shows strong specia specificity unlike hsp promoter. We have also shown, that copia promoter is heat shock regulated in transiently transfected cells. As seen in Fig. 2 (B & D), the copia-CAT1 is transcriptionally activated by heat shock when transfected into both 67j25D and 79f7Dv3g cells and display a temperature response similar to the hsp-CAT1 transfected cells.

These results are consistent with the results of Strand & McDonald (1985) obtained for flies. Copia's ability to be induced by stress is correlated with the presence of sequences homologus sequence which appear to be appropriately positioned within the

Figure 2. CAT synthesis assayed in Drosophila cells transfected with copia-CAT1 plasmid DNA. Cells were kept at 25°C (A,C) or heat shocked at 37°C for 30 min and allowed to recover for 60 min (B,D). A, B = 67j25D and C, D = 79f7Dv3g cells.

to the heat shock promoter consensus element's long terminal repeat.

References: Braude-Zolotarjova, T.Ya., V.T. Kakpakov & N.G. Schuppe 1986, In vitro 22:481-484; Di Nocera, P.P. & I.B. Dawid 1983, PNAS USA 80:7095-7098; Gorman, C.M., L.F. Moffat & B.H. Howard 1982, Mol. Cell. Biol. 2:1044-1051; Kakpakov, V.T., V.A. Gvozdev, T.P. Platova et al. 1969, Genetika (USSR) 5:67-75; Strand, D.J. & J.F. McDonald 1985, Nucleic Acids Res. 13:4401-4410.

Campbell, J. and A.F. Sherald. George Mason University, Fairfax, Virginia. Sensitivity of the suppressor of black mutation of **D.melanogaster** to dietary beta-alanine.

Tan pigment in **D.melanogaster** is synthesized by oxidation of N-beta-alanyl-dopamine, a compound derived by conjugation of dopamine with beta-alamine (BALA) (Black, in press). The black mutation (b, 2-48.5) has decreased levels of BALA (Hodgetts 1972; Hodgetts & Choi 1974; Sherald 1981), and the consequent oxidation of free dopamine results in the formation of black melanin. The lesion responsible for decreased BALA is still unknown; however, the X-linked, recessive suppressor of black (su(b), 1-0) cures the coloration phenotype of black and restores wild type levels of BALA (Sherald 1981). In the absence of black, su(b) results in an increase of over 60% in BALA concentration suggesting that the suppressor may produce a defect in BALA catabolism which allows black to accumulate normal levels of this amino acid despite its decreased synthesis.

In mammals, the first enzyme in BALA catabolism is BALA transaminase, which is also responsible for catabolism of gamma-amino-butyric-acid (GABA) (Wu 1976). Since both BALA and GABA are inhibitory neurotransmitters, sensitivity to dietary administration of these compounds would provide suggestive evidence for defective catabolism in su(b).

Flies were mated in vials made with modified Carpenter's (1950) media containing no addition, or 0.2M of either BALA or GABA. Strains tested included the five alleles of su(b) reported by Sherald (1981), which are maintained both in attached-X stocks, and homo- and hemizygous. An independently isolated allele, su(b)^{DK}, supplied by Erik Bahn was also tested, as was the non-allelic X-linked, semidominant Suppressor of black (Su(b), 1-55.5) reported by Pedersen (1982). All strains contain black except su(b)^{31} which is also maintained homo- and hemizygous without the black mutation.

Table 1. Mean progeny/5 vials from **D.melanogaster** strains on control media and containing 0.2M beta-alanine or 0.2M gamma-amino-butyric acid.

Strain	Control male	Control female	BALA 0.2 M male	BALA 0.2 M female	GABA 0.2 M male	GABA 0.2 M female
su(b)^{31}	27	22	0	0	4	7
su(b)^{31};b	42	38	0	0	44	30
su(b)^{14};b	97	81	0	0	63	57
su(b)^{13};b	58	43	0	0	49	43
su(b)^{12};b	57	56	0	0	55	49
su(b)^{11};b	72	61	0	0	73	55
su(b)^{DK};b	63	59	0	0	NA*	NA
Su(b);b	75	59	41	40	37	27
C(1)DX,yf/Y;bXsu(b)^{31}Y;b	67	39	0	27	34	21
C(1)DX,yf/Y;bXsu(b)^{14}Y;b	29	27	0	17	8	8
C(1)DX,yf/Y;bXsu(b)^{13}Y;b	33	34	0	16	1	6
C(1)DX,yf/Y;bXsu(b)^{12}Y;b	19	25	0	24	30	13
C(1)DX,yf/Y;bXsu(b)^{11}Y;b	18	55	0	23	NA	NA
b	65	42	59	43	47	36
e	101	87	57	49	36	26
s (sable)	85	5	27	13	63	40
Oregon	65	40	46	25	42	21
Canton	35	23	20	19	18	8

* Strain not tested on gamma-amino-butyric acid.

The results shown in Table 1 confirm that su(b) is sensitive to BALA. Virtually 100% lethality was obtained for all su(b) progeny at a concentration of 0.2M. The effect is especially obvious in the attached-X stocks where only males carry the mutation. Since the black mutation alone shows no sensitivity, while su(b)^{31} maintained without black exhibits lethality, the effect must result entirely from su(b). Observation of the vials indicated that death occurred very early in development. There were few first instar larvae, and the large numbers of unhatched eggs suggest that a maternal contribution of the compound might be sufficient to inhibit development in offspring homo- or hemizygous for the suppressor. Interestingly, the unhatched eggs never melanized.

The non-allelic, semidominant Su(b) mutation showed no sensitivity suggesting that it suppresses black by a different mechanism. Ebony which normally accumulates BALA (Jacobs & Brubaker 1963; Hodgetts & Choi 1974) also failed to exhibit sensitivity. However, ebony is deficient in beta-alanyl dopamine synthetase which catalyzes the conjugation of dopamine and BALA (Black, in press). Presumably, catabolism is normal and it does not accumulate sufficient BALA for lethality.

At least at this concentration, there is no evidence of differential sensitivity to GABA. It may be that the concentration tested was insufficient, but it is also possible that, if su(b) is deficient in catabolism, the enzymatic defect may not be the transaminase, or that this enzyme differs in substrate specificity between Drosophila and mammals.

References: Black, B., Insect Biochem. in press; Carpenter, J.M. 1950, DIS 24:96-97; Hodgetts, R.B. 1972, J. Insect Physiol. 18:937-947; ———— & A. Choi 1974, Nature (Lond.) 252:710-711; Jacobs, M.E. & K.K. Brubaker 1963, Science 139:1282-1283; Pedersen, M. 1982, Carlsberg Res. Commun. 47:391-400; Sherald, A.F. 1981, Mol. Gen. Genet. 183:102-106; Wu, J. 1976, in: GABA in Nervous System Function, Roberts et al. (ed.), Raven Press.

Chatterjee, S. and B.N. Singh. Banaras Hindu University, Varanasi, India. Pattern of mating between light and dark forms of **Drosophila ananassae.**

Figure 1. Photograph showing light and dark males and females of **D.ananassae.**

Drosophila ananassae is a cosmopolitan and domestic species. It possesses many cytological and genetical peculiarities (Singh 1985). Futch (1966) reported the existence of ethological isolation between light and dark forms of this species from Samoa which were found to be two different sibling species (**D.ananassae** and **D.pallidosa**) of **D.ananassae** complex (Futch 1973). A number of investigations on behaviour genetics of **D.ananassae** have been carried out by the present authors (Singh & Chatterjee 1985a, b; Singh et al. 1985; Singh & Chatterjee 1986a, b; Chatterjee & Singh 1986; Chatterjee 1986). The results of these studies have shown that there is existence of sexual isolation within **D.ananassae.** Futhermore, there is significant variation in mating propensity when various wild type and mutant strains are compared. Mating ability of inversion karyotypes also varies, and there is positive correlation between inversion frequency and mating propensity in natural populations of **D.ananassae.** The results on the whole suggest that males are more subject to intrasexual selection than females. During the present study we have tested the pattern of mating betwen light and dark forms of **D.ananassae** collected from a natural population in India.

D.ananassae flies were collected from Birlapur, West Bengal, in October and November 1985. The isofemale lines raised from the females of this population show variation in body colour. Two lines, one showing dark flies and another light flies, were maintained for more than ten generations in the laboratory. Some variation has also been observed within each line in body colouration. Figure 1 shows the light and dark forms of **D.ananassae.**

To test the pattern of mating between light (L) and dark (D) forms, mating success between these two types of flies was studied in an Elens-Wattiaux (1964) mating chamber at approx. 24°C temperature under normal laboratory light condition (10 lux - lux range 10,000). Both multiple- and male-choice techniques were employed. Virgin females and males from both the lines were collected and aged for seven days in small batches. In multiple-choice experiment 15 flies of each sex of each type were introduced in mating chamber and number of pairs mated were scored during one hour duration. Thus in each set 60 flies were introduced in mating chamber, and in total five replicates were run. In male-choice experiments both types of males were tested separately. In first experiment 15 light males and 15 females of each of the two types were introduced in mating chamber, and number of pairs mated was scored during one hour duration. Thus, in each set 45 flies were introduced in mating chamber, and in total five replicates were run. In the same way, male-choice experiment was conducted with dark males.

The number of matings in multiple-choice experiment is shown in Table 1. Table 2 shows the X^2 values calculated to assess the relative sexual activity of light and dark forms. The results of male-choice experiments are given in Table 3. In the same table, the values of X^2 calculated to measure the difference between homogamic and heterogamic matings and isolation index (I.I.) are also shown. The values of I.I. and isolation estimate and insignificant difference between homogamic and heterogamic matings indicate that there is no evidence of selective mating between light and dark forms of **D.ananassae.** Furthermore, the X^2 values calculated on marginal totals to assess the relative mating

Table 1. Results of multiple-choice experiment involving light and dark forms of **D.ananassae.**

L♀ x L♂	L♀ x D♂	D♀ x L♂	D♀ x D♂	Isolation estimate
33	20	26	33	0.69

Table 2. X^2 for 1:1 ratios on marginal totals to assess the relative mating propensity of light and dark forms of both sexes based on multiple-choice experiments.

♀ \ ♂	L	D	total
L	33	20	53
D	26	33	59
Total:	59	53	

X^2 L,D female 0.32 P>0.50
X^2 L,D male   0.32 P>0.50

Table 3. Results of male-choice experiments involving light and dark forms of **Drosophila ananassae.**

L male				D male			
Homo.	Hetero.	X^2	I.I.	Homo.	Hetero.	X^2	I.I.
28	36	1.0	-0.125	34	33	0.014	0.015

Table 4. X^2 for 1:1 ratios on marginal totals to assess the relative mating propensity of light and dark forms of both sexes based on male-choice experiments.

♀ \ ♂	L	D	total
L	28	33	61
D	36	34	70
Total:	64	67	

X^2 L,D female 0.6 P>0.30
X^2 L,D male   0.06 P>0.70

propensity of two types of males and females, based on the results of both multiple- and male-choice experiments, suggest that the flies differing in body colouration have similar mating propensity. Thus, it may be concluded that light and dark forms of **D.ananassae** show random mating and similar mating activity.

References: Chatterjee, S. 1986, Studies on behavioral genetics in **Drosophila ananassae**, PhD thesis, Banaras Hindu Univ.; Chatterjee, S. & B.N. Singh 1986, Ind. J. Exp. Biol. 24:195-196; Elens, A.A. & J.M. Wattiaux 1964, DIS 39:118-119; Futch, D.G. 1966, Univ. Texas Publ. 6615: 79-120; _____ 1973, Evolution 27:456-467; Singh, B.N. 1985, Nucleus 28:169-176; Singh, B.N. & S. Chatterjee 1985a, Braz. J. Genetics 8:457-463; _____ 1985b, Can. J. Genet. Cytol. 27:405-409; _____ 1986a, Heredity 57:75-78; _____ 1986b, Genetica, accepted; Singh, B.N., S. Chatterjee & S. Roy 1985, Ind. J. Exp. Biol. 23:661-662.

Cintrón, C.N. and Y. Pérez-Chiesa. University of Puerto Rico, Rio Piedras. Induction of dominant lethals in **Drosophila melanogaster** treated with fagaronine.

Fagaronine chloride (CAS 225965) is a strong antileukemic alkaloid isolated from **Fagara zanthoxyloides** (Mesmer et al. 1972; Tin-Wa et al. 1974). The drug binds to DNA and double stranded RNA (Pezzuto et al. 1983); it inhibits nucleic acid synthesis and protein synthesis in mammalian systems (Sethi & Sethi 1975; Sethi 1976, 1979; Casiano-Torres & Baez 1986), and the activity of reverse transcriptase activity of RNA oncogenic viruses (Sethi & Sethi 1975). Fagaronine was shown to induce sex-linked recessive lethals and partial male sterility in **D.melanogaster** (Perez-Chiesa & Cintron 1986). In this work, we tested its potency for the induction of dominant lethals in **D.melanogaster** males.

The flies were reared in banana medium under laboratory conditions at 25°C. Twenty **D.melanogaster** males with a ring-X-chromosome and a genetically marked Y-chromosome, R(1),2, y B/B^{S}Yy$^+$, 3 to 4 days old, were exposed for 48 hr to a 5% sucrose solution containing 0.0 mM (Control), 1.5 mM or 3.0 mM of fagaronine chloride (NSC 157995), as described in Perez-Chiesa & Cintron (1986). The fagaronine was dissolved first in dimethylsulfoxide (DMSO) and then in the sucrose solution. After treatment, each male was crossed individually to just one y w sn^3 virgin female. Three successive broods were collected by transferring the males without anesthesia to a new virgin female every three days. The females, in turn, were subcultured every day for seven consecutive days. The ratio of the number of eggs producing live adults to the total number of eggs, was computed as percentage relative eclosion. For each brood, treatments were compared to their controls and among themselves using simple contingency tests. In making the comparison, those eggs of broods producing no adult progeny were not counted and the male was considered sterile for that brood.

The percenatage of dominant lethals was calculated as follows: % DL - 100% - % relative eclosion. The results are shown in Table 1. The low progeny yield obtained in all groups and broods was unexpected. Even among controls, an average of only 58% of the eggs reached adulthood. Since there were few larvae in each vial (an average of 17 eggs per vial) the competition for food could not have been a major cause of mortality during larval development. We suspect the low yield is explained by lowered Darwinian fitness due either to continued inbreeding of the stocks used or to effects of the gene markers used. However, this problem does not affect the results since the percenteage of dominant lethal is calculated relative to control.

The relative frequency of dominant lethals in every brood for each treatment is high and significantly different from the control. However, the difference between the frequencies of dominant lethals in the 1.5 mM and 3.0 mM concentrations is not significant (16.9% vs 18.8%). Thus, no dose effect is observed. No significant difference is found either, when individual broods are considered. The same pattern of brood sensitivity is found for both, the 1.5 mM and the 3.0 mM concentration. That is, mortality in brood 2 was higher and significantly different from that of brood 1 (P<0.05 for 1.5 mM; P<0.01 for 3.0 mM) but not from that of brood 3. Brood 1 corresponds to sperm exposure to the treatment. If there is no inhibition of spermiogenesis, brood 2 corresponds to spermatid exposure and brood 3 to spermatocyte and spermatogonia exposure. Therefore, it appears as if spermatids are more susceptible to treatment than sperms.

Table 1. Dominant lethals in the germ cells of **D.melano-gaster** males after exposure to fagaronine for 48 hr.

Conc. mM	Brood	No. of eggs	No. of flies	% eclosion	% relative eclosion	% dominant lethals
0.0	1	2039	1207	59.20	100.0	---
1.5	1	1613	812	50.34	85.0	15.0[a]
3.0	1	1974	979	49.59	83.8	16.2[a,c]
0.0	1	2045	1151	56.28	100.0	---
1.5	2	1228	562	45.77	81.3	18.7[a,d]
3.0	2	2700	1224	45.33	80.5	19.5[b,c,e]
0	3	4268	2515	58.93	100.0	---
1.5	3	3430	1667	48.60	82.5	17.5[b]
3.0	3	3138	1498	47.73	81.0	19.0[b,c]
0	1-3	8352	4873	58.34	100.0	---
1.5	1-3	6271	3041	48.49	83.1	16.9[b]
3.0	1-3	7812	3701	47.38	81.2	18.8[b,c]

a=significantly different from its control (P<0.01).
b=significantly different from its control (P<0.001).
c=not significantly different from 1.5 mM.
d=signif. diff. from 1.5 mM, brood 1 (X^2=5.66, P<0.05).
e=signif. diff. from 3.0 mM, brood 1 (X^2=8.14, P<0.01).

The sensitivity of each brood is not necessarily or exclusively of a genetic nature. The term dominant lethal is an operational one. True dominant lethals, mainly chromosomal aberrations which in hetrozygous condition do not allow for normal development, may be mimicked by unfertilized eggs or by insemination with non-functional sperm (Brink 1969). Therefore, it is possible that besides a genetic effect the drug has a spermicidal effect and/or spermiogenesis may be somewhat inhibited resulting in fewer sperms being produced. This is supported by the fact that in the second brood there were more sterile males among those treated with 3.0 mM than among those treated with 1.5 mM or the control (7/18, 0/18, and 1/18, respectively). In our previous work (Perez-Chiesa & Cintron 1986), partial sterility of males treated with 3.0 mM was detected in all broods. Furthermore, only with the 1.5 mM concentration was the frequency of sex-linked recessive lethals significantly higher than the control. Therefore, exposure of males to fagaronine at the two concentrations used could induce different frequencies of true dominant lethals but the dose effect may be masked or a brood-pattern sensitivity may be seen because of the toxic effects of the drug. The capacity of fagaronine and related compounds, to induce chromosome breaks and loss is now under investigation.

References: Brink, N.G. 1969, Mutation Res. 8;139-146; Casiano-Torres, C.A. & A. Baez 1986, Biochem. Pharmac. 35:679-685; Messmer, W.M., M. Tin-Wa, H. Fong, C. Bevelle, N. Farnsworth, D. Abraham & J. Trojanek 1972, J. Pharm. Sci. 61:1858-1859; Perez-Chiesa, Y. & C.N. Cintron 1986, Mutation Res. 173:193-196; Pezzuto, J.M., S.K. Antosiak, W.M. Messmer, M.B. Slaytor & G.R. Honig 1983, Chem. Biol. Interact. 43:323-339; Sethi, M.L. 1979, J. Natl. Prod. 42:187-196; Sethi, V.S. 1976, Cancer Res. 36:2390-2395; ____ & M.L. Sethi 1975, Biochem. Biophys. Res. Commun. 63:1070-1076; Tin-Wa, M., C.L. Bell, C. Bevelle, H. Fong & N.R. Farnsworth 1974, J. Pharm. Sci. 63:1476-1477.

Daniels, S.B., I.A. Boussy, A. Tukey, M. Carrillo and M.G. Kidwell. University of Arizona, Tucson. Variability among "true M" lines in P-M gonadal dysgenesis potential.

"True M" lines of **D.melanogaster** are lines that completely lack any P element-hybridizing sequences in their genomes (Engels 1984; Kidwell 1985). Since all lines collected from the wild within the last ten years have been found to carry P elements (D. Anxolabehere, M.G. Kidwell & G. Periquet, unpubl. data), it seems that true M lines are laboratory relicts of pre-P element-invasion populations of **D.melanogaster.**

It has generally been assumed that the various tests of P-M hybrid dysgenesis using traits such as gonadal dysgenesis (GD) are measuring properties of the P element system. As a corollary, it has been assumed that different true M lines are equivalent. We report here tests of a variety of true M lines suggesting that this is not always correct, and that true M lines can differ in their responses to P lines.

The true M lines we have tested are as follows (AC indicates a stock in the laboratory of Arthur Chovnick, University of Connecticut, Storrs, Connecticut; MGK indicates the laboratory of Margaret G. Kidwell, University of Arizona, Tucson, Arizona): **Canton-S** (AC): originally collected in the 1930's in Canton, Ohio; **0231.1** (AC): obtained from Mid-America Drosophila Stock Center at Bowling Green State University, Bowling Green, Ohio; originally collected at Ica, Peru, in 1956; **ry^{42}** (AC): X-ray induced point mutation at XDH locus, 1964; Oregon-R background; **ry^{506}** (AC): derived from ry^{+5}; **Kalahari** (AC): received from the University of South Africa, Johannesburg, South Africa, in the early 1970's; **Limpopo** (AC): received from the University of South Africa, South Africa, in the early 1970's; **Df ry^{36}/MKRS** (AC): Df(3R)ry^{36}, ry^{36}/Tp(3;3)MKRS, M(3)S34 Kar ry^2 Sb; **CyO;TM2(ry)/ Xa** (AC): In(2LR)O, Cy pr cn^2;

Table 1. Mean percent dysgenic ovaries (± standard error) among F1 females of crosses between females of the true M lines listed and males of the moderate P line Agana, reared at 25°C and 29°C, and among female offspring of intrastrain crosses of the same lines, reared at 29°C. All means are of two broods of two replicates (four progenies; 50 females scored from each) unless followed by a number in parentheses, in which case the number of progenies scored is given.

| | Percent dysgenic ovaries among progenies | | |
| | x Agana males | | Intrastrain |
True M line	25°C	29°C	29°C
CyO TM2(ry)/Xa	10.0 ± 10.0 (2)	28.8 ± 6.3	1.3 ± 0.7 (3)
CyO TM2(ry$^+$)/Xa	13.5 ± 3.5 (2)	32.0 ± 5.6	0.3 ± 0.3
CyO MKRS/Xa	20.0 ± 2.8 (2)	40.3 ± 7.2	2.7 ± 0.7 (3)
0231.1	5.5 ± 1.0	43.3 ± 3.3	0 ± 0 (2)
Cy/Pm;DcxF/Sb	42.5 ± 9.5 (2)	50.0 ± 5.8	0 ± 0
CyO TM6/Xa	36.7 ± 11.9 (3)	60.3 ± 3.6	0 ± 0
ry^{42}	21.8 ± 7.4	66.0 ± 3.3	0 ± 0 (2)
Kalahari	30.3 ± 2.5	66.0 ± 3.8	1.3 ± 0.3
Canton-S	18.0 ± 5.7	67.8 ± 0.9	1.7 ± 0.3 (3)
Df ry^{36}/MKRS	71.0 ± 8.4	82.8 ± 3.9	6.5 ± 0.3
ry^{506}	53.5 ± 4.2	84.3 ± 2.2 (3)	0 ± 0
H41 (IM)	60.0 ± 5.0 (2)	86.8 ± 3.3	0 ± 0
Limpopo	63.8 ± 7.2	89.3 ± 2.6	19.0 ± 4.4

1)
$$\frac{X^{Bal}}{X^{Bal}} \quad \frac{CyO\ TM2(ry^+),\ Ubx}{Xa} \quad \times \quad \frac{X^L}{Y^L} \quad \frac{2^L}{2^L} \quad \frac{3^L}{3^L} \qquad \text{single}\ \sigma$$

2)
$$\frac{X^{Bal}}{X^{Bal}} \quad \frac{CyO\ TM2(ry^+),\ Ubx}{Xa} \quad \times \quad \frac{X^{Bal}}{Y^L} \quad \frac{CyO}{2^L} \quad \frac{TM2(ry^+),\ Ubx}{3^L}$$

3)
$$\frac{X^{Bal}}{X^{Bal}} \quad \frac{2^L\ TM2(ry^+),\ Ubx}{Xa}$$
$$\frac{X^{Bal}}{X^{Bal}} \quad \frac{CyO \qquad 3^L}{Xa}$$
$$\frac{X^{Bal}}{X^{Bal}} \quad \frac{2^L \qquad 3^L}{Xa}$$
$$\frac{X^{Bal}}{X^{Bal}} \quad \frac{CyO\ TM2(ry^+),\ Ubx}{Xa}$$
$$\frac{X^{Bal}}{X} \quad \frac{2^L \qquad 3^L}{CyO\ TM2(ry^+),\ Ubx}$$

Females of each assortment type were crossed to Agana males and to males of the same assortment type (inter se).

F1 females reared at 29°C were dissected and scored for development of ovaries.

Dysgenesis scores were separately recorded for the different F1 phenotypes.

In(3LR)Ubx^{130}, Ubx^{130} e^s ry/T(2;3)ap^{Xa}, ap^{Xa}; **CyO;TM2(ry$^+$)/Xa** (AC): In(2LR)O, Cy pr cn^2; In(3LR)Ubx^{130} e^s/T(2;3)ap^{Xa}, ap^{Xa}; **CyO;MKRS/Xa** (AC): In(2LR)O, Cy pr cn^2; Tp(3;3)MKRS, m(3)S34 Kar cy^2 Sb/T(2;3)ap^{Xa}, ap^{Xa}; **CyO;TM6/Xa** (MGK): In(2LR)O, Cy pr cn^2;In(3LR)TM6, Ubx^{67b} e^s/T(2;3)ap^{Xa}, ap^{Xa}; courtesy of Ms Susan Shepherd, University of California, Davis, California; **H41(IM)** (MGK): Basc(In (1)sc^{S1L}sc^{8R}+S, sc^{S1} sc^8 w^a); SM1 (In(2LR)SM1, al^{2}Cy cn^2 sp^2)/ In(2LR)bw^{V1}, ds^{33K} dp b bw^{V1}; TM2 (In(3LR)Ubx^{130}, Ubx^{130} e^s)/ Sb; spa^{pol}; see Kidwell 1981; **Cy/Pm;DcxF/Sb** (MGK): In(2L+2R)Cy, Cy/ bw^{V1}; In(3LR)DcxF, ru h D/Sb; see Bucheton & Bregliano 1982.

All lines were tested for complete absence of P element-hybridizing DNA by Southern blot analysis of genomic DNA, using as a probe the plasmid pπ25.7bwc which carries nucleotides 40-2885 of the complete 2907 bp P element. No hybridization was detected in any of the lines (data not shown).

Table 1 shows the results of GD tests of the true M lines using a moderately strong P line (**Agana**, from Agana, Guam, collected in the 1960's). Twenty females of each line were crossed to twenty males of Agana, and fifty F1 females from two sequential broods were dissected and scored for the presence or absence of developed ovaries. This was repeated once for each cross. Crosses were made and the progenies reared at the temperatures indicated in Table (25°C or 29°C). The means and standard errors of the percent undeveloped ovaries presented in Table 1
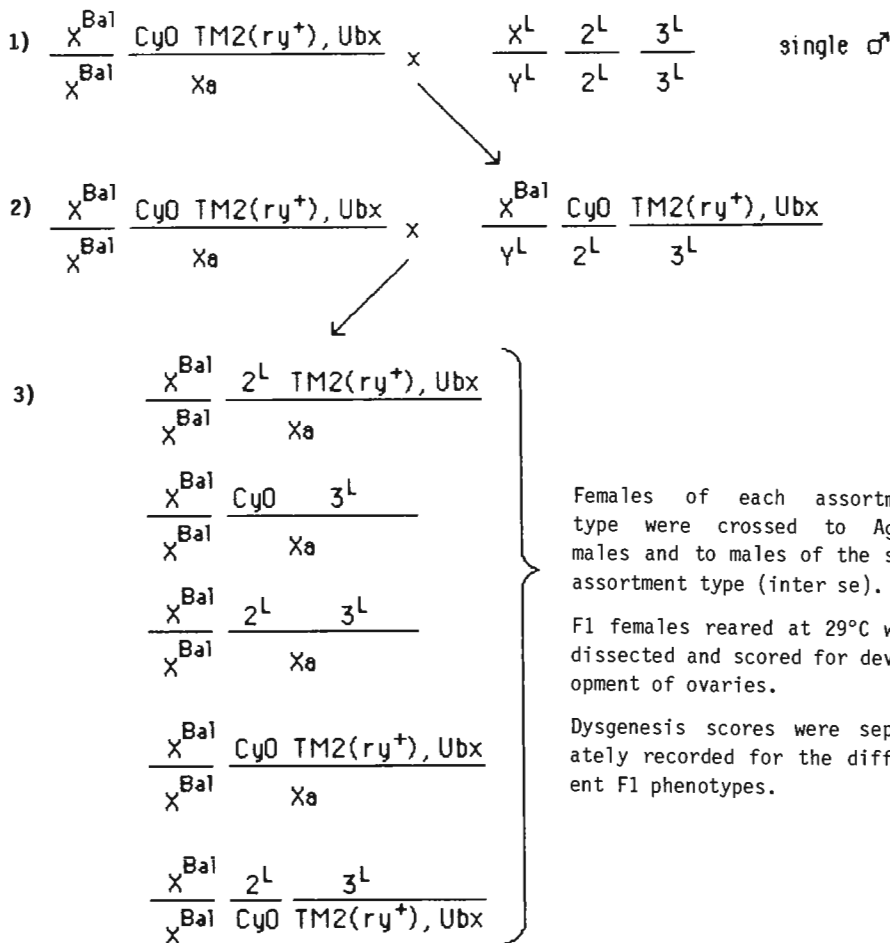
Figure 1. Crosses to evaluate autosomal contributions to the intrastrain and P-M GD sterilities of Limpopo. The superscript L indicates a chromosome from Limpopo. The CyO TM2(ry$^+$)/Xa balancer stock is described in the text; X^{Bal} is the X chromosome from the balancer stock. Crosses 1 and 2 were carried out at 23°C and 25°C, respectively.

Table 2. Percent GD sterilities of various combinations of Limpopo second (2^L) and third (3^L) chromosomes with balancer chromosomes, in crosses to Agana males and inter se, reared at 29°C. In each case the X chromosome is derived from the balancer stock. All means and standard errors are of three replicates each. Note that in crosses involving the translocation Xa there was the possibility of recombination involving the left arms of both autosomes (see Figure 1).

| Genotype of female parent | Crosses with Agana males | | Inter se crosses | |
	Genotype of offspring	mean GD sterility	Genotype of offspring	mean GD sterility
Cy;Ubx/Xa	Cy;Ubx	48.3±10.5	Cy;Ubx/Xa	0 ± 0
	Xa	48.7± 2.0		
2^L;Ubx/Xa	2^L;Ubx	41.0± 5.9	2^LUbx/Xa	0 ± 0
	Xa	42.3± 4.5		
Cy;3^L/Xa	Cy;3^L	77.7± 5.5	Cy;3^L/Xa	2.3±1.5
	Xa	49.0± 8.5		
2^L;3^L/Xa	2^L;3^L	78.3± 1.9	$2^L/2^L$;$3^L/3^L$	16.1±8.0
	Xa	53.7± 1.5	2^L;3^L/Xa	5.3±2.9
2^L/Cy;3^L/Ubx	Cy;Ubx	83.0± 7.8	2^L/Cy;3^L/Ubx	14.3±0.9
	2^L;Ubx	79.0± 3.5	$2^L/2^L$;3^L/Ubx	19.0±2.9
	Cy;3^L	95.3± 3.3	2^L/Cy;$3^L/3^L$	19.4±7.0
	2^L;3^L	89.0± 3.6	$2^L/2^L$;$3^L/3^L$	19.6±3.3

are thus of four progenies (two broods of two replicates), except as noted. At 29°C, the usual temperature for cross A and A* testing of lines (Engels & Preston 1980; Kidwell, Frydryk & Novy 1983), mean sterility scores ranged from 29% to 89%.

Table 1 also shows intrastrain sterility tests of the same lines at 29°C. Most lines showed low intrastrain sterility (3% or less). Limpopo's intrastrain GD sterility (at 25°C, 7.0 ± 1.9% in females; at 29°C, 19% in females and 16.5 ± 2.5% in males) is exceptionally high. Figure 1 presents a set of crosses designed to evaluate the autosomal contributions of Limpopo to GD sterilities in intrastrain and P-M reference crosses by constructing individuals carrying various combinations of autosomes from Limpopo and the balancer stock. Table 2 presents the results, which show that GD sterility in the P-M reference cross, relative to the "basal" effects of the balancer chromosomes used, is due largely to effects of the Limpopo third chromosome. In contrast, the intrastrain GD sterility appears to be due largely to an interaction

between the second and third chromosomes. Therefore, Limpopo's high intrastrain sterility, while interesting in itself, does not seem to completely account for the high GD sterility seen in the P-M reference crosses.

A series of tests similar to those reported in Table 1 was also done with a weak P line, Chepachet (collected at Chepachet, Rhode Island, in 1974). The results produced a similar ranking of the true M lines for GD sterility at both 25°C and 29°C (data not shown). Chepachet, however, is anomalous in consistently producing more GD sterility at 25°C than at 29°C, a result at variance with other P lines whose temperature-dependent characteristics have been investigated.

We conclude from these experiments that all true M lines are not equal, at least with respect to their susceptibility to gonadal dysgenesis in crosses with P lines. We do not know whether the differences are due to genetic differences between lines (in the sense of different alleles at loci affecting susceptibility to P activity; Slatko et al. 1983) or to effects of other transposable element systems, whether interacting with the P element system (Gerasimova et al. 1984), or acting independently to produce GD sterility (Blackman et al. 1987; Yannopoulos et al. 1987).

References: Blackman, R.K., R. Grimaila, M. Macy, D. Koehler & W.M. Gelbart 1987, Cell 49:497-505; Bucheton, A. & J.-C. Bregliano 1982, Biol. Cell 46:123-132; Engels, W.R. 1984, Science 226:1194-1196; —— & C.R. Preston 1980, Genetics 92:161-174; Gerasimova, T.I., L.M. Mizrokhi & G.P. Georgiev 1984, Nature 309:714-716; Kidwell, M.G. 1981, Genetics 98:275-290; —— 1985, Genetics 111:337-350; ——, T. Frydryk & J.B. Novy 1983, DIS 59:63-69; Slatko, B., S. Hanlon & R.C. Woodruff 1983, DIS 59:115-117; Yannooulos, G., N. Stamans, M. Monastirioti, P. Hatzopoulos & C. Lewis 1987, Cell 49:487-495.

David, J.R., P. Capy, S. McEvey, K. Daly and F. Borai.* Lab. de Biologie et Génétique Evolutives, CNRS, Gif-sur-Yvette, France; * Dept. of Genetics, Fac. of Agriculture, Alexandria, Egypt. Latitudinal clines of Adh gene frequencies in **Drosophila melanogaster**: a Mediterranean instability.

In **D.melanogaster**, the Adh gene (alcohol dehydrogenase) has two widespread alleles, F and S, found in all natural populations but with highly different frequencies. These geographic variations are not random but exhibit marked clinal tendencies, i.e., an increase of the F allele when moving from lower to higher latitudes.

Such clines have been observed in various parts of the world, in North America and Australia (Johnson & Schaffer 1973; Oakeshott et al. 1982), between Europe and equatorial Africa, and between the equator and southern Africa (David et al. 1986). These different continents harbor populations with very different histories of colonization. Ancestral populations are found in the Afrotropical region. Colonization of North Africa and Eurasia presumably occurred some millennia ago, and independently of human transport (see Lachaise et al. 1987). A similar hypothesis can be made for the colonization of southern Africa. In America and Australia, on the other hand, **D.melanogaster** is likely to have been introduced more recently, within the last few centuries, due to the activities of man (David & Tsacas 1981).

American populations were presumably founded by several independent introductions, from tropical Africa to tropical America, during the slave trade period, and more recently from Europe. The history of Australian colonization is more conjectural, although an introduction from southeast Asia can be excluded (David & Tsacas 1981): a mixture of African and European populations seems a likely hypothesis.

An independent occurrence of parallel latitudinal clines on different continents is a powerful, but not conclusive, argument that the clines have some adaptive significance. It could be proposed, for example, that the difference between Afrotropical and European populations is due mainly to genetic drift during the northward extension of the species range. Transfers, within the same latitudes, of populations from Africa and Europe to Central and North America, respectively, could have led to a similar clinal pattern. For a better analysis of these geographic clines, it seemed interesting to consider, not only the latitudinal tendencies and correlations, but the absolute gene frequencies in different parts of the world. A preliminary report is presented here.

In the European-African study (David et al. 1986), we noticed that the cline was not linear but divided into three parts, with a very steep increase between 30°-40° of latitude (see Figure 1). In that study, all populations from these intermediate latitudes were collected in countries with a Mediterranean climate, i.e., a hot, dry summer and a mild, rainy winter. In the world, other places have such climatic features, such as the southeastern coast of Australia, California and Chile. Australian values (Oakeshott et al. 1982) have been plotted in Figure 1, as well as some data from California (Smith et al. 1984). Remarkably these values are included within the general sigmoid cline found between Europe and Africa, thus steep latitudinal variation occurs in various countries of the world which have Mediterranean climates.

Places between 30° and 40° latitude do not always have Mediterranean climates, for example, the East Coast of U.S.A. In this area a detailed set of Adh data is available (Johnson & Schaffer 1973). As shown in Figure 2, this cline is much more regular and almost linear, and quite different from "Mediterranean" populations for latitudes between 30° and 40°. A few populations, taken fron non-Mediterranean localities on the West Coast (Smith et al. 1984) also fit this linear cline.

It was previously known that, at similar temperate latitude, Adh frequencies could be different on different continents (Oakeshott et al. 1982; David et al. 1986). We now get a more coherent overall pattern if we take into consideration the various climates which may exist at intermediate latitudes. The very steep cline which occurs in Mediterranean countries indicates great variations over short geographic range. Other data, most of which are unpublished, show that significant variations can be observed in the same locality, either during different seasons of the year or in the same season over successive years. All these observations justify our use of the term "Mediterranean instability". But what is the significance of that instability?

A first hypothesis is that the cline is not adaptive but is mainly due to gene flow and diffusion between two stable, tropical or temperate, populations. We could, for example, assume that some kind of selection maintains a very high frequency of the S allele in the tropics and of the F allele in cold temperate countries, while no equilibrium is reached in intermediate Mediterranean places. However, some geographic features argue against this hypothesis: North African countries are separated from tropical places by the Sahara desert which prevents any gene flow between the two. Another interpretation based on the interaction between selective and stochastic processes seems, for the moment, more likely. Summer climate in Mediterranean countries is very harsh for Drosophila populations, because of heat, aridity and lack of resources. In many places, summer populations are likely to disappear, while new colonizations occur in winter. Such a succession of extinctions and colonizations will lead to founder effects, i.e., a classical means for producing large but unpredictable genetic fluctuations. Oakeshott et al. (1982) pointed
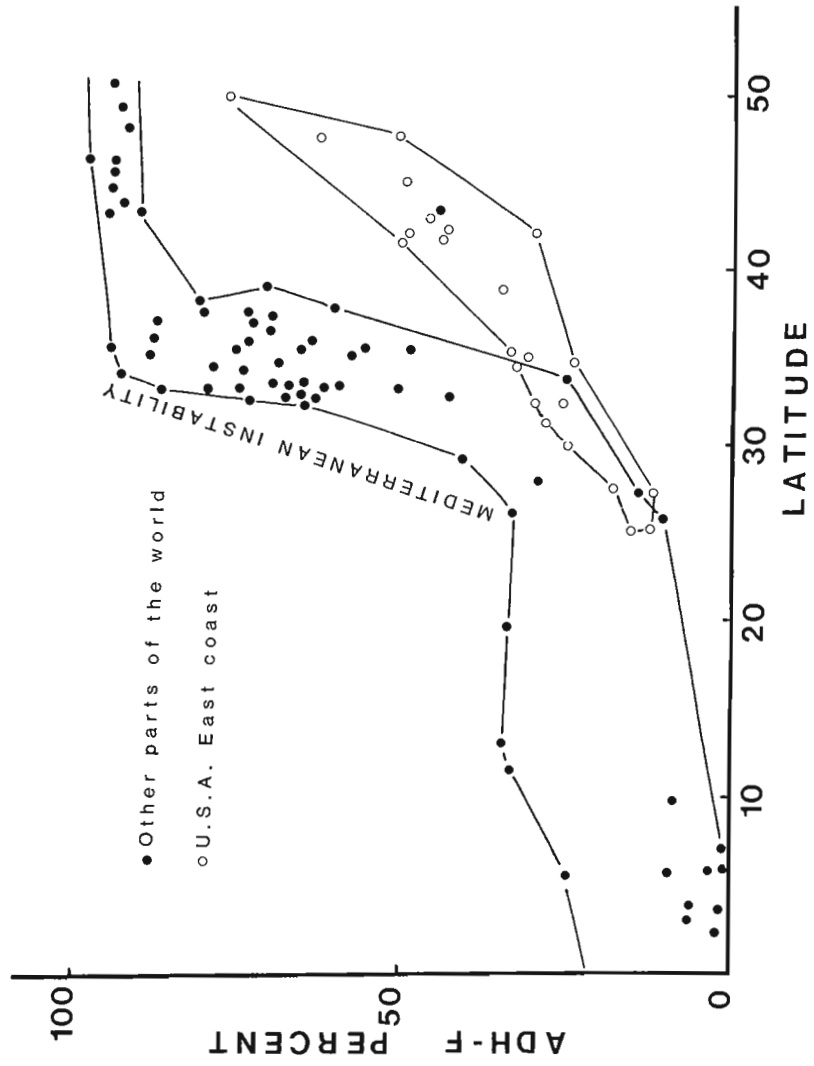
Figure 2. Relationship between frequency of Adh^F allele and latitude of origin. On the East Coast of U.S.A., where tropcial conditions are progressively replaced by temperate, humid conditions, the cline is approximately linear. In other parts of the world (same values as in Figure 1), where the transition between tropical and cool temperate conditions occurs through a Mediterranean climate, a non-linear, sigmoid cline is observed with a genetic instability in Mediterranean countries.
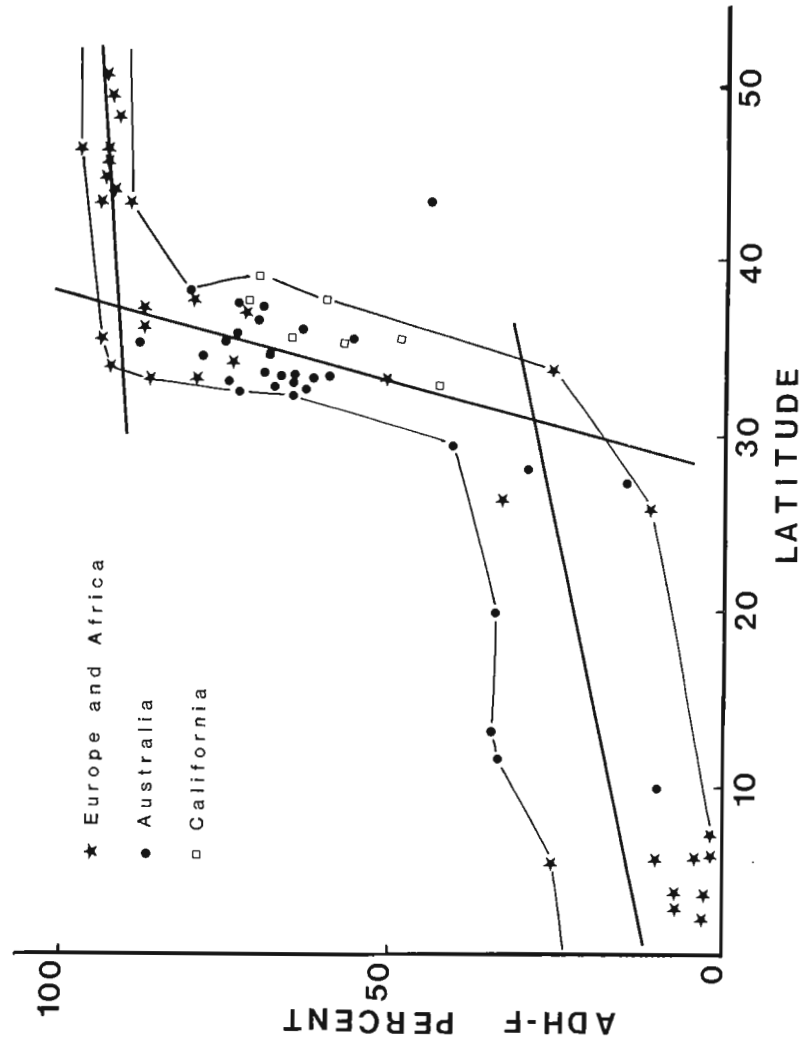
Figure 1. Relationship between frequency of Adh^F allele and latitude of original of natural populations from Europe, Africa, Australia and California. Note the non-linearity of the cline. Between approximately 30° and 40° of latitude, populations live under Mediterranean climates.

out that most Adh latitudinal variations could be accounted for by the amount of rain falling during the most rainy month of the year, in the cold season. This makes sense with our observations if we consider that winter rain in such countries will determine the peak of abundance of populations, but also that this amount of rain is quite variable according to local geographic conditions and also between years. Selective pressures could therefore exist in winter but would be quite variable over short geographic range or successive years.

In places like the East Coast of America, tropical conditions are progressively replaced by a temperate humid climate without a Mediterranean intermediate, Drosophila populations are likely to be bigger in more stable environments which could explain the linearity of the cline. Finally, in spite of numerous field investigations, much remains to be understood with respect to geographic variations of Adh frequencies. Populations from countries with typically unstable Mediterranean climates now appear to be more worthy of attention in order to gain a better understanding of the selective or stochastic processes which may operate.

References: David, J.R. & L. Tsacas 1981, C.R. Soc. Biogeog. 57:12-26; David, J.F., H. Mercot, P. Capy, S.F. McEvey & J. Van Herrewege 1986, Gen. Selec. Evol. 18:405-416; Lachaise, D., M.L. Cariou, J.R. David, F. Lemeunier, L. Tsacas & M. Ashburner, Evol. Biology (in press); Johnson, F.M. & H.E. Schaffer 1973, Biochem. Genet. 10:149-163; Oakeshott, J.G., J.B. Gibson, P.R. Anderson, W.R. Knibb, D.G. Anderson & G.K. Chambers 1982, Evolution 36:86-96; Smith, M.R., G.K. Chambers, L.D. Brooks, F.M. Cohan & S.C. Cohan 1984, DIS 60:188-189.

Dhingra, G. and N.K. Vijayakumar.
Haryana Agricultural University, Hisar, India.
Genotoxic studies in Drosophila treated with dimethoate insecticide.

Organophosphorous (OP) and carbamate insecticides have conspicuously replaced organochlorines in agricultural practices. The acute toxicity of OP for insects and mammals is mainly due to the blocking of the cholinesterase enzyme by its phosphorylation. However, there are confounding reports as to whether OP insecticides alter the genetic material or not. Dimethoate, one such compound, has been known to cause chromosomal aberrations in lymphocytes of persons exposed to it (Czeizel et al. 1973; Yoder et al. 1973) as well as in plants (Ma et al. 1984; Kaur & Grover 1985). In bacteria, both positive and negative mutagenicity of dimethoate are known (Hanna & Dyer 1975; Shirasu et al. 1976; Moriya et al. 1983).

In the present study, the Oregon-K strain of **D.melanogaster** maintained at 25±2°C, was used. Dimethoate, dissolved in acetone, was administered by exposing the flies to standard cornmeal-yeast agar medium containing its varying concentrations. The concentrations, at which enough progeny could be raised, were selected for DL (0.25, 0.15, 0.10 ppm) and SLRL (0.25, 0.15 ppm) tests. For DL studies, the adult male progeny were individually crossed with two Or-K virgins and a score of the hatched and unhatched eggs, after a suitable incubation time, was made. The data are presented in Table 1 and analysis of variance (ANOVA) demonstrated that the dimethoate-induced dominant lethality was non-significant from both, normal and solvent controls.

Table 1. Dominant lethal mutations induced by dimethoate in **D.melanogaster**.

Conc. (ppm)	Eggs tested	Unhatched eggs	Lethality (%)
0.25	1596	811	50.81 ± 14.65
0.15	862	605	70.19 ± 7.19
0.10	2950	545	18.47 ± 2.22
A*	1337	381	28.50 ± 4.81
C**	903	213	23.59 ± 0.43

*Acetone(5.00 ppm) **normal control

Table 2. Sex-linked recessive lethal mutations induced by dimethoate in **D.melanogaster**.

Conc. (ppm)	Chromosomes tested	SLRL	Lethality (%)
0.25	711	2	0.28 ± 0.199
0.15	691	1	0.14 ± 0.199
A*	747	6	0.80 ± 0.13
C**	581	2	0.34 ± 0.28

*Acetone(5.00 ppm) **normal control

For SLRL studies, the Basc test protocol was followed. The treated males were crossed with M-5 virgins and data for induced lethality were recorded (Table 2). As for the DL test, ANOVA and the two-way contingency tests failed to reveal any significant deviation from the controls, thereby demonstrating the inability of dimethoate to cause lethal mutations at the concentrations tested. In the meiotic cells of grasshoppers also, dimethoate did not affect the sensitivity of the centromeric regions (Majumdar & Hall 1973). Neither DL nor chromosomal aberrations in bone-marrow and spermatogonial cells of mice were induced by dimethoate (Degraeve et al. 1983; Fishbein 1983). In Drosophila also, it had not been found to increase the frequency of lethals (Valencia 1977) and partial chromosomal loss (Woodruff et al. 1983). These corroborate the present findings and indicate that dimethoate fails to cause mutations in germ cells of mice and Drosophila.

References. Czeizel, A., Trinh van Bao, I. Szabo & P. Ruzicska 1973, Mutation Res. 21:187-188; Degraeve,N., M. Chollet, J. Moutschen-Dahmen, J. Moutschen, J. Gilhot-Delhalle & A. Collizi 1983, Mutation Res. 113:244; Fishbein, L. 1983, Proc. XV Int. Cong. Genetics, New Delhi, India, Dec. 12-21, p99; Hanna, P.J. & K.F. Dyer 1975, Mutation Res. 28:405-420; Kaur, P. & I.S. Grover 1985, Cytologia 50:187-197; Ma, Te-Hsiu, M.M. Harris, Y.A. Anderson, I.

Ahmed, K. Mohammad, J.L. Hare & G. Lin 1984, Mutation Res. 138:157-167; Majumdar, S.K. & R.C. Hall 1973, J. Heredity 64:213-216; Moriya, M., T. Ohta, K. Watanabe, T. Miyazawa, K. Kato & Y. Shirasu 1983, Mutation Res. 116:185-216; Shirasu, Y., M. Moriya, K. Kato & T. Kada 1976, Mutation Res. 40:19-30; Valencia, R. 1977, Report EPA 68-01-2474, pp70; Woodruff, R.C., J.P. Phillips & D. Irwin 1983, Environ. Mutagenesis 5:835-846; Yoder, J., M. Watson & W.W. Benson 1973, Mutation Res. 21:335-340.

Dhingra, G. and N.K. Vijayakumar. Haryana Agricultural University, Hisar, India. Mutagenic evaluation of lindane in **D.melanogaster.**

Table 1. Dominant lethal mutations induced by lindane in **D.melanogaster.**

Conc. (ppm)	Eggs tested	Unhatched eggs	Lethality (%)
5.00	1085	285	26.27 ± 9.32
4.00	1361	505	37.11 ± 11.43
3.00	1071	385	35.95 ± 10.54
2.00	2381	307	12.89 ± 5.13
A*	1337	381	28.50 ± 4.81
C**	903	213	23.59 ± 0.43

*Acetone(5.00 ppm) **normal control

Table 2. Sex-linked recessive lethal mutations induced by lindane in **D.melanogaster.**

Conc. (ppm)	Chromo-somes tested	SLRL	Lethality (%)
5.00	500	1	0.20 ± 0.13
4.00	649	1	0.15 ± 0.09
3.00	584	2	0.34 ± 0.16
2.00	659	2	0.30 ± 0.19
A*	747	6	0.80 ± 0.13
C**	581	2	0.34 ± 0.28

*Acetone(5.00 ppm) **normal control

Lindane, an organochlorine insecticide, though banned or under restricted usage in most western countries, is still widely used in India, though there are a number of reports about its carcinogenicity (Ishidate & Odashima 1977; Ward et al. 1979) and its inhibitory effect on RNA, DNA and protein synthesis in algal cell cultures (Jeanne 1979) and on growth in **Neurospora crassa** and **Saccharomyces cerevisiae** (Srivasan & Radhakrishnamurthy 1978). It has also been found to induce morphological mutants in blue-green algae (Das & Singh 1978), aberrant genocytes in quail (Lutz-Ostertag 1981), respiratory adaptation mutants in yeast (Pasupathy et al. 1981) and chromosomal aberrations in rat fibroblasts in vivo and in vitro (Zimonyic et al. 1981). However, because of limited knowledge about its effect on germ-cell mutagenesis, lindane was tested for the induction of DL and SLRL mutations in the Oregon-K strain of **D.melanogaster.**

Preliminary studies on the toxicity of lindane, dissolved in acetone, and mixed in the standard cornmeal-yeast-agar medium were conducted to assess its effect on the fertility of the fly. The maximum dose (5.0 ppm) which gave enough progeny and three lower doses (4.0, 3.0, 2.0 ppm) were selected for genotoxicity evaluation. Adult male flies raised on medium containing varying concentrations of lindane were individually crossed with Or-K virgins for DL and with M-5 virgins for SLRL studies.

A total of about 1000 eggs per dose were scored for unhatchability (Table 1). Statistical analysis of per cent lethality by analysis of variance (ANOVA) in comparison with control values, both normal and solvent, showed non-significant effects at all the four concentrations tested.

For SLRL tests, the Muller-5 test protocol was adopted. On an average, about 600 X-chromosomes per dose were scored for sex-linked recessive-lethals. The data (Table 2) were analysed using ANOVA and the Chi-square contingency tests. The induced frequencies were non-significant from those of control values. However, for the former test, the control showed higher SLRL frequencies. Probably the aberrant sperms were naturally screened out in the treated samples, and so only the spontaneous frequency of lethals could be scored for.

Earlier studies by Epstein et al. (1972) also showed no effect of lindane on DL frequency in mice. Other reports on the non-mutagenic effect of lindane include: lack of UDS (±MA) in cultured human fibroblasts (Ahmed et al. 1977), chromosomal aberrations (CA) in CH cultured cells (Ishidate & Odashima 1977), CA in lymphocytes of workers formulating lindane (Kiraly et al. 1979), and gene conversion in yeast (Murthy 1979). However, the fact that lindane can alter aflatoxin B_1 mutagenesis in rats and mice by modifying the metabolizing enzymes of the liver (Decloitre & Hamon 1979), the carcinogenic tendencies of this compound and its inhibitory effect on DNA, RNA and protein synthesis, as mentioned above, clearly depict the potent detrimental nature of lindane. Hence, it should be used with utmost caution along with appropriate precautionary measures.

References. Ahmed, F.E., R.W. Hart & N.J. Lewis 1977, Mutation Res. 42:161-174; Das, B. & P.K. Singh 1978, Microbios Lett. 5:103-107; Decloitre, F.& G. Hamon 1979, Mutation Res. 64:130-131; Epstein, S.S., E. Arnold, J. Andrea, W. Bass & Y. Bishop 1972, Toxicol. Appl. Pharmacol. 23:288-325; Ishidate, Jr., M. & S. Odashima 1977, Mutation Res. 48:337-354; Jeanne, M. 1979, Can. J. Bot. 57:1464-1472; Kiraly, J., I. Szentesi, M. Ruzicska & A. Czeine 1979, Arch. Environ. Contam. Toxicol. 8:309-319; Lutz-Ostertag, Y. 1981, Arch. Anat. Microsc. Morphol. Exp. 70:161-188; Murthy, M.S.S. 1979, Mutation Res. 64:1-17; Pasupathy, K., K.G. Chetty & D.S. Pradhan 1981, Abstracts VI Ann. EMSI Conf., Saha Inst. Nucl. Phys., Calcutta, India, Jan. 27-29, 1981, pp42-43; Srivasan, K. & R. Radhakrishnamurthy 1978, Ind. J. Microbiol. 18:113-115; Ward, J.M., R.A. Griesemer & E.K. Weisburger 1979, Toxicol. Appl. Pharmacol. 51:389-397; Zimonyic, D., B. Soldatovic, M.A.N. Haidary & N. Dujin 1981, Mutation Res. 85:285.

Dhingra, G. and N.K. Vijayakumar.
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Toxic and lethal effects of lindane on the life
cycle of **Drosophila melanogaster.**

Lindane is an agriculturally important compound which has been registered for use on 67 crop plants (Thompson 1982). It is the gamma isomer of hexachlorocyclohexane (HCH,BHC), an organochlorine insecticide, and it was tested for toxicity and sterility induction in the Oregon-K strain of **D.melanogaster.** The insecticide was dissolved in acetone and varying concentrations of it were mixed in the standard cornmeal-yeast-agar medium. Treatments to eggs, third instar larvae and adult flies were given by exposing them to medium containing lindane. For the treatment to eggs, nine concentrations of lindane were tested. Eggs laid by untreated flies were transferred to lindane-containing medium and were scored for their hatchability 24 hr later; and for fly emergence, 9-10 days later. Percent egg hatchability and fly emergence was calculated. Similarly, third instar larvae were transferred to medium containing 10 concentrations of lindane. Percent pupa formation and fly emergence was recorded after appropriate incubation times. The data were plotted on a graph and a best eye-fitted line was drawn for each of the above parameters. Solvent (acetone) and normal (untreated) controls were simultaneously conducted for comparison purposes.

For adult flies, 3-4 day old flies (males and females separately) were exposed for 15 hr to medium containing 12 concentrations of lindane. Data on percent mortality for the flies were plotted on a probit-logarithm graph for estimating the LD_{50} values (Gupta 1980).

In order to assess whether sterility was induced by lindane, adult males raised on medium containing the insecticide were individually crossed with a Muller-5 virgin female, and the progeny count for each cross was recorded. Solvent (5.0 ppm acetone) and normal controls were simultaneously conducted and statistical analysis using analysis of variance (ANOVA) carried out to assess any significant deviation.
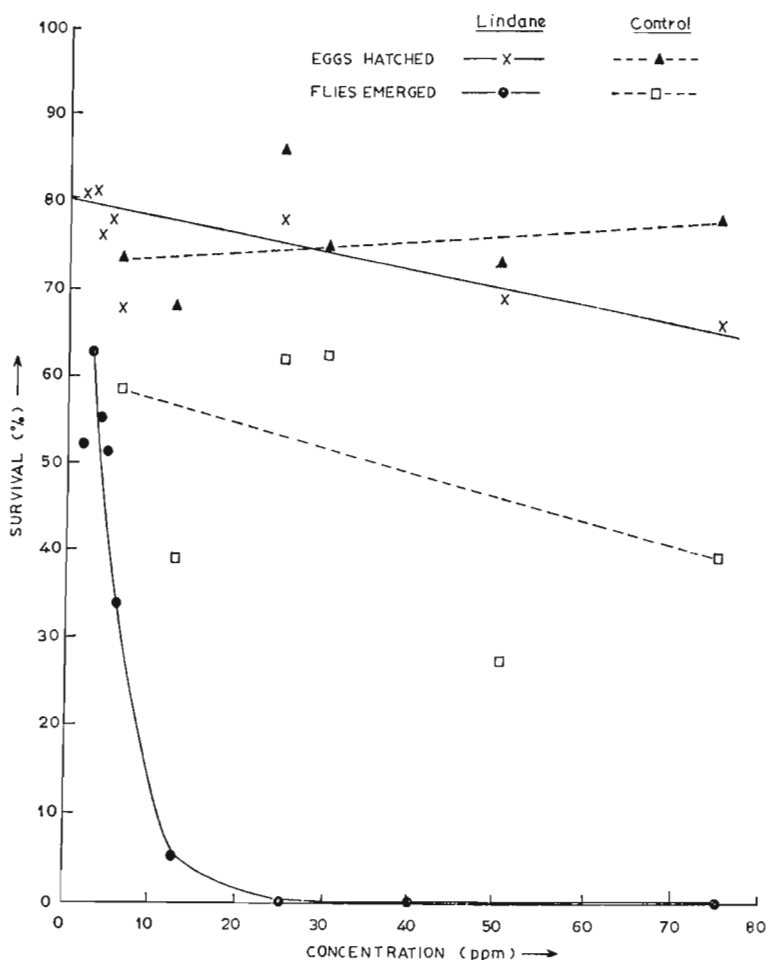
Fig. 1. Effect of lindane on eggs of D. melanogaster.

Table 1. Effect of lindane on eggs of **D.melanogaster.**

Conc. (ppm)	Eggs treated	Eggs hatched	% Hatchability	% Fly emergence
75.0	150	100	66.67	0.00
50.0	150	104	69.33	0.00
25.0	150	117	78.00	0.00
12.5	150	117	78.00	5.33
6.3	150	102	68.00	34.00
5.0	150	117	78.00	51.33
4.0	150	114	76.00	55.33
3.0	150	122	81.33	63.00
2.0	145	117	80.70	52.41
A*	150	113	75.33	62.67
C**	105	86	81.90	75.74

* 30 ppm acetone in the medium
** untreated medium

Table 2. Effect of lindane on the third instar larvae of **D.melanogaster.**

Conc. (ppm)	Larvae treated	% Pupa formation	% Fly emergence
75.0	80	7.50	0.00
50.0	80	23.75	0.00
25.0	180	42.22	1.11
20.0	100	56.00	12.00
15.0	101	85.15	51.49
12.5	80	67.50	36.25
10.0	100	78.00	57.00
6.3	80	88.75	63.75
5.0	101	95.00	69.00
1.0	51	94.12	54.90
A*	303	83.33	54.79
C**	303	83.33	63.70

* 30 ppm acetone in the medium
** untreated medium
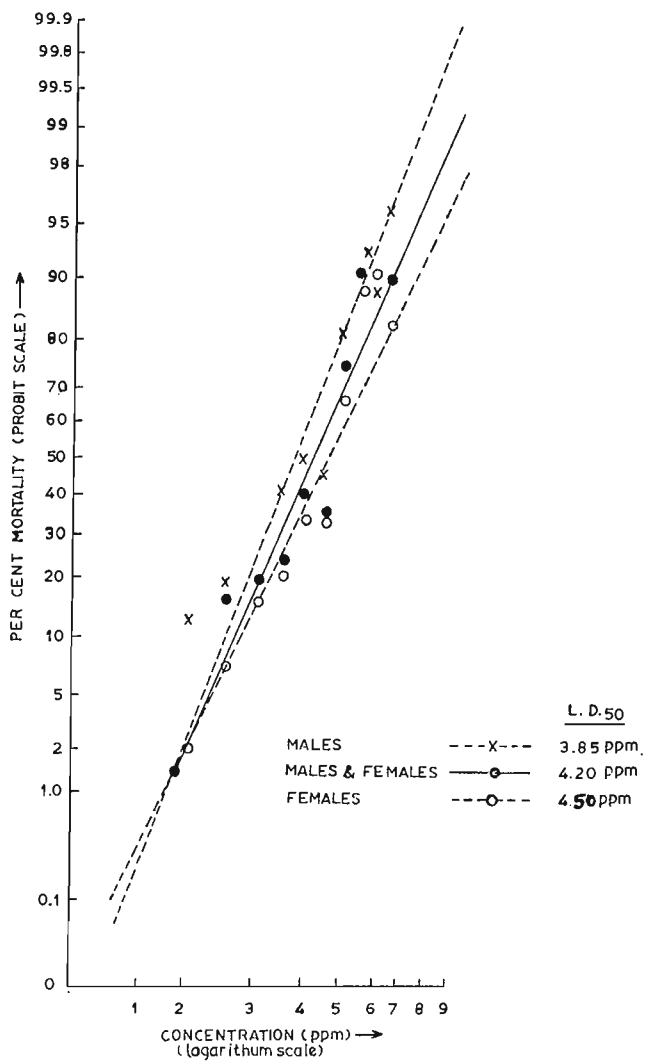
Figure 3. Effect of lindane cn the adult flies of **D.melanogaster** (15 hr treatment).
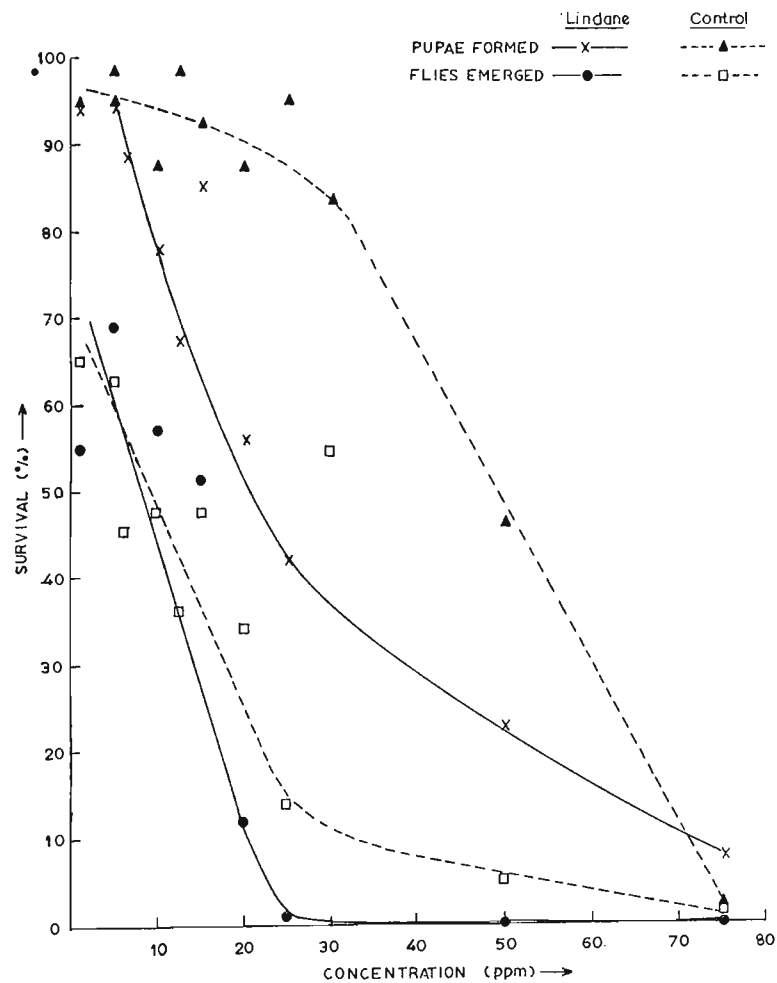
Figure 2. Effect of lindane on third instar larvae of **D.melanogaster**.

Table 3. Effect of lindane on the survival of adult **D.melanogaster** (15 hr treatment).

Conc.	percent mortality		
(ppm)	females	males	combined
6.5	82.00	96.00	89.00
6.0	90.00	88.00	89.00
5.5	88.00	92.86	90.00
5.0	65.52	80.62	73.86
4.5	31.20	45.71	32.40
4.0	31.57	50.11	38.99
3.5	20.00	40.57	23.60
3.0	15.29	25.00	18.94
2.5	7.20	19.89	16.61
2.0	12.00	13.68	12.71
A*	0.00	5.00	3.33

* 6.50 ppm acetone in the medium

Table 4. Effect of lindane on sterility induction in **D.melanogaster** males.

Conc. (ppm)	No. of crosses	Male progeny	Male progeny treated male	Female progeny	Female progeny/ treated male	Total progeny	Progeny/ treated male
5.00	43	617	14	1318	31	2167	50
4.00	61	1024	17	1598	26	3708	64
3.00	86	1401	16	2439	28	3920	52
2.00	83	2071	25	3617	44	6055	80
A*	59	1320	22	2173	37	3870	60
C**	49	936	19	1598	33	2794	58

* 5.00 pm acetone in the medium; ** untreated medium

Table 1 depicts the concentrations from 2.0-75.0 ppm and the data recorded for percent hatchability and fly emergence when eggs were exposed to lindane. At all the concentrations, lindane failed to affect the hatchability of eggs, but concentrations above 6.3 ppm showed a drastic reduction in fly emergence as compared to controls (Fig. 1).

In Table 2 and Fig. 2, the result of treatments to third instar larvae with lindane are shown. Up to 15.0 ppm, percent pupae formation was comparable to control, but from 15.0-25.0 ppm, a reduction in their formation was observed. Similarly, from 1.0-10.0 ppm, the effects were comparable to controls for fly emergence, while with concentrations from 15.0-25.0 ppm, fly emergence decreased, and above 25.0 ppm, no flies emerged at all.

Adult male flies were found to be more susceptible than females (Table 3). When the data were plotted (Fig. 3), the LD_{50} values were 3.85, 4.50 and 4.20 ppm for males, females and for both, respectively. At concentrations from 5.0-75.0 ppm, percent mortality rose from about 73 to 100.

The data on sterility induction is shown in Table 4. At all the four concentrations tested, the progeny per treated male decreased steadily from 80 at 2.0 ppm to 50 at 5.0 ppm; yet statistical analysis showed this to be non-significant from the control values.

The effect of lindane on percent hatchability was found to be negligible, though the effect on the larvae (those emerging from eggs on the treated medium and those directly transferred to treated medium) was more pronounced as pupae formation and fly emergence almost followed a dose-dependent pattern. Smith et al. (1970) had also shown that chlorinated hydrocarbons like lindane and aldrin failed to affect the hatchability of eggs of hens, though the developmental processes were found to be definitely affected in various organisms like weevils (Ivbijaro 1977) and white fly (Sharaf 1978). Repeated spray treatments with lindane to eggs, embryos, chicken and adult quails result in an increase in abnormal genocytes, and some of the cells degenerated during meiotic prophase (Lutz-Ostertag 1981). Singh et al. (1985) also found that the Bihar hairy caterpillar was susceptible to treatments with lindane. Lindane also showed a higher toxicity to males as compared to females in the present study. Such differences in sensitivity based on sex, age and size of an organism have been well documented (Gupta 1985).

Thus, lindane has been found to affect the various developmental states of Drosophila, especially at the higher concentrations. It has been found to be an effective larvicide rather than an ovicide, and so its use against larvae of pests of other members of the order Diptera is suggested. However, lindane has not been found to affect the fertility of the treated flies, thereby demonstrating that either the insecticide failed to penetrate the germ cells, or that it could not cause any metabolic or genetic imbalance by which a sperm-disfunctioning could occur.

References. Gupta, D.S. 1980, Residue analysis of insecticides, H.A.U., Hisar, India; Gupta, P.K. 1985, Modern Toxicology, v.1, Metropolitan Book Co. Pvt. Ltd., N.Delhi, India; Ivbijaro, M.F. 1977, Ind. J. Exp. Biol. 15:1236-1238; Lutz-Ostertag, Y. 1981, Arch. Anat. Microsc. Morphol. Exp. 70:161-188; Sharaf, N.S. 1978, Z. Pflanzenkr. Pflanzen Schutz 85:509-512; Singh, D.S., P. Sircar & S. Dhingra 1985, J. Ent. Res. 9:15-18; Smith, S.I., C.W. Weber & B.L. Reid 1970, Toxicol. Appl. Pharmacol. 16:179-185; Thompson, W.T. 1982, Agricultural Chemicals, Book 1: Insecticides, Thompson, Fresno, Calif. USA.

Di Pasquale Paladino, A., P. Cavolina, G. Romano and R. Ribaudo. Università di Palermo, Italy. Further observations on the tu-pb genome of **Drosophila melanogaster.**

Previous genetic analysis indicated that the tu-pb phenotype of **Drosophila melanogaster** depends from at least two different loci: one, recessive, on the 3rd chromosome, localized in the region between st (62) and e^s (70.7) (Di Pasquale Paladino & Cavolina 1983); the other one, apparently semidominant, on the 2nd chromosome. Attempts to localize the latter were unsuccessful.

Further investigations have been undertaken to elucidate the participation of the 2nd chromosome tu-pb factor/s in tumor manifestation. BIL^2/CyO (or SM5); TM3, Ser, Sb/D males were mated to tu-pb females (tu-pb/tu-pb; tu-pb/tu-pb) and F_1 heterozygous males backcrossed to tu-pb females. Progenies, showing different genotypical combinations of tu-pb 2nd and 3rd chromosomes, were scored for tumor incidence (Table 1). The results obtained emphasize the participation of both chromosomes. As regards the effect of the 2nd tu-pb chromosome, even if only one is sufficient to allow tumor manifestation, in flies heterozygotes the incidence of the trait appears strongly reduced in comparison with homozygotes (which exhibit the same tumor percentage of the tumor strain). On the other hand, about one-half of the homozygous 2nd chromosome combinations seems to elicit lethality, in consideration of the reduced number of progenies found in homozygous genotypes in comparison with the heterozygous ones. Yet, this effect is less evident comparing genetic combinations which include dominant markers on 2nd and 3rd chromosomes.

These findings are in agreement with cytological observations made on 2R salivary chromosomes of the tu-pb strain showing the occurrence of a heterozygous inversion ring in the pairing of homologues which extends from 51 E-F to 56 D-E-F regions (map units

Table 1. Tumor incidence in progenies from crosses a, b, c.

	♀♀		♂♂		
	% tu	N.	% tu	N.	Total
Cross a: ♂♂ tu-pb/CyO; tu-pb/D or TM3 x ♀♀ tu-pb/tu-pb; tu-pb/tu-pb					
tu-pb/CyO; tu-pb/D or TM3	0.0	340	0.0	352	692
tu-pb/CyO; tu-pb/tu-pb	9.7	474	1.7	421	895
tu-pb/tu-pb; tu-pb/D or TM3	0.0	206	0.0	216	422
tu-pb/tu-pb; tu-pb/tu-pb	39.4	226	14.4	125	351
		1246		1114	2360
Cross b: ♂♂ tu-pb/SM5; tu-pb/D or TM3 x ♀♀ tu-pb/tu-pb; tu-pb/tu-pb					
tu-pb/SM5; tu-pb/D or TM3	0.0	352	0.0	424	776
tu-pb/SM5; tu-pb/tu-pb	4.9	469	1.3	369	838
tu-pb/tu-pb; tu-pb/D or TM3	0.0	246	0.0	222	468
tu-pb/tu-pb; tu-pb/tu-pb	36.2	232	7.2	222	454
		1299		1237	2536
Cross c: ♂♂ tu-pb/BIL^2; tu-pb/D or TM3 x ♀♀ tu-pb/tu-pb; tu-pb/tu-pb					
tu-pb/BIL^2; tu-pb/D or TM3	0.0	567	0.0	607	1174
tu-pb/BIL^2; tu-pb/tu-pb	14.9	652	4.8	566	1218
tu-pb/tu-pb; tu-pb/D or TM3	0.0	320	0.0	325	645
tu-pb/tu-pb; tu-pb/tu-pb	38.5	348	10.5	323	671
		1887		1821	3708

Table 2. Progeny from ♀♀ Bc, Elp, px/tu-pb; tu-pb/tu-pb x ♂♂ tu-pb/tu-pb; tu-pb/tu-pb.

Parentals	Recombinants Bc-Elp	Recombinants Elp-px	Total
390	37 (8.5%)	8 (1.8%)	435

Figure 1. Pairing pattern of the 2R chromosome in the tu-pb strain.

83-98 ≃) (Figure 1). Since this pairing pattern is present in all nuclei of all individuals examined (63), it should be maintained through a balanced lethal mechanism, and the observations made on 2R polytene chromosomes of heterozygous tu-pb/Oregon-R larvae confirm this hypothesis (Di Pasquale Paladino et al. 1986).

Some genetic data are also consistent with the presence of a heterozygous inversion extending from 83 to 98 of the 2R chromosome: the reduced frequency of exchange between Bc (80.2) and Elp (99.0), but not between Elp and px (100.5) found in the progeny of the cross ♀♀ Bc, Elp, px/tu-pb; tu-pb/tu-pb; x ♂♂ tu-pb/tu-pb; tu-pb/tu-pb (Table 2) (Di Pasquale Paladino et al. 1986).

In conclusion, two 2nd chromosomes differing for the presence of an inversion and at least for some loci are kept in heterozygous conditions in the tu-pb genome. Only one or both of them may be carriers of tumor factor/s.

References: Di Pasquale Paladino, A. & P. Cavolina 1983, DIS 59:31; _____, _____, G. Romano & R. Ribaudo 1986, Atti A.G.I. v.XXXII:69.

Doane, W.W. Arizona State University, Tempe, Arizona. Location of the map control gene relative to Amy and nw in **D.melanogaster.**

Kikkawa (1964) was the first to use electrophoretic variants of amylase to locate its structural gene (Amy) on the genetic map of chromosome 2R at 2-78.1. Subsequent estimates of the position of Amy, using different genetic markers (cf. Lindsley & Grell 1968; and Lindsley & Zimm 1985), are in general agreement with this: Bahn (1967) and Klarenberg et al. (1986) place Amy at 2-77.8, using curved (c, 2-75.5) and welt (wt, 2-82) as markers, and Doane (1969) estimates its position is at 2-77.3, using the markers c and adipose^{60} (adp^{60}, 2-83.4). Here data are presented that indicate the closest visible marker which is centromere-distal to Amy is narrow (nw). This is based on revised estimates which have located nw at 2-79.0 with respect to c or at 2-79.2 relative to adp^{60} (Doane & Clark 1984), or at 2-79.6 when the markers M(2)S7 and Bc (2-77.5 and 2-80.6, respectively; see Lindsley & Grell 1968) are used. Pooling these estimates and weighting by sample size suggests that nw is at 2-79.3 ± 0.2 cM (Doane & Clark 1984), i.e., approximately 1.5 map units distal to the Amy locus.

The midgut activity pattern (map) gene, a _trans_-acting regulator for tissue-specific amylase expression in the posterior midgut (PMG) of flies, was located by Abraham & Doane (1978) at about 2-80±, and more precisely by Klarenberg et al. (1986) at 2-79.0. The position of map relative to nw has been revealed in an attempt to synthesize a strain of flies carrying Amy^{1,6} map^A and nw^D. My homozygous Amy^{1,6} map^A strain was crossed to a balanced stock containing a second chromosome with multiple dominant markers and the Amy^1 map^C alleles. Progeny of F$_1$ females were screened for double recombinants that expressed appropriate markers to indicate whether map lies to the left (proximal) or right (distal) of nw. The Amy^{1,6} map^A stock carries a duplication at the Amy locus, with Amy^1 the proximal gene copy and Amy^6 the distal copy (Gemmill et al. 1986). These genes code, respectively, for amylase isozyme no. 1 and no. 6, which are expressed throughout most of the PMG in map^A flies reared on standard cornmeal-molasses-yeast-agar medium. By contrast, Amy^1 map^C homozygotes produce only isozyme no. 1 and, on the same diet, display little or no amylase activity in the PMG. A Cy/Pm balancer stock, In(2LR)SM1, al^2 Cy cn^2 sp^2/ In(2LR)bw^{V1}, dp b ds^{33k}, which carries Amy^1 map^C alleles on each second chromosome was used in this experiment. It is not known whether Amy is duplicated in these balancers, but there is evidence for two Amy gene copies in other Amy^1 strains (Gemmill et al. 1986; Boer & Hickey 1986).

The following cross was made: Cy/Pm males were mated to females of the heterozygous genotype S Sp Tft Amy^1 map^C nw^D Pin^{Yt}/S$^+$ Sp$^+$ Tft$^+$ Amy^{1,6} map^A nw$^+$ Pin$^+$. (The S Sp Tft nw^D Pin^{Yt} chromosome containing Amy^1 map^C was provided by T.A. Grigliatti.) Their progeny (total = 958) were selected for expression of narrow dominant (nw^D), a recessive lethal, and the nw^D flies were analyzed electrophoretically for amylase isozymes. Of a total of 463 nw^D flies, only two produced both isozyme no. 1 and no. 6. These flies were heterozygous for a balancer chromosome containing Amy^1 map^C and presumably carried Amy^6 (and probably Amy^1), nw^D, map^A or map^C, and wild type alleles at all other loci. Each double recombinant chromosome was isolated by backcrossing to Cy/Pm and establishing a subline balanced with the Cy chromosome. The recombinant chromosome from each subline was then tested for the presence of Amy^1 and for its map allele by outcrossing to a c Amy^{2,3} map^C stock. Non-Cy progeny were analyzed electrophoretically in polyacrylamide disk gels and classified according to their midgut activity patterns for amylase activity. Both double recombinant chromosomes carried Amy^{1,6} map^A nw^D, i.e., neither carried a map^C allele. The gene order thus appears to be Amy-map-nw, which is consistent with data presented above concerning distances between Amy and nw (Doane & Clark 1984) and between Amy and map (Abraham & Doane 1978; Klarenberg et al. 1980). It also appears that the map and nw loci are tightly linked.

References: Abraham, I. & W.W. Doane 1978, PNAS USA 75:4446; Bahn, E. 1967, Hereditas 58:1; Boer, P.H. & D.A. Hickey 1986, Nucl. Acids Res. 14:8399; Doane, W.W. 1969, J. Exp. Zool. 171:321; _____ & A.G. Clark 1984, DIS 60:234; Gemmill, R.M., P.E. Schwartz & W.W. Doane 1986, Nucl. Acids Res. 14:5337; Kikkawa, H. 1960, Jpn. J. Genet. 39:401; Klarenberg, A.J., A.J.S. Visser, M.F.M. Willemse & W. Scharloo 1986, Genetics 114:1131; Lindsley, D.A. & E.H. Grell 1968, Carn. Inst. Wash. Publ. No. 627; _____ & G. Zimm 1985, DIS 62:1; Rizki, T.M., R.M. Rizki & E.H. Grell 1980, Roux's Arch. Dev. Biol. 188:91.

Doane, W.W. and F.E. Dumapias. Arizona State University, Tempe, Arizona. Deletion limits of the adp, map and nw loci in **D.melanogaster.**

Several lethal deficiencies on the right arm of chromosome 2 which are distal to the amylase (Amy) structural gene locus were tested to determine if they could uncover mutant phenotypes for recessive alleles at the adipose (adp), midgut activity pattern (map) and narrow (nw) gene loci. Flies heterozygous for a given deficiency chromosome and the homologous chromosome bearing one of the following mutant alleles were examined: adp^{fs}, map^C and nw^2. Deficiency heterozygotes carrying the dominant mutant nw^D were also examined. In addition, complementation tests which utilized adp^{fs} (Doane 1960), a recessive maternal effect lethal allele of the adp gene (see Lindsley & Zimm 1985), were performed to determine if any of a number of recessive female sterility factors located in the general vicinity of the adp locus could uncover the adipose fat body or female sterility phenotypes in mature heterozygous adults. The positions of adp, Amy, map and nw on the genetic map of chromosome 2R are summarized in an accompanying research note (Doane 1987).

Table 1 lists the deletions tested, their break points, and the source of each deficiency chromosome. Stocks are balanced over a CyO chromosome which, in the case of Df(2R)PC4, carries the dominant temperature sensitive lethal 1(2)DTS 513. Deficiency heterozygotes of both sexes with the genotype Df(2R)PC4/cn adp^{fs} bw developed the hypertrophied adult fat body characteristic of adp mutants; in addition, females of this genotype were sterile. None of the other deficiencies

Table 1. Deficiencies in chromosome 2R used in the trans configuration to test various mutant alleles in the Amy-adp region for allelsim.

Deficiency	Cytology	Reference	Source
Df(2R)PC4	Df(2R)55A;55F	Nusslein-Volhard et al. 1984	1
Df(2R)P29	Df(2R)55C1,2;56B1,2	Karp 1986	2
Df(2R)P34	Df(2R)55E2-4;56B2-C1 (or 55D2-E1/56B2)	Karp 1986	2
Df(2R)P66	Df(2R)55D2-E1;55E3,4	Karp 1986	2

1 = Trudi Schupbach, Dept. of Biology, Princeton University
2 = Robert Karp, The Biology Laboratories, Harvard University

were able to uncover these phenotypes, which resemble those found in adp^{fs} homozygotes. The breakpoints of the overlapping deficiencies in Table 1 lead to the conclusion that adp lies within the region of the cytological map encompassed by 55A through 55C1,2. None of the Df/nw^2 genotypes produced the characteristic narrow wings of nw^2 homozygotes; all of the Df/nw^D genotypes were viable and expressed a pronounced narrow phenotype. Because nw is approximately 4 cM left of the adp on the genetic map and roughly 1.5 cM right of Amy (see Doane 1987), its cytological location must lie within the limits of 54A and 55A, presumably toward the distal limit. The map locus, which is centromere-proximal to nw and tightly linked to it (Doane 1987), is about 1 cM right of Amy (Abraham & Doane 1978; Klarenberg et al. 1986). It, therefore, must lie within the region between 54A and 55A. In support of this, we found that flies of the genotype Df(2R)PC4/c Amy^{2,3} map^C produce amylase midgut activity patterns of the "A" type, indicating that the deficiency chromosome with the breakpoint nearest map and nw contains a map^A gene.

None of the 12 female sterility mutant stocks which were received from Dr. Trudi Schupbach appear to carry mutant alleles of either the adp or nw genes. Stocks tested include those referred to as DB48, DF6, HD53, HL54, PB28, PF24, PL63, PN5, QD68, QP71, QQ36 and RO64. Female sterility mutants in the DB48, DF6 and HL54 stocks are, respectively, halted (hal), fs(2)lto DF6 and staufen (stau). Those in the HD53 and PN5 stocks are lethal over Df(2R)PC4 which, at its proximal end, spans the region containing the adp gene. Other stocks had not been so fully described at the time they were received.

References: Abraham, I. & W.W. Doane 1978, PNAS USA 75:4446; Doane, W.W. 1960, J. Exp. Zool. 145:1; _____ 1987, DIS (this issue); Karp, R. 1986, pers. comm.; Klarenberg, A.J., A.J.S. Visser, M.F.M. Willemse & W. Scharloo 1986, Genetics 114:1131; Lindsley, D.L. & G. Zimm 1985, DIS 62:1; Nusslein-Volhard, C., E. Wieschaus & H. Kluding 1984, Roux's Arch. Dev. Biol. 193:267; Schupbach, T. 1986, pers. comm.

Duttagupta, A.K. and S. De. University of Calcutta, India. Studies on the heat shock loci in some Drosophila species.

A brief elevated temperature shock of about 37°C induces the synthesis of certain specific new puffs in the polytene chromosome of the larval salivary glands of **Drosophila melanogaster** and also represses the transcriptional activity of all other chromosomal regions (Ritossa 1962; Ashburner 1970). Ashburner & Bonner (1979) reported that the temperature shock response in different species of Drosophila is almost similar. The study of this phenomenon has been extended to various species of Drosophila other than **D.melanogaster** viz. **D.hydei** (Berendes et al. 1965), **D.pseudoobscura** (Pierce & Lucchesi 1980), **D.virilis** (see Ashburner & Bonner 1979), **D.ananassae, D.kikkwai, D.nasuta** (Lakhotia & Singh 1982).

In the present report, we have characterised the heat shock response in 4 species of Drosophila: **D.virilis, D.americana, D.mulleri** and **D.arizonensis** of which the first 2 species belong to the virilis group and the other two species belong to the repleta group of Drosophila. Though the heat shock induced puffing activity in **D.virilis** has been already reported by different workers, here we have presented our data on the heat shock puffing activity in **D.virilis** for comparison with that of its closely related species, **D.americana** and in the hybrid of **D.virilis** and **D.americana**.

The principal objectives of this study are (1) to localize the heat shock puffs in polytene chromosome arms in different species of Drosophila, (2) to find out the evolutionary homology of different arms in these species on the basis of their location, and (3) to draw a relationship between species of the same group.

To induce the heat shock puffs in these species, larvae grown at 20°±1°C were transferred to an Eppendorf tube and immersed at 38° to 40°C water bath for 40 min. Interestingly, we have noticed that in **D.mulleri** and **D.arizonensis**, the heat shock puffs are only induced when they are exposed to 45°C water bath instead of 37°C, the temperature at which heat shock puffs are induced in the majority of the Drosophila species.

Figure 1 a-d. Photographs showing the ^{3}H-uridine grain incorporation in different heat shock loci of (a) **D.virilis**, (b) **D.americana**, (c) hybrid of **D.americana** and **D.virilis**, and (d) **D.arizonensis**.

Table 1. Location of puff sites induced by heat shock in different species.

Species	IInd Chromosome	IIIrd Chromosome	IVth Chromosome
D.virilis	1BC, 2B & 25B	10C & 21BC	X
D.americana	1C, 2C & 26A	11A & 21C	X
D.mulleri	A1,C5 & E2	X	C1 & F4
D.arizonensis	A1,C5 & E2	X	D2, E2 & F1

Note: the position of similar heat shock puffs in **D.virilis** and **D.americana** are different because we have arbitrarily subdivided the chromosomal arms in these two species.

Salivary glands were dissected out from heat treated larvae of which some were used for preparing morphological slides using Aceto-Orcein stain and some for ^{3}H-uridine autoradiography.

Chromosomal preparations from the heat treated nuclei of the species reveal that:

(1)   In **D.virilis** and **D.americana:** Five major heat shock puffs have been induced on the basis of their puff size of which three are located in the IInd chromosome (element-E) and two in the IIIrd chromosome (element-D) (see Table 1). The location of different heat shock loci have been identified on the basis of polytene chromsome map (manuscript in preparation). Data on the heat shock puffs in the hybrid (**D.americana** ♀ x **D.virilis** ♂) reveals that all the five major heat shock puffs are equally expressed in both the homologs (Fig. 1C) suggesting that **D.virilis** heat shock loci remain unaltered in **D.americana** cytoplasm with regard to their puffing activity and transcriptional activity.

(2)   In **D.mulleri** and **D.arizonensis:** Five to six major heat shock puffs have been identified in both the species. The location of different heat shock loci have been identified on the basis of polytene chromosome maps prepared by Wasserman (1954). The position of these puffs have been presented in Table 1.

^{3}H-uridine autoradiography was performed to see the transcriptional activity of these heat induced puffs as well as other chromosomal sites in the heat treated nuclei. Data show that ^{3}H-uridine incorporation is specifically restricted to the temperature shocked puffs (which we have identified from the morphological slides), and the rest of the chromosomal regions show very little or no labelling at all in comparison to the control nuclei.

So the results on transcriptional activity of these heat shock puffs reaffirm their location in polytene chromosome (Fig. 1: a,b,c,d).

Thus, the specific induction of very similar number and pattern of heat shock puffs in the IInd and IIIrd chromosomal arms in both **D.virilis** and **D.americana** and in IInd and IVth chromosomal arms in **D.mulleri** and **D.arizonensis** confirms their close phylogenetic relationship. The induction of heat shock puffs in two chromosomal arms of **D.virilis** and **D.americana** which are homologous to the 3L and 3R arms of **D.melanogaster** indicate their evolutionary homology; i.e., these loci are conserved during evolution. But interestingly, three of major heat shock loci in **D.mulleri** and **D.arizonensis** (both belonging to the repleta group) are located in the IVth chromosome which is homologous to the left arm of IInd chromosome of **D.melanogaster**. This may be due to the fact that a reciprocal translocation between Vth and IVth chromosome has taken place in these species during evolution.

References. Ashburner, M. 1970, Chromosoma 31:356; ——— & J.J. Bonner 1979, Cell 17:241; Berendes et al. 1965, Chromosoma 16:35; Lakhotia, S.C. & A.K. Singh 1982, Chromosoma 86:265; Pierce, D.A. & J.C. Lucchesi 1980, Chromosoma 76:245; Ritossa, F. 1962, Experentia 18:571; Wasserman, M. 1954, Univ. Texas Publ. 5422:130.

Fogleman, J. and J. Williams. University of Denver, Colorado. Oviposition site preference of cactophilic Drosophila in the **eremophila** complex.

The recent discovery and description of a new species in the **eremophila** complex, **D.micromettleri** (Heed 1986) has led to several investigations designed to elucidate the phylogenetic and ecological relationships between this species and the other two species in the complex, **D.mettleri** and **D.eremophila**. Based on Heed's (1986) work on chromosome morphology and interspecific hybridization tests, **D.mettleri** and **D.micromettleri** are more similar to each other than either is to **D.eremophila**, and, in certain respects, **eremophila** appears to be the more derived species. Geographic distributions of the three species are non-overlapping and are as follows: **D.mettleri** - the Sonoran Desert (with disjunct populations in southern California and on Santa Catalina Island); **D.eremophila** - central and southeastern Mexico; and **D.micromettleri** - islands of Jamaica and Hispanola. Although polyphagic, a primary host plant of **D.mettleri** is the saguaro cactus (**Carnegiea gigantea**). **D.eremophila** has been found in association with prickly pear cactus (**Opuntia sps.**) and **D.micromettleri** is found in areas where the columnar cactus, **Stenocereus hystrix**, is abundant.

Table 1. Oviposition site preference (% eggs laid in each substrate of cactophilic Drosophila.

Drosophila species	Cactus species	Cactus		Soaked soil		Total eggs	Chi square	
		Light	Dark	Light	Dark		L vs D	C vs S
D.mettleri	C.gigantea	0.0	5.1	69.7	25.1	195	30.4	157.1
D.eremophila	C.gigantea	6.1	60.7	9.2	24.0	196	94.4	22.2
D.eremophila	O.ficus-indica	7.9	51.9	20.0	20.2	1876	367.2	72.2
D.micromettleri	C.gigantea	32.4	13.4	9.3	21.6	2823	21.3	107.6
D.micromettleri	S.hystrix	0.0	0.0	98.0	2.0	1142	1051.9	***
D.mojavensis	C.gigantea	24.9	75.1	0.0	0.0	3036	765.0	***
data from Fogleman et al. (1981):								
D.nigrospiracula	C.gigantea	94.1	5.9	0.0	0.0	12155	9470.3	***
D.mettleri	C.gigantea	1.1	1.8	73.3	23.7	7026	1682.3	6218.6

*** = oviposition in one substrate type only.

D.mettleri exhibits the novel behavioral characteristic of ovipositing in soil which has been soaked by cactus rot exudate (Heed 1977). Speculation that **D.eremophila** and **D.micromettleri** are also soil-breeders is based on the following information: (1) there are no records of either species having been reared from any cactus tissue (Heed 1986); (2) the larval mouth hooks of all three species are similar and distinctive; and (3) like **D.mettleri** (but to a lesser extent), **D.eremophila** can use soaked soil as a larval substrate in laboratory experiments (Fogleman 1984). Previous oviposition site preference (OSP) tests support the hypothesis that non-soil breeding Drosophila will not oviposit in soaked soil if suitable cactus substrate is available (Fogleman et al. 1981). This report extends the OSP tests to **D.eremophila** and **D.micromettleri** in an effort to establish whether these species, like **D.mettleri** are soil-breeders.

OSP tests were performed in a 50-liter rectangular aquarium which was placed on its side and equipped with a cardboard cover. Light and dark areas were produced in the aquarium by covering half of it with black construction paper. Lighting was provided by a 25 watt fluorescent lamp positioned 30 cm above the device. Plastic petri dishes (100 x 15 mm), containing either 25 g of necrotic cactus tissue or 40 g of soil soaked with the rot juices obtained from the same tissue, were placed in the corners of the aquarium such that one soil plate and one cactus plate were in each area. The weights used represent approximately equal volumes of the two substrates. Additional rot juice was periodically added to the soil plates in order to sustain the percent moisture content between 15 and 25% by weight. Each OSP test employed between 150 and 200 adults of a single species and ran for 24 to 72 hr. Ambient conditions were 20°C and 50% relative humidity. Strains of **D.mettleri** and **D.micromettleri** were obtained from Bill Heed's lab (Univ. of Arizona). **D.eremophila** was obtained from the Univ. of Texas Species Resource Center. At the end of each test, the 4 dishes were removed and the number of eggs in each dish was counted and recorded.

The results of the OSP tests are presented in Table 1. In these tests, **D.mettleri** females laid 94.8% of their eggs in soaked soil. Previously published data on the OSP of **D.mettleri** are included in the table for comparison (97% oviposition in soil) and demonstrate the consistency of the behavioral preference of this species. When saguaro is used as the substrate source, the oviposition preferences for soil of **D.eremophila** and **D.micromettleri** are 33.2% and 30.9%, respectively. Saguaro, however, is not a typical host plant for either species. The host plants utilized by **D.eremophila** in nature are unknown. Use of mission cactus (**Opuntia ficus-indica**) as a substrate source for **D.eremophila** increased the percent eggs laid in soil to 40.2%, but this increase is not statistically significant. Use of the presumed host plant, **S.hystrix**, in OSP tests for **D.micromettleri** results in 100% oviposition in soil. All Chi Square statistics based on the hypothesis of no preference are significant at the 0.001 level. Significant interspecific differences are shown in preferences for ovipositing in lighted versus darkened areas, but these differences are not correlated with soil-breeding.

The non-soil breeding species, **D.mojavensis** and **D.nigrospiracula**, did not lay any eggs in saguaro soaked soil. Necrotic saguaro tissue is a primary substrate of **D.nigrospiracular** in nature and is also used to some extent by **D.mojavensis** (Heed & Mangan 1986). Laboratory viability tests have demonstrated that larvae of these species are not viable in soaked soil substrates (Fogleman 1984).

Within this limited number of species, lab OSP tests can distinguish between soil-breeding Drosophila (up to 100% oviposition in soil) and species which are not soil-breeders in nature (0% oviposition in soil). The contention that both **D.eremophila** and **D.micromettleri** are soil-breeders is supported by our results. The degree to which a soil-breeding species prefers to oviposit on soil (versus cactus tissue) is dependent on the cactus species which serves as the source of the soil substrate.

References: Fogleman, J. 1984, DIS 60:105-107; ———, K.R. Hackbarth & W.B. Heed 1981, Am. Nat. 118:541-548; Heed, W.B. 1977, Proc. Ent. Soc. Wash. 79:649-654; ——— 1986, in: Genetics, Speciation and the Founder Principle (Giddings, Kaneshiro & Anderson, eds.), Oxford Univ. Press (in press); ——— & R.L. Mangan 1986, in: The Genetics and Biology of Drosophila (Ashburner, Carson & Thompson, Jr., eds.), Academic Press, London, 3e:311-345.

<u>**Froelich, A.* and R. Noethiger.**</u> University of
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alleles of sex-determining genes in **Drosophila
melanogaster.**

Previous investigations (Lewis 1949) revealed that
certain spontaneous mutations of **Drosophila melano-
gaster** are suppressed by su(Hw) (suppressor of Hairy-
wing, 3-54.8). At 10 different loci, the insertional
sequence gypsy was found in 19 alleles suppressible
by su(Hw)^2 (Modolell et al. 1983). The only suppres-

sible allele at the white locus, white apricot (w^a, 1-1.5), contains a sequence that shows homology to the
copia transposable element (Bingham & Judd 1981). These observations reveal a correlation between the
origin of a mutation (disruption of the wild type gene by an insertion) and suppressibility. Although the
mechanism of suppression has not yet been elucidated, its manifestation could serve as a tool to detect
mobile elements in interesting genes that could then be cloned using this spontaneous "transposon tagging".

We are interested in the genes that govern the process of sex determination. We have tested 9
spontaneous alleles of the 5 sex-determining genes Sex-lethal (Sxl), intersex (ix), transformer-2 (tra-2),
transformer (tra), and doublesex (dsx), for suppressibility by 4 different suppressors, namely su(Hw)^2, su(s)^2,
su(f), and su(w^a) (Lindsley & Grell 1968). The nine tested alleles were Sxl^{f1} (in X/X), Sxl^{M1} (in X/Y), ix,
tra-2, tra, tra^{Z4}, tra^{Z5}, dsx, dsx^D (dsx^D was not tested with su(Hw)). For effects of the various mutations
of the sex-determining genes, see Noethiger & Steinmann-Zwicky (1985); for the new alleles tra^{Z4} and
tra^{Z5}, see Butler et al. (1986).

All combinations were raised at 18°, 25° and 29°C, and for each combination, at least 13 flies, but
usually between 50 and 100, having the relevant genotype were analyzed. The result was disappointing:
none showed any sign of suppression.

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References: Bingham & Judd 1981, Cell 25:705; Butler, Pirrotta, Irminger-Finger, Noethiger 1986, EMBO J.
5:3607; Lewis, E.B. 1949, DIS 23:59; Lindsley & Grell 1968, Carnegie Inst. Wash. Publ. No 627; Modolell, Bender,
Meselson 1983, PNAS USA 80:1678; Noethiger & Steinmann-Zwicky 1985, Trends in Genetics 1:209.

<u>**Fuyama, Y.**</u> Tokyo Metropolitan University,
Tokyo, Japan. Haploid embryos of **Drosophila
melanogaster** are female.

The balance theory of sex determination in Droso-
phila predicts that haploid flies should be female.
However, as no haploid flies have ever emerged, evi-
dence for the theory has been obtained from the hap-
loid tissues in haplo-diploid mosaics (e.g., Bridges

1930; Santamaria & Gans 1980). I here present evidence that haploid embryos of **D.melanogaster** are
female.

Haploid embryos were produced using a male sterile mutant, ms(3)K81. The sperm produced by
homozygous ms(3)K81 males are defective in syngamy, but are still capable of activating eggs to initiate
gynogenetic development, which gives rise to haploid embryos having only the maternally transmitted
genome (Fuyama 1984). A considerable fraction of these haploid embryos continue to develop to reach the
final stages of embryogenesis producing larvae with differentiated cuticles, though they never hatch. To
know the sex of these haploid embryos, the ability of the haploids to develop in the egg cytolasm having
sex-specific lethal effects on normal diploid embryos, was examined. Eggs with androcidal effects were
obtained from a strain of **D.melanogaster** (ORNSR) that carries sex-ratio spiroplasmas (SRO) derived from
D.nebulosa, and those with gynocidal effects were obtained from the females homozygous for the
daughterless (da) mutation. The sex-specific killing effects of both the SRO and the da gene are known
to act very early in embryogenesis (Williamson & Poulson 1979; Sandler 1972). Therefore, we may reason
that, if haploids are female as predicted from the balance theory, very few of them will develop until later
stages of embryogenesis in the gynocidal eggs produced by homozygous da mothers, whereas their
development will be unaffected by the presence of the SRO.

To test for the effects of the SRO on the survival of haploid embryos, virgin females of the ORNSR
strain were mated with ms(3)K81 males, and the eggs produced were collected 24 to 48 hr after laying,
mounted in paraffin oil, and microscopically examined for development. As controls, females of a male
supplying stock (OR), which was genetically identical with ORNSR, but was free from the SRO, were used
as mothers. The effects of the da cytoplasm was tested with eggs laid by homozygous da females and, as
controls, with those obtained from da/In(2LR)Cy females. Development of haploid embryos was arrested
at various stages; however, only those embryos whose denticle belts were clearly visible were scored as
viable embryos. The presence of denticle belts suggested that these embryos had developed at least until
Stage 13 defined by Bownes (1975). All experiments were performed at 25°C.

Table 1. Development of haploid embryos in eggs with or without sex-specific lethal effects.

Mother	No. eggs examined	No. eggs developing	% developing ± S.E.*
OR	1144	74	6.47 ± 0.75
ORNSR	1110	68	6.13 ± 0.74
da/In(2LR)Cy	1680	62	3.69 ± 0.47
da/da	1570	0	0.0

* standard error obtained by assuming the Poisson distribution.

As shown in Table 1, the late stage embryos were found to occur in eggs carrying the SRO at almost the same rate as in the control, indicating that the SRO did not affect the development of the haploid embryos. In contrast, none of the eggs obtained from homozygous da females contained the late stage embryos, while an appreciable number of embryos were found in the eggs from heterozygous mothers. Haploid embryos, therefore, seem to be inviable in the da egg cytoplasm. The rate of occurrence of embryos in the eggs produced by da/In(2LR)Cy mothers was significantly lower than those in OR or ORNSR eggs; this was not unexpected considering that Cy is recessive lethal.

The results obtained above indicate that haploid embryos of **D.melanogaster** are genetically female, and also that the SRO and the da cytoplasm manifest their sex-specific lethal effects by assessing the X:autosome ratio, as it has been widely accepted based on several other lines of evidence (Williamson & Poulson 1979; Baker & Belote 1983). Thus, the balance theory of sex-determination seems to be valid for haploid embryos, too.

References: Baker, B.S. & J.M. Belote 1983, Ann. Rev. Genet. 17:345-393; Bownes, M. 1975, J. Embryol. Exp. Morph. 33:789-801; Bridges, C.B. 1930, Science 72:405-406; Fuyama, Y. 1984, Jpn. J. Genet. 59:91-96; Sandler, L. 1972, Genetics 70:264-274; Santamaria, P. & M. Gans 1980, Nature 287:143-144; Williamson, D.L. & D.F. Poulson 1979, in: The Mycoplasmas v3:175-208.

Ganter, P.F. and W.T. Starmer. Syracuse University, Syracuse, New York. A note on the insects collected on a birch flux.

Table 1. The insects collected from a slime flux on a birch. O.=Order, SubO.=suborder, Sec.=section, SuperF.=superfamily, F.=family, SubF.=subfamily.

Taxon	No. species	No. individ. female	male
O. Diptera			
SubO. Nematocera			
F. Anisopodidae			
SubF. Anisopodinae	1		
SubO. Brachycera			
F. Dolichopodidae	1		
SubO. Cyclorrhapha			
Sec. Calyptratae			
F. Muscidae	5		
F. Calliphoridae	4		
Sec. Acalyptratae			
F. Tephritidae	1		
F. Drosophilidae	12		
Chymomyza amoena		3	3
Cinderella lampra			1
Drosophila			
affinis			41
algonquin			17
athabasca			242
affinis group females		159	
duncani		1	
hydei		4	5
immigrans		1	
macrospina macrospina			1
melanica		5	1
melanogaster		7	13
robusta		1	

Sap flowing from a wounded deciduous tree may become infected with bacteria, protozoa, and yeasts. Secondarily, the "slime flux" formed from the fermentation of the sap is food for a variety of animals. In the spring of 1986, a sap flow from a paper birch (**Betula papyrifera**) was initiated by the feeding of a yellow-bellied sapsucker (**Sphyrapicus varius varius**) in Syracuse, N.Y. We were able to observe the flux from the time that it was initiated in early April until sap stopped flowing in the fall. The sapsucker and ruby-throated hummingbirds (**Archilochus colubris**) continued to feed on the flux after fermentation started. Fermentation of the sap produced a large mass of microflora on the tree below the wound. This material was mostly yeast and extended more than a meter down the trunk as a large, foamy white mass. Samples of it were plated onto acidified yeast extract-malt extract agar and only one species of yeast (**Zygosaccharomyces bailii**, 86-700.1) was found,

Table 1(contin.):

Taxon	No. species	No. individ. female male
O. Hymenoptera		
SubO. Apocrita		
SuperF. Scolioidea		
F. Formicidae	2	
SuperF. Vespoidea		
F. Vespidae		
SubF. Vespinae	4	
SubF. Polistinae	1	
SuperF. Sphecoidea		
F. Sphecidae		
SubF. Nyssoninae	1	
Sphecius speciosus		2
SuperF. Apoidea		
F. Apidae		
SubF. Apinae	1	
Apis mellifera		2
SubF. Bombini	1	
O. Coleoptera		
F. Staphylinidae	2	
F. Nitidulidae	1	

although other species may have been present at densities too low to detect. Table 1 lists the insects collected as they fed on the slime flux in June. The collection was dominated by **Drosophila**, in terms of number of species and individuals. The dominance in number of individuals is due to the large number of flies from the affinis group collected (the affinis group was the only group with more than one species represented).

Garcia, L., P. Casares and M.C. Carracedo.
Universidad de Oviedo, Spain. Success in
selecting for pupation height in D.melanogaster.

Selection for increasing larval pupation height in **Drosophila melanogaster** presents no problems, but failures for decreasing pupation height have been noticed in the same experiments (Mensua 1967; Markow 1979). Similar results occurred with **D.simulans** where only success for in-the-upward direction of selection was found (Casares & Carracedo 1986).

There are several possible explanations to these facts. Ringo & Wood (1983), in view of their failure to increase pupation height in **D.simulans**, argued for a long history of natural selection for low pupation sites in populations of this species, with few additive genes for this character. It is also possible that depending on the methodology used, the distribution of phenotypes may or may not permit the application of enough selective pressure to obtain a response over a few generations. On the other hand, Casares & Carracedo (1987) suggested the dependence between duration of larval development and pupation height as the probable cause of their failure to select low pupation sites.
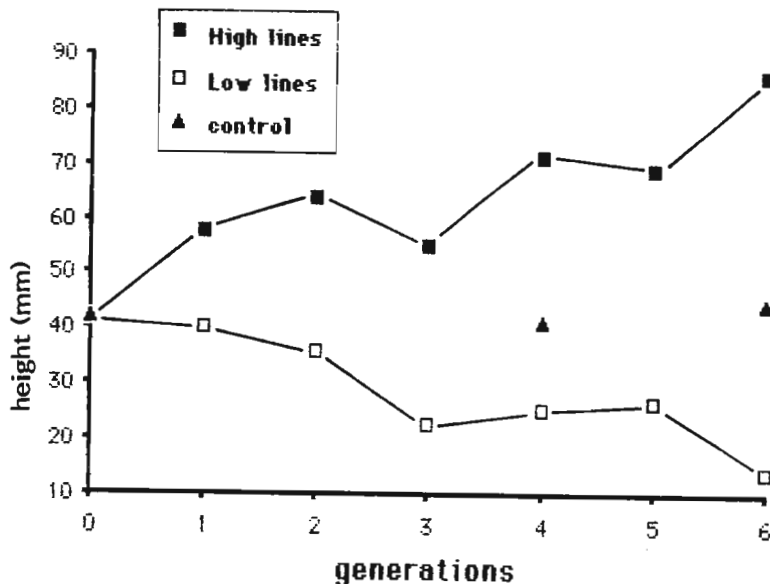
We have started a program of selection for pupation height in **D.melanogaster** with the same methodology as Casares & Carracedo (1986). Seventy-five first instar larvae were seeded in vials (18 x 200 mm) with food, and the localization of pupae was registered at the end of the preadult development. Pupae were classified into 16 height classes. The twelve highest and lowest pupae of each vial were selected and two "high" and "low" lines started with 8 replicates for each of the four lines. Controls were only carried out in some generations.

Figure 1. Response to selection for pupation height over generations.
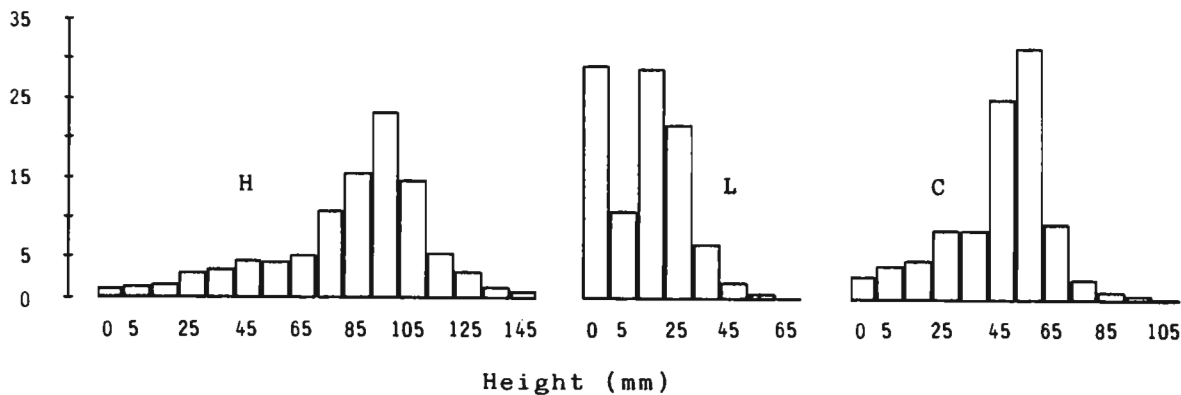
Figure 2. Distribution of pupae in height classes in the sixth generation of selection. C = control. H = high lines. L = low lines.

Figure 1 shows the response to selection over generations. Figure 2 shows the distribution of pupae in height classes for the control (C), high (two lines pooled) and low (pooled) lines in the sixth generation of selection. Response to selection was significant. Realized heritabilities were $h^2 = 0.18 \pm 0.04$ and $h^2 = 0.15 \pm 0.02$ for the high and low lines, respectively, two non-significantly different values. In our **D.melanogaster** population, therefore, additive variability exists for pupation height. This is the first time that successful selection for low pupation sites is shown.

We think that the failures reported by other authors basically lie in the experimental procedures used. Our methodology permits a broad phenotypic distribution for pupation height, which makes the selection of extreme phenotypes easy. The mean pupation height in our base population (x=4.3 mm, Figure 1) also permits the displacement of the entire population towards lower and lower pupation sites. This value is higher than those found by Mensua (1967), Markow (1979) and Casares & Carracedo (1986), all of which failed to select for low pupation sites, and who found means approximately 1.5, 3.5 and 2 cm, respectively, in their base populations.

In this respect, to succeed in selection experiments for decreasing pupation height, we think it is necessary to achieve a very high humidity level inside the vials, which makes the base population pupate some centimeters above the food. Humidity, in turn, will depend on larval density, the tendency of food to liquify as larvae work it, and the type of plug used through which the interchange of humidity between vials and the outside occurs. Finally, the above mentioned effect of duration of larval development in pupation height cannot be ignored, because in the present work we have also found slower larva-pupa development in the low lines as predicted by Casares & Carracedo (1987).

References: Casares, P. & M.C. Carracedo 1986, Experientia 42:1289; _____ & _____ 1987, Behav. Genet. 17 (in press); Markow, T.A. 1979, Behav. Genet. 9:209; Mensua, J.L. 1967, DIS 42:76; Ringo, J.M. & D. Wood 1983, Behav. Genet. 13:17.

Gebhardt, M.D., S.C. Stearns, H. Bachmann and T. Diggelmann. Zoology Institute, Basel, Switzerland. Plasticity of age and size at hatching in **D.mercatorum.**

In preparing a research program aimed at selection for reaction norms of compound life history traits, we performed experiments on isofemale lines of **D.mercatorum** with the following objectives: (1) to screen environmental factors for their effects on age and size at hatching; (2) to see whether interactions between environmental factors would occur; (3) to get a first impression of genetic variation for phenotypic plasticity by comparing the reaction norms of different isofemale stocks. The factors were temperature, relative humidity, food quantity and quality, and egg density.

The stocks derived from single inseminated females caught by Dr. A. Templeton at different sites near Kamuela, Hawaii, in June 1985. For each experiment, virgin females and males were mated in groups of about 30 flies for four or five days with about two males per female. Then the females were allowed to oviposit for six to eight hours in petri dishes (5 cm dia.) containing a standard medium (100 g cornmeal, 60 g sucrose, 15 g dried yeast and 13.8 g agar made up to 1 liter with distilled water and cooked for 1 hr). The egg laying medium was dyed black with active charcoal for better visibility of eggs and supplemented with a drop of living yeast suspension. Eggs were transferred to small tubes (16 mm dia.) containing the experimental medium and kept under constant experimental conditions as specified below until the last adults had emerged. Adults were collected at 12 hr intervals and preserved by deep freezing until
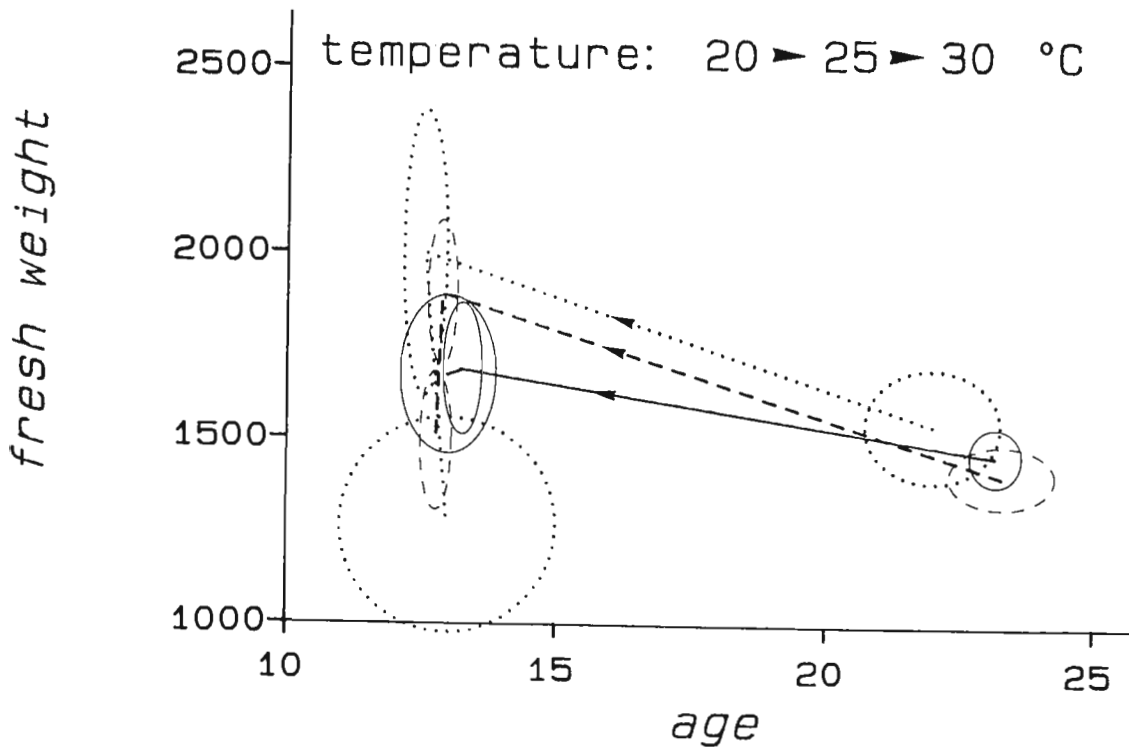
Figure 1. Reaction norms for age and fresh weight at eclosion along a temperature gradient for three egg densities (experiment a). The orientation (arrows) indicates increasing temperature. Ellipses around points represent 95% confidence areas. Age in days, fresh weight in micrograms.

measurements were taken. The difference between the midpoint of the egg laying period and the observation interval in which an adult fly had emerged served as our estimate of developmental time (= age). Fresh or dry weight (3 hr at 70°C) was determined on a Mettler microbalance to 0.01 mg.

　　Experiment a: Three egg densities (5, 10 and 15 eggs/tube) were tested at each of three temperatures (20, 25 and 30°C). For each combination, two replicate tubes were prepared with 2 ml of the standard medium.

　　As qualitative results were not affected by data transformations, we present ANOVAs on the original data in Table 1. Temperature had a strong and consistent effect at all densities on both age and fresh weight, whereas density did not affect either of these variables very much. The simultaneous effects on age and weight become visible by drawing the reaction norms as in Figure 1. Low temperature delayed development by up to 10 days. Raising the temperature above 25°C produced no further acceleration of development. Fresh weight had a maximum at 25°C and was significantly lower at both ends of the temperature spectrum.

　　This contrasts with the generally made observation in **D.melanogaster** that flies are bigger when developing at lower temperatures (Imai 1933), although it is known that body size decreases again when temperature drops below a fairly cool level (about 17°C, David & Clavel 1967). Comparable differences of temperature responses between twelve species of the **D.obscura** group have been found by Pfriem (1983) (although in another temperature range because these are temperate species), indicating that some differences of ecological adaptation between **D.mercatorum** and **D.melanogaster** might be involved.

　　Density had very weak effects compared to temperature, but it can be inferred from Figure 1 that development was slightly delayed and fresh weight was lower at higher densities. Separate analyses within temperature showed that these trends are marginally significant at 20°C and 25°C and not present at 30°C. Mortality was affected by both factors. It was substantially increased at 30°C ($P<0.005$, G-test) and slightly increased at both the lowest and the highest density ($P<0.05$, G-test). The density effect on mortality was enhanced by higher temperatures (Table 2).

　　Experiment b: Each combination of three different food qualities and five relative humidities (40, 50, 60, 70 and 80%) was created. Food qualities were: standard; high yeast, high sugar (45 g dried yeast, 120 g sucrose); and high yeast, no sugar. There were three replicate tubes per combination containing 2 ml of medium and 5 eggs per tube.
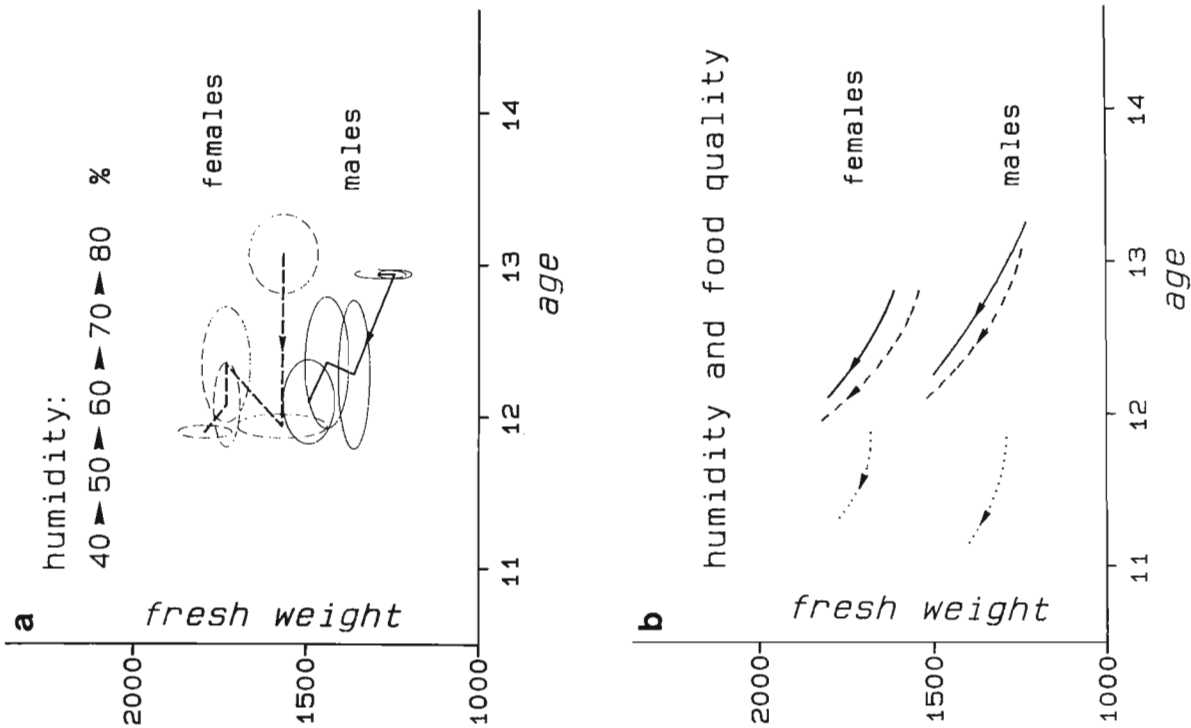
Figure 2. **(a)** Reaction norms for age and fresh weight along a relative humidity gradient for both sexes separately, obtained with standard food (experiment b). **(b)** Idealized reaction norms along the same relative humidity gradient as in (a), for both sexes separately, obtained on three different food media. These are: dotted trajectory = additional yeast, no sugar; broken trajectory = standard medium; solid trajectory = additional yeast, additional sugar. The orientation (arrows) indicates increasing humidity. Other symbols as in Fig. 1.
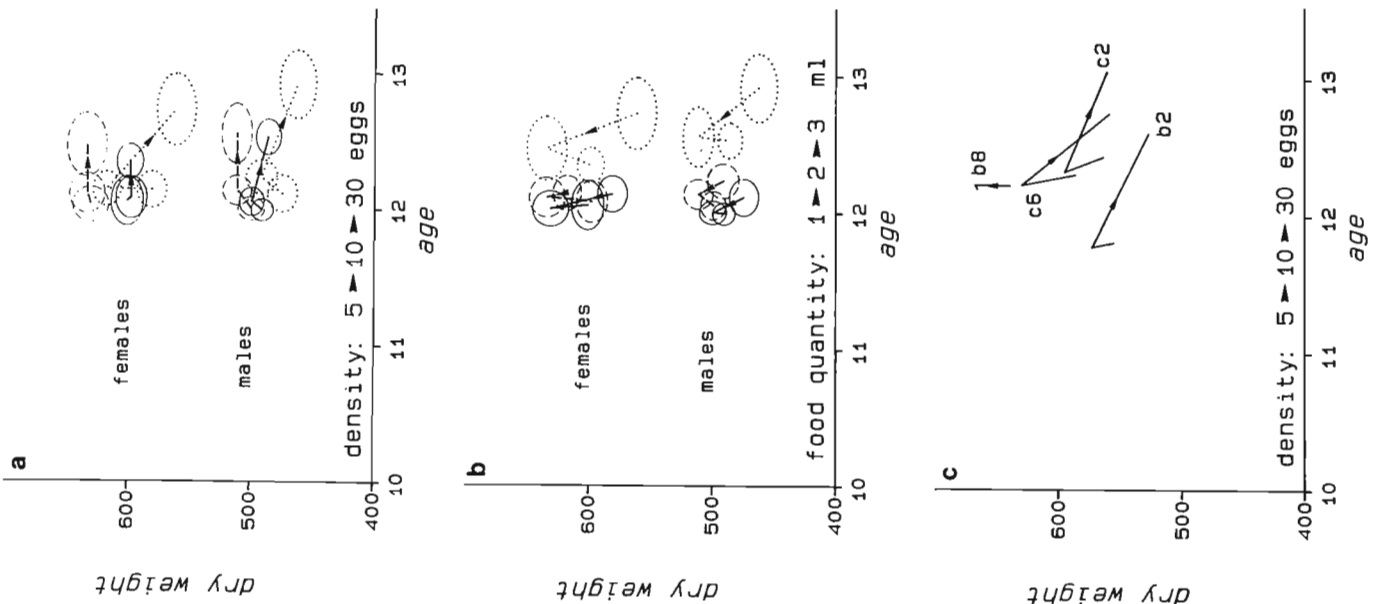
Figure 3. Reaction norms for age and dry weight along a gradient of egg density (a), and food quantity (b), for both sexes separately (experiment c). The data for all crosses have been pooled in (a) and (b). In (a), the dotted, broken, and solid trajectories were obtained with 1, 2, and 3 ml food, respectively. In (b), the dotted, broken, and solid trajectories were obtained with egg densities of 30, 10, and 5, respectively. In (c), the density reaction norms are plotted for females of the four isofemale lines separately, obtained at the lowest food level, without error ellipses for clarity. Symbols are as in Fig. 1.

Table 1. ANOVA tables for the effects of temperature and egg density in experiment (a) on age and fresh weight. The mean squares are based on type III sums of squares.

variable: source	df	mean square	P
age:			
model	16	146.75	<0.001
error	78	1.00	
temperature	2	852.50	<0.001
egg density	2	2.73	0.071
temp * dens	4	1.23	n.s.
replicates	8	2.67	0.012
fresh weight:			
model	16	0.288	<0.001
error	78	0.095	
temperature	2	1.320	<0.001
egg density	2	0.002	n.s.
temp * dens	4	0.172	n.s.
replicates	8	0.084	n.s.

Table 2. Hatchability (in %) at different temperature and density combinations in experiment (a).

egg density	5	10	15	mean
temperature:				
20	60	70	60	63
25	70	90	50	67
30	20	45	20	28
mean:	50	68	43	53

Table 3. ANOVA tables for the effects of humidity and food quality in experiment (b) on age and fresh weight. The mean squares are based on type III sums of squares.

variable: source	df	mean square	P
age:			
model	59	1.004	<0.001
error	101	0.115	
humidity	4	3.338	<0.001
food quality	2	9.434	<0.001
sex	1	0.330	0.094
hum*food*sex	22	0.179	0.033
replicates	30	0.212	0.013
fresh weight:			
model	59	0.090	<0.001
error	101	0.008	
humidity	4	0.111	<0.001
food quality	2	0.005	0.511
sex	1	2.402	<0.001
hum*food*sex	22	0.014	0.025
replicates	30	0.022	<0.001

Table 3 gives the results of ANOVAs performed on the original data, and Figure 2a shows the reaction norms for humidity (for females and males separately) that were obtained on the standard food medium. The reaction norms obtained with the other two food qualities (additional yeast and sugar, additional yeast but no sugar) showed the same pattern but differed in size and position. In Figure 2b these relationships are sketched by idealized trajectories without standard errors.

The relevant points are the following. Humidity: on each food quality and for both sexes desiccation leads to smaller, more slowly developing flies. Food quality: for both sexes, additional yeast did not appear to have an effect, but sugar had a delaying effect. Leaving out sugar shortened development by about one day, increasing it above the standard level had a slight (statistically not significant) delaying effect. Fresh weight was not affected by either factor. Sex: there were no consistent sex differences with regard to developmental rate, but the usual sexual dimorphism in fresh weight was apparent in all environments. An interaction between food and humidity existed; higher sugar levels made the flies more sensitive to the variation in humidity. (Figure 2b).

Experiment c: Four isofemale lines were tested at each of three food quantities (1, 2 and 3 ml/tube) and three egg densities (5, 10 and 30 eggs/tube). To keep the total egg number per combination constant, the number of replicates was adjusted to 12, 6 and 2 for the density series. The standard food medium was used.

In Table 4a the ANOVAs for the untransformed data of age and dry weight are presented. Apart from the sexual dimorphism for dry weight and the density effect on age, it appears that the differences between the lines exceeded the effects of the environmental variation on both age and dry weight. Many of the significant interactions involve lines and indicate differences in the plastic responses of the lines. In Figures 3a and 3b the reaction norms for age and dry weight due to egg density and food quantity have been drawn. To demonstrate the general effects of the environmental factors, the data of all the lines have been pooled and the mean values used. Higher egg densities clearly and consistently resulted in slower development for both sexes and at all food levels. A density effect on dry weight was also consistent but more complicated: the maximum generally did not occur at the lowest density, but at the intermediate level (10 eggs per tube, Figure 3a). Food quantity produced reaction norms similar to egg density, suggesting that they are comparable stress factors (less food corresponding to higher density): lowering the food quantity delayed development, and the intermediate food quantity produced the heaviest flies (Figure 3b). Sex differences existed as usual with regard to dry weight at all densities and food quantities, but not in a consistent way with regard to age. Some differences depended on the line or on the food level (see interaction terms in Table 4a), but are probably biologically unimportant (the greatest mean difference was three hours).

Differences among lines existed with respect to position (main effects in Table 4a) and shape (interaction terms) of the reaction norms. To elucidate these differences, we performed ANOVAs and drew reaction norms for each line separately. All the main environmental effects were in agreement, but there were differences in sensitivity. For example, line b2 was very sensitive to differences in food quantity with respect to both age and dry weight, whereas line c2 showed little response (Table 4b). This and the additional fact that the absolute differences between lines were large explains the significance of the interaction terms in Table 4a.

Table 4. (a) ANOVA tables for the effects of stock differences (line), food quantity and egg density in experiment (c) on age and dry weight. The mean squares are based on type III sums of squares. (b) Significances of effects within lines, calculated in separate ANOVAs.

a: **complete data set**

variable: source	df	mean square	P
age:			
model	41	4.430	<0.001
error	1463	0.431	
line	3	16.293	<0.001
food quantity	2	4.575	<0.001
egg density	2	31.111	<0.001
sex	1	1.051	0.119
line*food	6	2.447	<0.001
line*dens	6	1.391	0.004
line*sex	3	1.191	0.040
dens*sex	2	1.642	0.022
line*food*dens	16	1.218	<0.001
dry weight:			
model	29	0.226	<0.001
error	1474	0.002	
line	3	0.297	<0.001
food quantity	2	0.162	<0.001
egg density	2	0.031	<0.001
sex	1	4.847	<0.001
line*food	6	0.028	<0.001
line*sex	3	0.019	<0.001
food*dens	4	0.019	<0.001
food*sex	2	0.013	0.006
line*food*sex	6	0.006	0.018

Table 4 (contin.):
b: **separate analyses for lines**

line	b2	b8	c2	c6
age:				
food	***	***	n.s.	***
density	***	**	***	***
sex	***	n.s.	n.s.	*
food*sex	***	**	*	n.s.
dry weight:				
food	***	***	***	***
density	*	n.s.	n.s.	***
sex	***	***	***	***
food*sex	*	*	***	**

***=p<0.001; **=p<0.01; *=p<0.05.

Table 5. Hatchabilities (in % flies emerged) for the different lines, food quantity levels and egg densities.

factor	level	hatch- ability
line	b2	72
	b8	53
	c2	70
	c6	82
food	1	63
	2	67
	3	79
density	5	73
	10	74
	30	62
mean		70

As an example, Figure 3c depicts the response that females of the four lines showed to density variation at the lowest food quantity level. The standard error ellipses are left out for clarity, but they are quite large, so that the true shape of the reaction norms cannot be inferred unambiguously. It can be noted, however, that position and extension in the age-weight plane (= sensitivity to the environment) vary considerably and that the genetical differences (position of the reaction norms) tend to exceed the variation due to the environments (extension of the reaction norms).

Hatchability: Higher densities and lower food levels both reduced the probability that a fly hatched (P<0.02 and P<0.001, resp., G-tests), but the absolute differences between the lines were even greater (P<0.001). Apart from an abnormally low number of b2-flies hatching at the intermediate food level, there were no interactions. Table 5 gives the hatchabilities for each level of each factor, pooling the levels of the other factors.

Egg density and food quantity probably act through a similar mechanism (namely the food amount available to the individual larva), rather than by two separate effects. Genuine density effects have been reported in the literature (e.g., Mensua & Moya 1983; Scheiring et al. 1984) but only for densities about ten times as high as in our experiments. This is probably because competition between Drosophila larvae feeding on a limited resource scramble competition mediated by the reduced food availabe to the individual (Bakker 1961, 1969). Accordingly, reduction of food produced a similar qualitative response, although different in magnitude (Figure 3a, b), and the analysis of variance revealed no direct interactions between the two factors (Table 4).

Our results agree in most respects with the observations made by other authors (Lewontin 1955; Furuya & Mori 1961; Barker & Podger 1970). Reduction of the available food increases developmental time and there is an intermediate level where weight and probably also hatchability are at a maximum. Lewontin (1955) ascribed this phenomenon to facilitation among larvae at low densities, although this is not much more than giving a name to the phenomenon. Bakker (1969) argued that it can be explained in terms of competitive interactions of different genotypes in the same vial, but an improvement of the fresh food by a sort of pre-processing (mechanically by the larvae or chemically by bacteria introduced with the eggs) might also be involved.

References: Bakker, K. 1961, Arch. Neerl. Zool. 14:200-281; ——— 1969, Neth. J. Zool. 19:541-595; Barker, J.S.F. & R.N. Podger 1970, Ecology 51:170-189; David, J. & M.-F. Clavel 1967, J. Insect Physiol. 13:717-729; Furuya, Y. & S. Mori 1961, Jap. J. Ecol. 11:104-105; Imai, T. 1933, Wilhelm Roux' Arch. f. Entwicklungsmech. 128:634-660; Lewontin, R.C. 1955, Evolution 9:27-41; Mensua, J.L. & A. Moya 1983, Heredity 51:347-352; Pfriem, P. 1983, DIS 59:101-102; Scheiring, J.F., D.G. Davis, A. Ranasinghe & C.A. Teare 1984, Ann. Ent. Soc. Amer. 77:329-332.

Georgiev, P.G.,* S.E. Korochkina, V.A. Mogila and T.I. Gerasimova. N.I. Vavilov Institute of General Genetics, Moscow; *Inst. of Molecular Biology, Moscow, USSR. Mitomycin C induces transpositions of mobile elements in **Drosophila melanogaster** genome.

The transpositions of mobile genetic elements occur very rarely in usual conditions (Ananiev et al. 1978) comlicating studies on the features of transposition. Therefore, the search for the factors activating the migration of mobile elements in Drosophila genome seems to be important. We analyzed the influence on transposition of mitomycin C which was shown to activate the transpositions in prokaryotes (Kuban-

eishvili et al. 1983). Mitomycin C in concentration of 20 µg/ml was injected the abdomen of males of the $y^2sc^1w^a$ strain of **D.melanogaster**, obtained from M.D. Golubovsky (Inst. of Cytology & Genetics, Novosi-birsk). This strain contains three mutations. Two of them, y^2 and sc^1, are induced by mdg4 (gypsy) insertion into the loci yellow and scute (Modollel et al. 1983). We confirmed this by means of in situ hybridization of ^3H labeled mdg4 DNA with larval polytene chromosomes. The label was detected in 1AB region, where both the scute and yellow genes are located. Besides that the suppression of the mutations by the suppressor $su(Hw)^2$ was demonstrated. The flies with genotype "grey body, normal bristles" but "apricot eyes" were typical of mutation w^a. Thus, $su(Hw)^2$ suppresses y^2 and sc^1 mutations indicating the dependence of their induction by mdg4 (gypsy). The w^a mutation is not induced by copia, which could not be detected by in situ hybridization in the region of the white locus. According to preliminary data, it is induced by mdg element BEL. Finally, the cytotype of the strain $y^2sc^1w^a$ was found to be the M one on the basis of sterility analysis in crosses with P- and M-strains.

Table 1. Major mutation events in the offspring of $y^2sc^1w^a$ males injected with mitomycin C.

Locus	Mutation events	Number of mutations	Number of chromosomes analyzed	Mutation frequency ($\times 10^{-4}$)
yellow	$y^2 \to y^+$	4	58,000	0.7
scute	$sc^1 \to sc^+$	9	58,000	1.6
white	$w^a \to w^{ad}$	5	58,000	0.9
forked	$f^+ \to f^{MC}$	16	48,000	3.3

About 58,000 flies were analyzed in the offspring of injected males belonging to the $y^2sc^1w^a$ strain. We found $y^+sc^1w^a$ and $y^2sc^+w^a$ reversions appearing with the rate of 0.7×10^{-4} and 1.6×10^{-4}, respectively (Table 1). The appearance of these reversions shows the excision of mdg4 from the loci yellow and scute, respectively. Also, novel w-mutations were detected. The flies with dark apricot eyes (w^{ad}) appeared with the rate of 0.9×10^{-4}. They probably represent the partial reversions of w^a mutation. Finally, the mutations in the locus forked (I; 56.7) appeared in the offspring of crosses of mitomycin C injected $y^2sc^1w^a$ males and females C(1)RM,yf with attached X-chromosomes. Sixteen independent f^{MC}-mutations were

detected among 48,000 chromosomes analyzed. A high frequency of mutagenesis (3×10^{-4}) and its specificity strongly suggest dependence of the f^{MC} mutations on insertion of a certain, not yet identified, mobile element. The insertion nature of mutations was confirmed by frequent appearance of f^+ reversions in destabilized homozygous strains carrying f^{MC} mutations (Georgiev et al. 1986). In the control experiment, the males were injected with 0.14M NaCl without mitomycin C. No mutations were found among 30,000 X-chromosomes analyzed.

The next question arises whether the above mentioned mutations appear from single excision or insertion events or as a result of transposition explosions (Gerasimova et al. 1984). To answer the question, we analyzed the distribution of five different mobile elements (mdg1, mdg2, mdg3, mdg4, copia) in X-chromosomes of the original strains and six derivatives ($y^2sc^{+1MC}w^a$, $y^2sc^{+2MC}w^a$, $y^{+1MC}sc^1w^a$, $y^{+2MC}sc^1w^a$, $y^2sc^1w^af^{MC}1$ and $y^2sc^1w^{ad}1$). Five to ten larvae were taken from different generations in each case. No difference in mdg distribution between parented and daughter strains could be detected. Mdg1 was always located in 1B, 4B, 19A, 20A regions; mdg2, in 4D, 12G, 19A, 20D; mdg3, in 4G; mdg4, in 1AB; and copia in 4A, 5A, 7D, 9A, 19E and 20D. Thus, mitomycin C induced only single transposition events such as excision of mdg4 from the loci scute and yellow, partial excision of transposon from the white locus, and insertion of unidentified transposon into the locus forked.

References: Ananiev, E.V., Y.V. Ilyin, V.A. Gvozdev & G.P. Georgiev 1978, Chromosoma 65:359-371; Georgiev, P.G., S.E. Korochkina, V.A. Mogila & T.I. Gerasimova 1986, Dokl. Acad. Nauk SSSR (Russ.) 291:1492-1495; Gerasimova, T.I., L.Y. Mizrokhi & G.P. Georgiev 1984, Nature 309:714-716; Kubaneishvili, M.G., V.S. Smirnov & V.A. Tarasov 1983, Genetika (Russ.) 19:903-912; Modollel, J., W. Bender & M. Meselson 1983, PNAS USA 80:1678-1682.

Ghosh, A.K. and A.S. Mukherjee. University of Calcutta, India. Effect of trisomy for 3L on the expression of 87A and 87C heat shock loci in heat shocked trisomy for 3L nuclei.

Figure 1. Histogram showing the transcriptive activity of 87A and 87C heat shock puffs in Oregon R[+]; C(3L), F(3R); and Trisomy-3L strains of **D.mel-anogaster.** Grain incorporation at 95D heat shock puff has been used as internal control.

The duplicated heat shock loci 87A and 87C are responsible for coding 70K heat shock protein. 87A contains two genes of hsp70 whereas 87C contains three genes of hsp70 (Ashburner & Bonner 1979). It has been reported that 87C also contains $\alpha\beta$ repeated sequences whose transcription is induced by heat shock. Mukherjee & Lakhotia (1979) reported that in the wild type strain of **D.melanogaster** (Oregon-R[+]) 87A and 87C incorporate equal amounts of ^{3}H-uridine in heat shocked nuclei. But under different experimental conditions (Colchine + 37°C temperature shock, see Lakhotia & Mukherjee 1984; or heat shock followed by Benzamide, see Lakhotia & Mukherjee 1980), these two heat shock loci behave differentially. In this report, we present the transcriptional activity of 87A and 87C in a chromosomally imbalanced strain of **D.melanogaster** (trisomy for the whole left arm of 3rd chromosome-T3L).

To generate the trisomy for 3L of **D.melanogaster,** C(3L)VGI ru st, F(3R)VDI e[s] females were crossed with Oregon R[+] males or vice versa. The only surviving class of larvae will be trisomy for 3L. Transcriptive activity has been monitored by ^{3}H-uridine autoradiography (for details, see Lakhotia & Mukherjee 1969). Results revealed that after heat shock treatment 87A and 87C heat shock loci expressed differentially in trisomy-3L(T3L) stock. The 87C locus is transcriptionally 2.8 times more active than the 87A locus (87C/87A=2.8). The mean grain number on 87A in T3L is equal to that of Oregon R[+]. Data also revealed that in both Oregon R[+] and C(3L) VGI ru st, F(3R) VDI e[s] strains 87A and 87C heat shock loci expressed equally (see Figure 1). The total transcriptive activity of 87A and 87C loci in T3L strain is 1.5 times more than that in Oregon R[+]. Devlin et al. (1985) reported that in heat shocked nuclei, T3L condition has no influence on hsp 70 gene at the level of transcription. They have measured the level of hsp70 K transcript using filter hybridization technique by which they have not detected the level of expression of $\alpha\beta$ repeated sequences (at 87C) which are concurrently induced during heat shock treatment. As our present report reveals that 87C is nearly three-fold more active than 87A in trisomy-3L stock instead of an equal level of transcriptional activity (87C/87A=1), both being in diplo dose, presumably, the higher transcriptive activity of 87C found here (at the level of chromosomal RNA synthesis) might be due to an enhanced production of $\alpha\beta$ coded transcript in this region and the trisomy for 3L arm has a positive influence on the transcriptive activity of this region (87C) in heat shock treated T3L nuclei.

References. Ashburner, M. & J.J. Bonner 1979, Cell 17:241; Devlin et al. 1985, Dev. Genet. 6:39; Lakhotia, S.C. & A.S. Mukherjee 1969, Genet. Res. 14:137; Lakhotia, S.C. & T. Mukherjee 1980, Chromosoma 81:125; _____ & _____ 1984, IJEB 22:67; Mukherjee, T. & S.C. Lakhotia 1979, Chromosoma 74:75.
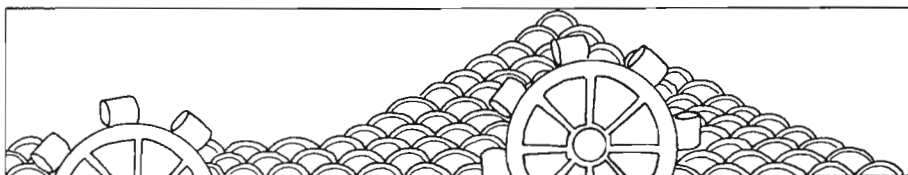
Ghosh, A.K. and A.S. Mukherjee. University of Calcutta, India. Lack of dosage compensation phenomenon in haplo-4 chromosome in **Drosophila melanogaster.**

The transcriptive behaviour of different hyperploid autosomal arms in **Drosophila melanogaster** has been reported (Devlin et al. 1984; Ghosh 1986). However, so far autosomal gene activity has not been measured in whole arm monosomic condition in Drosophila. The primary objective of this report is to present the results of measurement of transcriptional activity of whole 4R chromosome in haplo and diplo conditions.

To generate the monosomy-4R progeny, C(4)RM Tft/CyO females were crossed with Oregon R^+ males or vice-versa. To measure the transcriptive activity of 4-chromosome, we have used ^{3}H-uridine autoradiography.

Results reveal that the transcriptional activity of monosomy-4 is 50% of the activity of disomy-4 chromosome (M/D = 0.51, p<0.001).

Previous studies on autosomal gene activity in trisomic condition claimed the existence of autosomal dosage compensation in **D.melanogaster.** The result of the present report suggests that though the autosomal genes have the ability to equalize their products with that of the diploids in hyperploid condition, they do not undergo dosage compensation by hyper transcriptive activity in monosomic condition, like the haplo-X in male. This result strongly supports the idea, that the type of regulation of autosomal dosage compensation is different from that of haplo-male-X dosage compensation in Drosophila.

References. Devlin et al. 1984, Chromosoma 91:65; Ghosh, A.K. 1986, Ind. J. Exp. Biol. 24:555.

Fig 1a

Fig 1b

Ghosh, M. and A.S. Mukherjee. University of Calcutta, India. Effect of salivary gland extract on the X chromosome of salivary gland nuclei of **Drosophila melanogaster** under in vitro culture condition.

Larval salivary glands from In(1)BM^2 (rv) a modulator mutant stock and the wild type Oregon R^+ stock of **D.melanogaster** were grown in Drosophila culture medium (Schneider's medium minus yeast hydrolysate) with/without salivary gland extract of either stock. The purpose was to examine: (1) whether the extra inflated X chromosomal morphological conformation (Mukherjee & Ghosh 1986) in the male salivary gland of In(1)BM^2 (rv) is retained; (2) whether the three morphotypes of the X as reported by Mukherjee & Ghosh (1986) appear with a given frequency and (3) whether extract from Oregon R^+ larval salivary glands also possess the inducing property. For this purpose, the glands were cultured either for 24 hr or 48 hr at 18°C and then cytological preparations were performed and examined.

Figure 1a and b present the manifestation of the flabby in the mutant male gland without the extract and in the Oregon R^+ male gland exposed to the extract from the mutant. Results

Figures 1a and 1b. Photomicrographs showing (a) the flabby X chromosome from In(1)BM^2 (rv) male salivary gland cultured in in vitro condition without salivary gland extract; (b) induced flabby X chromosome from Oregon R^+ male salivary gland cultured in in vitro and was supplemented with In(1)BM^2 (rv) male salivary gland extract. Cultures were maintained at 18°C.
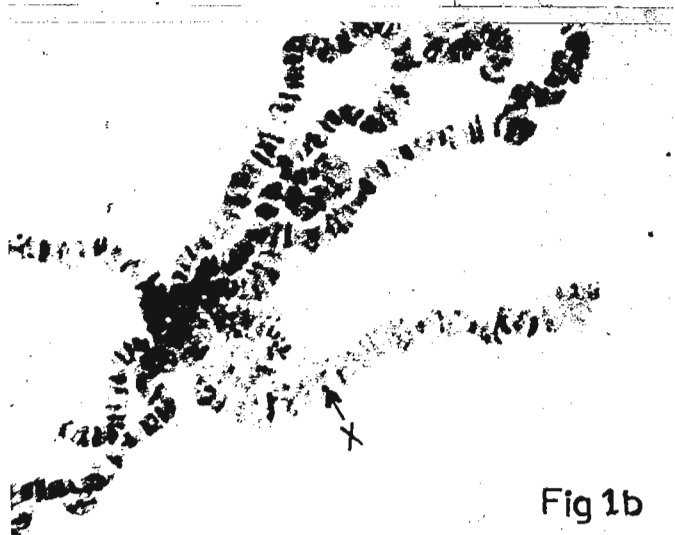
Table 1. Frequency (in %) of superactive flabby X chromosomes of **Drosophila melanogaster** salivary glands after culture in Schneider's medium (minus yeast hydrolysate) supplemented with/without salivary gland extract from Oregon R$^+$ or In(1)BM^2(rv) male.

Salivary gland culture	Salivary glands extract	Culture period	Total no. of chromosomes	No. of X chromosomes (Frequencies in %)		
				Flabby	Intermediate	Normal
In(1)BM^2(rv) male	-	24 hr	1610	620 (38.51)	440 (27.33)	550 (34.16)
In(1)BM^2(rv) male	-	48 hr	1339	530 (39.58)	397 (29.65)	412 (30.79)
In(1)BM^2(rv) male	Oregon R$^+$ male	24 hr	1908	249 (12.89)	630 (33.01)	1032 (54.09)
In(1)BM^2(rv) male	Oregon R$^+$ male	48 hr	1879	248 (13.20)	453 (24.11	1178 (62.69)
Oregon R$^+$ male	In(1)BM^2(rv) male	24 hr	1104	574 (50.82)	388 (36.65)	142 (13.53)
Oregon R$^+$ male	In(1)BM^2(rv) male	48 hr	1244	720 (57.88)	300 (24.12)	224 (18.00)

"-" indicates without salivary gland extract.

presented in Table 1 reveal that extracts from both Oregon R$^+$ male as well as from In(1)BM^2 (rv) male larval glands are able to evoke flabby type of X. However, as expected on the basis of the predicted quanta of the inhibitor/enhancer signals (Mukherjee & Ghosh 1986), the extract from Oregon R$^+$ male larval glands is relatively less efficient to maintain the supra-active flabby condition of the mutant X than the extract from the mutant gland.

References. Mukherjee, A.S. & M. Ghosh 1986, Genet. Res. 45:65-75.

Ghosh, S. and A.S. Mukherjee. University of Calcutta, India. Transcriptional and enzymatic analysis of segmental aneuploids of **Drosophila melanogaster** involving the proximal region of X-chromosome.

Stewart & Merriam (1975) showed that Drosophila females with a duplication for the proximal half of the X chromosome 2.5X's have a normal level of G6PD and an increased level of 6PGD. Males having duplications for different segments of the X-chromosome (that is > 1.0 X) showed control levels of G6PD and 6PGD except where the duplicated region included the structural gene for G6PD or 6PGD. This led them to the conclusion that dosage compensation and other levels of X chromosomal activity (other than normal) constitute a "Chromosomal phenomenon" in that the number of whole X chromosomes must vary before a change in level of transcriptional activity can be varied.

On the other hand, Maroni & Lucchesi (1980) using the technique of autoradiography showed that in Drosophila with duplications involving X chromosome, the overall rate of transcription of the X chromosome (relative to autosomal transcription) remains constant. This constancy was achieved by a uniform reduction of the rate of synthesis of the X chromosomal segments.

Table 1. Transcriptive activity of the X chromosome in relation to autosome in male and female flies aneuploid for different segments of X chromosome. No. in parentheses indicates no. of segments scored.

Strain	1-3C	1-8C	13F-18A	16F-20	8C-20	Entire X
Oregon R$^+$ male	.52(22) ±.02	1.39(20) ±.042	.53(22) ±.03	.62(20) ±.03	1.70(19) ±.06	3.25(18) ±.07
Dp(16F-20) male	.49(19) ±.02	1.34(14) ±.06	.64(12) ±.05	.94(18) ±.06	2.41(13) ±.10	3.63(18) ±.11
Dp(13F-18A) male	.47(22) ±.009	**1.24(25) ±.04	.79(25) ±.04	--	1.86(26) ±.15	3.12(27) ±.16
Dp(8C-20)	***.37(17) ±.02	***1.05(22) ±.03	--	--	1.78(22) ±.07	***2.87(23) ±.08
Oregon R$^+$ female	.55(18) ±.02	1.60(18) ±.06	.63(18) ±.03	--	2.00(18) ±.02	3.52(16) ±.14
Dp(13F-18A) female	***.40(24) ±.02	***1.19(19) ±.05	.84(21) ±.03	--	1.97(18) ±.08	3.29(20) ±.12

Values marked with asterisks are significantly lower than corresponding control values: **P<.01, ***P<.001.

Table 2. Specific activity of 6PGD and G6PD of diff. strains of Drosophila normalized against IDH. No. in paren. indicate no. of observations.

Strain	Gene dose	6PGD/IDH	Gene dose	G6PD/IDH
Oregon R$^+$ male	1	.151±.004 (8)	1	.213±.005 (8)
Dp(18A-20) male	1	.153±.003 (3)	2	.450±.003 (3)
Dp(16F-20) male	1	*.190±.012 (3)	2	.430±.020 (3)
Dp(15EF-20) male	1	.170±.020 ***(3)	2	.390±.020 (3)
Dp(13F-18A) male	1	.176±.003 (3)	1	.216±.003 (3)
Oregon R$^+$ female	2	.170±.014 **(4)	2	.237±.007 (4)
Dp(13F-18A) female	2	.252±.013 (4)	2	.305±.152 (4)

Values with * are significantly higher than corresp. values of control: *P<.05; **P<.01; ***P<.001.

Our transcriptional studies for aneuploids bearing duplications for the regions 16F-20, 13F-18A and 8C-20, of the X chromosome show that at the transcriptional level monitored by autoradiography (Lakhotia & Mukherjee 1969), the activity of the X chromosome (relative to autosome) segments is significantly reduced when the concerned segment is not included within the duplicated region (Table 1).

The activity of two X-coded enzymes, [G6PD (18DE) and 6PGD (2D3)] normalized against an autosomal enzyme - [NADP dependent IDH (66A-D)] was studied for the aneuploids for (18A-20), (16F-20), (15EF-20), and (13F-18A) regions of the X chromosome, according to the method of Lucchesi & Rawls (1973). The normalized 6PGD activity of those aneuploids duplicated for the proximal region of the X chromosome was not decreased when compared to control levels of Oregon R^+ male and female. Rather, a significant increase in the value was noted for Dp(16F-20) male, Dp(13F-18A) male and for Dp(13F-18A) female, even though they had the normal dose of 6PGD gene (Table 2). Thus, dosage compensation might involve the processing of X-coded transcripts at their translational level to vary the apparent number of enzyme molecules.

References. Lakhotia, S.C. & A.S. Mukherjee 1969, Genet. Res. 14:137-150; Lucchesi, J.C. & R.H. Rawls 1973, Biochem. Genet. 91:41-51; Maroni, G. & J.C. Lucchesi 1980, Chromosoma (Berl) 77:253-261; Stewart, B.R. & J.R. Merriam 1975, Genetics 79:635-647.

<u>**Gilbert, D.G.**</u> P.O. Box 302, Bloomington, Indiana. Estimating single gene effects on quantitative traits. 3. Test of the single population method with **D.melanogaster.**

A wild type population is examined for effects of 5 allozymes on 6 quantitative traits using the single population method (Gilbert 1985). Single or multiple locus genetic variation for each trait is demonstrated.

The population of **D.melanogaster** was collected in Hyde Park, Chicago, and maintained at more than 1000 flies until testing 2-7 months later. Flies were collected from timed egg batches, recording development time. Blocks of 10 male-female pairs were mated, for mating speed, copula duration, and progeny numbers. Each fly was scored for wing length, sternopleural bristle number and all allozymes, for a total sample of 220 flies in 11 blocks. The loci measured are Adh (II-50.1), Est-6 (E6, III-36.0), Pgm (III-43.4), Est-C (EC, III-51.7), and Ao (III-56.6), giving also a measure of heterozygosity for each fly. Electrophoretic slow (S) alleles are most common at all loci. Rare alleles are combined with the less common fast (F) allele for this analysis.

A selection of the results is portrayed in Figure 1. This figure shows the average trait z-score for each genotype and sex. Additive locus effects appear as a leftward or rightward trend of the symbols. Significantly different mean scores are determined by non-overlapping error bars.

Traits show influence of loci in the following manner: **Development time.** Est-C alleles show significant additive effects, SS < FS < FF, where SS flies develop fastest. An interesting sex by genotype interaction also appears, the common tendancy of male heterozygotes to develop faster than female heterozygotes, while homozygotes of both sexes tend to be similar (for Est-6, Pgm, Ao and Adh, see Figure 1). **Mate speed.** Est-6 SS males and FF females mate faster than the other genotypes. Est-C FF flies (rare, 4 of 208 flies) mate faster than the other genotypes. **Copula duration.** Adh shows an additive effect, SS > FS > FF. **Progeny.** Ao shows an additive effect, SS > FS > FF. **Wing length.** Pgm shows an additive effect, SS > FS > FF. **Bristle number.** Ao has a homozygote difference (SS > FF). Adh has a heterozygote effect (FS > FF), as does Pgm (SS > FS).

Est-6 is a known reproductive - pheromonal enzyme. Its allozymes have influenced mating speed in previous studies. The precise form of influence varies with temperature and base population characteristics. This is the first examination of Est-6 effects on mating speed within a segregating population, and it corroborates the earlier multiple population studies (Aslund & Rasmuson 1976; Gilbert 1985; Gilbert & Richmond 1982).

Loci affecting sternopleural bristle numbers have been mapped using other methods (Davies 1971). Several of these studies report loci at or very close to Pgm (within mapping resolution). At least one of these studies also report loci mapping in the Ao and Adh regions. Est-6 and Est-C fall outside of the reported bristle regions.

Based on correspondence with these previous studies, the single population method tested here is validated. This method of estimating single gene effects has many positive features that make it a very useful tool. It can be applied to any organism, including man, where mapped, polymorphic loci can be scored. It requires no artificial breeding schemes, and individuals can be sampled and scored directly from their natural population. Each individual can be scored for many traits and many loci at once. Thus, the cost and effort of this method is much less than for other methods.
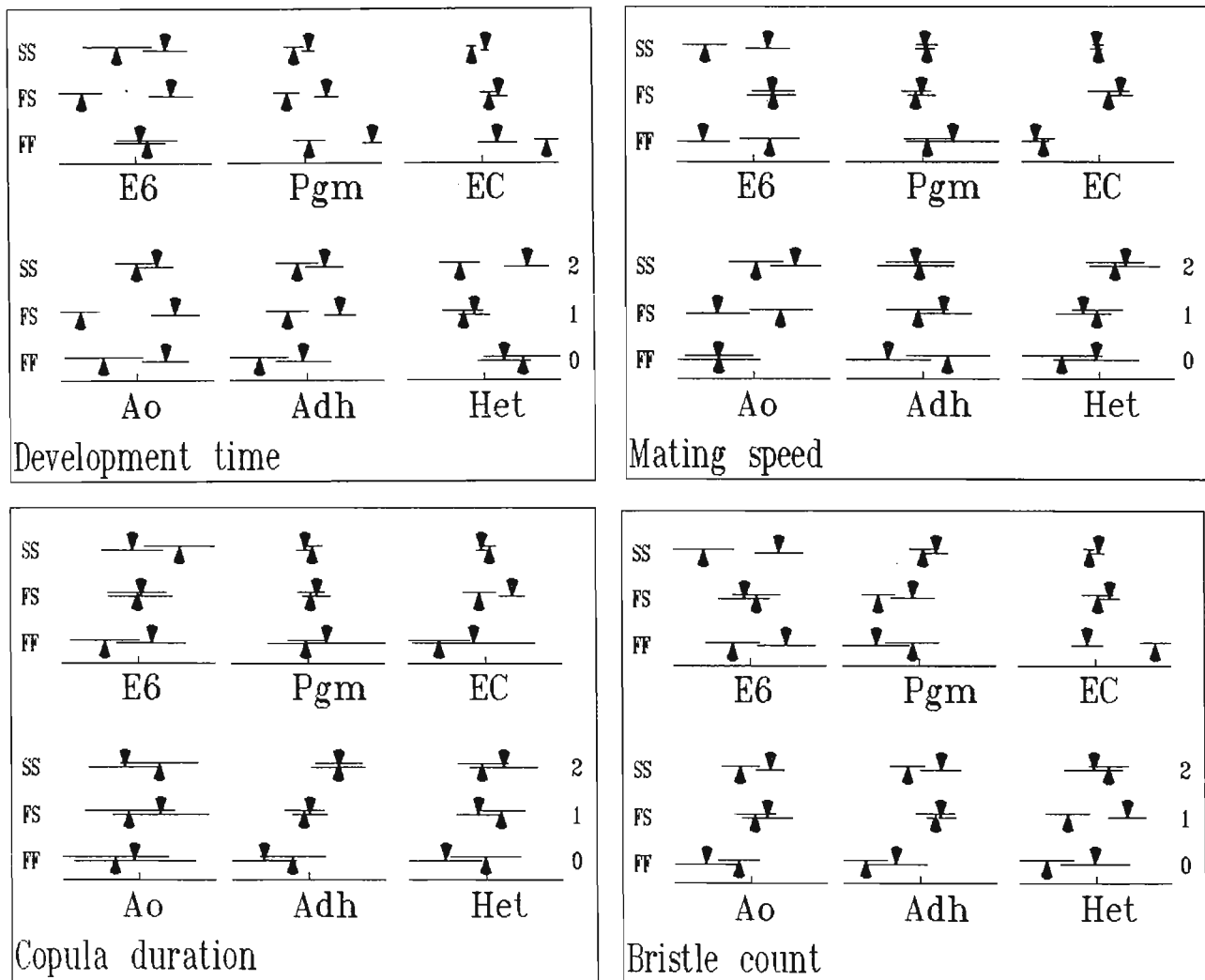
Figure 1. Average z-scores for each genotype and sex, plotted on a horizontal axis, with zero at the center. Female means are depicted with downward pointers, male means with upward pointers. The horizontal bars are the standard error of the mean. Trait means relative to the number of heterozygous loci per fly (0, 1 or 2) are shown in the subplots labeled "Het". The other subplots show scores for the three genotypes FF, FS and SS at each locus. Each locus subplot is scaled independently; the absolute dispersion is not comparable among subplots.

The use of a large, natural population minimizes error due to unknown loci linked to the measured ones. This single population method relies on the generally proven assumption that loci are in linkage equilibrium in the natural population under study. Also the method is currently limited to testing the effects of the scored loci only. A future paper will present statistical treatment and methods for applying this design to mapping quantitative trait loci at any map position.

References: Aslund, S. & M. Rasmuson 1976, Hereditas 62:175-178; Davies, R.W. 1971, Genetics 69:363-375; Gilbert, D.G. 1985, Theor. Appl. Genet. 69:625-629; ——— & R.C. Richmond 1982, PNAS 79:2962-2966.

Godoy-Herrera, R.[1] and J.C. Araneda.[2]
1-Universidad de Chile; 2-Inst. Profesional
de Chillán, Chile. The development of
spicules of cuticle in **Drosophila melanogaster**.

Drosophila melanogaster larvae pass by three instars during their life history. Larval growing occurs by loss of ancient cuticle with formation of a new one (Bodenstein 1950). The cuticle of **D.melanogaster** larvae has a variety of differentiations. One of them are spicules set up on the ventral zone of the larva. These structures are grouped into belts, one for each larval segment (see Figure 2; for further details, see Lohs-Schardin et al. 1979 and Araneda & Godoy-Herrera 1986). Spicules are part of the larval locomotor apparatus aiding these individuals to crawl on and burrowing into substrates where they breed (Agnew 1973; Casares et al. 1986; Godoy-Herrera 1986). New knowledge on the rhythms of growth of the larval denticles could be of interest to understand properly the characteristic behavior of **D.melanogaster** larvae of first, second and third instars (Godoy-Herrera op.cit.). Here we compared the rhythms of growth of spicules of the 3°, 7° and 11° (antero-posterior) belts of the wild type larva of **D.melanogaster** between 24 and 96 hr old.

We used the Oregon R-C strain of **D.melanogaster**. Eggs were incubated at 24°C. After hatching, successive samples of larvae of first (24 hr), second (48 hr), early third (72 hr) and late third (96 hr) instars of development were collected. Larvae of each group were fixed in an extended state by plunging them live into water at 70°C. Once dead, larvae were dried and deposited dorsally on a slide with a drop of xilol and faced with a cover slide. Before the spicular belts were examined under microscope, the preparations were squashed to draw the larvae off their internal organs. This procedure allows for making more conspicuous the larval spicular belts. Figure 1 shows a drawing of some denticles of the 7° belt of 72 hr larvae. It can be seen that spicules are arranged in rows. In Figure 2 is shown the general pattern of distribution of spicular belts of **D.melanogaster** larvae.

Table 1 shows the means of long and wide of spicules of the 3°, 7° and 11° belts of 24, 48, 72 and 96 hr larvae. Denticles of the 3° belt of 24 hr larvae are longer than those of the corresponding belts of 48 and 72 hr larvae but shorter than those of 96 hr larvae. The 7° spicular belt of 24 hr larvae is conformed by denticles longer than those shown by 48 hr larvae, but shorter than the denticles of 72 and 96 hr larvae. In contrast, spicules of the 11° belt of 24 and 48 hr larvae have a similar length which is shorter than that of spicules of 72 and 96 hr larvae.

Table 1 also shows the growing in width of spicules of the 3°, 7° and 11° belts between 24 and 96 hr of larval development. Denticles of the 3° belt of 24 hr larvae are wider than those of 48 hr larvae but smaller than spicules of the corresponding belts of 72 and 96 hr larvae. Denticles of the 7° belt of 24 hr larvae have a width greater than that exhibited by the denticles of 48 and 72 hr larvae, but smaller than that of spicules of larvae of 96 hr of development. Denticles conforming to the 11° belt of 24 hr larvae are wider than those shown by 72 hr larvae but smaller than the spicules of the 11° belts of 48 and 96 hr larvae.

Table 2 shows an index obtained as a ratio between length and width of spicules of the 3°, 7° and 11° belts (Table 1) between 24 and 96 hr of larval development. During the whole larval period, the length of spicules of the 3° belt is greater than the width of these structures. In contrast, in larvae of 24 and 48 hr of age, spicular width of the 7° and 11° belts is greater than the length of these differentiations. This last situation changes drastically after 72 hr of development, because in the larva of third instar the length of spicules of the 7° and 11° belts

Figure 1. Drawing of denticles of the 7° belt of 72 hr larvae. Magnified 1000x.
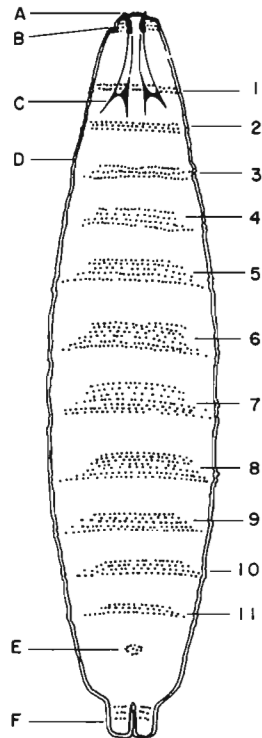
Figure 2. Pattern of distribution of spicular belts of **D.melanogaster** larvae (A) larval hooks; (B) antenno-maxilar complex; (C) cephalopharyngeal skeleton; (D) larval cuticle; (E) group of spicules; (F) spicules of the tracheal zone; 1-11 spicular belts.

Table 1. Means of length and width of spicules of the 3°, 7° and 11° larval belts at 24, 48, 72 and 96 hr of development (the Oregon R-C strain). In each larva tested, length and width of 10 spicules of each one of the belts studied were measured. Ten larvae of each age group (24 to 96 hr) were examined. (*p<0.05; df=198)

larval age (hr)	belt	mean length (um)	mean width (um)	t in respect to spicules of 24 hr larvae: length			width		
				belt: 3	7	11	3	7	11
24	3	3.42±0.06	2.07±0.08	-			-		
	7	3.78±0.07	4.35±0.03		-			-	
	11	2.88±0.02	4.26±0.06			-			-
48	3	2.88±0.06	1.68±0.02	6.136*			4.730*		
	7	3.20±0.06	3.30±0.03		6.444*			24.764*	
	11	2.80±0.04	4.64±0.06			1.789			4.470*
72	3	3.02±0.03	2.50±0.05	5.714*			4.669*		
	7	4.30±0.11	2.63±0.07		3.221*			22.572*	
	11	4.90±0.15	3.44±0.09			13.015*			7.578*
96	3	7.80±0.13	4.16±0.17	30.888*			11.850*		
	7	7.60±0.15	4.86±0.02		26.630*			14.170*	
	11	12.24±0.20	7.00±0.13			14.976*			19.134*

Table 2. Index length/width of spicules of 3°, 7° and 11° belts between 24 and 96 hr of larval development (see Table 1).

larval age (hr)	belt 3	belt 7	belt 11
24	1.65	0.86	0.67
48	1.71	0.97	0.60
72	1.21	1.63	1.42
96	1.87	1.56	1.75

predominates over the width of these structures.

The results of this research suggest that during the larval period of **D.melanogaster** there are changes in size and form of cuticular spicules of these preadults. These changes in size and form are relative to a particular larval belt. These findings seem to suggest that the genetic program controlling the development, for instance, of spicules of the 3° belt is different than those involved in the control of spicules of the 7° and 11° belts. On the other hand, the fact that spicules of a certain larval belt being different in size and form in respect to spicules of the other belts suggests that **D.melanogaster** larvae could use them in different ways during activitys such as locomotion and burrowing.

References. Agnew, J.D. 1973, DIS 50:51; Araneda,J.C. & R. Godoy-Herrera 1986, Rev. Chilena Ent. 14:81-86; Bodenstein, D. 1950, The Postembryonic Development of **Drosophila**, In: Biology of **Drosophila** (M. Demerec, ed.), John Wiley & Sons, NY, pp275-367; Casares, P., E. San Miguel & M.S. Carracedo 1986, DIS 63:40-41; Godoy-Herrera, R. 1986, Heredity 56:33-41; Lohs-Schardin, M., C. Cremer & C. Nusslein-Volhard 1979, Dev. Biol. 73:239-255.

Gómez, F. and C. Nájera. Universidad de Valencia, Spain. Polymorphism of ADH in natural populations of **D.melanogaster-D.simulans.**

Aiming at a study on the possible differences in the ADH allele frequencies in different natural populations, captures of the sibling species **D.melanogaster-D.simulans** were made in two different seasons of the year: Spring and Autumn.

In Spring, the captures were carried out in a cellar in Valencia, in seven more points around it (two in the north, at 500 and 1000 m from the cellar; two in the south, at the same distance; two in the west, at the same distance and one in the east at 500 m from the cellar, because of the proximity of the sea), and also in a pinewood placed in La Cañada (12 km from Valencia).

Autumn captures took place in a cellar in Cheste (a distance of 25 km from Valencia), its surroundings (two captures in each of the four cardinal points at 500 and 1000 m from the cellar) and in a pinewood in La Cañada as well.

The gene frequencies for either of the two species in each population can be observed in Table 1 (Spring) and 2 (Autumn). In both tables, a high frequency of allele F to the detriment of S is observed in every population of **D.melanogaster**, reaching this gap at its maximum point in those cellar environments, where allele S does not reach 5% in the capture of Valencia or 10% in that of Cheste. In the other populations, slight variations can be observed, which could be due to the different microniches settled in each point of capture.

Table 1. Spring frequencies of ADH alleles in natural populations. C=cellar; P=pinewood; E=east; S=south; N=north; W=west; 1=500m; 2=1000m.

	C	P	E	1S	2S	1N	2N	1W	2W
D.melanogaster:									
(males	0.968	0.829	0.801	0.847	0.958	0.817	0.871	0.881	0.882
F(females	0.940	0.843	0.821	0.800	0.795	0.779	0.824	0.847	0.843
(totals	0.953	0.838	0.814	0.821	0.832	0.797	0.845	0.862	0.845
(males	0.032	0.171	0.199	0.153	0.142	0.183	0.129	0.119	0.118
S(females	0.060	0.157	0.179	0.200	0.205	0.221	0.176	0.153	0.157
(totals	0.047	0.162	0.186	0.179	0.168	0.203	0.155	0.138	0.136
D.simulans:									
(males	--	--	0.004	--	0.007	0.009	0.035	0.016	0.012
F(females	--	0.017	--	0.022	0.019	--	--	--	0.007
(totals	--	0.008	0.002	0.011	0.011	0.005	0.014	0.009	0.010
(males	--	1.000	0.996	1.000	0.993	0.991	0.965	0.984	0.988
S(females	--	0.983	1.000	0.978	0.981	1.000	1.000	1.000	0.993
(totals	--	0.992	0.998	0.989	0.989	0.995	0.986	0.991	0.990

Table 2. Autumn frequencies of ADH alleles in natural populations. C=cellar; P=pinewood; E=east; S=south; N=north; W=west; 1=500m; 2=1000m.

	C	P	1E	2E	1S	2S	1N	2N	1W	2W
D.melanogaster:										
(males	0.913	0.852	0.692	0.831	0.900	0.885	0.918	0.844	0.900	0.871
F(females	0.903	0.740	0.795	0.828	0.750	0.872	0.770	0.759	0.748	0.798
(totals	0.909	0.805	0.740	0.829	0.840	0.879	0.818	0.795	0.829	0.833
(males	0.087	0.148	0.308	0.169	0.100	0.115	0.082	0.156	0.100	0.129
S(females	0.097	0.260	0.205	0.172	0.250	0.128	0.230	0.241	0.252	0.202
(totals	0.091	0.195	0.260	0.171	0.160	0.121	0.182	0.205	0.171	0.167
D.simulans:										
(males	--	--	--	--	0.005	0.005	--	0.005	--	--
F(females	--	--	--	0.003	0.007	0.002	0.004	--	--	0.003
(totals	--	--	--	0.002	0.006	0.002	0.002	0.002	--	0.002
(males	--	1.000	1.000	1.000	0.995	0.995	1.000	0.995	1.000	1.000
S(females	--	1.000	1.000	0.997	0.993	0.998	0.996	1.000	1.000	0.997
(totals	--	1.000	1.000	0.998	0.994	0.998	0.998	0.998	1.000	0.998

With regard to populations of **D.simulans**, a monomorphism in favour of allele S in all the Spring and Autumn populations can be practically noticed, without any really significant differences among the various points of capture. The frequency distribution is therefore homogenous.

It has been checked whether these populations are in Hardy-Weinberg equilibrium, finding out that, in the case of **D.melanogaster**, almost all the populations coming from the Spring capture (less activity in the cellar, normally only bottling) are in equilibrium, except 1S (males and total), E (males) and 1W (males). In the pinewood capture there is no equilibrium among the males either. On the contrary, in the Autumn capture (much more activity in the cellar and goods being moved to and fro), this equilibrium only occurs in 2W and 2S populations, whereas in the rest of them females are usually the ones that contribute to unbalance.

Regarding now **D.simulans**, all the Autumn captures are in Hardy-Weinberg equilibrium, whereas on the contrary, in Spring, the distance from equilibrium is well apparent except in those E and 1S populations.

We can deduce from these analyses concerening **D.melanogaster**, that there is a more or less homogeneous distribution of the ADH gene frequencies in the surroundings of a cellar, with the exception of special microniches, so they could be reduced to generic captures "surroundings of a cellar" compared to "inside of cellar". One also gathers that more activity in the cellar causes an alteration on the balances in those zones close to it.

Gómez, F. and C. Nájera. Universidad de Valencia, Spain. Proportion of **D.melanogaster-D.simulans** in natural populations.

Collections of the sibling species **D.melanogaster-D.simulans** have been carried out in two different seasons of the year 1986: Spring and Autumn. Both collections were carried out in three different habitats: (1) Cellar. In Spring the chosen cellar was in Valencia and in Autumn in Cheste (25 km away from Valencia). (2) Surroundings of cellar. This population was divided into 8 subpopulations, corresponding to 8 captures carried out at 500 and 1000 m from the cellar in North, South, East and West directions. In the cellar of Valencia, only one capture towards the East was made, due to the fact that the distance from the cellar to the sea was 500 m in that direction. (3) Pinewood in La Canada at 12 km from Valencia and 25 km from Cheste.

Table 1. Number of collected individuals and percentage of the sibling species **D.melanogaster-D.simulans** in the spring collection. *=p<0.05; **=p<0.01; ***=p<0.001.

| | D.simulans | | | | D.melanogaster | | | |
| | males | females | | % sim- | males | females | | % melano- |
	no. (%)	no. (%)	X²	ulans	no. (%)	no. (%)	X²	gaster
Pinewood	284(51.36)	269(48.64)	0.35	63.27	108(33.64)	213(66.36)	33.69***	36.73
East (500m)	124(44.77)	153(55.23)	2.89	58.19	68(34.17)	131(65.83)	19.32***	41.81
South(500m)	45(50.00)	45(50.00)	0.00	23.44	134(45.58)	160(54.42)	2.12	76.56
South(1000m)	217(62.60)	104(32.40)	39.08***	50.87	183(59.03)	127(40.97)	9.76**	49.13
North(500m)	115(55.56)	92(44.44)	1.39	76.38	30(46.88)	34(53.12)	0.14	23.62
North(1000m)	100(40.49)	147(59.51)	8.57**	55.26	89(44.50)	111(55.50)	2.20	44.74
West (500m)	218(56.04)	171(43.96)	5.44*	68.61	80(44.94)	98(55.06)	1.62	31.39
West (1000m)	241(61.64)	150(38.36)	20.72***	62.46	127(54.04)	108(45.96)	1.38	37.54

Table 2. Number of collected individuals and percentage of the sibling species **D.melanogaster-D.simulans** in the autumn collection. ***p<0.001.

| | D.simulans | | | | D.melanogaster | | | |
| | males | females | | % sim- | males | females | | % melano- |
	no. (%)	no. (%)	X²	ulans	no. (%)	no. (%)	X²	gaster
Pinewood	735(62.82)	435(37.18)	75.08***	82.39	112(44.80)	138(55.20)	2.50	17.61
East (500m)	375(53.50)	326(46.50)	3.28	82.96	78(54.17)	66(45.83)	0.84	17.04
East (1000m)	175(48.75)	184(51.25)	0.18	58.95	110(44.00)	140(56.00)	3.36	41.05
South(500m)	147(35.17)	271(64.83)	36.20***	41.88	277(47.76)	303(52.24)	1.08	58.12
South(1000m)	453(48.09)	489(51.91)	1.30	78.37	126(48.46)	134(51.54)	0.18	21.63
North(500m)	286(65.45)	151(34.55)	41.08***	54.62	174(47.93)	189(52.07)	0.54	54.62
North(1000m)	165(40.94)	238(59.06)	12.54***	62.00	134(54.25)	113(45.75)	1.62	38.00
West (500m)	106(36.68)	183(63.32)	19.98***	39.11	186(41.53)	264(58.67)	13.18***	60.89
West (1000m)	219(38.56)	349(61.44)	29.30***	66.90	94(33.45)	187(66.55)	30.12***	33.10

Males were identified by their genitalia (Sturtevant 1919) while females were identified by the genital differences of their male progeny.

In the collections made inside the cellars, no individuals of **D.simulans**, neither male nor female, were found. The number of collected flies was 350 females (41.27%) and 498 males (58.73%) in the Autumn population and 199 females (51.69%) and 186 males (48.31%) in the Spring one. There is, therefore, a higher number of individuals in Autumn (time of the vintage), although the cellar had a more suitable temperature in Spring.

Regarding the sex ratio in the cellar of Cheste, there was an excess of males (X^2 = 25.48, p < 0.01) while in the cellar of Valencia the sex ratio did not differ significantly from unity (X^2 = 0.37).

The number of males and females collected from each of the two sibling species (contrasted by means of a X^2 test) as well as the percentage for each sex and the total percentage of each species, are reflected in Table 1 for the Spring population and in Table 2 for the Autumn ones. The number of individuals was higher in the Autumn collections for both species (5287 **D.simulans**, 2825 **D.melanogaster**) than in the Spring ones (2475 **D.simulans** and 1801 **D.melanogaster**).

In both seasons, a predominance of **D.simulans** (57.88% in Spring and 65.18% in Autumn) can be observed, although there are some populations (South 500 m in Spring and South 500 m, North 500 m, West 500 m in Autumn) with a higher proportion of **D.melanogaster**, these populations being the closest to the cellar.

As we regard the sex ratio, in the Spring collections for **D.melanogaster** three populations are unbalanced, with advantage for the females in two of them and of the males in the other, while for **D.simulans**, four of the populations are unbalanced, with advantage for the males in three of them and for the females in the other.

In the Autumn collection, for **D.melanogaster**, only two populations are unbalanced with advantage of the females, while for **D.simulans**, six of the nine populations are unbalanced, four in favour of the females and two of the males. It is noticeable that the West populations are unbalanced in favour of the females for both species, being moreover the only ones with a sex ratio different from unity for **D.melanogaster** in this season.

References. Sturtevant, A.H. 1919, Psyche 26:153-155.

González, A. and J.L. Ménsua. Universidad de Valencia, Spain. Evidence for coadaptation in a population of **Drosophila melanogaster.**

Third chromosomes of two **Drosophila melanogaster** populations from the same locality (Requena-Valencia-Spain) were compared in the homozygous and in the heterozygous condition. The habitats of the two populations were relatively distinct: the inside and outside of a wine cellar. The outside location (vineyard) was 4 km from the cellar. All flies were captured at the end of October, one month after the grape harvest.

The component used to measure fitness of the chromosomes was chromosome viability. Their extraction and maintenance were carried out with the TM2 h ca/CSb strain.

The homozygote and heterozygote relative viabilities in the two populations were estimated according to Watanabe et al. (1976) and were analysed by means of two level nested ANOVAs (Table 1). The lethal-carrying chromosomes were excluded from the homozygous analysis.

The genetic component of these distributions' total variance of relative viability (σ_G) was estimated, in each of the four cases, from the difference between mean squares (MS chromosomes-MS error), divided by the mean number of replicates (two in all the cases). Approximate confidence intervals for variance components (Schefeé 1959) were used to compare the genetic components within and between populations (Table 2).

The genetic component of the homozygotes' relative viabilities was higher than that of the heterozygotes in both populations, the differences being highly significant.

The genetic components of relative viabilities of the homozygotes from the cellar and vineyard do not present significant differences. However, a higher genetic component has been observed in the heterozygotes' relative viabilities in the cellar than in the vineyard.

In the grape harvest period there is a massive entrance of flies into the cellar from outside (the flies are carried passiveley or are attracted by the culture medium) and the migrant genotypes can interact with the new population. Therefore, at the moment of these captures, the **Drosophila melanogaster** population from the cellar would be made up of flies with genotypes adapted to this habitat and of offspring of flies from the vineyard (migrated into the cellar) which would either conserve their own genetic constitution or would be genetically the result of crosses with cellar flies. Instead, the vineyard population would only have genotypes from the outside habitat.

On analysing the chromosomes in the homozygous state, both populations present a similar genetic component of the variance of relative viability; that is to say, the chromosomes of the two populations are similarly variable in their genetic constitution with respect to viability.

On analysing the vineyard chromosomes in a heterozygous condition, only one type of combination will be found according to the origin of the chromosomes which make up this population. However, with respect to the cellar chromosomes, two possible types of heterozygous combinations can occur:

(1) Heterozygous combinations, either between chromosomes with genetic architectures from the cellar, or between chromosomes with genetic architectures from the vineyard (from flies which had migrated into the cellar).

(2) Heterozygous combinations between chromosomes from the cellar and vineyard, or between chromosomes with hybrid genetic architectures from both populations.

The possible second type of heterozygous combinations will be responsible for the genetic component of the variance of the heterozygotes' relative viability in the cellar being higher than in the vineyard, since this genetic component is similar in both populations in a homozygous state.

Table 1. Two level nested ANOVAs of the relative viabilities of third chromosomes in cellar and vineyard populations.

source of variation	homozygote viability				heterozygote viability			
	df	SS	MS	Fs	df	SS	MS	Fs
CELLAR								
Cycles[a]	9	2.8204	0.3134	2.26*	9	1.4×10^{-5}	1.5×10^{-6}	ns
Chromosomes[b]	107	14.8047	0.1384	28.83**	145	2.6522	0.0183	1.73*
Error[c]	117	0.5662	0.0048		155	1.6411	0.0106	
Total	233	18.1913			309	4.2933		
VINEYARD								
Cycles[a]	8	1.7114	0.2139	ns	8	7.8×10^{-6}	9.7×10^{-7}	ns
Chromosomes[b]	96	13.7918	0.1437	16.90**	136	2.0556	0.0151	1.33*
Error[c]	105	0.8900	0.0085		145	1.6432	0.0113	
Total	209	16.3922			289	3.6988		

*=p<0.05; **=p<0.01; a=groups of chromosomes analysed simultaneously; b=within cycles; c=replicates within cycles.

Table 2. Genetic components of the variance of the relative viabilities (σ_G^2) and their approx. confidence intervals (95%).

	Cellar σ_G^2	Vineyard σ_G^2
homozygote viability	0.0520<0.0668<0.0890	0.0530<0.0676<0.0890
heterozygote viability	0.0030<0.0038<0.0050	0.0015<0.0019<0.0025

This fact is interpreted as reflecting that in nature gene complexes are coadapted at the between-chromosomes level, within populations. The hybrid chromosomal combinations which are formed in the cellar after the grape harvest, will be responsible for the breakdown of the highly integrated genotypes which evolved within this population, being the results obtained here and reflecting it.

References: Schefeé, H. 1959, The Analysis of Variance, Wiley & Sons, NY; Watanabe, T., O. Yamaguchi & T. Mukai 1976, Genetics 82:63-82.

Hägele, K. and H.A. Ranganath. Inst. für Genetik, Bochum, Ruhr-Universität, Fed. Rep. of Germany. The microchromosomes of five members of the **Drosophila nasuta** subgroup.

The **Drosophila nasuta** subgroup of the **immigrans** species group of Drosophila (Wilson et al. 1969) is divided into three morphophenotypic complexes on the basis of silvery markings on the frons of the males. One of these complexes, the orbital sheen complex, includes five members, namely **D.sulfurigaster sulfurigaster, D.s.albostrigata, D.s.bilimbata, D.s.neonasuta** and **D.pulaua** (Sajjan & Krishnamurthy 1972). The karyotypes of these morphologically nearly indistinguishable and phylogenetically very closely related forms consists of 2n = 8 chromosomes (Sajjan & Krishnamurthy 1974; Ushakumari & Ranganath 1986). Studies of the chromosomal differentiation in the members of the orbital sheen complex showed that the mitotic chromosome complements of **D.s.bilimbata** and **D.s.sulfurigaster** are almost indistinguishable, whereas **D.s.neonasuta, D.s.albostrigata** and **D.pulaua** have notable differences in their karyotypic organization (Ushakumari & Ranganath 1986). Investigations of the polytene chromosomes demonstrated that the long arms of **D.s.sulfurigaster, D.s.bilimbata** and **D.pulaua** are homosequential, while **D.s.neonasuta** and **D.s.albostrigata** carry inversions on chromosome arm 2L (Rajasekarasetty et al. 1980).

The polytene microchromosomes (chromosomes 4) of the Drosophila forms mentioned above have not yet been studied systematically. These chromosomes are small in all five members of the orbital sheen complex and rarely appear well suitable for banding analysis. The salivary gland microchromosomes of the five Drosophila members have in common that their diameter is about twice of the diameter of the other chromosomes in the same nucleus (Figure 1). This particularity is caused by specific arrangements and banding sequences. The analysis of the banding pattern showed that the microchromosomes of all five members carry a duplicated region of five large bands. One of the duplicated region is located close to the distal chromosome end, whereas the second duplication is near the

Figure 1a-g. Salivary gland microchromosomes of (a,b) **D.s.neonasuta** (stock 209.2), (c) **D.s. sulfurigaster** (no. 3019.8), (d) **D.s.albostrigata** (S-11), (e) **D.s.bilimbata** (GUM-8) and (f,g) **D.pulaua** (V-6). In (b,g) the loop structure of the chromosome is visible, in (a, c-f) intimate pairing of the duplications (bracket) and tight synapsis of non-homologous bands hides largely the loop structure. Arrows point to the apex of the loops where the bands bend down.

a
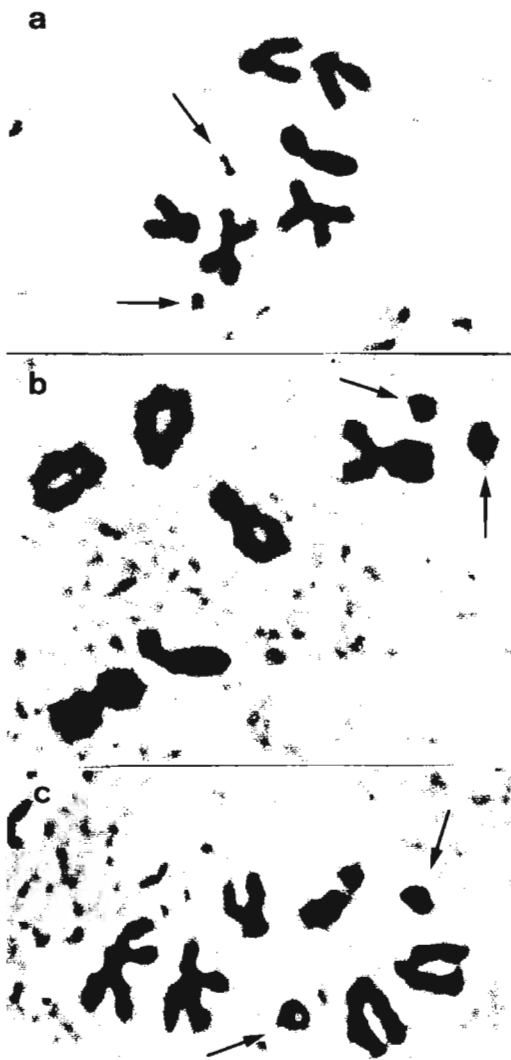
Figure 2a-c. Mitotic metaphase plates of males of (a) **D.s.sulfur-igaster**, (b) **D.s.neonasuta** and (c) **D.pulaua**. Arrows point to the dots. Note in (b) heterozygosity of the dots.

proximal end. The two duplicated regions are oriented to one another in an inverted sense. In salivary gland nuclei these duplicated regions pair. Hereby the chromosome bends down forming a small loop. In cases where the duplicated proximal and distal bands are only loosely attached to one another, the loop configuration can clearly be seen (Figure 1b, g). Mostly, however, pairing of the duplications is very intimate, and also non-homologous bands are tightly lying side by side, and, therefore, the loop configuration is no longer recognizable (Figure 1a, c-f). Thus, this organization is responsible for the larger diameter of the microchromosomes in comparison to the other polytenes.

As far as the bending of the microchromosomes and the diffuse appearance of bands in the duplicated regions allow analysis of the banding pattern, it is obvious that these chromosomes are homosequential in all five Drosophila members and that they have the same number of bands. In contrast to these polytene microchromosomes the sizes of the mitotic microchromosomes (dots) differ between the members (Ushakumari & Ranganath 1986). The smallest dots are present in **D.s.sulfurigaster** (Figure 2a), **D.s.bilimbata** and **D.s.albostrigata**. In **D.s.neonasuta** two types of dots have been recorded (Figure 2b). One type is somewhat larger than the small dots in the above mentioned three members; the other type appears similar to the large dots of **D.pulaua** (Figure 2c). The size difference between the dots may only be due to differences in alpha-heterochromatin. This type of heterochromatin is not replicated in polytene chromosomes (Rudkin 1969) and, therefore, the sizes of the polytene microchromosomes are similar in all five Drosophila members.

Interestingly, in **D.n.albomicana** which is a member of the frontal sheen complex of the **nasuta** subgroup, the polytene microchromosomes (Hagele & Ranganath 1982) show the same chromosome organization as in the five members of the orbital sheen complex. Thus, **D.n.albomicana** and the members of the orbital sheen complex share, in addition to a common inversion in chromosome 2L (Rajasekarasetty et al. 1980), also a common inverted duplication in the microchromosome. This supports the assumption of Rajasekarasetty et al. (1980) and Ranganath & Hagele (1981) that **D.n.albomicana** and the five members of the orbital sheen complex diverged from a common ancestor.

References: Hagele, K. & H.A. Ranganath 1982, Chromosoma 85:215-220; Rajasekarasetty, M.R., S.R. Ramesh & N.B. Krishnamurthy 1980, Entomon 5:1-12; Ranganath, H.A. & K. Hagele 1981, Naturwiss. 68:527; Rudkin, G.T. 1969, Genetics 61:227-238; Sajjan, S.N. & N.B. Krishnamurthy 1972, DIS 49:60; Sajjan, S.N. & N.B. Krishnamurthy 1973-74, J. Mys. Univ. 26:162-167; Ushakumari, A. & H.A. Ranganath 1986, Current Sci. 55:745-747; Wilson, F., M.R. Wheeler, M. Harget & M. Kambysellis 1969, Univ. Texas Publ. 6918:207-253.

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Experimental design and remating in
Drosophila melanogaster.

There are differences between experimental designs in which once-mated **Drosophila melanogaster** females are continuously confined with a second male or are periodically confined (Newport & Gromko 1984). During continuous confinement remating occurs rapidly, there is no relationship between remating rate and first mating productivity, and progeny production by the first male is considerably reduced by remating. In the periodic confinement design, females are less likely to remate, there is a negative correlation between first mating productivity and the probability of remating, and remating does not greatly reduce the number of progeny produced by the first male. In other studies Gromko & Pyle (1978) and Gromko, Newport & Kortier (1984) have explored the relationship between first mating productivity and time to remating of once-mated females periodically confined for two hours with second males. Gromko, Newport & Kortier (1984) have argued that periodic confinement designs are more representative of the natural reproductive biology of **Drosophila melanogaster.**

There is a potential problem with periodic confinement experiments in that once-mated females are actively transferred and the males are aspirated just prior to the remating period. Handling could disturb the flies resulting in a lower probability of remating during the following two hours of observation. This hypothesis is suggested by the results of Manning (1961) who found that flies selected for faster virgin mating speed were less active than control flies. Remating and initial mating are independent behaviors in some respects (Pyle & Gromko 1978). Nevertheless, if the flies are disturbed by transfer between vials and are more active as a result, then they may be less likely to remate.

The goal of this study was to test the effect of handling on remating. The experimental method was to actively transfer flies to vials for remating or to combine males and females with a minimum of disturbance. The stock used was collected at Putah Creek near the Davis campus approximately three years before the experiment and subsequently maintained in mass culture. Virgins were collected with CO_2 anaesthesia. Virgin females and males were held (100 per bottle) for several days with no live yeast on the food medium surface. The first mating was obtained by randomly combining single females and males in 8 dram vials with live yeast on the food surface. Mating was rapid and 94% of the females mated within two hours. The once-mated females and a set of virgin males were held separately (15 per bottle) with live yeast on the medium surface and transferred daily for 4 days before being used in the experiment.

In the studies by Gromko and co-workers the time of day remating was tested is not always stated. A mid-morning period (9 - 11 a.m.) was used in our experiments. In one treatment two females and two males were placed in separate 8 dram vials with 10 ml of food and live yeast on the medium surface. The openings of the vials faced each other, with males and females separated by a cardboard strip. The vials were held together by a rubber band and placed on a table under fluorescent ceiling lights. In the other treatment two males were placed in one vial and two females in the other. Both vials were plugged with cotton and faced each other in pairs. These vials alternated with the vials divided by cardboard. After 24 hr males and females were combined either by removing the cardboard divider as gently as possible or by aspirating males and females from the separated vials back into the same vials, which were now unplugged and joined with a rubber band. Immediately thereafter the flies were observed continously for 2 hr and all matings were recorded. The remating proportion in the treatment where flies were not handled was 0.45 (total number of females = 40). The proportion remating in vials where flies were aspirated was 0.57 (total number of females = 42). The males and females combined by aspiration initially appeared to be more active, yet there was no significant difference (G = 1.20, 1 d.f.) in the frequency of remating.

References: Gromko, M.H., M.E. Newport & M.G. Kortier 1984, Evolution 38:1273-1282; _____ & D.W. Pyle 1978, Evolution 32:588-593; Manning, A. 1961, Anim. Behav. 9:82-92; Newport, M.E. & M.H. Gromko 1984, Evolution 38:1261-1272; Pyle, D.W. & M.W. Gromko 1981, Am. Nat. 117:133-146.
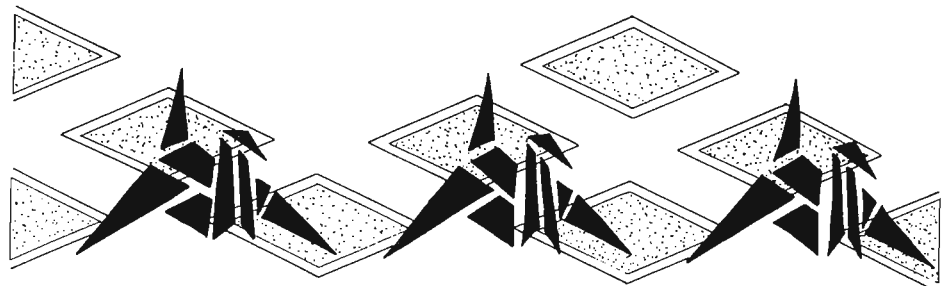
HuKai and H.L. Carson*. Hainan University,
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Republic of China; *-University of Hawaii,
Honolulu, Hawaii. Interspecific copulation
between far distant Drosophila species.

While visiting Carson's laboratory in Hawaii from
February 5 to April 10, 1986, I had some very
interesting field trips collecting picture-winged flies.
I experienced an unusual environment, the rainforest
of Hawaii, in which the beautiful Hawaiian flies are
evolving. After returning with flies from one of
these trips, I found a strange interspecific copulation

between far distant Drosophila species. The male was from a rather familiar species **Drosophila suzukii**,
with its beautiful wing spots; the female was of a kind of Hawaiian fly, **Drosophila tanythrix.**

Drosophila suzukii belongs to the subgenus Sophophora and is a member of the **melanogaster** species
group, specifically belonging to the Asian **suzukii** subgroup. It is a small fly about 2.0 mm long.
D.tanythrix, on the other hand, is a Hawaiian species found only on the "big island" of Hawaii. It belongs
to a distinctive group of 15 species in the Hawaiian islands; it is a large fly, with a body size about 6.0 mm
in length. It has a rufous color, with wings that are subhyaline, faintly tinged with brown. **D.tanythrix**
displays a striking sexual dimorphism; the male has unusual, highly modified antennae and aristae and has
forelegs with long bristles; these characters are used in mating behavior (Spieth 1968). The systematic
position of these species has been a subject of controversy. The elongated first and second antennal joints,
especially elaborate in males, are characters unique in the family Drosophilidae and at one time were used
to place such flies in a separate genus (Antopocerus). This was later reduced to a subgenus (Hardy 1977;
see also the treatment of Wheeler 1981). Kaneshiro (1976), however, feels that this procedure obscures
the fact that other characters relate these flies very closely to the subgenus Drosophila. He has pointed
to other groups of Hawaiian drosophilids that also show bizarre secondary sexual characters in males; he
thus prefers to consider the "Antopocerus" flies only as a species group within the large subgenus
Drosophila.

During the collections made on the island of Hawaii at the Olaa Tract fern forest, Carson
concentrated on collecting picture-winged flies, whereas I collected some others. Before the journey back
to the laboratory, Carson labelled the non-picture-winged flies as "junk" and dumped them all together in
a large sugar-vial and marked them as flies for HuKai to study.

At the lab, I put this vial in the 18-degree incubator; it included some **suzukii** and **tanythrix** males
and females as well as some other species, such as immigrans group, **D.simulans,** and miscellaneous small
and large Hawaiian fungus-eating drosophilids, all crowded together.

We collected the flies February 11-15. On the afternoon of February 21, I etherized this whole vial
of junk flies. Examining them, I found a pair (**suzukii** male: **tanythrix** female) sticking together in
copulation. I showed this pair to Carson and to T.W. Lyttle. They were overetherized and died, so I pinned
them as a specimen (see Figure 1).

Checking the literature, I found that the
suzukii subgroup is considered the most primi-
ive in the **melanogaster** group (Lemeunier
et al. 1986). It has a wide distribution, being
known from India, Japan and China (I have
found the species in Northern China), as well
as in the Afrotropical and Australasian reg-
ions. The species was found in Hawaii for the
first time in 1980 and now has spread over all
the islands.

I decided to try to get them copulating
again, using newly-collected flies brought from
the Big Island March 19 by Carson. I separated
the males from the females and combined **tan-
ythrix** females with **suzukii** males and put the
vials on my desk at about 25°C. The flies
seemed not active. A few days later, I put
the vials into the 18° incubator. It seemed
to me that it is not easy to get them copulat-

Figure 1. A copulating pair consisting of **Droso-
phila tanythrix** female (left) and **Drosophila
suzukii** male. Photograph of a pinned specimen by
Stephen W. Michael.

ing again; I felt embarrassed to do such an experiment with no result, thinking that people would laugh at me.

The next morning, however, was March 28 and I took the vials out of the incubator at 9 a.m. I found a pair that was copulating. I was excited and thought that no one would believe if I told them. At that time, a graduate student, Robert Wisotzkey was in the lab and I asked him to be an eye witness. This was not necessary since Carson said he believed me. Later, while I was out for collections on the island of Maui, he made some evening experiments on April 1-3. After he found no courtship or tapping under an infra-red light, he turned on the overhead light; the room temperature was about 20°. Males and females were combined in one large vial en masse. Immediate active courtship, tapping and "shivering" was begun by many males. Females were seen tapping and approaching males. Males were seen to attempt copulation by rushing the female from behind but the females seemed to move frantically to escape. Males began courting in less than one minute after being brought into white light. One copulation was observed at 8:53 p.m. and at 8:56 another was found. One pair separated very soon, and the other pair broke apart during aspiration.

In summary, we have observed four firm copulations of **suzukii** males and **tanythrix** females, two apparently in darkness and two under conditions of light. Based on chromosomal studies, Carson & Yoon (1982) suggest that Antopocerus-like flies arose no more than 1.8 million years ago. This is the approximate age of the high Molokai volcano, the area proposed for the origin of the subgroup. Clearly evolved from an older group of Hawaiian drosophilids, their lineage must be separated from their common ancestor with the Sophophora by at least the age of the oldest high island, Kauai, that is, about six million years. This is surely only a minimum estimate of the time-distance between the species.

Why should distant species behave in this manner? We suggest that the secondary sexual characters of both of these species serve the primary function of an intrapopulational process, namely sexual selection. Such characters may or may not serve the secondary function of sexual isolation. In this case, a small behavioral compatibility may exist due merely to chance.

References. Carson, H.L. & J.S. Yoon 1982, Genetics & Biology of Drosophila, M. Ashburner et al., eds. 3b, 277-304, Academic Press; Hardy, D.E. 1977, Proc. Entomol. Soc. Wash. 79:82-95; Kaneshiro, K.Y. 1976, Proc. Hawaii. Ent. Soc. 22:255-278; Lemeunier, F. et al. 1986, Genetics & Biology of Drosophila 3e: 148-239, Academic Press; Spieth, H.T. 1968, Univ. Texas Publ. 6818:319-333; Wheeler, M.R. 1981, Genetics & Biology of Drosophila, M. Ashburner et al., eds. 3a:1-97, Academic Press.

Imasheva, A.G., D.B. Kholodenko and L.A. Zhivotovsky. N.I. Vavilov Inst. of General Genetics, Moscow, USSR. The change in variation of metric wing characters in experimental lines of **Drosophila melanogaster.**

Stabilizing selection, i.e., selection favoring intermediate phenotypes, plays an important role in natural populations. Such selection has been investigated experimentally in Drosophila (see, e.g., Prout 1962; Scharloo 1964; Gibson & Bradley 1974), but in all these studies selection was conducted on one trait only. The present experiment was started with the view of studying the effects of stabilizing selection on a complex of correlated characters in **D.melanogaster.**

The details of experimental procedure are described elsewhere (Imasheva et al. 1986). The foundation stock came from a large random-breeding cage population kept at the Biological Dept. of the Moscow State University for about 6 months prior to the start of the experiment. Selection was conducted in 2 replicate lines; 2 other lines were maintained without selection and served as controls. Nine quantitative characters of the wing were used for selection (Figure 1). Measurements were performed on detached wings using an eyepiece micrometer.

Selection was conducted only on females with the coefficient of 50% (15 flies selected out of 30 measured). Females selected as parents for the next generation had minimal distance from the "average" phenotype (Zhivotovsky & Altukhov 1980); 15 males used as parents were sampled at random. Flies were kept in bottles on standard yeast medium at 25°C. The experiment continued for 15 generations.

The results of the experiment are presented graphically in Figure 2, where the change of generalized variance (Zhivotovsky 1984) of wing characters in time is shown. There was no substantial difference in variance between the selected and control lines. Thus, artificial stabilizing selection in the context of our experiment proved to be ineffective (which may be due to the low selection intensity). However, as can be seen from the graph, the amount of variation of wing characters decreased steadily both in control and selected lines.

Such a loss in phenotypic variance can be attributed to the action of the following factors: (1) natural stabilizing selection; (2) inbreeding, as only 15 pairs of parent were used each generation; (3) both these factors at once.
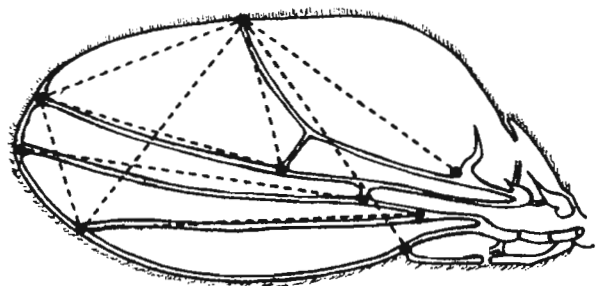
Figure 1. Wing measurements.

Figure 2. Generalized variance in the selected (S) and control (C) lines.

Table 1. Generalized variance and coefficient of variation at the end of the experiment (15th generation).

Population	Variance	Coefficent of variation
Selected line I	1.255	1.481
Selected line II	0.972	1.306
Control line I	1.071	1.367
Control line II	1.282	1.498
Base population	1.308	1.528

In order to choose from the above possibilities, we have measured the variability of wing characters in the base cage population of **D.melanogaster**, from which our lines were derived. The similar loss in variance revealed in the base population (Table 1) excluded inbreeding as a relevant factor in these changes.

We suggest that the observed decrease in phenotypic variance of wing characters is due to the pressure of natural stabilizing selection occurring in the laboratory conditions with the much greater intensity than the experimentally applied stabilizing selection. There is evidence that the modal phenotypes for different quantitative characters have superior fitness (Tishkin & Glotov 1983). Our results indicate that natural selection in constant laboratory environment favors flies with average parameters of the wing.

References: Gibson, J.B. & B.P. Bradley 1974, Heredity 33:293-302; Imasheva, A.G., D.B. Kholodenko & L.A. Zhivotovsky 1986, Genetika (USSR) 22:2291-2295; Prout, T. 1962, Genet. Res. 3:364-382; Scharloo, W. 1964, Genetics 50:553-562; Tishkin, V.V. & N.V. Glotov 1983, Genetika (USSR) 19:622-627; Zhivotovsky, L.A. 1984, in: Integration of polygenic systems in populations, "Nauka" Publ. House, Moscow.

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The instability of a P element insertion mutation
is affected by chromosomes derived paternally
from a pseudo-M strain of **D.melanogaster.**

In the P-M system of hybrid dysgenesis, some M cytotype strains possess P elements (Bingham, Kidwell & Rubin 1982). These so-called M' or pseudo-M strains may in fact be widespread in some parts of the world (Kidwell 1983; Anxolabehere et al. 1985). In addition to this populational significance, these strains have been useful in studies of P element activity (Kidwell 1985; Simmons & Bucholz 1985). The experiments we report here are in this vein.

A stock of the wild-type strain called Sexi was obtained from Margaret Kidwell in 1985. Previous characterizations by Kidwell (1985) had indicated that this strain had P elements, but that they were not able to induce GD sterility. This is a feature of Q strains in the P-M system; however, Sexi is distinguished from these by variation in its ability to suppress GD sterility. Both P and Q strains possess the P cytotype, a condition that is transmitted maternally and that suppresses hybrid GD sterility whenever females from these strains are crossed to strong P males. Kidwell (1985) pointed out that some females from the Sexi strain appeared to have the P cytotype, while others had the M cytotype. Still other females seemed to

Table 1. GD sterility in Sexi hybrid females.

Cross	No. Crosses	No. females	No. sterile	Unweigh- ted %
A	27	295	11	3.8
B	31	361	1	0.3
A*	35	402	135	33.4

Figure 1. Distribution of GD sterility among the hybrid daughters of Sexi females x π_2 males (Cross A*).
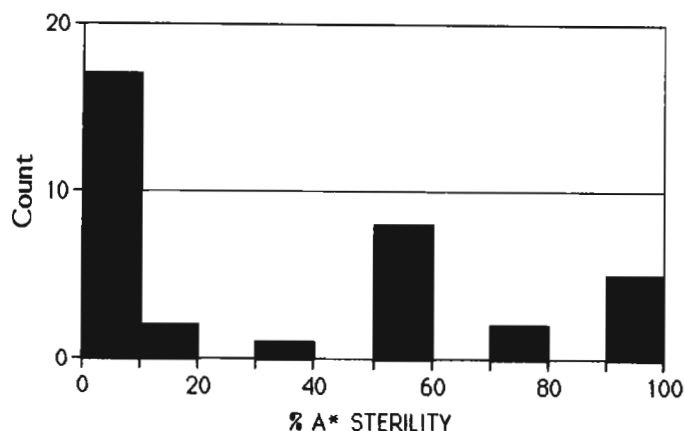
have an intermediate cytotype, since in crosses with strong P males, roughly half of their hybrid daughters showed GD sterility. Kidwell (1985) also noted that when Sexi was crossed with H-41, a true M strain, the F_1 progeny exhibited variation in the degree of suppression, no matter which way the cross was performed. In this respect, the progeny of the two reciprocal crosses were indistinguishable; both types exhibited some ability to suppress GD sterility, but not nearly as much as the Sexi females themselves. All these facts suggest that Sexi possesses few, if any, autonomous P elements, and that it is partially able to regulate the activity of these elements when they are introduced by a dysgenic cross. Furthermore, the equivalence of the reciprocal hybrids between Sexi and H-41 suggests that this partial regulation is due to chromosomal factors rather than to maternal inheritance.

Kidwell's (1985) analysis of the Sexi strain dealt only with its ability to induce or suppress GD sterility. We have investigated another dysgenic trait, the hypermutability of a P element insertion mutation of the singed bristle locus. This mutation, sn^W (singed weak), was discovered by Engels (1979), who found that in the M cytotype it is unstable. This instability arises from the excision of one or the other of two nonautonomous P elements inserted in the mutation (H. Roiha, K. O'Hare & G. Rubin, pers. comm.). However, excision of either of these elements depends absolutely on the P transposase, the product of the gene present in autonomous P elements (Engels 1984; Karess & Rubin 1984; Rio, Laski & Rubin 1986). Thus, sn^W can be used to assay for the transposase, as well as for the M cytotype which permits its action. Simmons & Bucholz (1985) used this mutation for still another purpose, to determine the extent to which transposase activity was affected by P elements derived paternally from an M' strain. In their experiments, the instability of sn^W was reduced when these elements were present, suggesting that the available transposase was titrated by the P elements from the M' strain.

As a prelude to the study of Sexi's effect on sn^W hypermutability, we tested its ability to induce and suppress GD sterility using standard crosses. In one cross (A), Sexi males were mated individually to bw; st females from a true M strain; this was to determine if the Sexi males could induce GD sterility. In another cross (B), individual Sexi females were mated to bw; st males to check for background sterility, and in still another cross (A*), individual Sexi females were mated to males from the strong P strain π_2 so that the cytotype of these females could be determined. All these crosses were performed at 29° and 8-12 hybrid daughters from each were examined for GD sterility. These examinations were done by squashing the females in colored water between two glass slides so that any eggs they contained would be extruded into the surrounding fluid. Females that did not extrude eggs were classified as having GD sterility.

The results are shown in Table 1. It is clear that crosses A and B produced very few sterile females, whereas cross A* produced a moderate frequency of them. The low frequency of sterile females from cross A corroborates Kidwell's finding that Sexi does not induce much, if any, GD sterility. In comparison, a strong P strain such as π_2 would induce 100% sterility in this cross. Only one sterile female was found among the 361 derived from cross B, which serves as the control. This very low frequency is less than the frequency from the reciprocal cross (A), but not significantly so.

The females derived from cross A* exhibited a mean sterility frequency of 33.4%. However, the actual distribution of sterility among the daughters of the Sexi females who were te ted indicates quite a bit of scatter about this mean. This is shown in Figure 1, from which it is apparent that many Sexi females had fewer than 10% sterile daughters, suggesting that they had the P cytotype. These results are consistent with those obtained by Kidwell (1985), who showed that among females from the Sexi strain, there is variation in the ability to suppress hybrid GD sterility.

The ability of Sexi flies to induce P element action was tested further by using the sn^W assay. Sexi males were mated to y sn^W; bw; st females in mass cultures at 25°. The only P elements that were present in the sn^W strain were located in the vicinity of the singed locus and these were all nonautonomous (Engels

Table 2. sn^W hypermutability in hybrid males and females from Sexi males X y sn^W;bw;st females.

Hybrid	No. cultures	sn^W	sn$^+$	sn^e	Total	mutation rates u	v
males	178	4,455	18	32	4,505	1.10	0.74
females	100	3,515	--	3	3,518	--	0.15

The mutation rates were calculated as unweighted percentages; u=percent sn$^+$ + sn^e among total; v=percent sn^e among sn^e + sn^W.

Table 3. sn^W hypermutability in males with different genetic backgrounds.

age	No. cultures	sn^W	sn$^+$	sn^e	Total	mutation rates (%) unweighted	weighted
Canton S:							
young	241	6,824	294	262	7,380	10.23 ± 0.92	7.53
old	191	4,701	226	139	5,066	10.19 ± 1.02	7.20
combined	432	11,525	488	433	12,446	10.21 ± 0.69	7.40
Sexi:							
young	220	6,205	99	172	6,476	4.23 ± 0.47	4.18
old	190	4,468	89	125	4,682	4.51 ± 0.51	4.57
combined	310	10,673	188	297	11,158	4.37 ± 0.35	4.35

1984): this strain also had the M cytotype. The y sn^W/Sexi daughters that emerged from these cultures were mated individually to y sn^3 v males and their progeny were scored for the singed weak and singed extreme phenotypes. The singed extreme flies were genotypically sn^e/sn^3 and resulted from mutations of sn^W to sn^e occurring in the germ lines of their mothers; these mutations are manifestations of transposase-catalyzed sn^W instability. The y sn^W sons from the initial crosses were also tested for sn^W instability. The procedure was to mate these sons individually to C(1)DX, y f females so that germ line mutations of sn^W could be detected in the male progeny. In this case, however, sn$^+$ males were also scored since there was no confusion between the sn$^+$ derivatives of sn^W and the sn$^+$ allele carried by the Sexi X chromosome. The tests of the y sn^W/Sexi females enabled us to screen the Sexi X chromosome and autosomes for transposase-producing P elements, while the tests of the sn^W males enabled us to screen the Sexi Y chromosome and autosomes. In both cases, the progeny of the tested flies were scored on the 14th and 16th days after mating. All cultures were reared at 25°C.

The results are shown in Table 2. About one percent of the progeny of the tested males were either sn$^+$ or sn^e, indicating some transposase activity. Only 0.1% of the progeny of the tested females were sn^e, suggesting either a much lower level of transposase activity in females or a sex chromosome difference in the distribution of transposase-producing elements. It is clear, however, that at least some Sexi flies carried autonomous P elements.

Do the P elements in the Sexi flies titrate the transposase, as suggested for another M' strain (Simmons & Bucholz 1985)? To answer this question, the methodology of Simmons & Bucholz was employed. Males from 13 different sn^W stocks were mated individually to Muller-5 (M5) females from a stock without any P elements. Each of these sn^W stocks had an X chromosome with at least one autonomous P element. The derivation and propagation of these stocks was described by Simmons & Bucholz (1985). Each cross was replicated twice. M5/sn^W daughters were collected from each of the cultures, divided into two groups and then mated individually to either Sexi or Canton S males, the latter coming from a pure M stock. In the next generation, sn^W males with either the Sexi or Canton S background were selected and mated individually to three C(1)DX, y f; bw; st females. As many as 20 males from each cross, replicate and background were mated, but not at the same time. Rather, half of the males were mated as soon as they were collected and the other half were mated seven days later. The sons of these matings were scored for bristle phenotype on the 14th and 16th days after mating. All cultures were reared at 25°C.

Table 3 shows the results of this experiment. The unweighted mutation rate that is given is the average percentage of singed extreme and wild-type flies calculated over the 13 crosses. The weighted rate is simply the sum of the singed extreme and wild-type flies divided by the total flies scored. The weighted mutation rates in the Canton S background are somewhat less than the unweighted rates because males from two of the crosses with this background were exceptionally mutable. In the unweighted calculation, these crosses contributed equally to the average, rather than in proportion to the actual number of flies counted. In the Canton S background, the unweighted mutation rate for the combined data was 10.21%, whereas in the Sexi background it was only 4.37%. By a t-test, this difference is significant. In neither background was there a difference between the mutation rates of young and old males. The consistency of results across ages strengthens the conclusion that the mutability of sn^W was reduced in the Sexi background.

A more conservative comparison of backgrounds over crosses and ages was done by pooling the results across cultures and replicates. For the young flies, the average mutation rate in the Canton S background exceeded that in the Sexi background ten times; for the old flies, this happened nine times.

Overall, there were 2 x 13 = 26 comparisons. On the hypothesis that there is no effect of background on the mutation rate, the probability of these or more extreme results is 0.015 by the sign test, which is significant. Additional experiments are underway to clarify the effect of the Sexi genetic background on the mutability of sn^W.

The apparent reduction in sn^W mutability reported here cannot be attributed to a cytoplasmic factor, since the Sexi strain was used paternally in the crosses. Rather, this reduction would seem to be due to factors on the Sexi chromosomes, quite possibly the P elements they carry. Qualitatively, these results are consistent with those obtained by Simmons & Bucholz (1985), who studied sn^W hypermutability in the presence of chromosomes derived paternally from another M' strain. The titration hypothesis they advanced may therefore explain the effects of the Sexi chromosomes as well.

References: Anxolabehere, D., D. Nouaud, G. Periquet & P. Tchen 1985, PNAS USA 82:5418-5422; Bingham, P.M., M.G. Kidwell & G.M. Rubin 1982, Cell 29:995-1004; Engels, W.R. 1979, PNAS USA 76:4011-4015; _____ 1984, Science 226:1194-1196; Karess, R. & G.M. Rubin 1984, Cell 38:135-146; Kidwell, M.G. 1983, PNAS USA 80:1655-1659; _____ 1985, Genetics 111:337-350; Rio, D.C., F.A. Laski & G.M. Rubin 1986, Cell 44:21-32; Simmons, M.J. & L. Bucholz 1985, PNAS USA 82:8119-8123.

Kamping, A. and W. van Delden. University of Groningen, Haren, Netherlands. The relationship between tolerance to starvation and tolerance to alcohols.

Drosophila melanogaster strains homozygous for the Adh^F allele (FF) or the Adh^S allele (SS) have continously been kept on food medium to which one of the following alcohols had been added: methanol (M), ethanol (E), propanol (P), isopropanol (IP), butanol (B), hexanol (H), octanol (O) and glycerol (G). Some of the strains have previously been tested for alcohol tolerance and showed increased tolerance to the specific alcohol they had been exposed to for about 100 generations, while also cross-tolerance to some other alcohols has been observed (Van Delden & Kamping 1983; Kerver & Van Delden 1985). The increased tolerance was in some cases accompanied by increased ADH activity, but it was concluded that other factors than ADH activity were also involved in the development of alcohol resistance.

In view of the observation that regular FF and SS strains differ in their resistance to starvation (Van Delden & Kamping 1986), it was decided to test the alcohol strains for their tolerance to starvation. Prior to the starvation test, the alcohol strains were cultured for one generation on regular food. For the tests 20 flies, 6-7 days old, were put into vials with 9 ml agar (18 g agar in 1 liter water). The sexes were separated; the number of replicates per sex and strain was five. Mortality was recorded at intervals.

Table 1 gives the mortalities of the alcohol strains relative to the mortalities of the control strains (SSN and FFN) which had regularly been kept on normal food and were simultaneously tested for their tolerance to starvation. Due to great differences in tolerance to starvation among the sexes and Adh genotypes, the mortalities are given for different periods of starvation. For comparison: mortality percentages at the given times were 81, 69, 64 and 66 for SSN ♀♀, FFN ♀♀, SSN ♂♂ and FFN ♂♂, respectively. Females are more tolerant to starvation than males. A striking difference exists between SS and FF strains. All SS alcohol strains have a lower mortality than the SSN control strain and are consequently more tolerant to starvation. The FF alcohol strains on the other hand are less or equally tolerant than the FFN control strain, except for the FF strains regularly kept on hexanol and octanol, as the females of the butanol strain also have a higher tolerance to starvation. This difference between SS and FF strains may be related to the difference in the mechanism of alcohol adaptation observed by Kerver & Van Delden (1985). The finding that the hexanol and octanol tolerant strains, FFH and FFO, behave differently from the other alcohol strains, may be associated with the different mechanism of adaptation found in the strains kept on higher alcohols, compared with strains kept on alcohols with a lower number of C-atoms.

The relationship between the mechanisms involved in the development of tolerance to starvation and tolerance to alcohols is the subject of further study.

References: Kerver, J.W.M. & W. van Delden 1985, Heredity 55:355-367; Van Delden, W. & A. Kamping 1983, Entomol. Exp. Appl. 33:97-102; _____ & _____ 1986, DIS 63:134-135.

Table 1. Mortalities of alcohol strains in starvation test, expressed relative to mortalities of control strains.

Adh genotype	SS	SS	FF	FF
Sex:	♀	♂	♀	♂
Starvation period (h):	50	31	96	72
Strain:				
control	1.00	1.00	1.00	1.00
methanol	0.37	0.97	1.16	1.30
ethanol	0.38	0.80	1.28	1.27
propanol	0.51	0.53	1.43	1.52
isopropanol	0.62	0.77	1.29	1.18
glycerol	0.42	0.47	1.16	1.44
butanol	0.11	0.47	0.90	1.17
hexanol	0.21	0.14	0.22	0.89
octanol	0.48	0.58	0.33	0.92

Kidwell, M.G. University of Arizona, Tucson.
A survey of success rates using P element
mutagenesis in **Drosophila melanogaster.**

The P-M system of hybrid dysgenesis in **D.melanogaster** is being widely used to attempt to generate many types of mutations. These include mutations resulting from P element insertions within specific loci which may provide a molecular tag for the locus of interest. The induction of P element mutations resulting in various general classes of phenotyes (e.g., imaginal disc defects) and the secondary mutagenesis of P elements already resident at specific chromosomal locations have also been used as mutagenesis strategies.

In order to evaluate the method of P element mutagenesis, a survey of both published and unpublished results has been carried out. These results are summarized in Table 1. In reading this table several qualifications should be noted. The survey covers only a limited sample of experiences with this technique and is not intended to be exhaustive. It should be used only as a rough guide of general trends in the types and frequencies of mutagenesis that have been attempted. In particular, too much weight should not be placed on observed frequencies in individual experiments. Methods of scoring and reporting frequencies vary widely, e.g., the total number of mutant individuals, uncorrected for premeiotic events, are reported in some studies, but the number of independent mutations are provided in others. In some instances, P element insertions have been directly implicated in mutation induction. In others, different insertions have been implicated, or the specific mechanism has not been identified.

Overall, the results suggest that P element mobilization under conditions of P-M hybrid dysgenesis is a promising method of mutagenesis for some loci and for a number of purposes. However, it should be noted that there are apparently a number of cold spots in the genome (e.g., the Adh, nap and st loci) resulting from site specificity of P element insertions. Unfortunately, it has not been possible a priori to identify such loci. There is also some evidence for differential success rates resulting from the use of different P and M stocks in the initial cross.

Secondary P element mutagenesis has the potential for being useful for a number of purposes such as the study of gene regulation. Very high mutation frequencies of resident P element insertions have been observed in several studies, for example, the secondary mutagenesis of P elements located in sequences affecting regulation of the rudimentary locus (Tsubota & Schedl 1986).

For further information on the background, methodology and sources of stocks suitable for P element mutagenesis, see Engels (1985a and b) and Kidwell (1986).

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References: Engels, W.R. 1985a, DIS 61:1; _____ 1985b, DIS 61:70; Kidwell, M.G. 1986, P-M mutagenesis, pp59-81 in: Drosophila - A Practical Approach (D.A. Roberts, ed.), IRL Press, Oxford; Tsubota, S. & P. Schedl 1986, Genetics 114:165-182. All references to specific experiments are included in the body of Table 1.

Table 1. Results of P element mutagenesis survey. The meanings of column title abbreviations are as follows: **p/s** = whether primary (p) or secondary (s) mutation was involved; **TT** = whether (Y) or not (N) transposon tagging was of interest; **P** = the P strains used as the source of P elements for mutagenesis; **M** = the M strains used in the initial cross; **Cyt** = the cytotype (P or M) in which the mutations were maintained after induction; **F1 sex** = whether mutations were induced in females (F) or males (M); **No.** = number of mutants recovered; **Tot.** = total number of progeny or chromosomes scored; **Ref.** = see list of References at end of Table.

Target	p/s	TT	P	M	Cyt	F1-Sex	No.	Tot.	%	Ref	Comments
Adh (alcohol dehydrogenase)	p						0	10,000,000	0.0000	11	
Aldox (Aldehyde oxidase)	p	Y	π2	ry+5	M	♂	3	1,021	0.2938	18	1 of 3 identified as a P insertion
bib (big brain)	p	Y	Harwich	seF8	P	♂+♀	0	6,000	0.0000	2,59	Same screen used for mam mutants
Bx (Beadex)	p	N	π2	C(1)DX,yf	M	♂	32	7,989	0.4006	47	No mutations recovered using a Q strain
ca (claret)	p	Y	π2	y^2 wb f (true M)	M	♀	1	11,372	0.0088	16	Frequency of ca using an M' strain was 3/31,278 (0.0096%)
Chromosome 3 imaginal disc defects	p	Y	Cage 3, 78.25, 78.1, 8.31.15, π2	m w h red e	P	♂	1	4,680	0.0214	13	1 P element insertion mutation (awd) has been sequenced and is located at 100C-D

Target	p/s	TT	P	M	Cyt	F1-Sex	No.	Tot.	%	Ref	Comments
Chromosome 3 lethals	p	N	Cage 3, 78.25 78.1, 8.31.15, π_2	m w h red e	P	♂	390	4,680	8.3333	13	Embryonic lethals = 165(3.52%); Late larval lethals = 20 (0.43%)
Chromosome 3 lethals	p	?	Harwich, π_2	red e	M	♂	206	8,000	2.5750	30	
Comments: Embryonic lethals = 143; post-embryonic lethals = 163; min. no. lethal events = 152											
cin	p	Y	π_2	$In(1)sc^8$	M	♂	1	67,884	0.0015	18	Mutant does not contain a P element
cn (cinnabar)	p	N	MR-h12, n1, T-007, OK1	pr cn	M	♂	107	311,027	0.0344	25	Deletions
Df(1) mal 12 (including runt and fog)	p	Y	π_2	$In(1)sc^8$	M	♀	32	10,500	0.3048	60	
Dhod (Dihydroorotate dehydrogenase)	p	Y	Kerbinou, Harwich, Luminy, π_2	Charolles, Cocka-ponsett Forest, Agana	M	♂	4	20,000	0.0200	41	
Dl (Delta)	p	Y	Harwich	se^{F8}, se ss k e^s ro	P	♂	5	70,000	0.0071	39	1 mutant identfied by P element homology, 4 by mapping
dvr (divers)	p	N	23.5/Cy L^4	dp b cn bw;ve	M	♂	0	2,376	0.0000	58	
dvr (divers)	p	N	31.1/Cy L^4	dp b cn bw;ve	M	♂	1	6,197	0.0161	58	
E (spl) (Enhancer of split)	p	Y	Harwich	se^{F8}, se ss k e^s ro	P	♂	0	70,000	0.0000	39	
eag (ether-a-go-go)	p	Y	N366.2 (π_2)	C(1)DX,yf (CantonS)	P	♂	3	28,000	0.0107	20	
eag (ether-a-go-go)	p	Y	N366.2 (π_2)	g sd f (CantonS)	P	♀+♂	24	86,300	0.0278	20	
Comments: Majority of mutants not yet analyzed but at least 1 is a P insert. 21/24 mutations were induced in females and 3/24 in males.											
fs (female sterile) in 7F-8A	p	Y	Various	$sn^3 lz^{50e30}$	M	♀	2	2,942	0.0680	38	
fs (female sterile) in 7F-8A	p	Y	Various	C(1)Ay; Dp(12)FN107/Cy	P	♂	4	1,781	0.2246	38	
fz (frizzled)	p	Y	Harwich	Oregon-R	M	♀+♂	6	75,000	0.0080	1	Probably only 1 of 6 mutations is a P insertion
Gl (Glued) revertants	p	Y	Cranston, Harwich Kerbinou	Gl st/TM3 Sb Ser	M	♂	18	20,000	0.0900	23	In some Glued revertants, normal Glued functions were restored by P insertions.
h (hairy)	p	Y	π_2; 25	Oregon-R	M	♂	8	22,991	0.0348	27	0/18,593 mutants were observed with the use of a Q strain
Lethals in 7F-8A	p	N	Various	$sn^3 lz^{50e30}$	M	♀	0	2,942	0.0000	38	
Lethals in 85DE	p	Y	π_2 IC3	m w h red e	P	♂	9	7,093	0.1269	33,10	IC3 produced a higher frequency of mutants than π_2
Lethals in cSOD microregion (68A2-C1)	p	N	Kerbinou	Canton-S	M	♀	73	4,741	1.5398	8	
Comments: Mutations recovered in 5 out of 11 functional groups, incl. lxd; 85% of all single site mutants fell in a single group											
lt (light)	p	Y	π_2	Sd-5,Pin,CantonS	M	♂	6	60,000	0.0100	26	Wide variation in reversion freq.
lxd (low xanthine dehydrogenase)	p	Y	Harwich		M	♂	1	6,000	0.0167	18	Mutants unstable in M cytotype but no P insertions identified
lxd (low xanthine dehydrogenase)	p	Y	Inhaca		M	♂	0	12,000	0.0000	18	
lxd (low xanthine dehydrogenase)	p	Y	π_2		M	♂	19	21,000	0.0905	18	Mutants unstable in M cytotype but no P insertions identified
lz (lozenge)	p	Y	Harwich	$In(1)sc^8$, sc^8 wa	?		3	41,592	0.0072	5	
lz (lozenge)	p	Y	Harwich	H-40 balancer	M	♀	5	75,198	0.0066	32	

Target	p/s	TT	P	M	Cyt	Fl-Sex	No.	Tot.	%	Ref	Comments
M (Minute)	p	?	Harwich, π_2	Canton-S (M') derived	M	♂	28	62,627	0.0447	19	Minute females were usually sterile
m (miniature)		N	Various MR strains				1	84,348	0.0012	15	Freq. given are for indep. events
m (minuature) lethals	p	N	π_2	C(1)DX,yf	M	♂	13	60,662	0.0214	47	No mutations in 18,516 chromosomes using a Q strain
mal (maroonlike)	p	Y	π_2	In(1)sc^8	M	♂	3	67,884	0.0044	18	1 of 3 identified as a P insertion
Male sterile genes on Y chromosome	p	N	Harwich, Inhaca Oxford	Ysx.YL Binscy	M	♂	13	2,417	0.5379	46	Problems with reversion and non-disjunction
mam (mastermind)	p	Y	Harwich	se^{F8}	P	♂	8	6,000	0.1333	59	4 of 8 mutants associated with chromosome rearrangement
mei-41	p	Y	Harwich, T-007, OK-1, π_2	f;bw;e sd;bw;e	M	♂	3	42,266	0.0071	57	
mei-9	p	Y	Harwich, T-007, OK-1, π_2	f;bw;e sd;bw;e	M	♂	4	42,266	0.0095	57	
ms1-1, mle, ms1-2	p	Y	π_2	Attached-X		♂	1	23,000	0.0043	34	
mus(1)101	p	Y	Harwich, T-007, OK-1, π_2	f;bw;e sd;bw;e	M	♂	0	42,266	0.0000	57	
mus(1)102	p	Y	Harwich, T-007, OK-1, π	f;bw;e sd;bw;e	M	♂	1	42,266	0.0024	57	
nap (no action potential)	p	Y	Inbred cage 3	C(1)DX,yf; cn bw (Canton-S)	P	♂	2	53,837	0.0037	20	**Comments:** Neither mutant carried a P element insertion. No other temperature-sensitive paralytic phenotypes were observed in the screen
nap (no action potential)	p	Y	N366.2 (π_2)	g sd f (CantonS)	P	♀+♂	0	109,800	0.0000	20	
net	p	Y	MR-h12	Oregon-R	M	♂+♀	30	27,400	0.1095	22	1(0.0036%) 1(2)gl; 2(0.0073%) net + lethal on chromosome 2
net 1(2)gl & 1(2)gl	p	N	Cy/MR-h12	pr cn	M	♂	4	3,442	0.1162	25	
net revertant	s	Y	net H121	netp25/Harwich	M	♂+♀	3	5,868	0.0511	22	
ninaE	p	Y	Harwich, π_2	w	M	♂	1	88,000	0.0011	42	Mutant contained neither a P nor a copia element
para (paralytic)	p	Y	N366.2 (π_2)	g sd f (CantonS)	P	♀	3	63,000	0.0048	20	**Comments:** Unknown whether the mutants carry P insertions. In one experiment any other temperature paralytic would have been observed. None were in 15,417.
para (paralytic)	p	Y	N366.2 (π_2)	g sd f (CantonS)	P	♀	2	109,800	0.0046	20,21	**Comments:** 5 ts-paralytic mutations were recovered altogether. Of these, 2 were para alleles and 3 were not. Of the 2 para alleles, 1 had a P insert, the other had an unknown insert. Nothing more is known of the 3 non-para mutations. (See also, nap 2nd entry.)
Pgd (Phosphogluconate dehydrogenase)	p	Y	Harwich,Inhaca,π_2 Luminy, ν^6,Kerbinou	wev4-wev8	M	♀	0	4,000	0.0000	40	
polycomb type regulatory genes	p	Y	Mcp (Harwich)	ppMcp (CantonS)	M	♀	8	30,000	0.0267	7	**Comments:** 1/8 mutants (an esc allele) was a P insertion. Other mutants: 1 ssa; 1 1(4)29; 1 polyhomeotic + 4 previously unidentified polycomb-type genes.
pr (purple)	p	N	MR-h12, n1, T-007, OK1	pr cn	M	♂	17	311,027	0.0055	25	Deletions
r (rudimentary) 5' end primary mutations	p	Y	Harwich, π_2	Various	M	♂	5	27,700	0.0181	51	
r (rudimentary) 5' end secondary mutations	s	N	Harwich, π_2	Various	M	♂	19	8,807	0.2157	52	

Target	p/s	TT	P	M	Cyt	F1-Sex	No.	Tot.	%	Ref	Comments
ras (raspberry)		N	Various MR strains				14	84,348	0.0166	15	Freq. given are for indep. events
ras (raspberry)	p	N	23.5/Cy L^4	dp b cn bw;ve	M	♂	0	2,376	0.0000	58	
ras (raspberry)	p	N	31.1/CyL^4	dp b cn bw;ve	M	♂	6	6,197	0.0968	58	
ras (raspberry)	p	N	Basc/+; MR/+	y^2 sn^3 ras^2 v	M	♂+♀	9	71,478	0.0126	48	The no. of mutation events was 9
ras (raspberry)	p	N	MR-h12	Berlin-K	M	♂	6	40,930	0.0147	15	
Reversion of Ant73b (small inversion)	p	Y	π	Balanced marker	P	♂	2	23,278	0.0086	31	
Rpll 215	p	Y	π$_2$;bw;st	FM7/C4	M	♂	9	40,180	0.0224	53	

Comments: 16 alleles actually recovered but some may represent clusters. Revertants were common.

Target	p/s	TT	P	M	Cyt	F1-Sex	No.	Tot.	%	Ref	Comments
Rudimentary-like in 93B	p	Y	Kerbinou,Harwich, Charolles, Cocka- Luminy, π$_2$ ponset Forest, Agana	M		♂	0	25,000	0.0000	41	
sd (scalloped)	p	N	23.5/Cy L^4	dp b cn bw;ve	M	♂	0	2,376	0.0000	58	
sd (scalloped)	p	N	31.1/CyL^4	dp b cn bw;ve	M	♂	1	6,197	0.0161	58	
sd (scalloped)	p	Y	Harwich	H-40 balancer	M	♀	10	16,042	0.0623	32	sd appeared to be a "warm spot" for mutation when Harwich was P strain
sd (scalloped)	p	Y	IC3,π$_2$,1978-100, 1978-68, 8.36-15	C(1)RM	M	♂	0	40,000	0.0000	43	
sd ry+destabilization	s		Harwich P18/MKRS	sd ry$^+$; ry^{506}	M	♂	261	3,074	8.4906	12	
Sensitivity to MMS	p	Y	T-007, OK-1 Kerbinou, π$_2$	w or sn^3	P	♂	2	10,392	0.0192	6	Both mutants were P inserts and unstable in P cytotype
Sensitivity to MMS	p	Y	T-007, OK-1 Kerbinou, π$_2$	f;bw;e	M	♀	0	5,244	0.0000	6	
Sensitivity to MMS	p	Y	T-007, OK-1 Kerbinou, π$_2$	f;bw;e	M	♂	0	1,584	0.0000	6	
sev (sevenless)	p	Y	25A	C(1)DX,yf in a CantonS background	M	♂	3	23,500	0.0128	4	25A has a P element near sev (at 10A). Mutation rates may be underestimated due to screening difficulties.
Sh (Shaker)	p	Y	π$_2$	C(1)DX,yf	P	♂	8	24,000	0.0333	28,50	

Comments: Contrary to the cited report, P inserts were not identified in any of the induced mutations (Timpe, L., pers. comm.)

Target	p/s	TT	P	M	Cyt	F1-Sex	No.	Tot.	%	Ref	Comments
shi (shibire)	p	Y	IC3,π$_2$,1978-100, 1978-68, 8.36-15	C(1)RM	M	♂	2	40,000	0.0050	43	1 P element insertion mutation confirmed.
sn (singed)	p	N	23.5/Cy L^4	dp b cn bw;ve	M	♂	2	2,376	0.0842	58	
sn (singed)	p	N	31.1/CyL^4	dp b cn bw;ve	M	♂	13	6,197	0.2098	58	
sn (singed)	p	N	Basc/+;MR/+	y^2 sn^3 ras^2 v	M	♂+♀	28	71,478	0.0392	48	
sn (singed)	p	N	MR-h12	Berlin K	M	♂	21	40,930	0.0513	15	
sn (singed)	p	N	Various MR strains				36	84,348	0.0427	15	Freq. given are for indep. events
sn (singed)	p	N	π$_2$	C(1)DX,yf	M	♂	39	7,989	0.4882	47	Mutation freq. using Q strain lower
sn^w (singed-weak) hypermutable allele	s	N	π$_2$	C(1)DX,yf;cn bw	M	♂	1748	2,810	62.2064	17	
st (scarlet)	p	Y	Cranston, Harwich, π$_2$	Canton-S	M	♀	0	28,200	0.0000	36	

Target	p/s	TT	P	M	Cyt	F1-Sex	No.	Tot.	%	Ref	Comments
st (scarlet)	p	Y	Several from W. Engels	Several from W. Engels	M	♂	0	60,000	0.0000	3	2 deletions but no insertions were observed
su(f)(suppressor of fork)	p	Y	π_2	C(1)DX,yf	M	♂	2	4,590	0.0436	37	
Comments: The 2 su(f) alleles were identified in a screen of 109 previously induced X chromosome lethals (M. Simmons, pers. comm.)											
su(s)(suppressor of sable)		Y					1			9	1 su(s) P insertion allele discovered in a screen for another locus
Sxl increase of function	s	N	Harwich	Sxl^{fPb}/B (M')	M	♂	70	1,000	7.0000	45	
Comments: % of mutations with reduced Sxl function was 0.01. The M' strain had a 1.1kb insert in Sxl and 11 other P inserts in the genome.											
v (vermillion)	p	N	Basc/+;MR/+	y^2 sn^3 ras^2 v	M	♂+♀	9	71,478	0.0000	48	
v (vermillion)	p	Y	Harwich	H-40 balancer	M	♀	1	75,198	0.0013	32	
vg (vestigial)	p	Y	π_2	Oregon R	M	♂	1	900,000	0.0001	55	
vg (vestigial)	s	N	21(P)mutant vg stock	Oregon R	M	♂	6	3,600	0.1667	54	
Comments: 2 lethals were deletions. 4 fertile alleles were either P element internal deletions (3), or the result of a possible P conversion event (1).											
w (white)		N	Various MR strains				1	84,348	0.0012	15	Freq. given are for indep. events
w (white)	p	Y	Harwich	H-40 balancer	M	♀	8	75,198	0.0106	44	Min. events per genome = 6×10^{-5}. Reversion rates = or > 1×10^{-3}.
w (white)	p	N	π_2	C(1)DX,yf	M	♂	9	131,544	0.0068	47	No mutations recovered in 42,000 chromosomes using a Q strain
w (white)	p	Y	π_2	$In(1)sc^8$	M	♂	7	67,884	0.0103	18	No information about P insertions. 24 other sex-linked mutants obsvd.
X-lethals	?	N	π_2	C(1)DX,yf	M	♂	127	5,534	2.2949	47	Lethal freq. using a Q strain lower.
X-lethals	p	N	Harwich	Cy/Prm; Sb/D	M	♂	45	1,354	3.3235	35	
X-lethals	p	N	Harwich, π_2	$In(1)sc^8$	M	♀	131	1,511	8.6698	60	Note that lethals induced in F1 ♀
X-lethals	p	N	Harwich, π_2	$In(1)sc^8$	M	♂	22	883	2.4915	60	
X-lethals	p	N	MR-h12	Berlin K	M	♂	114	10,024	1.1373	15	Range of 1.06-2.75% X-lethals obtained with other P & M strains
X-lethals	p	N	Various MR strains				274	19,246	1.4237	49,15	
Comments: Lethal frequencies for different P strains differed between 2.7% (T-007) and 1.1%											
X-lethals (to study larval neoplasms)	p	N	π_2	y w	M	♂	600			14	High revertant rate in P cytotype
X-linked loci involved in egg shell formation	p	Y	Harwich	?	P	♂	375	4,000	9.3750	29	>20% with weak phenotypes
y (yellow)		N	Various MR strains				4	84,348	0.0047	15	Freq. given are for indep. events
y (yellow)	p	N	Basc/+; MR/+	y^2 sn^3 ras^2 v	M	♂+♀	18	71,478	0.0252	48	Actual no. of mutation events = 4
y (yellow)	p	N	Cy/MR-h12	Cy/bwv	M	♂	4	23,299	0.0172	24	
Comments: Mutants at a number of X chromosome visible loci were also scored using a Maxy X chromosome.											
y (yellow)	p	Y	Harwich,Inhaca,π_2 Luminy, v^6,Kerbinou	wev4-wev8	M	♀	6	68,875	0.0087	40	
z (zeste)	p	Y	Harwich	Canton-S	M	♂	0	30,600	0.0000	56	
Zw	p	Y	Harwich,Inhaca,π_2 Luminy, v^6,Kerbinou	wev4-wev8	M	♀	2	68,875	0.0029	40	Comments: Both Zw mutants were induced with the Harwich P stock giving
a frequency of 1.4×10^{-4}. Both were nulls and had P insertions localized to regions 18 and 18C-D.											
zw (zeste-white) lethals	p	N	π_2	C(1)DX,yf	M	♂	28	70,882	0.0395	47	Mutation freq. using Q strain lower
zw+ to null	p	Y	π_2	Compound X	?	♂	0	82,000	0.000	34	

Table 1 (contin.) - References:

1. Adler, P.M. 1984, Genetics 107:s1.
2. Artavis-Tsakonas, S. (pers.comm.).
3. Baker, B. & M. McKeown (pers.comm.).
4. Banerjee, U. 1986, Dros. Conf. Abstracts.
5. Batterham, P. (pers.comm.).
6. Brodberg, R.K. & J. Boyd (pers.comm.).
7. Burgess, E., I. Duncan & U. Banerjee 1986, Dros. Conf. Abstracts.
8. Campbell, S., A. Hilliker & J.P. Phillips 1986, Genetics 112:205-215.
9. Chang, D.Y. et al. 1986, Mol.Cell Biol. 6:1520-1528.
10. Cheney, C.M. (pers.comm.).
11. Craymer, L. & Meyerowitz (cited by W.R. Engels 1983, Ann. Rev. Genet. 17:315-44).
12. Daniels, S.B. et al. 1985, Genetics 109:95-117.
13. Dearolf, C. (pers.comm.).
14. Denell, H. (pers.comm.).
15. Eeken, J.C.J. & F. Sobels (pers.comm.).
16. Endow, S.A. (pers.comm.).
17. Engels, W.R. 1979, PNAS 76:4011-4015
18. Finnerty, V. & C.K. Warner (pers.comm.).
19. Frisardi, M.C. & M. Rosbash (pers.comm.).
20. Ganetzky, B. & R.A. Kreber (pers.comm.).
21. Ganetzky, B. et al. 1986, Ann. N.Y. Acad. Sci. 479:325-337.
22. Ganot, D., Z. Kimchie & Z. Lev (pers.comm.).
23. Garen, A., B.R. Miller & M.L. Paco-Larson 1984, Genetics 107:645-655.
24. Green, M.M. 1977, PNAS 74:3490-3493.
25. Green, M.M. & S.H.Y. Shepherd 1979, Genetics 92:823-832.
26. Holm, D.G. (pers.comm.).
27. Ingham, P. et al. 1985, Genetics 111:463-486.
28. Jan, J.L. et al. 1983, Cold Spr. Harb. Symp. 48:233-245.
29. Kafatos, F.C. et al. 1985, in: Molecular Developmental Biology (L. Bogorat, ed.), Alan Liss.
30. Karess, R. (pers.comm.).
31. Kaufman, T. & S. Korikami (pers.comm.).
32. Kidwell, M.G. (unpublished results).
33. Lang, T. & C.M. Cheney 1986, Dros. Conf. Abstracts
34. Lucchesi, J. (pers.comm.).
35. Margulies, L. (pers.comm.).
36. Marsh, J.L. (pers.comm.).
37. Mitchelson, A. (pers.comm.).
38. Mohler, D. (pers.comm.).
39. Muskavitch, M.A.T. (pers.comm.).
40. Nero, D., PhD thesis, Cornell Univ. Library.
41. Osgood, C. (pers.comm.).
42. Pak, W.L. & J. O'Tousa (pers.comm.).
43. Poodry, C. (pers.comm.).
44. Rubin, G.M., M.G. Kidwell & P.M. Bingham 1982, Cell 29:987-994.
45. Salz, H.K. (pers.comm.); Salz, H.K., T.W. Cline & P. Schedl 1987, Genetics 117:221-231.
46. Schafer, U. & J. Nahmias 1985, Mol. Gen. Genet. 200:182-184.
47. Simmons, M.J. et al. 1984, Genetics 106:85-94.
48. Sinclair, D.A.R. & M.M. Green 1979, Mol. Gen. Genet. 170:219-224.
49. Sobels, F. & J.C.J. Eeken 1981, Mut.Res. 83:201-206.
50. Timpe, L. (pers.comm.).
51. Tsubota, S., M. Ashburner, M. & P. Schedl 1985, Mol. Cell Biol. 5:226.
52. Tsubota, S. & P. Schedl 1986, Genetics 114:165-182.
53. Voelker, R.A. et al. 1985, Mol. Gen. Genet. 201:437-445.
54. Williams, J. (pers.comm.).
55. Williams, J. & J. Bell 1986, Dros. Conf. Abstracts.
56. Wu, C. (pers.comm.).
57. Yamamoto, A.H. (pers.comm.).
58. Yannopoulos, G. (pers.comm.).
59. Yedvobnick, B. et al. 1985, Cold Spring Harb. Symp. Quant. Biol.
60. Zusman, S., D. Coulter & J.P. Gergen 1985, DIS 61:217.

Kjaer, T. University of Copenhagen, Denmark. dp^{ovh} is not synonymous to dp^h.

Lindsley & Zimm (1985) considered in their manuscript to the new redbook of mutants in **Drosophila melanogaster** that dp^{ovh} and dp^h were synonymes of the same dp-allele. From our work on the biochemistry of dp (Kjaer 1987), we have found that dp^{ovh} does not show the same phenotypical expression in trans-configuration with dp^{ov} as reported in dp^h (Lindsley & Grell 1968). Here, dp^h was considered a very extreme dp^{ov} allele, showing strongly oblique wings and strong thoracic invaginations.

In Figure 1, dp^{ov}/dp^{ovh} shows no deviation from the wild type. This could indicate that the dp allele was lost in the stock (dp^{ovh} Sp cn bw/In(2L)Cy, Cy dp^{lvl} cn bw from Bowling Green), especially since this heterozygote shows no thoracic effects either. To check this possibility, two test crosses were made: dp^{ov}/dp^h b bw and dp^{ovh} Sp cn bw/dp^h b bw. Figure 2 shows the expected very extreme oblique wing in dp^{ov}/dp^h. For comparison, Figure 3 shows the wing phenotype of dp^{ovh}/dp^h, and here it is clearly seen that although dp^{ovh} must be considered as a weaker allele than dp^{ov}, the dp oblique effect is not lost!

From these results, we conclude that dp^h and dp^{ovh} are not synonymous, but dp^{ovh} is to be considered a very weak ov allele like dp^{cm2}, although dp^{ov}/dp^{cm2} shows strong thoracic effects unlike dp^{ov}/dp^{ovh}. Furthermore, we consider it to be of the utmost importance to avoid any confusion in the already very obscure context in the dp locus.

Figure 1. dp^{ov}/dp^{ovh}.

Figure 3. dp^{ovh}/dp^{h}.

Figure 2. dp^{ov}/dp^{h}.
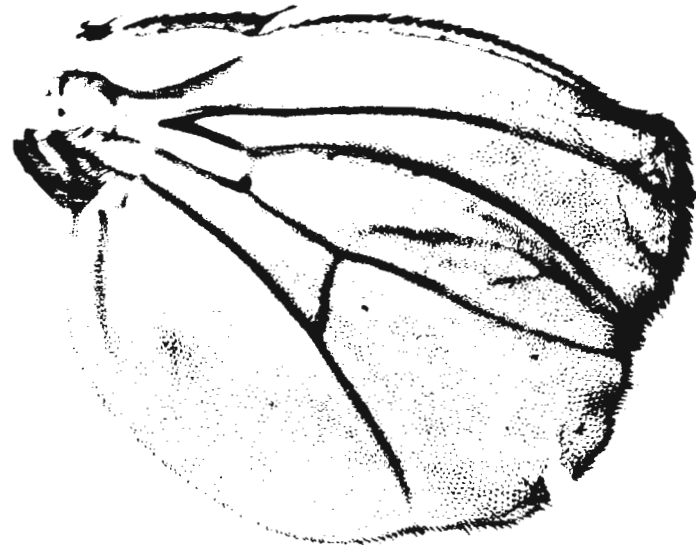
References: Kjaer, T. 1987, Hereditas 107 (in press); Lindsley, D.L. & E.H. Grell 1968, Carnegie Inst. Wash. Publ. No. 627; Lindsley, D.L. & G. Zimm 1985, DIS 62:92.

Kochieva, E.Z. and T.I. Gerasimova. N.I. Vavilov Inst. of General Genetics, Moscow, USSR. Repeated mutations and transposition memory in unstable **Drosophila melanogaster** stocks.

The phenomenon of transposition memory has been demonstrated for the cut locus (Mizrokhi et al. 1985). The process of reversions and repeated mutagenesis always correlated with the excision and re-insertion of mdg4 (gypsy) into the cut locus (Gerasimova et al. 1985). This work was aimed at finding out how the transposition memory was realized: whether the repeated mutagenesis resulted from the excision and re-insertion of a single transposable element, or repeated mutations occurred during transposition explosions -- mass simultaneous transpositions of many different mobile elements.

We have analyzed an unstable stock $cm^{MR17}ct^{MRpN17}$ which originated earlier from the stock ct^{MR2}. This stock was characterized by the high frequency of double reversions cm^+ct^+, unstable ones inclusive, which gave rise to repeated double mutations $cm\ ct^{pN}$ (Table 1).

Thus, we could follow a series of five successive transpositions: mutation → reversion → repeated mutation. The frequency of repeated mutagenesis was high and mutations occurred simultaneously in both loci.

The ct^{MRpN17} mutation depends on the insertion of an mdg4 copy with jockey within it (Mizrokhi et al. 1985). Unfortunately, the molecular nature of the transposon at the carmine locus is unknown. As followed from in situ hybridization experiments $ct^{MRpN} \rightarrow ct^+ \rightarrow ct^{MRpN}$ transitions are accompanied with an excision of mdg4 and jockey from the 7B3-4 region of the cut locus. Analysis of the distribution of different mobile elements (mdg1, mdg2 or 412, mdg3) on the X-chromosome of unstable revertants and repeated mutants showed that there were transpositions not only of mdg4 with jockey, but also of all the tested mobile elements (Table 1).

Table 1. Results of in situ hybridization of different mobile elements DNA with polytene chromosomes of the $cm^{MR17}ct^{MRpN17}$ and its derivatives.

Stocks	Freq. of mutational transitions	Localization of mobile elements on the X-chromosome				No. of transpositions
		mdg1	mdg2	mdg3	mdg4 + jockey	
$cm^{MR17a}ct^{MRpN17a}$		1C,8A,19E;	2B,3E,4C,20A;	13A,20D;	7B	
	3×10^{-3}					
$cm^{+1}ct^{+1}$		3C,7A,9B;	3E,4C,5A,20A;	13A,20D;	--	10
	0.7×10^{-3}					
$cm^{MR17b}ct^{MR17b}$		1C,8A,11C,17D,20A,20C;	2B,3E,4C,12D,18D,20A; -, -;		7B	16
	3×10^{-2}					
$cm^{+2}ct^{+2}$		1C,17D,20A,20C;	2B,3E,6A,19A,20A;	13A,20D;	--	11
	0.3-0.5					
$cm^{MR17c}ct^{MR17c}$		1C,8A,17D,20A,20C;	2B,3E,4C,12D,19A,20A; -, -;		7B	9
	5×10^{-2}					
$cm^{+3}ct^{+3}$		3C,11C,20A;	4C,12D,17D,19A,20A;	13A,20D;	--	14

Note: the reverse transpositions of mdg-elements are underlined.

The number of transposition events amounted to 24 per one mutation transition. In addition, repeated insertions combined with the majority of new transpositions. For example, with $cm^{MR17b}ct^{MRpN17b}$ four out of the nine new transpositions were re-insertions, and with $cm^{MR17c}ct^{MRpN17c}$ four out of the five transpositions were re-insertions.

The molecular basis of transposition memory has been explained for the cut locus and mdg4 (Mizrokhi et al. 1985). There remains a solo LTR after the excision of mdg4 from the cut locus. Similar data were described for the scute and yellow loci (Compuzano et al. 1985).

One may propose that the repeated transpositions of other mobile elements are also based on the inexact excision of a mobile element and it stems apparently from a homologous recombination between the extrachromosomal mdg circles containing a single LTR and genomic solo LTR.

Thus, we have arrived at the conclusion that the repeated mutations ct^{MRpN} result from re-insertions of mdg4 with jockey into the cut locus and these events always correlate with the mass transpositions of all tested mobile elements.

Therefore, transposition explosions are primarily scale changes which include not only actual transpositions of mobile elements but also their homologus recombination.

References: Compuzano, S., L. Carramolino, C.B. Carberera, M. Ruiz-Gomes, R. Villares, A. Boronat & J. Modollel 1985, Cell 40:327-338; Gerasimova, T.I., L.V. Matujnina, L.J. Mizrokhi & G.P. Georgiev 1985, The EMBO J. 4:3773-3779; Mizrokhi, L.J., L.A. Obolenkova, A.F. Priimägi, Y.V. Ilyin & G.P. Georgiev 1985, The EMBO J. 4:3781-3797.

Lamooza, S.B. Nuclear Research Center, Baghdad, Iraq. Genetic monitoring on irradiated population of Drosophila collected from Iraq.

The use of ionizing radiations has its genetic effects regarding future generations. These radiations induce gene mutations, the majority of which have deleterious effects on individuals carrying them. Therefore, the effect of radiation on a population will change its genetic constitution and probably affect the biological fitness (Dyer 1969a, 1969b). Changing in frequency of gene or chromosomal mutations in **Drosophila melanogaster** could be taken as a good measure for changing in radiation levels (Wurgler et al. 1976). The present investigation reports the spontaneous and induced dominant lethal mutations in **D.melanogaster** populations collected from three different localities in Iraq (Tuwaitha, Basrah, Mosul) (the nature of collections and collecting procedure as described by Lamooza et al. 1985) and for Oregon-k; the data are shown in Table 1.

Determination of dominant lethal mutations was carried out using a standard method by which eggs were collected daily from each single mated female on agar medium blackened with charcoal; after 2 days incubation the eggs, number of hatched and and unhatched eggs were determined. It is clearly seen from this table that the differences in the spontaneous level of Dominant lethal Mutation were statistically insignificant at two years of collection. On the other hand, Table 2a and 2b show the induction of dominant lethal mutations by different doses of gamma radiation (0.05, 0.1, 0.2 and 0.4 kGy) in males of the above mentioned populations in comparison with Oregon-k. Statistical analysis (two-way analysis of variance, $P<0.05$) showed no significant differences between the three populations and Oregon-k in their sensitivity to gamma radiation for two years of collection: 1980, 1981. The slight variation

Table 1. Natural frequency of dominant lethal mutation in the males of Drosophila collected from different localities in Iraq.

Drosophila population	Total no. of eggs examined	% Dominant lethals
Tuwaitha 1980	887	9.57
Tuwaitha 1981	2013	8.39
Basrah 1980	3109	9.93
Basrah 1981	2316	8.04
Mosul 1980	2761	9.05
Mosul 1981	1900	7.41
Oregon-K	5798	8.46

Table 2. (a) Frequency of dominant lethal mutations induced by different doses of gamma radiation in males of natural Drosophila populations from three localities in Iraq from 1980.

Dose (kGy)	Tuwaitha Total no eggs laid	% D.L.	Basrah Total no eggs laid	% D.L.	Mosul Total no eggs laid	% D.L.	Oregon-K Total no eggs laid	% D.L.
0.0*	887	13.07	3136	12.94	2875	8.86	1882	11.74
0.05	907	16.64	1629	15.89	1737	11.91	2087	17.29
0.1	865	21.04	2450	23.79	2387	17.39	2392	19.64
0.2	536	24.25	1932	37.93	2693	22.91	2435	28.50
0.4	577	44.36	2093	41.33	1931	52.04	2274	33.81
(b) Repeated for 1981:								
0.0*	2013	8.04	2316	7.05	1900	7.41	2660	8.39
0.05	1905	16.50	2192	15.39	2138	17.63	2556	16.17
0.1	1859	19.98	2432	19.41	2020	21.13	2460	18.17
0.2	1900	32.03	2112	35.97	2418	33.49	2455	37.98
0.4	2054	60.63	2313	61.55	2377	61.20	2527	61.39

D.L. = % of dominant lethals; * = control.

in the induction of dominant lethal mutations that was shown could be explained as a result of collecting these populations recently from the field, and they include a high degree of hybrid vigor as a result of genetic variation between individuals, but such variation disappeared in Oregon-k population due to brother sister interbreeding.

In conclusion, Tuwaitha population of **D.melanogaster** showed the same response in comparison with the other populations in spite of living in an area with possible radiation activities. Such studies are considered important to provide some information regarding the genetic changes that might occur in the environmental population with long-term low level exposure to radiation in order to compare them in different times and to correlate them with any increase in radiation level if any.

References. Dyer, K.F. 1969a, Genetics 61:275-291; _____ 1969b, Genetics 61:227-244; Lamooza, S.B. et al. 1985, Biochem. Genet. 23(3/4):321-328; Wurgler, F.E. et al. 1976, In: Genetics and Biology of Drosophila (Ashburner & Novitski, eds.), 1c:1268-1298.

Lechien, J., M. Libion-Mannaert and A. Elens. FNDP, Namur, Belgium. Food and frequency dependent sexual activity.

The problem of the "rare male advantage" in Drosophila is often investigated by the way of "multiple choice mating" experiments, using the "observation chambers" described many years ago (Elens 1957, 1958; Elens & Wattiaux 1964). Ordinarily, such observations have been done "without food" in the chamber. However, in some experiments concerning **Drosophila subobscura**, it has been found that the sexual activity was higher if a little quantity of lukewarm food had been dropped on the paper filter used as bottom for the observation chamber (Wattiaux 1968). Could the presence of food in the "copulation chamber" influence differently the sexual activity level of the various **Drosophila melanogaster** strains? If yes, it could influence also the so-called "rare male advantage" so frequently observed in the competition between such strains.

In the present work, we study the effect of food (or of the absence of it) on the competition between w and gt w^a D.melanogaster flies. A total of 30 pairs of virgin flies, 4-5 days old, were used for each test, but the relative frequencies of both genotypes differed. If p is the frequency of the gt w^a genotype and q the frequency of the w genotype, the studied ratios are: p/q = 3/27, 6/24, 9/21, 12/18, 15/15/, 18/12, 21/9, 24/6, 27/3 (the frequencies being the same for both sexes of the same type). Twenty-four hr before the test, groups of 30 flies of the same sex, assembled in numbers corresponding to the above proportions, were stored in separate vials. The total number of flies per vial was always the same; such a habit avoids any bias which could result from differences in the number of stored flies. As controls, separate groups of 30 virgin pairs of each genotype were similarly prepared. The tests were always done under the same conditions, at 25°C, 1000 lux, and 40-60% relative humidity. From 8 o'clock a.m., the 11 chambers were under constant control, until the end of the 5 hr observation time: the observation intervals (3-5 min) must, of course, be shorter than the duration of copulation, but the observed copulations are totalized and registered every 15 min only. Five repetitions have been done. It has been impossible to do at the same time the tests with and without food, but they have always been done during the same week, on alternate days.

The male sexual activities of both genotypes at various relative frequencies are shown, as cumulated percentages, in Figures 1 (in presence of food) and 2 (without food), for only 5 of the 9 relative frequencies tested (p/q = 3/27, 6/24, 15/15, 24/6, 27/3) as it was impossible to figure the whole of it; but the figures show also the cumulated percentage of copulations recorded at the same time for the 30 virgin pairs of the same genotype, observed as a control in a separate chamber.

The collected data have been analyzed by three methods. The Petit's "male sexual fitness coefficient" K_M (Petit 1951, 1958), with his standard error, has been determined 1, 2, 3, 4, and 5 hr after the beginning of experiments (Table 1 gives only the results for the p/q = 3/27, 6/24, 15/15/, 24/6, and 27/6 relative frequencies, and only for the 1st, 3rd, and 5th observation hr). According to Ayala, frequency-dependent mating success can be measured by the regression of the logarithm of the ratios (p/q) at which the individuals are present (Ayala 1972; Ayala & Campbell 1974). Such regression lines have been determined after 1, 2, 3, 4, and 5 hr of continuous observation (Figures 1 and 2 show the regression lines for the 1st, 3rd, and 5th hr only). The third method is a modification of Wattiaux's equation (Wattiaux 1964). In the original equation, ln dx/N - x, x was the number of individuals of one type having already copulated before the considered interval of time and N was the total number of these types individuals. In the present case, x has been replaced by x/N, a "mating index". The formula becomes: ln dx/N /(N - x/N). This equation

Table 1. Male sexual fitness coefficient, K_M, for some relative frequencies, determined after 1, 3, and 5 hours observation.

Relative frequencies	3 gt w^a/27 w K_M	SEM	6 gt w^a/24 w K_M	SEM	15 gt w^a/15 w K_M	SEM	24 gt w^a/6 w K_M	SEM	27 gt w^a/3 w K_M	SEM
in presence of food:										
gt w^a: 1st hr	0.62	0.4532	1.27	0.5510	1.00	0.4263	0.30	0.1348	0.26	0.1167
3rd hr	0.58	0.2761	0.75	0.2465	0.82	0.2262	0.57	0.1490	0.37	0.2257
5th hr	0.76	0.2992	0.92	0.2634	0.95	0.2151	0.52	0.1264	0.30	0.0729
w: 1st hr	1.61	1.1780	0.78	0.3384	1.00	0.4263	3.24	1.4561	3.70	1.6612
3rd hr	1.72	0.8872	1.31	0.4307	1.20	0.3310	1.72	0.3138	2.61	0.7920
5th hr	1.29	0.5078	1.08	0.3092	1.05	0.7520	1.88	0.4237	3.15	0.7857
without food:										
gt w^a: 1st hr	3.00	1.4141	0.21	0.2154	1.00	0.6324	0.41	0.2114	0.06	0.0742
3rd hr	2.42	1.0304	0.64	0.3083	0.71	0.2939	0.21	0.0692	0.14	0.0498
5th hr	2.10	0.8812	0.90	0.4458	0.64	0.2476	0.19	0.0649	0.17	0.0580
w: 1st hr	0.33	0.1555	4.75	4.8733	1.00	0.6324	2.40	1.2591	15.00	10.9541
3rd hr	0.41	0.1745	1.55	0.7469	1.40	0.5796	4.70	1.5502	7.00	2.4926
5th hr	0.47	0.1972	1.10	0.5426	1.54	0.5958	5.17	1.6688	5.72	1.9548
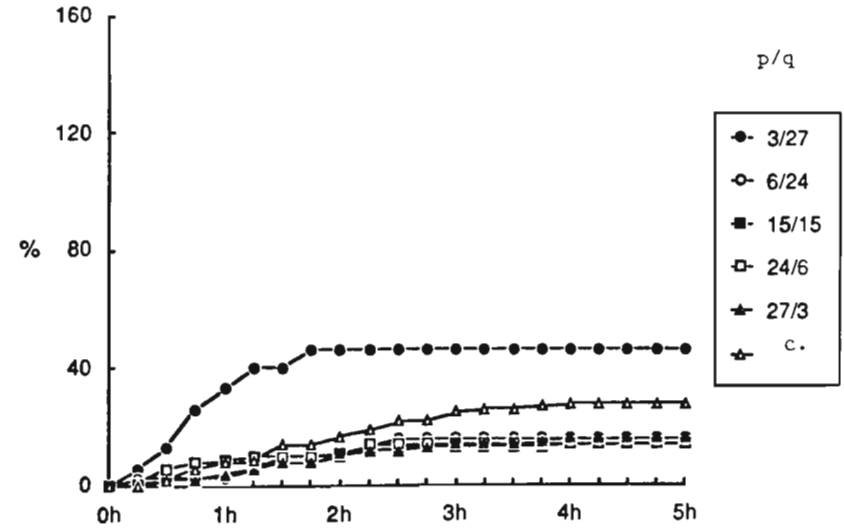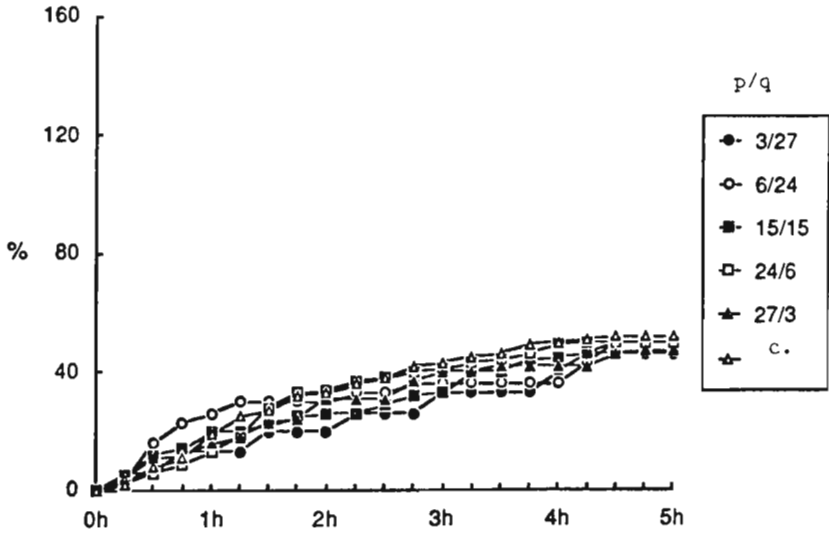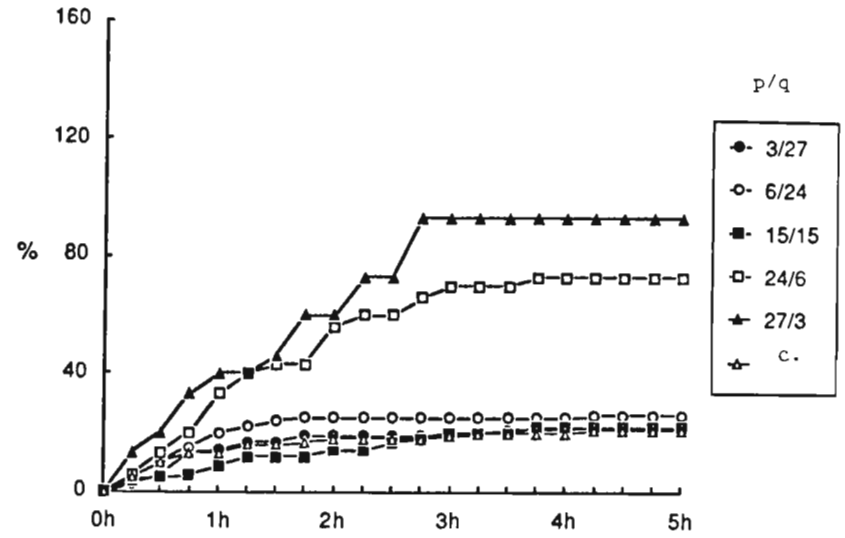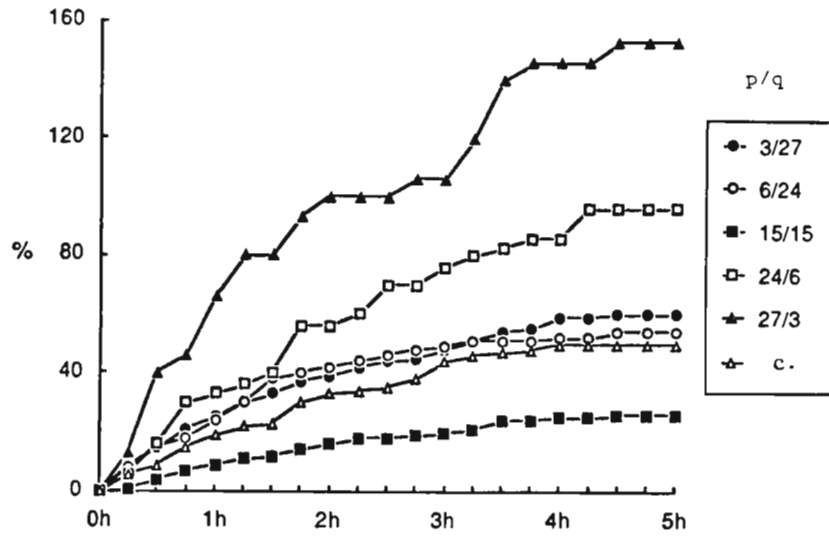
Figure 1. In presence of food. Cumulated percentage of the male sexual activity, at various relative frequencies (given as ratio of gt w^a on w: p/q = 3/27, 6/24, 15/15, 24/6, 27/3), in function of time in hours. The cumulated percentage of copulation is also given for the "control" test. Higher part: w male activity. Lower part: gt w^a male activity.

Figure 2. Without food. Cumulated percentage of the male sexual activity, at various relative frequencies (given as ratio of gt w^a on w: p/q = 3/27, 6/24, 15/15/, 24/6, 27/3), in function of time in hours. The cumulated percentage of copulation is also given for the "control" test. Higher part: w male activity. Lower part: gt w^a male activity.
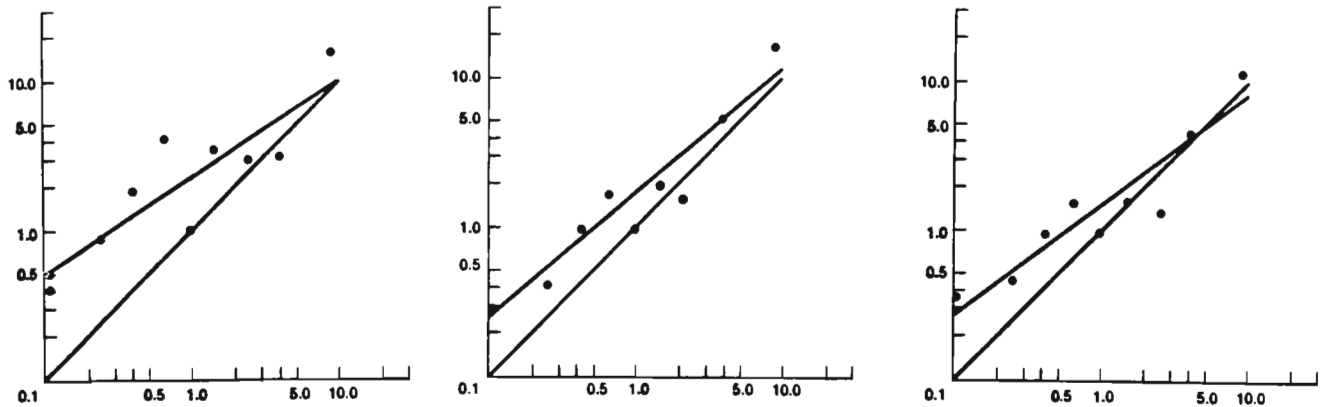
Figure 3. In presence of food. Linear regression of the logarithm of the ratio of males that mated (P_M/Q_M) on the ratio at which the males w and gt w^a are present (p_M/q_M). From left to right, the regression equations are: $Y = 4.20 + 0.67 X$ (1st hr), $Y = 2.34 + 0.83 X$ (3rd hr), $Y = 2.74 + 0.72 X$ (5th hr).
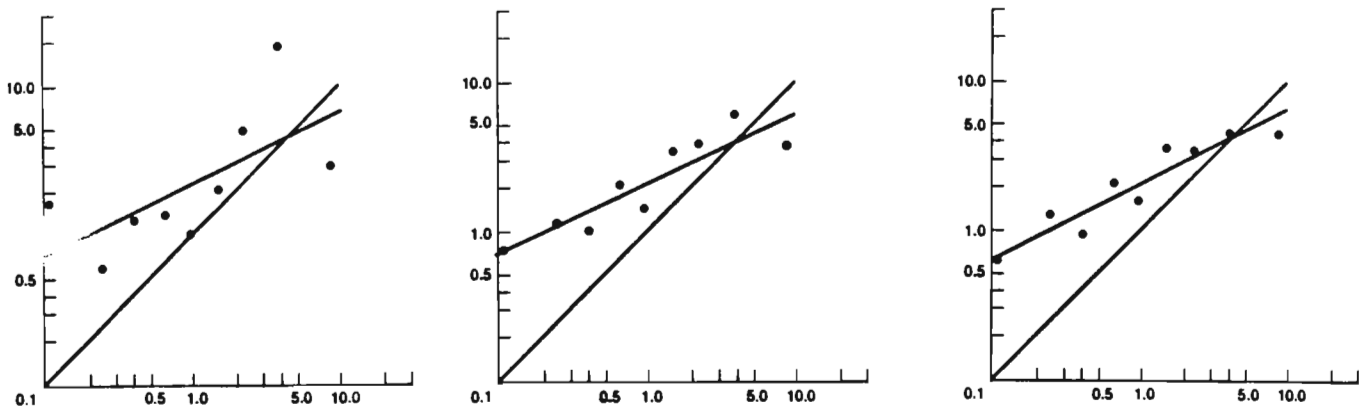
Figure 4. Without food. Linear regression of the logarithm of the ratio of males that mated (P_M/Q_M) on the ratio at which the males w and gt w^a are present (p_M/q_M). From left to right, the regression equations are: $Y = 5.27 + 0.49 X$ (1st hr), $Y = 5.33 + 0.47 X$ (3rd hr), $Y = 5.07 + 0.50 X$ (5th hr).

has been applied to the "male sexual activities" of both genotypes at their 9 various relative frequencies. However, Figures 3 and 4 show the regression lines corresponding to only some relative frequencies: p/q = 3/27, 6/24, 15/15, 24/6, and 27/3.

Table 1, calculated according to Petit, shows that the K_M coefficients, $(q_M/p_M)(P_M/Q_M)$, differ with the time at which they are calculated. At the 5th observation hr, "rare male advantage" is evident in the case of the white genotype (in good agreement with the observations of Petit). It is even more marked in absence of food. But, in absence of food, even gt w^a males seem advantaged when they are rare.

Similarly, Figure 4 shows that the frequency-dependence of the male sexual activity, calculated according to Ayala for the white flies, is more evident in absence of food (Figure 4). In presence of food, the slope of the regression line differs from 1 only after 5 observation hr (Figure 3).

In presence of food, the modified Wattiaux's equation shows that the w males are much more active when they are "rare" (27 gt w^a for 3 w) than at all the other relative frequencies: for the gt w^a males, the differences are never significant (Figure 1 and 5). In absence of food, one finds a significant difference between the activity of the w males at the two lower frequencies (24 gt w^a/6 w and 27 gt w^a/3 w) and at the other ones; the gt w^a males also are significantly more active when they are "rare" (3 gt w^a/27 w) than at all the other relative frequencies (Figures 2 and 6).

The three methods lead to the same conclusion: a "rare male advantage" is evident in our experiments, and not only for the white genotype.

References: Ayala, F.J. 1972, Behav. Genet. 2:85-91; _____ & C.A. Campbell 1974, Anim. Rev. Ecology 5:115-138; Elens, A. 1958, Experientia 14:274-276; _____ & J.M. Wattiaux 1964, DIS 39:118-119; Petit, C. 1951, Bull. Biol. France Belgique 85:392-418; Petit, C. 1958, Bull. Biol. France Belgique 92:248-418; Wattiaux, J.M. 1967, Z. Vererbungsl. 95:10-16; _____ 1968, Evolution 22:406-421.
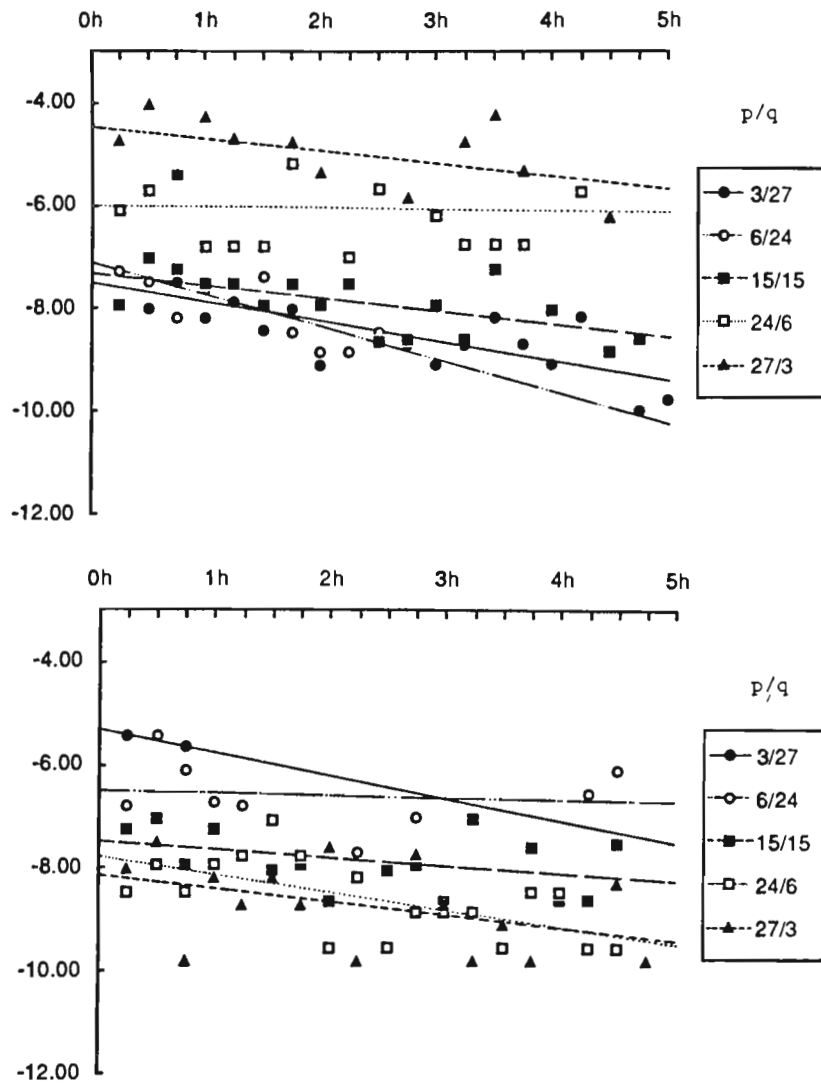
Figure 5. In presence of food. Relationship between the competing males gt w^a and w, when their relative frequencies differ (they are given as ratio of gt w^a on w: p/q = 3/27, 6/24, 15/15, 24/6, 27/3). Abscissa: time in hours. Ordinate: ln dx/N / (N-x/N). Higher part: w male activity. Lower part: gt w^a male activity.
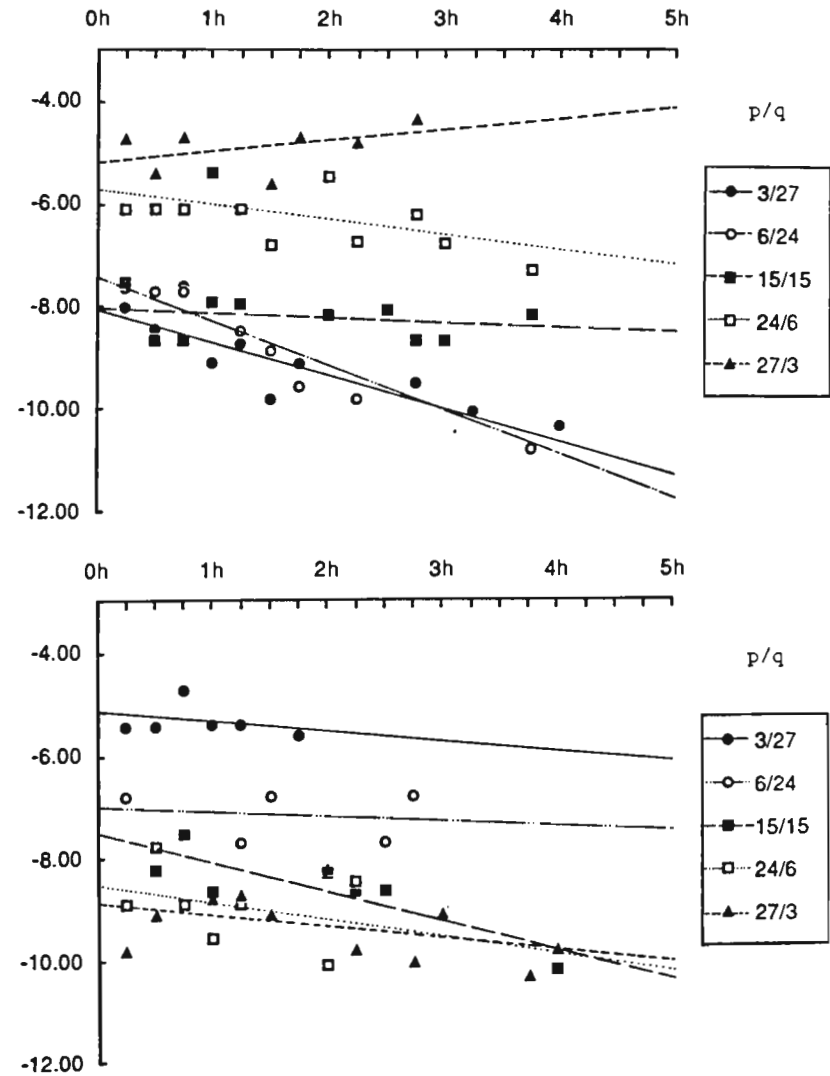
Figure 6. Without food. Relationship between the competing males gt w^a and w, when their relative frequencies differ (they are given as ratio of gt w^a on w: p/q = 3/27, 6/24, 15/15/, 24/6, 27/3). Abscissa: time in hours. Ordinate: ln dx/N / (N-x/N). Higher part: w male activity. Lower part: gt w^a male activity.

Lee, T.J. and G.J. Jhoo. Chung-ang University, Seoul, Korea. Genetic similarity among five species of the **D.quinaria** group.

Genetic similarity and phyletic relationship among five species, **D.angularis, D.brachynephros, D.unispina, D.curvispina, D.nigromaculata** were investigated by polyacrylamide gel electrophoresis (PAGE), SDS polyacrylamide gel electrophoresis (SDS PAGE) and two-dimensional gel electrophoresis (TDE).

Table 1. Similarities of proteins among five species of **D.quinaria** group obtained by PAGE.

	D.a.	D.b.	D.c.	D.u.
D.brachynephros	0.8750			
D.curvispina	0.7200	0.7600		
D.unispina	0.7308	0.6429	0.6296	
D.nigromaculata	0.3704	0.3571	0.3333	0.4074

In the similarities among five species obtained by PAGE, the highest similarity was 0.875 between **D.angularis** and **D.brachynephros**. The similarity between **D.curvispina** and **D.brachynephros** was 0.76, and **D.unispina** and **D.brachynephros** was 0.643. The similarities of **D.nigromaculata** to the other four species (Table 1) was very low.

Soluble protein patterns of **D.angularis** and **D.brachynephros** by SDS PAGE appeared highly similar. **D.curvispina** showed affinity with **D.unispina** in their protein patterns, and slightly differed from the patterns of **D.angularis** and **D.brachynephros**. The protein pattern of **D.nigromaculata** was different from the other four species.

Table 2. Genetic distance among five species of **D.quinaria** group obtained by TDE.

	D.a.	D.b.	D.c.	D.u.
D.brachynephros	0.1575			
D.curvispina	0.1969	0.2278		
D.unispina	0.2747	0.2978	0.2636	
D.nigromaculata	0.3230	0.3333	0.3767	0.4468

Genetic distance among five species were obtained by means of TDE. The genetic distance between **D.angularis** and **D.brachynephros** was 0.1575, lowest of all. The genetic distance between **D.angularis** and **D.curvispina** was 0.1969, and between **D.angularis** and **D.unispina** it was 0.2749. The genetic distance between **D.unispina** and **D.nigromaculata** was 0.4468, highest of all (Table 2).

References: Aquadro, C.F. & J.C. Avise 1981, PNAS 78:3784-3788; Lee, T.J. & S.S. Choi 1985, Rev. Tech. & Sci. Chung-ang Univ. 15:21-34.

Loreto, E.L.da S. and A.K. Oliveira. Universidade Federal do Rio Grande do Sul, Porto Alegre, Brasil. New leucine aminopeptidase larval isozymes of **Drosophila melanogaster.**

Beckman & Johnson (1964) identified six different bands showing LAP activity in pupae of **D.melanogaster**, but only two zones of activity have been observed in larvae (Sakai et al. 1969). By using a modified micromethod of starch gel electrophoresis, Muhs (1973) found another four zones between B and D

Beckman & Johnson's zones. It has been suggested that these enzymes are "pupal enzymes" responsible for histolysis of larval tissues (Muhs 1975). In contrast to this, Walker & Williamson (1980) measured the LAP activity and observed that it is higher in young larvae. They suggest that in larvae these enzymes may function in hydrolysis of digestive proteins and help to maintain osmotic pressure and supply amino acids for protein anabolism.

Figure. 0 = eggs; 1 = first instar larvae; 2 = second instar; 3 = third instar; W = white pupae; Y = yellow pupae; B = black pupae; A = adult (male).

We have studied the LAP isozymes in the development of Oregon-R stock, through acrilamida gel and tris-citric acid buffer pH 8.6 (38 mM tris; 2.5 mM citric acid in gel and 340 mM tris; 78 mM citric acid in electrode buffer). We found six zones in larvae: LAP A and LAP D according to Sakai et al. (1969) and more LAP B (weak), and three other bands denominated according to Muhs (1973) as LAP-C_1, LAP-C_2 and LAP-C_3. We did not observe the zones E and F described by Beckman & Johnson (1964) in Oregon-R stock, but we found these zones in pupae of the other stock, such as Leningrad and Buenos Aires.

We suggest that the larval isozymes here described are in accordance with the highest activity observed in young larvae by Walker & Williamson (1980).

References. Beckman, L. & F.M. Johnson 1964, Hereditas 51:221-230; Muhs, H.J. 1973, DIS 50:200-201; _____ 1975, Theor. and App. Gen. 46:215-220; Sakai, R.K., D.A. Tung & J.G. Scandalius 1969, Mol. Gen. Genetics 105:24-29; Walker, V.K. & J.H. Williamson 1980, Insect. Biochem. 10:535-541.

Markow, T.A. Arizona State University, Tempe.
Nocturnal emissions in **Drosophila simulans.**

Table 1. Number of flies aspirated from four clusters of fallen citrus, pooled from five days.

condition time	singles				mating pairs	
	D.m.		**D.s.**		**D.m.**	**D.s.**
	females	males	females	males		
D 6:00 AM	49	38	61	53	1	11
L 7:00 AM	77	60	48	46	3	6
L 8:00 AM	94	77	63	61	4	3
L 9:00 AM	65	55	81	52	17	6
L 10:00 AM	68	62	53	30	14	5
L 11:00 AM	61	58	44	47	7	1
L 12:00 AM	74	71	41	27	5	0
L 1:00 PM	41	37	24	26	5	0
L 2:00 PM	42	21	26	26	3	1
L 3:00 PM	36	39	20	14	1	0
L 4:00 PM	48	41	29	16	12	8
L 5:00 PM	81	57	38	41	24	16
L 6:00 PM	60	49	47	20	21	15
D 7:00 PM	54	51	59	63	9	9
D 8:00 PM	41	28	38	41	1	16
D 9:00 PM	19	22	19	23	0	10
D 10:00 PM	17	12	37	21	0	12
D 11:00 PM	24	18	21	19	0	8
D 12:00 PM	18	26	43	47	0	19
D 1:00 AM	20	9	35	26	0	17
D 2:00 AM	25	18	31	34	0	11
D 3:00 AM	16	3	44	51	0	11
D 4:00 AM	29	22	53	47	1	3
D 5:00 AM	31	38	50	47	1	7

Mating behavior of Drosophila species has been shown to be influenced by a number of environmental variables. In the laboratory, Hardeland (1972) has examined courtship activity over a 24 hr, 12:12 LD cycle in a number of species of the **melanogaster** group. **D.melanogaster** was observed to court most actively during the pre-light on hours while its sibling, **D.simulans**, only courted during the light hours. The data of Grossfield (1966, 1970) agree, showing that when pairs of flies are confined under conditions of continuous light or darkness, a high proportion of **D.melanogaster** females are inseminated under either condition, but copulation in **D.simulans** is greatly reduced in darkness.

Both **D.melanogaster** and **D.simulans** are found in Tempe, Arizona, throughout most of the year where they are associated with various fruits. I made hourly collections of these species from four patches of fallen citrus on five days in late November 1986 and recorded the number of single individuals and mating pairs recovered at each collection time. The data appear in Table 1. Mating pairs of **D.melanogaster** were more common than **D.simulans** during the daylight hours but during darkness, the only pairs mating were **D.simulans**. These observations differ dramatically from the earlier ones mentioned above. Possibly flies in nature rely on other cues in addition to light, such as temperature, to time their mating behaviors. Alternatively, strains of a given species may differ with respect to their light dependence (Grossfield, pers. comm.) and such strain differences may account for the contrast between the present study and the earlier ones. In any case, it no longer seems valid to generalize about light dependence in **D.simulans** courtship and mating. Furthermore, the value of studying natural populations in attempting to understand the ecological significance of Drosophila behavior cannot be overemphasized.

References: Grossfield, J. 1966, Univ. Texas Publ. 6615:147-176; _____ 1970, Am. Nat. 104:307-309; Hardeland, R. 1972, Anim. Behav. 20:170-174.

Mason, J.M. and L. Champion. NIEHS, Research Triangle Park, North Carolina. Y-autosome translocations may carry an allele of mu-2.

A number of laboratories have experienced difficulties maintaining the Y-autosome translocations generated by Lindsley et al. (1972). In many of the stocks the dominant markers appended to the progenitor Y chromosome tend to be lost with high frequency. Although we have not examined this problem directly, a number of observations in our laboratory suggest an explanation for the instabiity of these chromosomes. The relevant observations derive from an experiment designed to map a mutator. The mutator is semidominant (Table 1), and potentiates the recovery of terminal deficiencies (Mason et al. 1984). It may exhibit mutator activity in the presence or absence of x-irradiation, although a dose of x-rays as low as 100R may increase the frequency of y mutations 6X (Mason et al. 1984).

The mutator maps near the left end of chromosome 3, very close to r (0.0). In order to establish the cytological position of mu-2, we generated segmental aneuploid attached-X; mu-2/mu-2/DP(3L) females carrying the tip of 3L attached to a Y chromosome centromere (Lindsley et al. 1972). Females were irradiated and mutator activity was monitored by scoring for breakdown of the attached-X. None of the duplications showed evidence of complementing mu-2. However, the heterozygous translocations that served as controls did not complement mu-2 either (Table 2). Although the frequency of attached-X breakdowns in the euploid heterozygous translocation females is elevated, it is not as high as in the homozygous mu-2 females. The overall frequency in the translocation bearing females (2.05%) is 3X higher than in the heterozygous mu-2/TM6 females, and 3X lower than in the homozygous mu-2 females. The lack of complementation seen here suggests the possibility that the translocations used all carry an allele of mu-2, albeit a relatively weak allele. If true, the mutator allele may have been present on the third chromosome that was used as the progenitor of these translocations.

The irradiated females in Table 2 that were not heterozygous for a T(Y;3) carried a B^SYy^+ chromosome similar to the one used to generate the translocations. This Y chromosome acted as a pairing partner for the attached-X, as did the Y^P3^D chromosome in the translocation heterozygotes. It is possible, therefore, to monitor partial Y loss in the females carrying the B^SYy^+ chromosome. In homozygous mu-2 females the frequency of partial Y loss is 5.99%, similar to the frequency of attached-X breakdown.

Likewise, the frequency of partial Y loss is similar to the frequency of attached-X breakdown in the other two genotypes. The frequency of partial Y loss is greater in heterozygous females than in wild type females.

The pattern is less clear in the unirradiated females. There are no differences between heterozygous and wild type females in the frequencies of either attached-X breakdown or partial Y loss, although the numbers are small. Surprisingly, there are relatively small differences in the frequencies of attached-X breakdown and partial Y loss in homozygous mu-2 females with and without irradiation, and no differences in these two parameters between treated and untreated wild type females. On the other hand, the frequencies of these parameters are substantially higher in irradiated than unirradiated mu-2/TM6 females. Irradiation increases the frequency of y mutations in both homozygous mu-2 and wild type females (Mason et al. 1984; Table 1). It is not clear why the same is not the case for attached-X breakdown and partial Y loss.

Table 1. Mutation frequencies in females carrying free unmarked X chromosomes crossed with y w/y² sc Y males. The y w males result from X chromosome loss, y progeny from terminal deficiencies, and w males have lost the majority of the maternal X and carry only the tip (see Mason et al. 1984).

Female Genotype	X-ray dose	Progeny +	y w ♂	%	y	%	w ♂	%
mu-2/mu-2	500	73892	2296	3.01	177	0.239	38	0.051
mu-2/mu-2	0	69180	544	0.78	9	0.013	0	---
mu-2/+	500	59940	258	0.43	15	0.025	8	0.013
+/+	500	170877	460	0.27	17	0.010	20	0.012
+/+	0	54394	40	0.07	2	0.004	1	0.002

Table 2. Attached-X breakdown in mu-2/T(Y;3) females crossed with y pn males.

Female genotype (Chromosome 3)	T(Y;3) name	Break Point	X-ray dose	Maternal X Chrom. XX	0	X	Total	% X Exceptions
mu-2/T(Y;3)	S50	61A	500	1579	2091	95	3765	2.52
mu-2/T(Y;3)	B130	61DE	500	109	218	8	335	2.39
mu-2/T(Y;3)	A114	61F	500	1165	1945	43	3153	1.36
mu-2/T(Y;3)	D8	62A	500	97	285	11	393	2.80
Combination of 4 T(Y;3)s			500	2950	4539	157	7646	2.05
mu-2/mu-2	--	--	500	1659	2114	253	4026	6.28
mu-2/TM6	--	--	500	3204	3372	48	6624	0.72
+/+	--	--	500	5847	6773	47	12667	0.37
mu-2/mu-2	--	--	0	2002	1997	214	4213	5.13
mu-2/TM6	--	--	0	2871	2644	10	5525	0.18
+/+	--	--	0	5115	5694	38	10847	0.35

Table 3. Partial Y chrom. loss in attached-X/B^S+Yy$^+$ females crossed with y pn males.

Maternal genotype	X-ray dose	Maternal Y Chrom.				% Y Exceptions
		Y	0	Y^S+Y^L	Total	
mu-2/mu-2	500	2001	1784	241	4026	5.99
mu-2/TM6	500	3252	3330	42	6624	0.63
+/+	500	6721	5899	47	12667	0.37
mu-2/mu-2	0	1957	2052	204	4213	4.84
mu-2/TM6	0	2583	2932	10	5525	0.18
+/+	0	5674	5142	31	10847	0.29

In summary, a number of observations when taken together suggest that the instability of the Y-autosome translocations may be attributed to the presence of a mu-2 allele on these chromosomes. First, mu-2 potentiates the recovery of terminal deficiencies and other chromosomal aberrations. Second, mu-2 is semidominant, at least when irradiated. We have not made a serious effort to examine semidominance in the absence of irradiation. Third, unirradiated mu-2 females exhibit enhanced mutator activity. Fourth, the Y-autosome translocations fail to complement the mutator activity of mu-2. We should caution, however, that we have made no attempt to examine the stability of any of the Y-autosome translocations.

References: Lindsley, D.L., L. Sandler, B.S. Baker, A.T.C. Carpenter, R.E. Denell, J.C. Hall, P.A. Jacobs, G.L.G. Miklos, B.K. Davis, R.C. Gethman, R.W. Hardy, A. Hessler, S.M. Miller, H. Nozawa, D.M. Parry & M. Gould-Somero 1972, Genetics 71:157-184; Mason, J.M., E. Strobel & M.M. Green 1984, PNAS 81:6090-6094.

Meera Rao, P.M., B.V. Shyamala and H.A. Ranganath. University of Mysore, India. Metaphase karyotype of **D.formosana.**

Inter and intra-specific karyotypic variation is a well documented phemonenon among the Drosophilids. **D.formosana** was described by Duda (1926). It belongs to the **immigrans** species subgroup of the **immigrans** species group of Drosophila. We herein report the metaphase karyotype of **D.formosana** collected from Mysore.

Permanent air dry preparations of the larval neural ganglion were made as described by Ramachandra & Ranganath (1985). The male and female karyotypes are presented in Figures 1a and 1b. The metaphase karyotype reveals a pair of metacentric chromosomes and three pairs of acrocentric chromosomes, the diploid chromosome number being 2n=8. The X chromosomes are rod shaped and one of them is replaced by a darkly stained Y chromosome in males.

This karyotype of **D.formosana** differs from the basic karyotype of the **immigrans** species group in that the fourth (dot) chromosome is a "long rod". Wakahama et al. (1983) have given a diagramatic representation of the karyotype of **D.formosana** from Taiwan. From the comparison it appears that the dot chromosomes are relatively longer in the strain reported here.

Acknowledgements: The authors are grateful to Prof. N.B. Krishnamurthy, Chairman, Dept. of Studies in Zoology, for his help and encouragement; to Prof. T. Okada, for his advice in identifying the species; to Mr. M.G. Vasudeva Rao for preparing the photographs and to the U.G.C., New Delhi for providing the research grants.

References: Duda, O. 1926, Fauna sumatrensis, Beitrag 26, Drosophilidae (Dipt.) Suppl. Ent. Berlin 14:42-116; Ramachandra, N.B. & H.A. Ranganath 1985, Experientia 41:680-681; Wakahama, K., T. Shinohara, M. Hatsumi, S. Uchida & O. Kitagawa 1983, Jpn. J. Genet. 57:315-326.

Figure 1a-b: Metaphase chromosomes of **D.formosana** female (1a) and male (1b).

Mercot, H. and D. Higuet. CNRS, University of Paris 6, France. ADH activity in two **Drosophila melanogaster** strains from selection on body weight.

In **Drosophila melanogaster**, some authors have emphasized that both the amount of alcohol dehydrogenase (ADH) protein per mg body weight (Clarke et al. 1979) and ADH activity per mg body weight (Kamping & Van Delden 1978; Van Dijk 1981; Van Delden & Kamping 1983, 1985) increase with increasing body weight. In particular, Van Delden & Kamping (1983) have shown that strains, either homozygous for the Adh^F or the Adh^S allele, and kept for ninety generations in food supplemented with hexanol, showed an increase in both body weight and ADH activity.

Using two strains of **Drosophila melanogaster** genetically different for body weight, we have analyzed their ADH activity. These two strains, heavy (H) and light (L), were obtained from the Furnace Creek (FC) population by fifteen generations of selection for body weight (Higuet 1986). The H strain was polymorphic for Adh (Adh^F = 0.80); the L strain was monomorphic for the Adh^F allele. A HF strain was constituted from twenty-four pairs with an Adh^{FF} genotype issued from the H strain. Thus, we had at our disposal a heavy strain, homozygous for the Adh^F allele, in order to eliminate the ADH activity variation linked to the different Adh genotypes (Lewis & Gibson 1978; McDonald et al. 1980; Van Delden & Kamping 1980). All these strains were kept on cornmeal axenic medium (cornmeal 96 g, killed yeast 96 g, agar 15 g, Nipagine 6 g, water 1 liter).

ADH activity was assayed in adult males. Samples of 15 males (7-9 days old) were weighed and homogenized in 1 ml 0.1M tris HCl buffer, pH 8.6. The centrifuging was done at 15,000 rpm for 25 min at 4°C. The assays mixture consisted of 0.1 ml of supernatant, 0.8 ml 0.1 M tris HCl with 10% ethanol (v/v), 0.1 ml 0.01M NAD+. The initial increase in absorbance at 340 nm in the assay mixture was measured in a Perkin-Elmer Lambda 1 spectrophotometer at 25°C for 90 s. One unit of ADH activity is defined as an increase in absorbance of 0.001/min (equivalent to 1.61×10^{-4} moles NADH produced per min). Two series of measurements were made: the first with males obtained from axenic sucrose medium (sucrose 70 g, killed yeast 70 g, agar 20 g, Nipagine 6 g, water 1 liter) (four samples assayed twice); the second with males obtained from cornmeal medium (six samples assayed twice). All males were obtained from uncrowded cultures at 25°C. The variation of ADH activity per fly or per mg of fresh weight was analyzed with regard to strains (genetic effect) and medium (environmental effect) using analysis of variance.

Table 1 gives the ADH activity for the three strains (H, HF, L) from the two media. The results of the two analyses of variance are given in Table 2.

A strain effect is observed for ADH activity per fly as well as per mg of fresh weight. However, considering only the L and HF strains (i.e., the 2 strains monomorphic for the Adh^F allele), the results are the following: the HF strain displays a greater ADH activity per fly for both media (HF vs L: sucrose medium, t=3.81; p<0.001 - cornmeal medium, t=5.45; p<0.001). While no difference is evident when ADH activity is expressed per unit weight (HF vs L: sucrose medium, t=1.19; ns - cornmeal medium, t=0.01; ns).

For the medium effect, ADH activity per fly as per unit weight is greater for males developing from larvae fed with cornmeal medium. In addition, the cornmeal medium induces a greater weight (Tables 1, 2), but this weight increase due to the cornmeal medium is significant only for the L strain. As a matter of fact, the weight variation in this strain between the 2 media is associated with variation of ADH activity per unit weight.

In regard to the relationship between body weight and ADH activity per unit weight, two cases must be distinguished: either environmental variation of weight, or genetic variation of weight.

In the former case, we can classify the experiments of Clarke et al. (1979), Van Dijk (1981), and Van Delden and Kamping (1985) who observed an increase in ADH activity per unit weight as body weight increased; the weight variation was obtained through varying levels of crowding or amounts of yeast. In our experiment, we observed the same relationship for the L strain but used two different larval media, without crowding. However, the H and HF strains, the weights of which vary only slightly between the 2 media, show a significant variation of ADH activity from one medium to the other (Table 1). Such a relationship between ADH activity and nutritive factors have been described previously (Gibson 1970; Schwartz & Sofer 1976; Papel et al. 1979; McKechnie & Geer 1984).

In the latter case (genetic variation of weight), we observed no difference in ADH activity per unit weight between our 2 strains of extreme weight, in contrast to Van Delden & Kamping (1983), whose strain showed less difference in weight. These authors question whether there has been selection for body weight with a concomitant increase in ADH activity, or vice versa, or an interaction of both selective forces. For our part, this argument does not hold true if we express the ADH activity per unit weight. As for Anderson & Gibson (1985), they do not observe a correlation between weight and ADH activity per unit weight in six out of seven Australian populations. The importance of taking body weight into account when measuring ADH activity has been contested on two previous occasions. Birley & Marson (1981) have argued that all evidence suggests that genotype-environment interaction in ADH activity is independent of genotype-environment interaction for body weight. In this case, genetic variation in ADH activity would

Table 1. Mean weight per male (w), ADH activity per male and per mg of fresh weight for a total of 4 samples of 15 males from sucrose medium and 6 samples of 15 males from cornmeal medium. Standard errors are in parentheses.

	Sucrose medium			Cornmeal medium		
Strains:	H	HF	L	H	HF	L
w (mg):	1.07	1.00	0.70	1.09	1.03	0.79
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
ADH activity per male:	6.66	6.93	5.21	7.65	8.47	6.46
	(0.08)	(0.21)	(0.11)	(0.47)	(0.21)	(0.19)
ADH activity per mg of fresh weight:	6.21	6.90	7.46	7.03	8.21	8.21
	(0.07)	(0.19)	(0.09)	(0.47)	(0.18)	(0.27)

Table 2. Analysis of variance of ADH activity and body weight.

Source of variation	df	mean square	F	P
a. ADH activity/male:				
Mediums	1	11.52	28.35	<0.001
Strains	2	9.34	23.00	<0.001
M x S	2	0.16	0.39	n.s.
Error	24	0.41		
b. ADH activity/mg of fresh weight				
Mediums	1	6.62	15.41	<0.001
Strains	2	4.15	9.67	<0.001
M x S	2	0.22	0.51	n.s.
Error	24	0.43		
c. Body weight of males:				
Mediums	1	0.0150	30.00	<0.001
Strains	2	0.3089	617.80	<0.001
M x S	2	0.0034	6.80	<0.01
Error	24	0.0005		

merely reflect non-allelic gene action of body weight loci. This point of view is supported by our results since we observed a significant interaction between strains and medium for body weight but not for ADH activity. As for Maroni et al. (1982), their results, obtained with transferred chromosome lines, allow them to deem inappropriate the ratio ADH activity/body weight to obtain units per mg live weight. Such a ratio inflates the variance of results unnecessarily.

In this experiment, we have analysed simultaneously the variation of the ratio ADH activity/body weight when the body weight varied both genetically and environmentally. From our results, it follows that only the environmental variation of weight modified this ratio.

The whole of these observations points to the necessity for carefully controlled culture conditions when a determination of genetical variation in ADH activity is required, but undermines the requirement to express the ADH activity with respect to fresh weight.

References: Anderson, D.G. & J.B. Gibson 1985, Genetica 67:13-19; Birley, A.J. & A. Marson 1981, Heredity 46:427-441; Clarke, B., R.G. Camfield, A.M. Galvin & C.R. Pitts 1979, Nature 280:517-518; Gibson, J. 1970, Nature 227:959-961; Higuet, D. 1986, Evolution 40:272-278; Kamping, A. & W. van Delden 1978, Bioch. Genet. 16:541-551; Lewis, N. & J. Gibson 1978, Bioch. Genet. 16:159-170; Maroni, G., C.C. Laurie-Ahlberg, D.A. Adams & A.N. Wilton 1982, Genetics 101:431-446; McDonald, J.F., S.M. Anderson & M. Santos 1980, Genetics 95:1013-1022; McKechnie, S.W. & B.W. Geer 1984, Insect Biochem. 14:231-242; Papel, I., M. Henderson, J. Van Herrewege, J. David & W. Sofer 1979, Bioch. Genet. 17:553-564; Schwartz, M. & W. Sofer 1976, Nature 263:129-131; Van Delden, W. & A. Kamping 1980, Genetica 51:179-185; ⸻ & ⸻ 1983, Ent. Exp. Appl. 33:97-102; ⸻ & ⸻ 1985, DIS 61:178; Van Dijk, H. 1981, DIS 56:150-151.
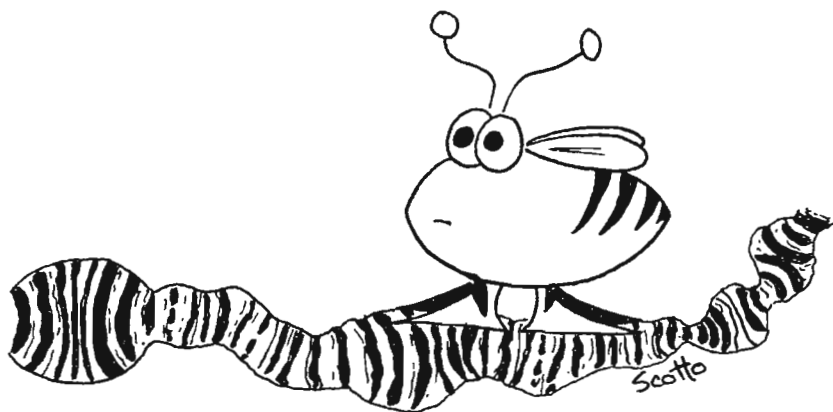
Mglinetz, V.A. and V.I. Ivanov. Dept. of Develop-
mental Genetics, Institute of Medical Genetics,
Moscow, USSR. Abdominal segment 7 in Drosophila
males following etherization of early embryos.

In contrast to females (having 7 abdominal segments),
adult **D.melanogaster** males have only 6. The 6th
segment in males is larger than all the preceding ones
and is equipped with two pairs of tracheas. So it was
considered to originate by fusion of the initial seg-
ments 6 and 7 (Ferris 1950). Following etherization
of embryos at 28°C (30 min) in imago males, well expressed mesothoracic transformation of metathorax
was usually accompanied by development of unilateral or bilateral signs of the 7th segment.

Poorly developed 7th segments were usually represented by three pigmented cuticle islands bearing
bristle patches. Two of the islands were located on dorsal surface of membranous tissue posterior to the
6th segment and the third one was located adjacent to the lower spiracle (trachea). The anterior medial
island produced the anterior part of the 7th tergite and the posterior lateral one produced the hind part
of the tergite, while the island adjacent to trachea produced the upper angle of the tergite. The 7th tergite
development was accompanied by the 7th sternite bearing 2 to 12 bristles. In this case the origin of the
7th tergite may be schematically presented as: Anterior and posterior dorsal nests of the histoblasts give
rise to the anterior and posterior compartments of the 7th tergite. Anterior ventral histoblast nest seems
to produce the spiracle and the tergite angle. The posterior ventral nest produces the 7th sternite.

We could not find any signs of division of the 6th segments in males into the 6th and 7th segments;
the latter seems to be simply absent in adult males, in spite of similar distribution of the four nests of his-
toblasts in the male and female larvae (Roseland 1976). It seems that in the males these anlagen stay in
the males in latent dormant condition taking no part in imaginal development, as is the case for the genital
disc (Epper & Nothiger 1982; Miglinetz & Tuguschi 1984). It may be that the etherization of embryos inter-
feres with the normal suppression of the histoblast nest.

References: Epper, F. & R. Nothiger 1982, Devel. Biol. 94:163; Ferris, G.F. 1950, in: "Biology of Drosophila"
(M. Demerec, ed.), J.Wiley, NY, pp368-419; Mglinetz, V.A. & M.G. Tugushi 1984, Ontogenez (Soviet J. Devel. Biol.)
15:486; Roseland, C.R., PhD thesis, U of California-Irvine.

Figures. Induction of bx-phenocopies (MT --- MS)
transformations and 7th abdominal segment development
(7T) in **D.melanogaster** males by ether treatment.
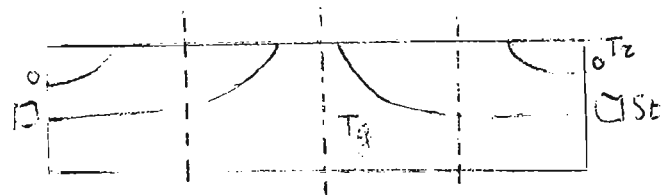
Miglani, G.S. and V. Mohindra. Punjab Agricultural University, Ludhiana, India. Genotypic verification of reccombinants induced with physical stress in males of **D.melanogaster.**

Physical stress. After 54 hr of egg deposition, the developing F_1 (Oregon K $\pm$) /dumpy (dp) black (b) cinnabar (cn) larvae were flushed out with water and then physically transferred with a camel-hair brush onto the standard food medium. After allowing the larvae to feed there for 32 hr, they were again flushed out and transferred onto the standard food medium. Each larva thus underwent two physical transfers during its lifetime which has been referred to as "physical stress" in this report. A control experiment was run simultaneously where transfers of larvae were not done. A two-day old F_1 male was crossed with 3 dp b cn females to get first test cross progeny (TC_1). Each of the TC_1 recombinants was again test crossed to obtain second test cross progeny (TC_2). A TC_1 recombinant was considered as genotypically verified if it produced recombinant type flies in addition to dp b cn type in TC_2 progeny. All the experiments were done at 25±1°C.

Induction of recombination in males. Out of 14 and 15 F_1 males randomly selected from control and physical stress experiments, 5 (35.7%) and 12 (80.0%) individuals, yielding progenies of 3475 and 5952, respectively, produced a total of 9 and 67 recombinants (Table 1). In all the experiments, the most prevalent male recombinant was + b cn. Non-reciprocal recombination was observed only in dp-b region. The number of + b cn male recombinants was strikingly higher than that of + b cn female recombinants. The overall percent recombinants (males plus females) in TC_1 progenies of control (0.267%) and physical stress (1.125%) experiments were significantly different from each other (p<0.0001).

Genotypic verification of recombinants. The percentages of TC_1 male recombinants verified in control (50%) and physical stress (62.5%) experiments differed significantly (p<0.01). The only class of male recombinants, all members of which were not verified was + b cn. Irrespective of the phenotype, almost all the TC_1 female recombinants further test crossed were verified. About those + b cn male recombinants that were not verified, it may be suggested that these individuals may not genotypically be + b cn, but they may have looked like + b cn flies because of certain developmental modifications.

Cytoplasmic influence. More than 90% of the TC_1 + b cn male recombinants, verified in TC_2 generation, did not produce + b cn and dp b cn flies in 1:1 ratio (Table 2). In fact, in an overwhelming majority (100% in control and 90.7% in physical stress experiments) of TC_1 + b cn male recombinants, recovery of recombinant type flies was below 15% in TC_2. However, almost all the TC_1 + b cn female recombinants verified, produced the two phenotypes in equal frequency (Table 2). In these reciprocal crosses, differences were noted with regard to proportion of recombinant type flies in TC_2. This suggests that cytoplasm may be playing some role in transmission of + b cn phenotype from male.

Table 1. Number of second chromosome recombinants recovered (R) in **D.melanogaster**, number of recombinants further test crossed (T) and verified (V).

	Control	Physical stress
F_1 males test crossed	14	15
Pooled test cross population size	3475	5952
F_1 males yielding recombinants	5	12
Recombinants recovered	9	67
Percent recombinants	0.267	1.125
Male recombinants	R – T – V	R – T – V
+ b cn	5 – 5 – 1	46 –37 –22
dp + +	3 – 3 – 3	2 – 2 – 2
dp b +	0 – 0 – 0	1 – 1 – 1
Total:	8 – 8 – 4	49 –40 –25
Female recombinants		
+ b cn	1 – 1 – 1	10 –1 – 1
dp + +	0 – 0 – 0	6 – 0 – 0
dp b +	0 – 0 – 0	2 – 0 – 0
Total:	1 – 1 – 1	18 –1 – 1

Table 2. Testing of TC_2 progenies of verified **D.melanogaster** TC_1 male recombinants for 1:1 ratio.

TC_1 phenotype	No. of TC_1 recomb. verified	No. of TC_2 progenies showing recombinants		
		= dp b cn	<dp b cn	>dp b cn
Control				
Males				
+ b cn	1	0	1	0
dp + +	3	2	1	0
Females				
+ b cn	1	1	0	0
Physical Stress				
Males				
+ b cn	22	1	20	1
dp + +	2	1	0	1
dp b +	1	0	1	0
Females				
+ b cn	1	1	0	0

Mittler, S. Northern Illinois University, DeKalb. Failure of food dyes to increase wing spots in somatic and recombination test in Drosophila.

The seven food dyes currently in use in the United States were tested for mutagencicity and recombinogenic activity using a somatic mutation and recombination test developed by Graf et al. (1984). The food dyes tested were FDC Red 3, FDC Red 40, FDC Blue 1, FDC Blue 2, FDC Green 3, FDC Yellow 5 and FDC Yellow 6. The female flr^3/TM3 Ser were mated to mwh/mwh and offspring reared from egg to adult on usually 4 to 5 different concentrations of the food dye mixed with 1 gram of Carolina finely ground 4-24 fly medium plus 4 ml of H_2O. The highest concentrations of dye that would permit growth were employed. At least 300 wings per concentration were examined for single spots usually clones of mwh and twin spots

Table 1.

Food Dye	Concentrations (g)	Results
FDC Red No. 3	0.005, 0.015, 0.025, 0.035, 0.45	Negative
FDC Red No. 40	0.1, 0.2, 0.3, 0.4, 0.5	Negative
FDC Blue No. 1	0.1, 0.2, 0.3, 0.4, 0.5	Negative
FDC Blue No. 2	0.1, 0.2, 0.3, 0.4, 0.5	Negative
FDC Green No. 3	0.05, 0.1, 0.2, 0.3	Negative
FDC Yellow No. 5	0.1, 0.2, 0.3, 0.4	Negative
FDC Yellow No. 6	0.25, 0.4, 0.6, 0.8	Negative

of mwh and flr^3 which represent induction of mutations and crossing over in the larvae wing anloge. All of the food dyes tested were negative when compared to controls.

In an attempt to increase the sensitivity of SMART, somatic mutation and recombination test, the larvae when they had fed on test media for two days were then subjected to 38°C for 1 hr and then permitted to feed until pupation. The heat shock did not increase significantly the wing spots in adults who as larvae were fed FDC Red no. 4 and FDC Blue No. 1 and FDC Blue No.2.

References: Graf, U. et al. 1984, Envir. Mutagen. 6:119-188.

Mittler, S. Northern Illinois University, DeKalb. The failure of video display terminals to induce somatic mutation and recombination.

Utilizing the somatic mutation and recombination test as described by Graf et al. (1984), larvae mwh/flr^3 Ser were exposed to video display terminals to determine whether these terminals induced mutations and recombination. Female flr^3/TM3 Ser mated to mwh were permitted to lay eggs for 24 hr in vials with cornmeal-molasses-agar media. These vials were taped on the front and back of Corona Data Systems, Inc., Model No. MT 14KS and IBM Displaywriter System Model 001 which were kept running for 4 days and the mwh/flr^3 adult wings were examined for induced wing spots. There was no significant increase by means of a 2x2 contingency table analysis in wing spots as a result of exposure to the front and back of video display terminals.

Table 1. Induction of wing spots in mwh/flr^3 after 4 day exposure to video display terminals.

Control	Total wings	Wings without spots	Twin spots	mwh <3	mwh >3
Control	120	107	1	7	6
IBM (Back)	200	183	3	12	5
IBM (Front)	280	264	0	8	8
Corona (Back)	260	230	3	23	7
Corona (Front)	240	227	0	10	3

There was a slight but not significant increase in small wing spots when the larvae were exposed to backs of the video display units where the temperature was a degree higher. Recently, Graf (1986) reported a temperature effect upon increase of small spots.

References: Graf, U. 1986, DIS 63:65; ——— et al. 1984, Envir. Mutagen. 6:119-188.

Molina, V., F. Gonzalez-Candelas and J.L. Mensua. Universidad de Valencia, Spain. Density- and frequency-dependent selection in **Drosophila melanogaster.**

Larval competition for food in Drosophila is a very important phenomenon in detecting frequency-dependent selection (Lewontin 1955). In order to detect frequency-dependent selection and density-dependent selection, and to check the relation between both types of selection, the following experiment was carried out:

Two strains of **Drosophila melanogaster** were used: Oregon-R (Or-R) and cardinal (cd, 3, 75.7). Larvae of both strains were seeded with the following genotype frequencies: 0.96/0.04, 0.84/0.16, 0.64/0.36, 0.36/0.64, 0.16/0.84 and 0.04/0.96. Two kinds of vials were used: large vial (7 x 1.3 ml) and small vial (5 x 0.8 ml). Three different amounts of food (Lewis' medium) were employed: 0.50, 0.75 and

Table 1. ANOVA for the angular transformation of viabilities.

Source of variation		df	cardinal strain MS	F	Oregon strain MS	F
Genotype frequency	(1)	5	1540.1	13.2 ***	492.2	4.3 ***
Density	(2)	4	48971.8	419.0 ***	29433.6	213.4 ***
Amount of food	(3)	2	4240.1	36.3 ***	5155.9	37.4 ***
Vial	(4)	1	2725.3	23.3 ***	37.4	0.3 n.s.
1 x 2		20	63.5	0.5 n.s.	180.7	1.3 n.s.
1 x 3		10	47.4	0.4 n.s.	38.3	0.3 n.s.
1 x 4		5	33.0	0.3 n.s.	69.7	0.5 n.s.
2 x 3		8	335.4	2.9 *	1269.7	9.2 ***
2 x 4		4	330.8	2.8 *	345.4	2.5 *
3 x 4		2	265.0	2.3 n.s.	744.9	5.4 **
1 x 2 x 3		40	45.2	0.4 n.s.	90.6	0.7 n.s.
1 x 2 x 4		20	51.6	0.4 n.s.	33.1	0.2 n.s.
1 x 3 x 4		10	35.1	0.3 n.s.	22.8	0.2 n.s.
2 x 3 x 4		8	86.5	0.7 n.s.	359.8	2.6 **
1 x 2 x 3 x 4		40	26.4	0.2 n.s.	121.5	0.9 n.s.
Error		360	116.9		137.9	
Total		539				

n.s. = not significant; * = $p \le 0.05$; ** = $p \le 0.01$; *** = $p \le 0.001$.

Table 2. ANOVA for the mean developmental time.

Source of variation		df	cardinal strain MS	F	Oregon strain MS	F
Genotype freuqency	(1)	5	4.7	2.0 n.s.	12.2	6.9 ***
Density	(2)	4	91.8	39.4 ***	129.3	73.0 ***
Amount of food	(3)	2	112.1	48.1 ***	116.7	65.9 ***
Vial	(4)	1	225.4	96.7 ***	216.6	122.3 ***
1 x 2		20	2.3	1.0 n.s.	1.7	1.0 n.s.
1 x 3		10	2.7	1.2 n.s.	1.4	0.8 n.s.
1 x 4		5	1.8	0.8 n.s.	2.3	1.3 n.s.
2 x 3		8	30.0	12.9 ***	14.1	7.9 ***
2 x 4		4	10.1	4.3 **	8.1	4.6 **
3 x 4		2	14.4	6.2 **	38.5	21.8 ***
1 x 2 x 3		40	2.4	1.0 n.s.	1.4	0.8 n.s.
1 x 2 x 4		20	3.7	1.6 n.s.	1.2	0.7 n.s.
1 x 3 x 4		10	3.0	1.3 n.s.	0.5	0.3 n.s.
2 x 3 x 4		8	10.4	4.5 ***	9.8	5.5 ***
1 x 2 x 3 x 4		40	3.0	1.3 n.s.	0.7	0.4 n.s.
Error		360	2.3		1.8	
Total		539				

n.s. = not significant; ** = $p \le 0.01$; *** = $p \le 0.001$.

1.00 ml. In each case, different amounts of larvae were seeded: 25, 50, 100, 150 and 200. A total of 180 (6 x 2 x 3 x 5) different cases were tested. In each case several repetitions were made, ranging from four to twelve. The parameters of fitness studied were viability and mean developmental time (MDT).

Table 1 shows the ANOVA of four factors (genotype frequency, larval density, amount of food and type of vial) for the angular transformation of the viabilities of cardinal and Oregon strains. Table 2 shows the ANOVA of four factors for the MDT.

With regard to viability it can be observed that the individual factors are significant in both strains, except for the type of vial in Oregon strain. Therefore, it can be said that both genotype frequency and larval density affect the viability of both strains.

The significant interactions are the same in both strains, except for the amount of food x type of vial and the density x amount of food x type of vial, which are significant in the Oregon strain but not in the cardinal one. This may reflect that the spatial disposition of food affects more the Oregon strain than the cardinal strain. This may be due to the fact that cardinal flies eat significantly less food than other normal strains of **Drosophila melanogaster** (Castro et al. 1986), and, thus the cardinal strain is less influenced by the spatial disposition of food.

It can also be observed that all the interactions in which appear simultaneously genotype frequency and density are not significant. This indicates that the variation of the viability with genotype frequency is the same in all the larval densities, and the increase of density only affects viability as a whole. Therefore, we think that independence between frequency-dependent selection and density-dependent selection exists, in opposition to some authors (De Benedictis 1977; Tosic & Ayala 1981).

In relation to the MDT, it can be observed that four individual factors are significant for the Oregon strain. In the cardinal strain the genotype frequency factor is not significant, and thus, cardinal strain does not present frequency-dependent selection in relation to the MDT. The other three factors are significant, and therefore, the larval density, the amount of food and the type of vial affect to the MDT of both strains.

The significant interactions are the same in both strains. This indicates that spatial disposition of food affects the MDT in both strains.

All the interactions in which genotype frequency and density appear simultaneously are not significant, as it occurred with the viability, supporting the hypothesis of independence between both types of selection.

References: Castro, J.A., L.M. Botella & J.L. Mensua 1986, Arch. Insect. Biochem. Physiol. 3:485-497; DeBenedictis, P.A. 1977, Genetics 87:343-356; Lewontin, R.C. 1955, Evolution 9:27-41; Tosic, M. & F.J. Ayala 1981, Genetics 97:697-701.

Molina, F., F. Gonzalez-Candelas and J.L. Mensua. Universidad de Valencia, Spain. Effect of the spatial disposition of food in larval competition of **D.melanogaster**.

Different models justifying frequency-dependent responses exist. Among these models there are some which use specific environmental and genetic parameters (DeJong 1976; Wallace 1981; Nunney 1983). These models show the importance of different factors such as number of food particles, time that one larva needs to eat a certain number of food particles, amount of food necessary for one larva to reach pupation, intrinsic viability and number of biological unit spaces.

None of these models refers to another factor: spatial disposition of food. The following experience was carried out to demonstrate the importance of this factor:

Two strains of **Drosophila melanogaster** were used: Oregon-R (OrR) and cardinal (cd: 3,75.7). Two kinds of vials were used: large vials (7 x 1.3 cm) and small vials (5 x 0.8 cm). Different amounts of larvae were seeded so that each larva had the same amount of food (Lewis' medium). We designate the ratio: "ml of food/number of larvae seeded" as level of competition. Two cases were studied: (1) 0.50/50 and 1.00/100, and (2) 0.50/100, 0.75/150 and 1.00/200.

The di-genotype cultures had the following genotype frequencies: 0.96/0.04, 0.84/0.16, 0.64/0.36, 0.36/0.64, 0.16/0.84 and 0.04/0.96. In each case the total viability of di-culture and the mean developmental time (MDT) were calculated.

Table 1 shows the ANOVA of three factors (genetic composition, level of competition and type of vial) for the angular transformation of viabilities. Table 2 shows the ANOVA for the MDT.

The level of competition is significative in three of the four analyses; this result coincides with the results of Mather & Caligari (1981). They found that there is not a single relation between level of food and viability, since it cannot be affirmed that a given number of eggs in a certain amount of food is necessarily equal to the half of eggs in the half of food. In our work, we have shown that viability decreases and MDT increases when the number of larvae increases, for the same amount of food per larva.

In any case, significant differences in the interaction level of competition x type of vial have also been detected. The same amount of food will have more surface available for the larvae in the large vial than in the small one, and thus, this amount of food will be more easily available for the larvae. But it can also occur that the desiccation of medium be higher in the large vial than in the small one. Therefore, in this case, the viability will be higher in the small vial than in the large one.

References: DeJong, G. 1976, Am. Nat. 112:155-175; Mather, K. & P.D.S. Caligari 1981, Heredity 46:239-254; Nunney, L. 1983, Am. Nat. 121:67-93; Wallace, B. 1981, Basic Population Genetics, Columbia U. Pr, New York.

Table 1. ANOVA for the angular transformation of viabilities.

Source of variation		df	MS	F
Case 1:				
Genetic composition	(1)	5	7.684	0.062 n.s.
Level of competition	(2)	1	545.406	4.379 *
Vial	(3)	1	539.250	4.329 *
1 x 2		5	12.063	0.097 n.s.
1 x 3		5	19.894	0.160 n.s.
2 x 3		1	746.031	5.989 *
1 x 2 x 3		5	6.613	0.053 n.s.
Error		48	124.562	
Total		71		
Case 2:				
Genetic composition	(1)	5	20.772	0.533 n.s.
Level of competition	(2)	2	50.997	1.309 n.s.
Vial	(3)	1	180.359	4.632 *
1 x 2		10	30.842	0.792 n.s.
1 x 3		5	55.266	1.419 n.s.
2 x 3		2	111.629	2.867 n.s.
1 x 2 x 3		10	18.241	0.468 n.s.
Error		72	38.939	
Total		107		

n.s.= not significant; *=$p \leq 0.05$.

Table 2. ANOVA for the mean developmental times.

Source of variation		df	MS	F
Case 1:				
Genetic composition	(1)	5	1.285	0.575 n.s.
Level of competition	(2)	1	35.238	15.774 **
Vial	(3)	1	64.808	29.010 ***
1 x 2		5	0.033	0.015 n.s.
1 x 3		5	0.375	0.168 n.s.
2 x 3		1	0.699	0.313 n.s.
1 x 2 x 3		5	0.167	0.075 n.s.
Error		48	2.234	
Total		71		
Case 2:				
Genetic composition	(1)	5	1.268	0.860 n.s.
Level of competition	(2)	2	48.867	33.040 ***
Vial	(3)	1	50.121	34.013 ***
1 x 2		10	0.427	0.290 n.s.
1 x 3		5	0.548	0.372 n.s.
2 x 3		2	21.641	14.686 ***
1 x 2 x 3		10	0.487	0.330 n.s.
Error		72		
Total		107		

n.s.=not significant; **=$p \leq 0.01$; ***=$p \leq 0.001$.

Molto, M.D., R.de Frutos and M.J. Martinez-Sebastian. Universidad de Valencia, Spain. Characteristic puffing patterns of **Drosophila guanche.**

Puffing activity patterns of the five large polytene chromosomes of **Drosophila guanche** were studied during the late third-instar larvae and through the prepupal period. A total of 151 loci, active in some of the seventeen developmental stages studied, were described. The distribution of these active loci per chromosome is the following: 21 on chromosome A, 30 on chromosome J, 25 on chromosome U, 39 on chromosome E and 36 on chromosome O.

All puffs described in **D.guanche** did not show the same level of activity. The group of puffs which appear at a frequency equal or superior to 75%, in at least one of the stages studied, has been taken as the characteristic puffing pattern of this species. A total of 23 loci (15% of the puffs described in this species) form the characteristic puffing pattern of **D.guanche.** These loci have been grouped into four types according to their moments of highest activity during development (Figure 1): (1) Third-instar larvae puffs: 4A, 13BC and 60AB. (2) Beginning of prepupation puffs: 35AB, 50D, 51D, 52AC, 57BC, 54E-55A, 60AB, 86A, 85AB and 98C. (3) Prepupation puffs: 4A, 16B, 18C, 41BD, 47BD, 61AC, 69B, 68DE and 97DE. (4) End of prepupation puffs: 26, 60AB, 90A, 97DE and 99BC.
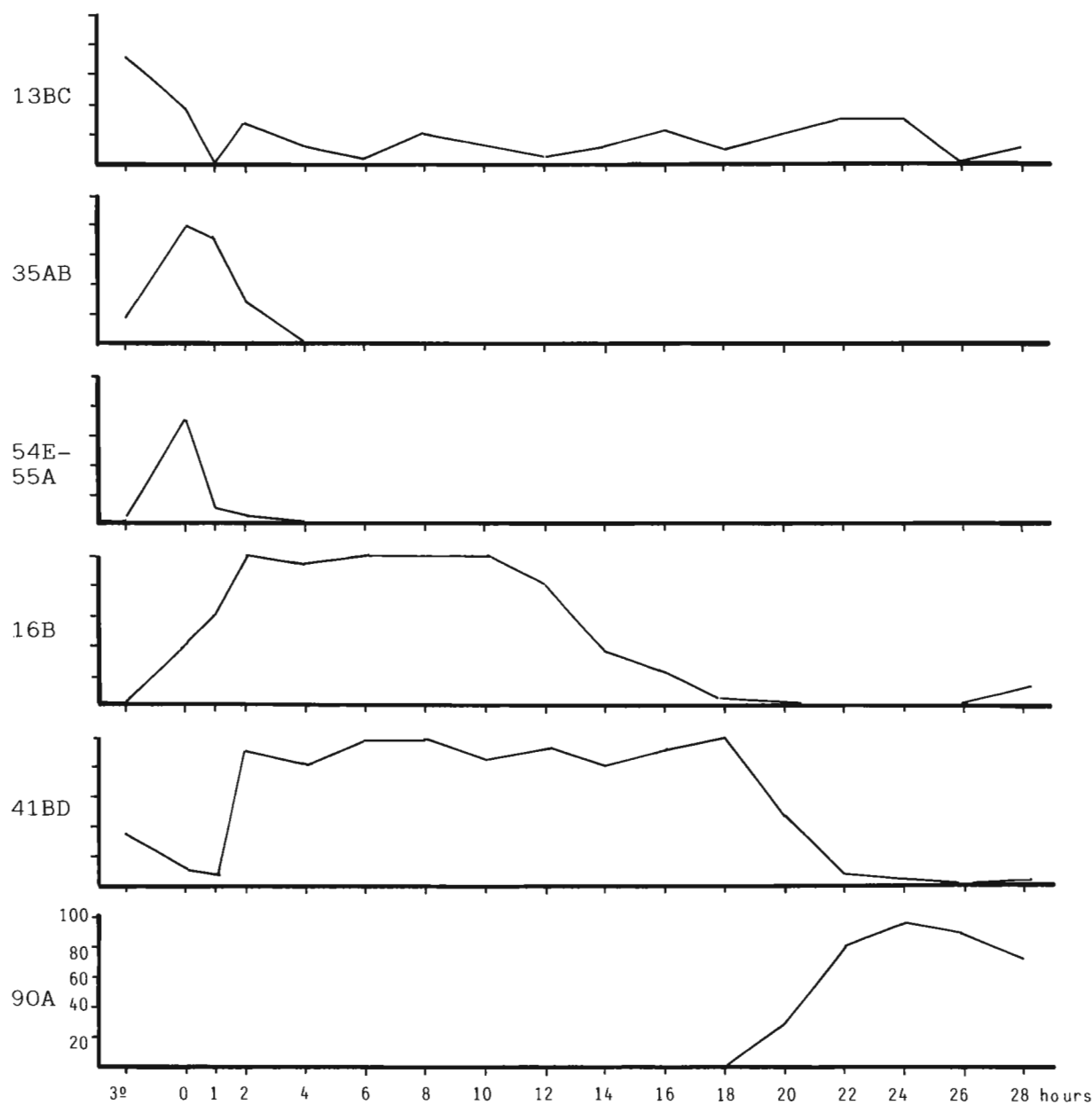
Figure 1. Behaviour during development of some puffs which form the characteristic pattern of **D.guanche.** The time of study and the degree of expression of these puffs are given on the abscissa and the ordinate, respectively.

Mutsuddi, D., M. Mutsuddi (Das) and A.K. Duttagupta. University of Calcutta, India.
The effect of a sex-specific lethal gene daughterless (da) on the X chromosome of **D.melanogaster.**

In **D.melanogaster**, the 2nd chromosome gene daughterless (da; Bell 1954; location 2-41.5, Cline 1978) is a temperature senstive, sex-specific lethal mutation. At a temperature of 22°C or above, da/da mothers leave behind only male progeny (XY or XO) because their eggs cannot support female (XX) development (Sandler 1972; Cline 1976, 1978). At 18°C, about 25% female progeny did survive the da maternal effect. These "escaper" females, however, display "morphological abnormalities in a variety of adult cuticular structure, characteristic of cell death during development" (Cline 1976). This temperature sensitivity of the da phenotype indicates that "the mutation has rendered the da^+ product thermally unstable" (Cline 1978).

Subsequently, it was proposed that the da locus affects the viability of the female progeny by regulating the X chromosomal transcription through Sx1 locus (location 1-19, Cline 1978; Lucchesi & Skripski 1981). The $Sx1^+$ product was stated to be essential for viability in females, but is lethal in males. Activation of $Sx1^+$ locus (through $Sx1^M$ mutation) would reduce the X chromosomal transcription rate in males (XY) and its inactivation (through da or $Sx1^f$ mutations) would make both the X chromosomes hyperactive in females (XX) (Lucchesi & Skripski 1981). As all previous works point to a concomitant association between X chromosomal transcription rate and its chromatin condensation, the morphology of the X chromosome in daughters of da/da mothers was examined at both permissive (18°C) and restrictive (20°C and 22°C) temperature. Furthermore, as the activation of the Sxl locus by the da^+ maternal factor also depends on the X/A ratio, the effect of da on different genotypes with intermediate X/A ratios (between .5 and 1) was also examined.

For the study of X chromosomes in females of da/da mothers, da/da female virgins (stock obtained through the kind courtesy of Dr. T.W. Cline) were crossed to wild type Oregon R^+ males and were maintained at 18°C, 20°C and 22°C. For cytological purposes, salivary gland chromosomes from third instar larvae were prepared following usual techniques (Das et al. 1982). At 18°C, female larvae from da/da mothers were recovered and were subjected to cytological observations. As evident from a representative photomicrograph (Figure 1), the X chromosome of such "escaper" females represents somewhat wider diameter than the paired autosomal regions of the same complement. Interestingly, the distal tip of the X chromosome appears to be the most affected region and shows a large swelling (Figure 1). This observation indicates that even at 18°C, the da product has a considerable influence on the X chromosome of "escaper" females. However, at both 20°C and 22°C, not a single female larvas from da/da mothers was recovered.

To determine the effect of da on different X chromosomal segmental aneuploids between 1X and 2X, da/da female virgins were crossed to males of any one of the following X;Y translocation stocks (obtained through the kind courtesy of Dr. J.R. Merriam), viz. B29(3E), J8(8C), B44(11A) and B10(15EF), and were maintained at 22°C. For control experiments, yellow (y) virgin females were crossed to males of the aforesaid translocation stocks and were maintained at the same temperature. Four types of progeny were expected from control experiment: (i) euploid males, (ii) euploid females, (iii) aneuploids with distal duplications, and (iv) aneuploids with proximal duplications (Maroni & Lucchesi 1980; Mutsuddi et al. 1983). Among these four classes, aneuploids with distal duplications does not appear in culture. However, aneuploids with proximal duplications survived in the larval stage, and in such larvae with up to 1.62 X
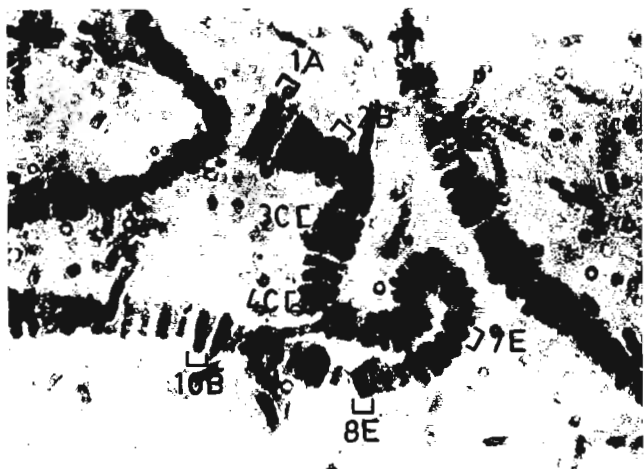
Figure 1. Photomicrograph depicting morphology of the X chromosome (labelled one) in daughters of da/da mothers at 18°C. Note that the distalmost tip of the X chromosome is remarkably dilated and does not have the typical configuration of that of the normal female-X chromosome.

chromosomal segments (duplications from 8D-20F), each part of the X chromosome shows all the characteristics of a typical male-X chromosome. In contrast, in aneuploid larvae with 85% proximal duplication (3E-20F), both the homologues behave like a typical female-X chromosome (Mutsuddi et al. 1983).

However, among the progeny of da/da mothers, aneuploid larvae with proximal duplications would survive if they can escape the lethal effect of da maternal factor. As evident from our experimental observations, 1X individuals with up to 62% proximal duplications did survive the da maternal effect, and each part of the X chromosome, once again, displayed all the characteristics of a typical male-X chromosome. In contrast, da maternal factor did not support the development of individuals with 85% proximal duplications, indicating that da factor only can act on progenies having the X/A ratio above 0.8.

References. Bell, A.E. 1954, Genetics 39:958-959; Cline, T.W. 1976, Genetics 84:723-742; ——— 1978, Genetics 90:683-698; Das, M., D. Mutsuddi, A.K. Duttagupta & A.S. Mukherjee 1982, Chromosoma 82:373-388; Lucchesi, J.C & T. Skripski 1981, Chromosoma 82:217-227; Maroni, G. & J.C. Lucchesi 1980, Chromosoma 77:253-261; Mutsuddi, D., M. Mutsuddi, A.K. Duttagupta & A.S. Mukherjee 1983, XV Int. Cong. Genet. Abstr. no. 207:118; Sandler, L. 1972, Genetics 70:261-274.

Mutsuddi, D., M. Mutsuddi (Das), and A.K. Duttagupta. University of Calcutta, India.
Effect of sex-transforming mutants on X chromosomal transcription in **D.melanogaster**.

In **D.melanogaster**, sex determination is under the control of X chromosome/autosome ratio (Bridges 1932); yet a number of mendelian gene mutations are known to change the sex of a fly (Baker & Ridge 1980). Using these sex-transforming mutants, attempts were made to examine the role of altered sexual physiology on dosage compensation (Muller 1950; Komma 1966; Smith & Lucchesi 1969) and X chromosomal replication pattern (Mutsuddi et al. 1985). Information gathered thus far from these experiments resolved that sexual physiology has little role in regulation of these two events. Nevertheless, the activities of a number of X-linked and autosomal genes (Muller 1950; Komma 1966; Smith & Lucchesi 1969) as well as replication pattern of various X chromosomal and autosomal sites (Mutsuddi et al. 1985) were reported to be influenced by these genes. As all earlier experiments on gene activity were based on a few loci on the X chromosome, a detail sitewise analysis of ^{3}H-UR incorporation pattern would provide information regarding the effect of these mutants on transcription over the whole X chromosome. Furthermore, as replication and transcription patterns were proposed to be causally connected, it is of special interest to enquire the transcription pattern of these chromosomal sites, which showed altered replication pattern in changed environmental condition (Mutsuddi et al. 1985). Five mutants, viz., sex-combless (sx), doublesex (dsx), doublesex dominant (dsx[D]), intersex (ix) and transformer-2 (tra-2), were used in the present investigation to examine the X chromosomal transcription (by ^{3}H-UR autoradiography of salivary gland cells) under six genotypic conditions, viz., sx/Y, dsx/dsx;XY, dsx/dsx;XX, dsx[D]/+;XX, ix/ix;XX and tra-2/tra-2;XX.

Table 1. Sitewise analysis of ^{3}H-UR incorporation pattern on the X chromosome in different sex-transforming mutants, showing sites which have remarkable alteration in transcription patterns as compared to those of wild type males (a and b) or females (c to f).

Site	X/A Male	Mutant	Female	Site	X/A Male	Mutant	Female
(a) sx/Y				**(d) (contin.)**			
1A	47	95	60	4DEF	241	120	282
1DEF	230	180	240	5CD	321	230	320
3F	100	62	82	7C	40	90	49
5CD	321	166	320	7D	200	300	190
8ABC	385	264	380	8D	90	250	98
11DEF	160	333	239	9EF	43	160	48
(b) dsx/dsx;XY				10DEF	160	140	239
1A	47	80	60	**(e) ix/ix;XX**			
1DEF	230	289	240	2AB	260	500	250
2EF	160	278	170	2CD	170	770	149
3A	80	135	80	2EF	160	320	170
3B	100	159	108	3A	80	180	80
4DEF	241	305	282	3F	100	20	82
5CD	321	254	320	4BC	173	106	176
(c) dsx/dsx;XX				4DEF	241	87	282
1DEF	230	190	240	5A	107	43	106
2CD	170	90	143	5CD	321	110	320
2EF	160	94	170	7D	200	280	190
4DEF	241	320	282	8D	90	260	98
7A	70	125	79	8E	40	136	50
8ABC	385	230	380	**(f) tra-2/tra-2;XX**			
10BC	140	325	140	1A	47	90	60
10DEF	160	600	239	2CD	170	295	149
(d) dsx[D]/+;XX				4DEF	241	105	282
2CD	170	330	143	5A	107	56	106
3DE	221	130	206	8ABC	385	280	380
--------continued next col.-							

Our results resolved that, in general, the total transcription occurring over the whole X chromosomal in these six genotype differs from that of normal males and females in an insignificant manner (p > 0.05 in each case). These observations strongly attested the notion that sexual physiology played little role in regulation of X-chromosomal gene activity. However, at the same time, reproducible alterations in ^{3}H-uridine uptake capacity on a good measure of sites on the X chromosome in each genotype, as compared to that of normal males and females, were also accounted (Table 1). The most prominent X chromosomal sites showing significant alteration in ^{3}H-UR incorporation pattern in comparison to that of wild type males (in case of sx/Y and dsx/dsx;XY) or females (in case of remaining genotypes) are 1A, 1DEF, 3F, 5CD, 8ABC and 11DEF in sx/Y; 1A, 1DEF, 2EF, 3A, 3B, 4DEF and 5CD in dsx/dsx;XY; 1DEF, 2CD, 2EF, 4DEF, 5CD, 7A, 8ABC, 10BC and 10DEF in dsx/dsx;XX; 2CD, 3DE, 4DEF, 5CD, 7C, 7D, 8D, 9EF and 10DEF in dsx^D/+;XX; 2AB, 2CD, 2EF, 3A, 3F, 4BC, 4DEF, 5A, 5CD, 7D, 8D, 8E in ix/ix;XX; and 1A, 2CD, 4DEF, 5CD and 8ABC in tra-2/tra-2;XX. Interestingly, though in each genotype there are some sites (like 1A in sx/Y, 1A and 4DEF in dsx/dsx;XY, 1DEF in dsx/dsx;XX, 2AB in ix/ix;XX, and 1A and 8ABC in tra-2/tra-2;XX) which show reproducible alterations in both of their replication and transcription patterns, a good measure of sites (like 1DEF, 3F, 5CD, etc., in sx/Y; 1DEF, 2EF, 3A, etc., in dsx/dsx;XY; etc.) show altered transcription pattern without representing any change in their replication patterns. The reverse situation, i.e., sites showing marked alteration in replication pattern without affecting their transcription patterns (like 9A in dsx/dsx;XX, 1DEF and 10A in ix/ix;XX, etc.) have also been encountered (see also Mutsuddi et al. 1985). The present experimental observations, together with those of earlier workers (Muller 1950; Komma 1966; Smith & Lucchesi 1969; Mutsuddi et al. 1985), substantiate the notion that sexual physiology cannot be accounted as a regulatory factor controlling X chromosomal replication and transcription; nevertheless, it can influence the replication and transcription pattern of a number of X chromosomal sites.

References. Baker, B.S. & K.A. Ridge 1980, Genetics 91:383-423; Bridges, C.B. 1932, in: Sex and Internal Secretions, Williams & Williams, pp 55-63; Komma, D.J. 1966, Genetics 54:497-503; Muller, H.J. 1950, Harvey Lecture Series 43:165-229; Mutsuddi, D., M. Mutsuddi & A.K. Duttagupta 1985, Ind. Jrl. Expt. Biol. 23:8-16; Smith, P.D. & J.C. Lucchesi 1969, Genetics 61:607-618.

Mutsuddi(Das), M., D. Mutsuddi and A.K. Duttagupta. University of Calcutta, India. Conservation of the early, moderately late and late replicating sites in three nearctic species of obscura group in Drosophila.

In the polytene system of Drosophila, all the chromosomal arms display their characteristic banding patterns, specific for a species. Analysis of relative distribution of silver grains over the chromosomal subunits (i.e., bands, interbands, puffs, etc.), after ^{3}H-TdR autoradiography, unanimously documents that dark bands take much longer time in terminating their replication than interbands and puffs (Lakhotia & Mukherjee 1970; Das et al. 1982). It is, therefore, of special interest to investigate the relative number of early, moderately late and late replicating sites over different chromosomal arms in related species, as well as the degree of conservation of replication patterns over different chromosomal subunits in the background of evolutionary relationship among different species.

Three nearctic species of obscura group, viz., **D.pseudoobscura, D.persimilis** and **D.miranda,** for their nearly similar chromosome complements [except the C element, which is an autosome (3rd chromosome) in the former two species, and has been transformed into a sex chromosome (X_2) in **D.miranda**] (Sturtevant & Novitski 1941; Patterson & Stone 1952; Das et al. 1982) and almost completely homologus banding patterns (Dobzhansky & Tan 1936; Mutsuddi et al. 1984, 1985; Das 1986) appeared to be ideal for our study. The salivary gland chromosome arms are designated as XL, XR, X_2 or 3rd, 2nd and 4th chromosome. Comparative study of replication patterns of different sites on the XL, XR and X_2 or 3rd chromosome, with respect to those of an autosomal segment, in these three species were performed following the conventional ^{3}H-TdR autoradiographic technique (Das et al. 1982).

Results, as summarized in Table 1, demonstrate that despite the evolutionary divergence and intra-chromosomal rearrangements, the total number of replicating sites on the XL and XR, as well as the percentage of early, moderately late and late replicating sites on each chromosome have almost been maintained in females of these three species. In males, despite that the X chromosomes exhibit faster replicating property and possess a distinctly higher number of early replicating sites (in comparison to that of females), the percentage of each kind of replicating site, once again, is found to be almost constant in these three species (Table 2). The C element (i.e., the X_2 in **D.miranda** and the 3rd chromosome in **D.persimilis** and **D.pseudoobscura**), on the other hand, being extensively rearranged in these three species, is somewhat shorter in length in the latter two species and has about 75 replicating units against 78 units

Table 1. Percentage of ^{3}H-TdR labelling frequencies of early, moderately late and late replicating sites on the XL, XR and X_2/3rd chromosome of **D.pseudoob-scura** (a), **D.persimilis** (b), and **D.miranda** (c) females.

Ch. arm.		Labelling frequencies					Total R.U.
		1 -20%	21-40%	41-60%	61-80%	81-100%	
XL	(a)	19.9	24.9	6.2	21.4	27.6	51
	(b)	19.7	25.5	5.8	21.6	27.4	51
	(c)	15.68	17.66	17.66	21.6	27.4	51
XR	(a)	37.0	25.5	14.2	5.7	17.6	79
	(b)	36.7	25.3	14.0	5.0	19.0	79
	(c)	36.7	24.06	12.66	10.12	16.46	79
X_2 3rd	(a)	45.3	27.6	13.1	8.6	5.4	75
	(b)	45.3	28.0	13.3	8.0	5.4	75
	(c)	33.33	34.61	20.51	6.42	5.13	78

Table 2. Percentage of ^{3}H-TdR labelling frequencies of early, moderately late and late replicating sites on the XL, XR and X_2/3rd chromosome of **D.pseudoob-scura** (a), **D.persimilis** (b), and **D.miranda** (c) males.

Ch. arm.		Labelling frequencies					Total R.U.
		1 -20%	21-40%	41-60%	61-80%	81-100%	
XL	(a)	62.4	16.2	9.6	6.0	5.8	51
	(b)	62.8	15.8	9.8	5.8	5.8	51
	(c)	56.86	15.69	15.69	5.88	5.88	51
XR	(a)	73.4	11.0	10.5	3.8	1.3	79
	(b)	73.4	11.4	10.1	3.8	1.3	79
	(c)	68.36	15.19	12.66	2.53	1.26	79
X_2 3rd	(a)	41.8	31.8	13.0	8.0	5.4	75
	(b)	41.3	32.0	13.3	8.0	5.4	75
	(c)	65.39	24.36	7.69	1.28	1.28	78

in **D.miranda** (Das et al. 1982; Mutsuddi et al. 1985; Das 1986). Data, however, indicate that, like XL and XR, this chromosome also conserves the number of early, moderately late and late replicating sites in females of these three species, and separately in males of the two ancestral species. Interestingly, the C element is the most early replicating element and is the possessor of highest number of early replicating sites among all the chromosomes in females of these three species (Table 1), and males of **D.miranda**, whereas these are less in number on XL and XR in that order (Table 2). Notably, though the XL is shorter in length (about two-thirds) than XR and has only 51 replicating units against 79 units on XR (Das et al. 1982; Mutsuddi et al. 1985), it possesses almost equal number of late replicating sites and terminates the replication almost synchronously with the XR.

A detailed study on the homology of banding patterns in these three species indicates that, with a few exceptions, the homologous sites exhibit a strict conservation of their respective replicating property. Interestingly, while such exceptions are rare between the chromosomes of two sibling species, i.e., **D.pseudoobscura** and **D.persimilis** (viz. 29th and 37th replicating units of XR, 10th and 43rd units of the 3rd chromosome of **D.persimilis** and their corresponding sites of **D.pseudoobscura**, etc.), these are considerably higher between the chromosomes of two more distantly related species, i.e., **D.miranda** and **D.pseudoobscura/D.persimilis** (viz., 6th replicating unit of XL, 5th and 16th units of XR and 10th, 32nd, 56th and 73rd units of X_2 of **D.miranda** and their corresponding sites of the other two species, etc.) (for chromosomal subdivisions, see Das et al. 1982; Mutsuddi et al. 1984, 1985). An extensive cytological study in hybrid chromosomes demonstrates that while all the homologous sites are morphologically identical in these three species, the exceptional sites, which show altered replication behaviour, also represent dissimilar morphology with respect to the staining intensity, dimension of bands, etc.

The present observation, therefore, indicates that the chromosomes which have been involved with frequent structural rearrangements in these three species, might have been little altered with respect to the morphology and replication behaviour in the homologous sites. However, the alterations which have occurred in a small number of sites represents a direct relationship with the phylogenetic divergence of these three species of obscura group.

References. Das, M. 1986, PhD Thesis, C.U.; Das, M., D. Mutsuddi, A.K. Duttagupta & .A.S. Mukherjee 1982, Chromosoma 87:373-388; Dobzhansky, T. & C.C. Tan 1936, Z. indukt. Abstamm.-Vererb.-Lehre 72:89-114; Lakhotia, S.C. & A.S. Mukherjee 1970, J. Cell Biol. 47:18-33; Mutsuddi, M., D. Mutsuddi, A.S. Mukherjee & A.K. Duttagupta 1984, Chromosoma 89:55-62; _____, _____ & A.K. Duttagupta 1985, Ind. Jrl. Expt. Biol. 23:616-624; Patterson, J.I. & W.S. Stone 1952, Evolution in the genus Drosophila, MacMillan Press, NY; Sturtevant, A.H. & E. Novitski 1941, Genetics 26:517-541.
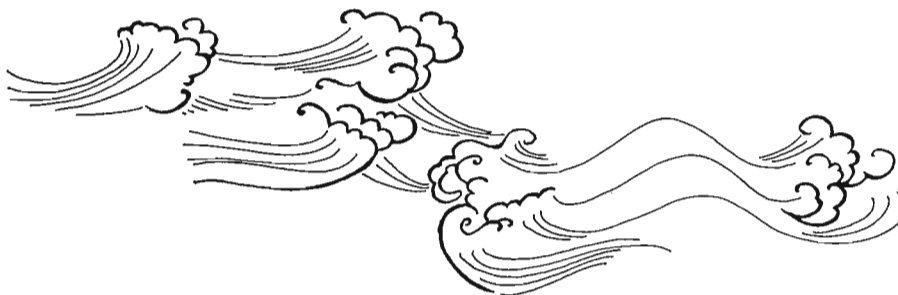
Mutsuddi(Das), M., D. Mutsuddi and A.K. Duttagupta. University of Calcutta,India. Inversion polymorphism of the 3rd chromosome of D.pseudoobscura in the laboratory culture.

The 3rd chromosome (C element) in two sibling species of obscura group, **D.pseudoobscura** and **D.persimilis**, displays a striking example of inversion polymorphism in natural populations. The frequency of such chromosomal rearrangements with respect to seasonal changes were analyzed and was predicted to have some adaptive biological role (Dobzhansky & Sturtevant 1938). An elegant study on gene sequence of the 3rd chromosome in different natural populations made it possible to successfully put different strains of these two species and **D.miranda** in a branching phylogenetic chart (Patterson & Stone 1952). The finding of spontaneous rearranements in gene sequence of the 3rd chromosome in **D.pseudoobscura** in our laboratory culture, in this respect, is exceedingly important and noteworthy.

The stock of **D.pseudoobscura** (obtained from National Drosophila Species Resource Center, Bowling Green, Ohio, USA) was reared in standard Drosophila culture medium at 18°C. Salivary gland chromosomes from mature third instar larvae were prepared following usual technique (Das et al. 1982). In cytological preparations, a large number of nuclei having the 3rd chromosome in the form of inversion heterozygotes (Figure 1a), in addition to the original chromosome sequence (Figure 1b), were observed. Estimation of relative distribution of above two types revealed that the nuclei with inversion heterozygotes, though present in large number, were outnumbered by the number of nuclei with normal chromosome sequence.

A more detailed analysis revealed that in inversion heterozygote condition, the pairing between two homologues of the 3rd chromosome is very rigid and complete; a band to band pairing has given the chromosome a distorted and non-linear configuration (Fig. 1a). An analysis of chromosome sequence, as shown schematically in Figure 2, indicates that at least five separate paracentric inversions in three steps were involved to give rise to the present inverted gene sequence. One of these inversions involved the whole middle part of the chromosome, leaving only the distal and a few proximal segments unaltered (Fig. 2). The remaining inversions might have been originated subsequently within the limit of this inversion (Fig. 2).

From earlier reports, it was evident that the rearrangements in the 3rd chromosome took place mostly at the distal-middle segments (Koller 1939; Patterson& Stone 1952; Strobel et al. 1978), ranging from about 23A to 29B subdivisions of **D.pseudoobscura** (for chromosomal subdivisions, see Mutsuddi et al. 1985) and their corresponding segments in other species; whereas the proximal segments were occasionally rearranged and appeared to be more conserved in nature. In the present case, the inversions extended on both middle-proximal and middle distal segments. The remaining unaltered segments at both ends included those segments (22A-22F and 33D-34F) which were always observed to keep their sequences conserved in all the strains of these two species. It is, therefore, interesting to inquire why some particular gene order of the 3rd chromosome has been strictly maintained in evolution, where the major segments showed frequent changes in their gene order in all the strains of these species.
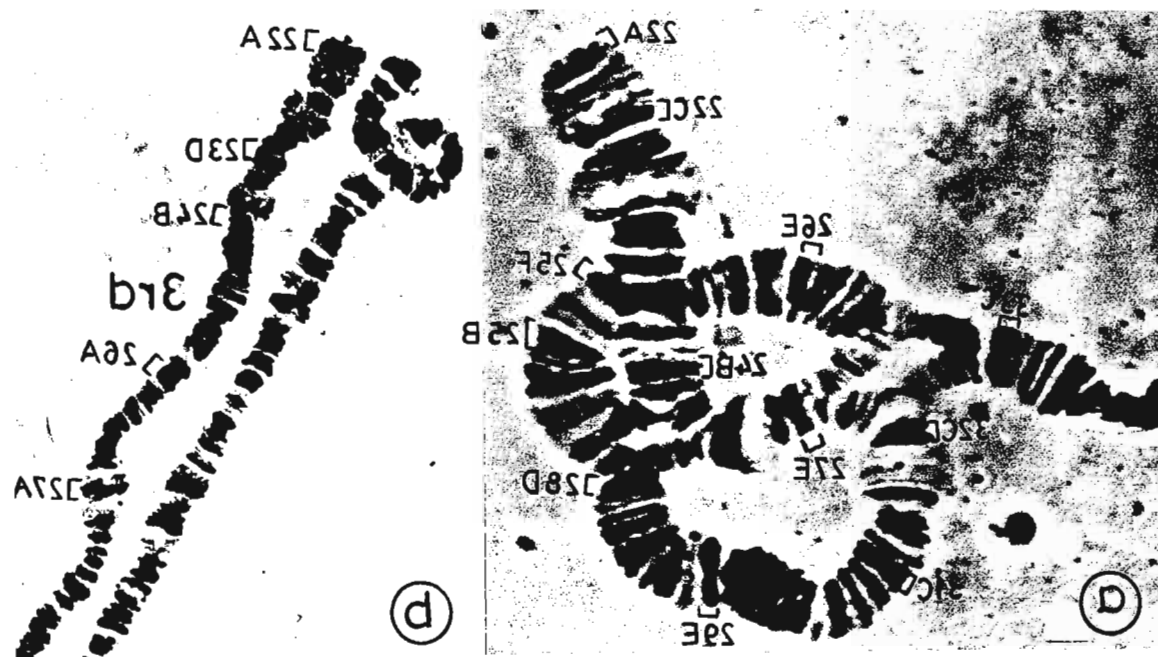
Figure 1. Photomicrograph showing the 3rd chromosome of **D.pseudoobscura** in inversion heterozygote (a) and homozygous normal (b) conditions. Chromosomal subdivisions in (a) represent the original gene sequence.
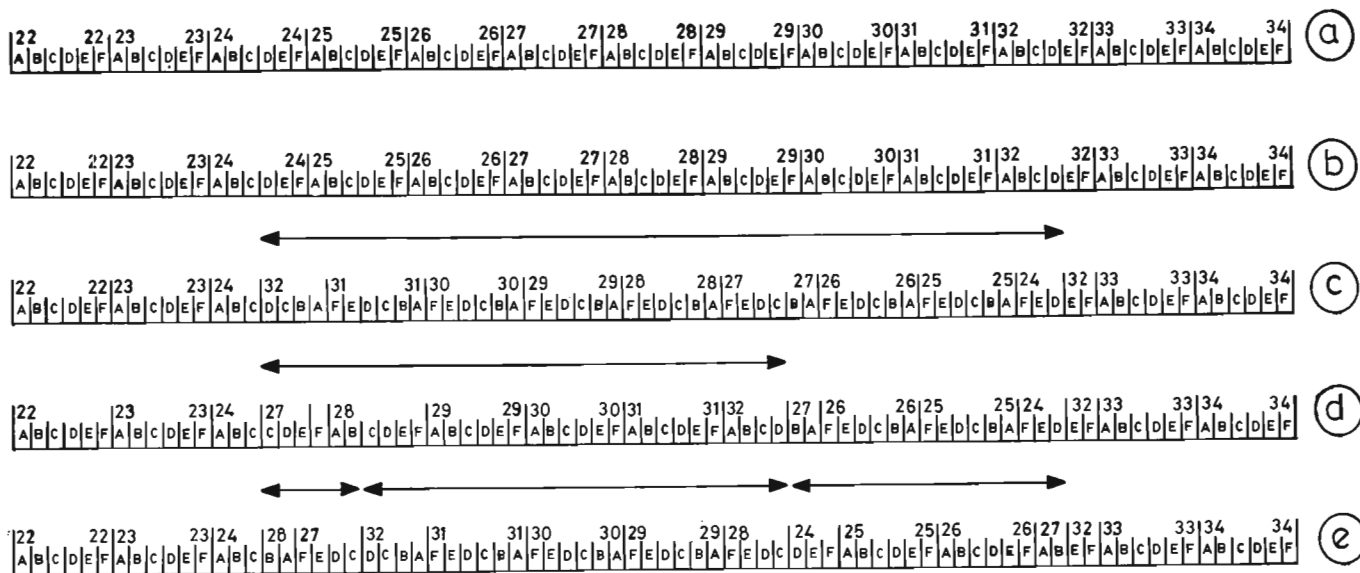
Figure 2. Schematic representation of gene sequence of the 3rd chromosome of **D.pseudoobscura** in original (a) and inverted (e) form. Figs. (b) to (d) represent the extent of different inversions (indicated by arrows) and different intermediate gene sequences between (a) and (e).

References. Das, M., D. Mutsuddi, A.K. Duttagupta & A.S. Mukherjee 1982, Chromosoma 87:373-388; Dobzhansky, T. & A.H. Sturtevant 1938, Genetics 23:28-64; Koller, P.C. 1939, Genetics 24:22-33; Mutsuddi, M., D. Mutsuddi & A.K. Duttagupta 1985, Ind. Jrl. Expt. Biol. 23:616-624; Patterson, J.I. & W.S. Stone 1952, Evolution in the genus Drosophila, MacMillan Press, NY; Strobel, E.C., C. Pelling & N. Arnheim 1978, Proc. Natl. Acad. Sci. 75:931-935.
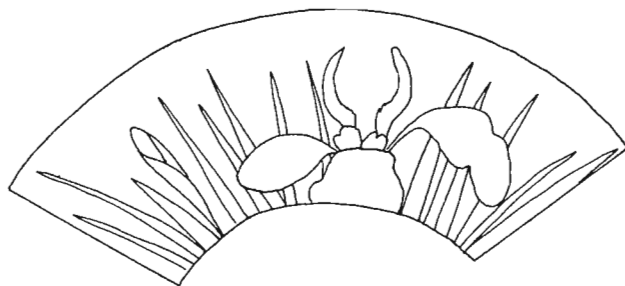
Orr, H.A. and J.A. Coyne. University of Maryland, College Park. Exceptions to Haldane's rule in Drosophila.

Haldane's rule states that "When in the F_1 offspring of two different animal races one sex is absent, rare, or sterile, that sex is the heterozygous [heterogametic] sex." In a recent review of 266 interspecific crosses in Drosophila (142 of which yield 1 fertile and 1 sterile sex), Bock (1984) found only one exception to this rule: the F_1 cross of **D.texana** X **D.lacicola** yields sterile females and fertile males.

Though Haldane's rule clearly holds for the vast majority of Drosophila hybridizations, we found several additional exceptions in the literature. Because analysis of the genetic bases of sterility or inviability in these rare cases could improve our understanding of the genetics of Drosophila speciation, we list here all putative exceptions. We hope this list will encourage such analyses when the necessary genetic markers become available: (1) **lacicola** ♂ X **texana** ♀ (F_1: sterile ♀, fertile ♂) (Throckmorton 1982); (2) **gaucha** ♂ X **pavani** ♀ (F_1: sterile ♀, occasional fertile ♂) (Brncic & Koref-Santibanez 1957); (3) **aldrichi-2** ♂ X **mulleri** ♀ (F_1: 9% ♀, 91% ♂) (Crow 1942); (4) **texana** ♂ X **montana** ♀ (F_1: absent ♀, sterile ♂) (Throckmorton 1982).

References: Bock, I.R. 1985, Evol. Biol. 18:41-70; Brncic, D. & S. Koref-Santibanez 1957, Evol. 11:300-310; Crow, J.F. 1942, UTP 4228:53-67; Throckmorton, L.H. 1982, The Genetics and Biology of Drosophila 3b:227-289.

Pérez, J.L., J.A. Castro and J.L. Mensua.
Universidad de Valencia, Spain. Larval
facilitation in **Drosophila melanogaster.**

Lewontin (1955) employed the term "genetic facilitation" to indicate a situation in which positive interactions among larvae of Drosophila can yield an improvement of viability. He also indicated the possibility that biotic residues excreted by the larvae could play an important role in the interpretation of larval facilitation as well as in the competitive processes in general. Lewontin (1955), Lewontin & Matsuo (1963) and Beardmore (1963) suggested that the positive interactions among larvae might also occur among different genotypes of the same species, being an important mechanism for maintaining genetic polymorphism. Budnik & Brncic (1976), Bos et al. (1977) and Bos (1979) have also studied these topics, finding larval facilitation among larvae of the same or different genotype. Though these authors considered biotic residues as the main cause to explain these phenomena, they do not explain which kind of metabolite can be responsible.

The purpose of the present work is to show larval facilitation at low densities in two strains of **Drosophila melanogaster,** considering viability and developmental time as the parameters.

The experiments were carried out with two strains of **Drosophila melanogaster:** a wild (+/+) and a cardinal (cd, III:75.7) strain. Both strains coming from the progeny of a single female captured in a cellar in Requena (Valencia, Spain) in October 1977 and maintained since then in the laboratory. Cultures were raised in 5 x 0.8 cm vials with 0.5 ml boiled medium (consisting of water, 1% agar, 10% sugar, 0.5% salt and 10% brewer's yeast). Newly emerged larvae (±2 hr) were sown into the vials. The cultures were maintained under constant light at a temperature of 25±1°C and at 60±5% relative humidity. The following larval densities were sown: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12, 14, 16, 18 and 20. Twenty replicates were made with 1 to 10 densities and 10 with the rest. Analyses of variance and regressions of the viabilities were performed with angular transformation.

Table 1 shows mean viabilities (V) and mean developmental times (MDT), with standard errors of the wild and cd strains. Table 2 reflects the best polynomial fits to V and MDT. Wild, in V, presents a constancy, while in MDT the best fit is four order polynomial regression. Cd, in V, fits a four order polynomial regression, while in MDT a linear fit is the most adequate.

The only case in which facilitation has been detected is in cd in V. A seeding density around 5 larvae gives the highest viability from a statistical point of view. In MDT, on the contrary, a progressive increase appears with density in both strains. We interpret these results in the following way: Biotic residues must play an important role, but in a negative way, because of the main presence of uric acid in the culture medium excreted by larvae (and also urea in a minor quantity), the effect of which has been demonstrated to be damaging to larvae (Botella et al. 1985; Castro et al. 1986). Nevertheless, we think that its harmful effect must be relevant only as larval density increases in the culture medium. We think that at low densities (perhaps 1-10 larvae) its effect cannot be very important, taking into consideration other factors. At very low densities, the difficulty in obtaining food must be high since culture medium cannot be sufficiently mixed by other larvae, the loss of humidity and the desiccation of the food being very

Table 1. Mean viabilities (V) (in percentages) and Mean developmental times (MDT) (in days) with standard errors of strains wild and cd. N = seeding density. a=only 15 in 20 replicates emerged adults; b=only 17 in 20; c=only 19 in 20.

	wild		cd	
N	V	MDT	V	MDT
1	75.00±9.93	10.93±0.21[a]	85.00±8.19	10.59±0.12[b]
2	72.50±6.76	11.21±0.11[c]	92.50±4.10	10.93±0.12
3	80.00±5.62	11.26±0.12	76.67±5.46	10.93±0.11
4	76.25±5.87	11.41±0.15	88.75±4.24	10.93±0.11
5	73.00±4.17	11.47±0.12	91.00±3.40	11.07±0.05
6	71.67±5.55	11.71±0.14	90.00±1.87	11.27±0.08
7	67.14±4.53	11.65±0.12	87.14±2.72	11.44±0.06
8	67.50±3.99	11.62±0.09	81.25±2.48	11.41±0.12
9	66.11±4.73	11.75±0.11	86.11±2.53	11.40±0.12
10	71.50±4.83	11.67±0.13	87.00±2.63	11.66±0.17
12	63.33±5.98	11.71±0.08	79.17±3.78	11.48±0.11
14	70.71±6.16	11.95±0.18	82.86±2.86	11.97±0.19
16	64.38±2.95	12.61±0.18	81.88±3.42	11.70±0.31
18	68.33±4.15	12.48±0.11	86.11±2.07	11.93±0.21
20	63.50±1.98	12.42±0.15	71.00±2.87	12.28±0.36

Table 2. Polynomial regression analyses for Viability (V) and Developmental Time (DT) of strains wild and cd. Viability has the arcsine transformation. a=intercept at origin; b1, b2, b3, b4 = respective coefficients of polynomial regression fits; c = mean value of all points when a polynomial regression could not be fitted. C.D. = coefficient of determination.

Strains	V	C.D.	DT	C.D.
wild	c=56.2511	---	a =10.4500	0.946
			b1= 0.5100	
			b2=-0.0850	
			b3= 0.0060	
			b4=-1.41x10^{-4}	
cd	a =49.8736	0.620	a =10.7234	0.903
	b1= 8.4656		b1= 0.0767	
	b2=-1.4270			
	b3= 0.0955			
	b4=-0.0022			

important, these factors affecting larvae negatively. When there are more larvae in the culture medium (5 in our case), negative interactions among them are practically null because they have space and food in abundance; a high degree of cooperation can be more easily found among them since culture medium has been mixed more and with a high degree of humidity because the living activities of these few larvae are enough to maintain the humidity; therefore, it is easier for larvae to obtain the food. As larval density increases, negative interactions among larvae become more important, with a more important struggle for exploitation (to obtain food) and an increasing importance of biotic residues. The conclusion is that at very low densities (1, 2 or 3 larvae) viability is not necessarily the highest.

Obviously, not all strains have the same resistance to humidity and uric acid, so their response will be different. Some strains have a high resistance to uric acid and desiccation (as wild strain), and detecting responses in these strains is difficult; in cd strain, on the contrary, it is detected since its sensitivity to uric acid and desiccation is known (Castro et al. 1986). Anyway, we think that the phenomenon of larval facilitation is a soft process because of its difficulty to be detected.

References: Beardmore, J.A. 1963, Am. Nat. 97:69; Bos, M. 1979, Evolution 3:768; ____, B. Burnet, R. Farrow & R.A. Woods 1977, Evolution 31:824; Botella, L.M., A. Moya, M.C. González & J.L. Ménsua 1985, J. Insect Physiol. 31:179; Budnik, M. & D. Brncic 1976, Evolution 29:777; Castro, J.A., L.M. Botella & J.L. Ménsua 1986, Arch. Insect Biochem. & Physiol. 3:485; Lewontin , R.C. 1955, Evolution 9:27; ____ & Y. Matsuo 1963, PNAS USA 49:270.

Ralchev, K.H. and B.H. Dunkov. University of Sofia, Bulgaria. Genetic localization of gene controlling diaphorase-1 in D.virilis by interspecific crossing.

Our previous study demonstrated the existence of at least three structural genes (Dia-1, Dia-2 and Dia-3) controlling the diaphorases (NAD(P)H: (acceptor) oxidoreductases in Drosophila (Ralchev et al. 1987). One of these loci, Dia-3 was recently mapped in D.virilis (Dunkov & Ralchev 1986). In the present paper, we report the localization of another one, Dia-1.

The investigation of different species of the virilis group (Figure 1) revealed that D.a.americana, D.a.texana, and D.novamexicana possess diaphorase-1 with slow mobility (DIA-1S), while the same enzyme of all other species is with fast mobility (DIA-1F). It is known that the species of this group can be crossed producing fertile progeny (Throckmorton 1982). This enabled the localization of Dia-1 gene. The crosses between D.virilis and D.novamexicana showed that the gene is X-linked (Figure 2). In order to map Dia-1 locus more precisely, D.novamexicana and D.virilis stock No127, cv - crossveinless, 1-25.0, w - white, 1-105.0 (Alexander 1976) were crossed according to the following scheme:

$$♀♀ \text{ D.virilis } 127 \frac{cv\ w\ (Dia\text{-}1F)}{cv\ w\ (Dia\text{-}1F)} \times ♂♂ \text{ D.novamexicana } \frac{+\ +\ (Dia\text{-}1S)}{}$$

$$♀♀\ F_1 \frac{cv\ w\ (Dia\text{-}1F)}{+\ +\ (Dia\text{-}1S)} \times ♂♂ \text{ D.virilis } 127 \frac{cv\ w\ (Dia\text{-}1F)}{}$$
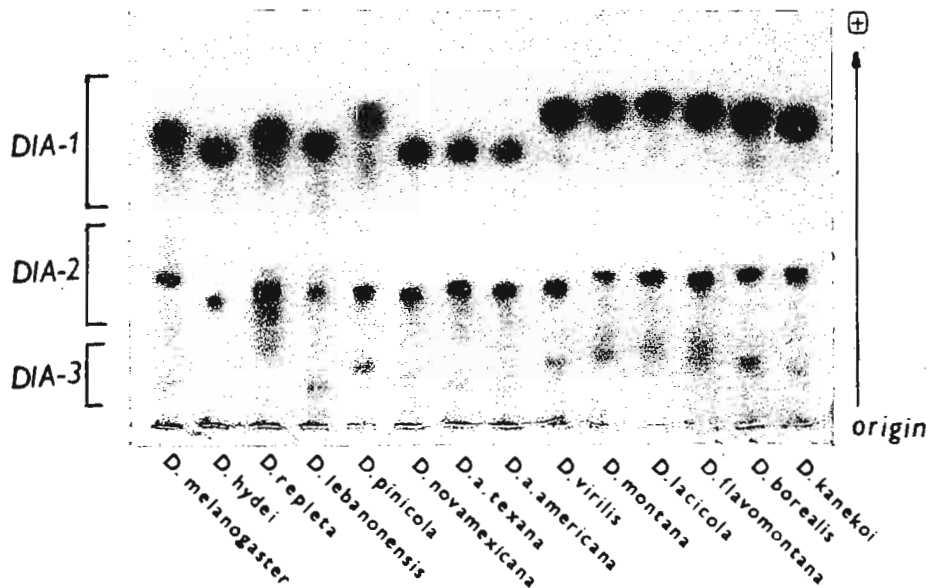
In the progeny thus obtained, 320 male flies recombinant by visible markers were selected and subjected to electrophoretic analysis in starch gel. The results obtained from the examination are presented in Table 1. The map position of Dia-1 calculated from these data is 1-101.7 ± 1.7. Thus, it turns out that Dia-1 locus

Figure 1. Interspecific polymorphism of diaphorases. Starch gel electrophoresis in the presence of NADH (Edwards et al. 1979), staining, as previously described (Dunkov & Ralchev 1986).
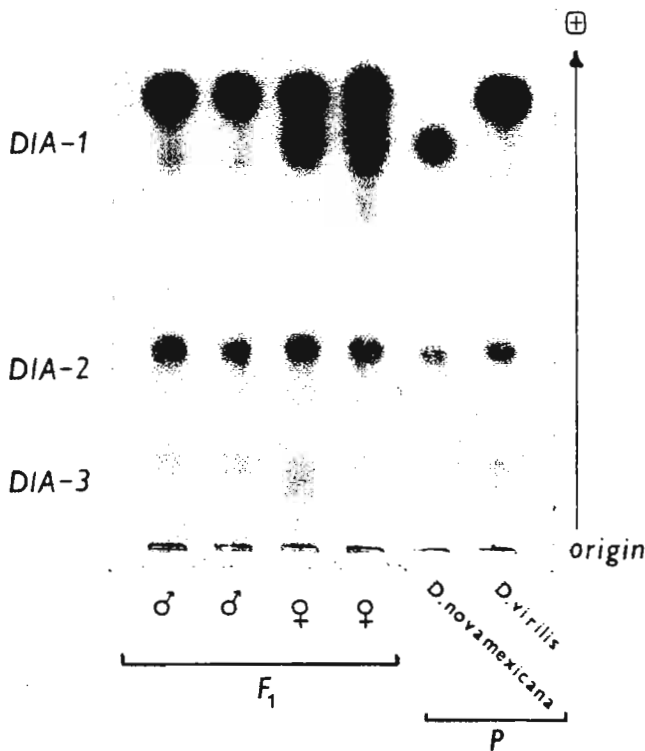
Figure 2. DIA-1 phenotype in F_1 progeny of cross: ♀♀ **D.virilis** (DIA-1F) × ♂♂ **D.novamexicana** (DIA-1S).

Table 1. Progeny analysis of the cross ♀♀ F_1 (**D.virilis** 127 × **D.novamexicana**) × ♂♂ **D.virilis** 127.

Recombinant males Phenotype of visible markers	Nr.	DIA-1 phenotype of recombinants DIA-1F	DIA-1S
cv +	160	5	155
+ w	160	152	8

is rather distant from the Dia-3 one which is localized at 1-65.3 ± 2.3 position.

In conclusion, it is worth noting that the sufficiently high fertility of the interspecific hybrids (as in the case of ♀♀ **D.virilis** × ♂♂ **D.nova-mexicana**) permits succesful mapping of genes also in the absence of intraspecific polymorphism.

References. Alexander, M.L. 1976, in: Genetics and Biology of Drosophila (Ashburner & Novitski, eds.) vlc:1365-1427, Academic Pr, NY; Dunkov, B.H. & K.H. Ralchev 1986, DIS 63:47; Edwards, Y.H., J.E. Potter & D.H. Hopkinson 1979, Ann. Hum. Genet. Lond. 42:293-302; Ralchev, K.H., B.H. Dunkov, A.S. Doichinov & M.J. Simeonovska 1987, Genetics and Breeding (Bulg. Acad. Sci.) 20(3):196-203; Throckmorton, L.H. 1982, in: Genetics and Biology of Drosophila (Ashburner, Carson & Thompson, eds.) v3b:227-296, Academic Pr NY.

Ramachandra, N.B. and H.A. Ranganath. University of Mysore, India. Adaptedness of a pair of chromosomal races: **Drosophila nasuta nasuta** and **D.n.albomicana** at three different temperatures.

D.n.nasuta (2n=8) and **D.n.albomicana** (2n=6) are a pair of morphologically indistinguishable cross fertile races belonging to the **nasuta** subgroup of the **immigrans** species group of Drosophila (Nirmala & Krishnamurthy 1972; Ranganath 1978). Extensive cytogenetic investigations on these races made by Ranaganath and his coworkers (1981; 1982a,b, 1983; 1986a, 1987) have shown the extent of similarities and differences between them. However, ecological genetics of these chromosomal races was less documented.

The success of a population depends on its adaptation to climatic conditions (Parsons 1982). Temperature is one of the important components of the ecology of a population. For ectothermic animals, such as Drosophila, temperature is certainly the most important factor of the environment. It affects all possible biological processes at the molecular, cellular and organismic levels (David et al. 1983). In view of this, the present report deals with the adaptedness of **D.n.nasuta** and **D.n.albomicana** at three different temperatures.

D.n.nasuta (2n=8) (Coorg, South India) and **D.n.albomicana** (2n=6) (Okinawa, Univ. of Texas Collections, USA, 3045.11) were employed for the present investigation. The adaptedness of these two races was assessed at three different but constant temperatures, namely at 18°C, 22°C and 26°C. The four important components of adaptedness namely, productivity, mortality, flies per bottle and population size were estimated. These cultures were maintained separated at three temperatures following the serial transfer technique of Ayala (1965) as reported by us (Ramachandra & Ranganath 1986b). Each experiment was conducted for a maximum period of 18 weeks. The analysis of variance test was computed to the mean values of population size to ascertain the extent of differences between these chromosomal races at three temperatures.

The adaptedness of a population to certain environments is a measure of its ability to survive and reproduce in these situations. Adaptedness is a cumulative assessment of four related parameters, namely, productivity, mortality, flies per bottle and population size (Ramachandra & Ranaganath 1986b).

Table 1. Four components of adaptedness of **D.n.nasuta** and **D.n.albomicana** at three different temperatures with the summary of the analysis of variance test.

Parameters	Temperatures		
	18°C	22°C	26°C
D.n.nasuta:			
Productivity	26.23±1.85	134.74±2.23	110.37±5.64
Mortality	17.89±1.74	110.46±2.67	92.89±6.67
Flies/bottle	26.56±0.76	59.05±2.11	40.37±2.07
Pop. size*	92.96±2.66	206.70±7.39	141.30±7.25
D.n.albomicana:			
Productivity	14.18±0.77	175.69±5.07	75.43±5.88
Mortality	12.33±0.75	152.18±7.81	68.92±4.50
Flies/bottle	12.38±1.04	70.46±2.19	28.63±1.47
Pop. size*	43.61±3.55	255.69±7.66	100.23±5.16

*analyis of variance test: F=180.28; df=5,18; CD=17.39. Significant at 5% level.

Table 1 gives the values of four components of adaptedness of **D.n.nasuta** and **D.n.albomicana** at three temperatures. The results reveal the following: (a) Both **D.n.nasuta** and **D.n.albomicana** were able to survive and reproduce at all three temperatures. (b) The 22°C appears to be more suitable for both the races wherein they have attained the highest adaptedness, while the 18°C seems to be less suitable for both the races with the least population size. (c) The analysis of variance test computed to compare the mean values of the population size has revealed that **D.n.nasuta** had significantly better adaptedness than **D.n.albomicana** at 18°C and 26°C, while **D.n.albomicana** had higher adaptedness than **D.n.nasuta** at 22°C.

The gene pool which maintains a larger population size may be said to be performing better than the one having smaller population size. In the present experiment, the ecological optimum for both **D.n.nasuta** and **D.n.albomicana** lies around 22°C wherein they have attained maximum adaptedness than at other temperatures. It can also be safely said that these cytologically distinct stocks have acted differently at these different but constant temperatures.

References. Ayala, F.J. 1965, Genetics 51:527; David, J.R., R. Allomand, Van Herrewege & Y. Cohet 1983, in: Genetics and Biology of Drosophila (Ashburner, Carson & Thompson, eds.) 3d:105, Academic Pr, NY; Hagele, K. & H.A. Ranganath 1983, Genetica 60:123; Nirmala, S.S. & N.B. Krishnamurthy 1972, DIS 49:60; Ramachandra, N.B. & H.A. Ranganath 1986a, Chromosoma 93:243; _____ & _____ 1986b, Ind. J. Exp. Biol. 24:137; _____ & _____ 1987, Ind. J. Exp. Biol. 25:55; Ranganath, H.A. 1978, Proc. Ind. Nat. Sci. Acad. (New Delhi), Science Academy Medals for Young Scientists, Lectures, 124; _____ & N.B. Krishnamurthy 1981, J. Hered. 72:19; _____ & K. Hagele 1982a, Chromosoma 85:83; _____, E.R. Schmidt & K. Hagele 1982b, Chromosoma 85:361; Parsons, P.A. 1982, Aust. J. Zool. 30:427.

Ramachandra, N.B., A. Ushakumari and H.A. Ranganath. University of Mysore, India.
Egg to adult rate of development and viability of **Drosophila nasuta nasuta** and **D.sulfurigaster neonasuta** in different media with different sugars.

D.nasuta nasuta and **D.sulfurigaster neonasuta** belong to the **nasuta** subgroup of the **immigrans** species group of Drosophila. Females of these species are indistinguishable, while males have slight differences (Nirmala & Krishnamurthy 1973; Ranganath & Krishnamurthy 1976). **D.n.nasuta** and **D.s.neonasuta** coexist in certain parts of South India (Siddaveere Gowda et al. 1977). The nature of ecological differentiation between these closely related sympatric species is not known. In view of this, an attempt has been made to understand their ecological differences or preferences under laboratory conditions. Experiments have been made to assess the response of **D.n.nasuta** and **D.s.neonasuta** to media containing one of the four types of sugars, namely, molasses, sucrose, glucose or fructose. Egg to adult rate of development and viability of **D.n.nasuta** and **D.s.neonasuta** were used as parameters for comparison.

Egg to adult rate of development is an important adaptive trait which determines the rate of increase or decrease of a population (Andrewartha & Birch 1954). It is the result of an interaction between the genotype and the environmental factors. In the present experiment, different sugar sources have acted as different types of environmental factors. The duration of development is defined as the time between egg laying and the emergence of the imago. The mean developmental time in days of **D.n.nasuta** and **D.s.neonasuta** is given in Table 1. The eggs of **D.n.nasuta** have experienced the fastest rate of development in the media with molasses, and the slowest in the media with glucose. Here, the mean rate of development ranges between 14.97 days to 18.87 days. The time consumed to get the adults of **D.n.nasuta** in different media can be represented as follows: molasses > fructose > sucrose > glucose.

On the other hand, the eggs of **D.s.neonasuta** have undergone the fastest and the slowest rate of devleopment in the media containing molasses and sucrose, respectively. The average rate of development ranges between 11.87 days to 14.91 days. The speed of development of **D.s.neonasuta** in different media can be represented as follows: molasses > glucose > fructose > sucrose.

Table 1. Mean developmental time in days of **D.n.nasuta** and **D.s.neonasuta** in four different media.

	Molasses	Wheat cream agar media with: Sucrose	Glucose	Fructose
D.n.nasuta	14.97±0.07	18.67±0.13	18.87±0.12	17.34±0.11
D.s.neonasuta	11.87±0.08	14.91±0.10	12.62±0.11	13.26±0.09

(Student-'t' test revealed that the difference between any two mean values was statistically sign., $P<0.01$).

Table 2. Egg to adult viability of **D.n.nasuta** and **D.s.neonasuta** in four different media with the summary of X^2 test.

Media with	Total no. of eggs placed	No. of adults emerged: D.n.nasuta	D.s.neonasuta
Molasses	600	417[a]	278[e]
Sucrose	600	361[b]	232[f]
Glucose	600	383[c]	204[g]
Fructose	600	296[d]	255[h]

	ΣX^2	df	p
a-d	21.41	3	<0.001
e-h	12.41	3	<0.01
a/e	27.40	1	<0.0001
b/f	27.62	1	<0.001
c/g	53.97	1	<0.0001
d/h	2.90	1	>0.05

The interspecies comparison has revealed that the eggs of **D.s.neonasuta** had significantly a faster rate of development than the eggs of **D.n.nasuta** in all the four types of media. Similarly, Ramachandra & Ranganath (1986) have reported significant differences in the rate of development of two sympatric domestic species, namely, **D.melanogaster** and **D.ananassae** in four different sugar media. These findings on the difference in the rate of development of a species in different media and of the differences between sympatric species can be an important component of variation of fitness of a species that feed constantly (Lewontin 1974).

Egg to adult viability is defined as the proportion of eggs reaching adulthood. Egg to adult viability of **D.n.nasuta** and **D.s.neonasuta** in four types of media is given in Table 2. The eggs of **D.n.nasuta** have achieved the highest viability in the media with molasses and the lowest in the media with fructose. The viability of **D.n.nasuta** in different media is as follows: molasses > glucose > sucrose > fructose. **D.s.neonasuta** had the highest and the lowest viability in the media with molasses and glucose, respectively. The egg to adult viability of **D.s.neonasuta** in different media is as follows: molasses > fructose > sucrose > glucose. The interspecies comparison showed that the eggs of **D.n.nasuta** had significantly a better viability than the eggs of **D.s.neonasuta** in all the media except in the media with fructose where the difference was insignificant.

By taking into cognizance of the egg to adult rate of development and viability of **D.n.nasuta** and **D.s.neonasuta** in different media, it can be mentioned that (a) the species under study have reacted differently to media with different sugars, and, therefore, each of the sugars under study had different impact on the performance of a species, and (b) in each case, the eggs of **D.n.nasuta** had a better viability and slower rate of development than the eggs of **D.s.neonasuta**.

Thus, these results reveal the differential ability of a species to exploit the media with different sugars and also the differences between two sympatric species to utilize a similar resource.

References. Andrewartha, H.G. & L.C. Birch 1954, The Distribution and Abundance of Animals, Univ Chicago Press; Lewontin, R.C. 1974, The Genetic Basis of Evolutionary Change, Columbia Univ Pr, NY; Nirmala, S.S. & N.B. Krishnamurthy 1973, Oriental Insects 7(2):267-270; Ramachandra, N.B. & H.A. Ranganath 1986, DIS 63:110-111; Ranganath, H.A. & N.B. Krishnamurthy 1976, Egypt. J. Genet. Cytol. 5:141-145; Siddaveere Gowda, L, M.R. Rajasekarasetty & N.B. Krishnamurthy 1977, DIS 52:35-37.

Ramesh, S.R. and W.-E. Kalisch. Ruhr-Universität Bochum, FR Germany. Salivary gland proteins in members of the **D.nasuta** subgroup.

The salivary gland proteins were investigated in **D.nasuta nasuta, D.n.albomicans, D.n.kepulauana, D.kohkoa, D.pulaua, D.sulfurigaster sulfurigaster, D.s.bilimbata,** and **D.s.albostrigata.** Three pairs of glands of mid third-instar larvae were used for the extraction of proteins following the method of Tissiéres et al. (1974), which were then subjected to 13.7% SDS-polyacrylamide gel electrophoresis (Laemmli 1970).

We found major and minor bands of protein fractions. Visible differences between males and females within each species do not exist, concerning the major bands in SDS gels. There are characteristic "sets" of major bands in all the species investigated which significantly differ in their mobility (Figure 1). On the other hand, the mobility of the minor bands in all the species investigated was found to be almost the same. This is in contrast to the results already found in different wildtype stocks of **D.melanogaster** (Velissariou & Ashburner 1980).

We also isolated the salivary plugs from 8 pairs of glands of late third-instar larvae after 95% ethanol fixation (Beckendorf & Kafatos 1976) in **D.n.nasuta.** We found that the major bands among others (labeled D in Figure 1) belong mainly to the glue proteins.

From hybrids of **D.n.nasuta** x **D.n.albomicans** (and vice versa) we know already that at least three of the major glue protein bands (labeled C in Figure 1) are produced by gene(s) located in the X chromosome. The details of this will be published elsewhere (in preparation).

In order to localize glycoproteins, PAS reaction was carried out after electrophoresis of glue proteins following the method of Segrest & Jackson (1972). At least five of the glue protein bands (labeled B in Figure 1) show PAS positive reaction which indicates post-translational modification of the concerned proteins. Finally, we could show that the amount of protein synthesis of at least two of the glue proteins (labelled A in Figure 1) is temperature-sensitive (Ramesh & Kalisch, in prep.).

The following experiments are in progress: (1) Chromosomal localisation of the remaining glue proteins of **D.n.nasuta** on the basis of the differences in mitotic chromosomes (Ranganath & Haegele 1982) between the subspecies **D.n.nasuta** and **D.n.albomicans.** (2) Composition of an electron microscopic map of the band-interband pattern (instead of the only existing light microscopic photo map based on chromosome squash preparations; Ranganath & Krishnamurthy 1974) for **D.n.nasuta.** (3) Analysis of the puff pattern in correlation to high and low temperatures on the basis of surface spread polytene (SSP) chromosomes (Kalisch et al. 1986) in **D.n.nasuta.**
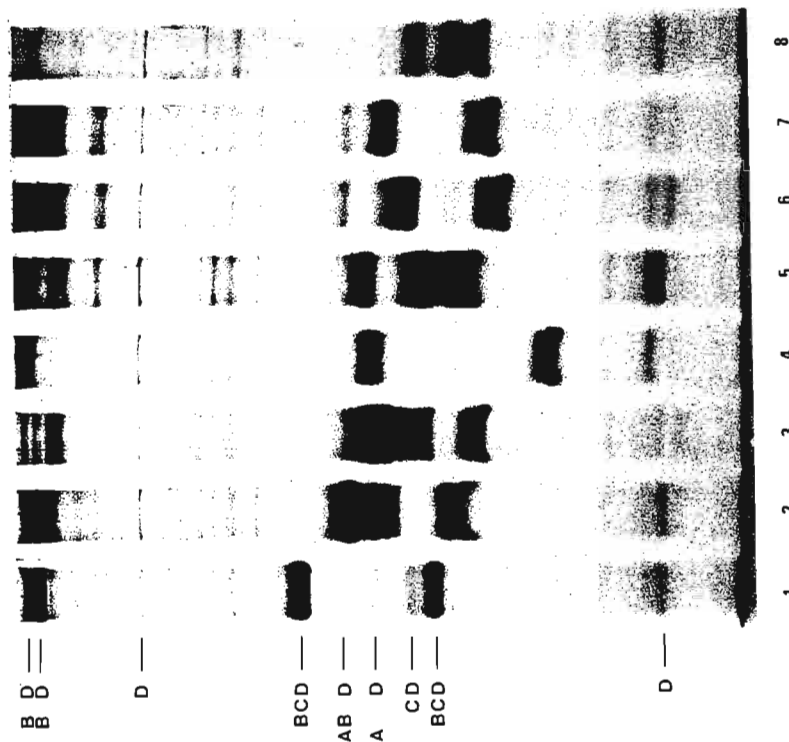
References: Beckendorf, S.K. & F.C. Kafatos 1976, Cell 9:365-373; Kalisch, W.-E., G. Schwitalla & T. Whitmore 1986, Chromosoma 93:387-392; Laemmli, U.K. 1970, Nature 227:680-685; Ranganath, H.A. & K. Hagele 1982, Chromosoma 85:83-92; Ranganath, H.A. & N.B. Krishnamurthy 1974, Sci. J. Mysore Univ. 26:65-69; Segrest, J.P. & R.L. Jackson 1972, in: Methods in Enzymol. (V. Ginsburg, ed.) v28:54-63, Acad. Pr, NY; Tissiéres, A., H.K. Mitchell & U.M. Tracy 1974, J. Mol. Biol. 84:389-398; Velissariou, V. & M. Ashburner 1980, Chromosoma 77:13-27.

Figure 1. SDS gel electrophoretic pattern of salivary gland proteins: (1) **D.n.nasuta**, (2) **D.n.albomicans**, (3) **D.n.kepulauana**, (4) **D.kohkoa**, (5) **D.pulaua**, (6) **D.sulfurigaster sulfurigaster**, (7) **D.s.bilimbata**, (8) **D.s.albostrigata**. For band labelling see text.

Remizova, E.J., V.K. Vikulova and V.A. Mglinetz.
Dept. of Developmental Genetics, Inst. of Medical
Genetics, Moscow, USSR. Prothoracic to meso-
thoracic leg transformation in **D.melanogaster**
following etherization of early embryos.

To induce bx-phenocopies 2.5 to 3 hr, **D.melanogaster**
embryos were treated with diethyl ether vapour at
17 and 28°C. In the course of leg morphology exami-
nation, the signs of mesothoracic transformation
were found not only in the metathoracic, but in pro-
thoracic legs as well. Most frequent were large
apical and preapical bristles on tibiae and two rows
of haete terminated by apical bristles on tarsi. Less frequent were certain signs of transformation in femur
(altered bristle pattern, absence of long bristles along the hind border), in coxa (deformation, altered bristle
pattern, additional joint), and in the region propleura, adjacent to the coxa base (development of
sternopleural bristles). Prothoracic leg transformation was observed less frequently than that of
metathoracic ones (see Table).

Transformations of prothoracic legs to mesothoracic ones were observed in some mutants (Lewis
et al. 1980; Struhl 1983); therefore, the cases described may be considered as phenocopies of such
mutations. No mesothoracic changes were observed in dorsal prothorax (humerus). However, several cases
of humerus duplication were found. In two cases partial symmetric duplications of prothoracic legs were
observed.

References: Lewis, R.A., T.C. Kaufman, R.E. Denell & P. Tallerico 1980, Genetics 95:367; Struhl, G. 1983, J.
Embryol. exp. Morphol. 76:293.

| Temperature | Rate of leg transformation, % | |
	Prothoracic	metathoracic
17°C	4.9 ± 0.9	11.1 ± 1.3
28°C	12.4 ± 1.4	41.8 ± 2.1

Figure. Mesothoracic transformation of pro- and metathoracic legs after etherization of Drosophila embryos. Signs
of transformation: apical and preapical bristles on tibiae (arrows); CxIII to CxII transformation; formation of
sternopleura (ST) instead of propleura.
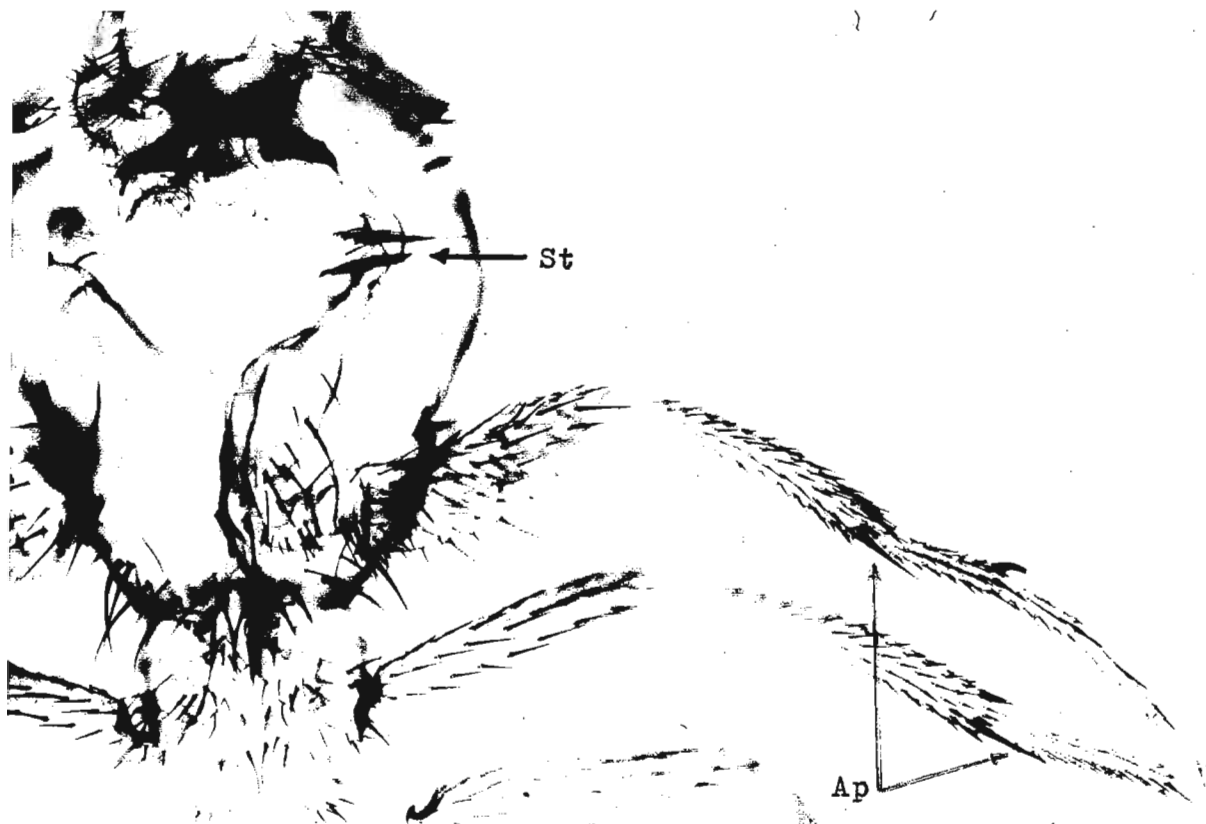
Reuter, G.[1] **and J. Szabad.**[2] 1-Martin-Luther University, Halle, DDR; 2-Biological Research Center, Szeged, Hungary. Disc autonomy of suppression and enhancement of position-effect variegation (PEV).

That PEV is suppressed by an additional Y chromosome has long been known. This type of suppression is disc and cell autonomous (Gearhart & MacIntyre 1971; Janning 1970). The dominant suppressor (Su-var) and enhancer (E-var) mutations of PEV provide excellent tools to study whether the suppression or enhancement of PEV is disc autonomous.

To investigate the above question, we made use of three mutations that alter PEV very strongly: Su-var(2)1^{01} (2-40.5; Reuter et al. 1982), Su-var(3)3^{03} (3-46.6; Reuter et al. 1986) and E-var(3)2^{01} (unpubl.). In the outlined experiments, all larvae and adults (except hosts in the control, Table 1) were homozygous for the strongly white variegating rearrangement In(1)w^{m4h} (=w^{m4h}), which provides an easily identifiable eye phenotype (Reuter & Wolff 1981; Reuter et al. 1983, 1986). The eyes of the flies that carry the Su-var or the E-var mutations are red and white, respectively.

Table 1. The autonomy of position-effect variegation in eye disc transplantations. Except the host in the control experiment, donor and host larvae were homozygous for the w^{4mh} rearrangement causing strong white-variegation. Donor and host carried either of the Su-var mutations or the wild-type alleles in homozygous condition.

	Donor	Host	Suppression of variegation Donor (implant)	Host	No. of implants
Control	+	wild-type	none	wild-type	8
Exper-imen-tal	+	Su-var(2)1^{01}	none	yes	10
	Su-var(2)1^{01}	+	yes	none	3
	+	Su-var(3)3^{03}	none	yes	9
	Su-var(3)3^{03}	+	yes	none	9

Experimental larvae were homozygous for either of the mutations Su-var(2)1^{01} or Su-var(3)3^{03}, heterozygous for E-var(3)2^{01}, or carried the wild-type alleles (Table 1). Eye discs were dissected from the donor and implanted into host larvae with appropriate genotype. After metamorphosis, the phenotype of the hosts and the recovered implants were inspected for white-variegation: to establish whether it was mottled, suppressed (red) or enhanced (white).

The results of the control experiment confirm that PEV is disc autonomous. Implants exhibited suppression or enhancement of variegation when Su-var(2)1^{01}, Su-var(3)3^{03} homozygous or E-var(3)2^{01} heterozygous discs were implanted into w^{m4h} homozygous hosts, while the eyes of the hosts were typically mottled (Table 1). In the reciprocal experiments, the implants were mottled and the eyes of the hosts were red or white due to the suppression or enhancement of PEV. There was no indication of the influence of the host genotype on the phenotype of the implants and vice versa. These results led us to conclude that suppression and enhancement of PEV (at least in the studied mutations) is a disc autonomous feature in Drosophila.

References. Gearhart, J. & R.J. MacIntyre 1971, P.N.A.C. 68:13; Janning, W. 1970, Mol. Gen. Genet. 107:128; Reuter, G. & I. Wolff 1981, Mol. Gen. Genet. 182:516; Reuter, G., R. Dorn & H.J. Hoffmann 1983, Mol. Gen. Genet. 188:480; Reuter, G., H.J. Hoffmann & I. Wolff 1983, Biol. Zbl. 102:281; Reuter, G., R. Dorn, G. Wustmann, B. Friede & G. Rauh 1986, Mol. Gen. Genet. 202:481.

Rzymkiewicz, D.M. Institute of Biochemistry and Biophysics, Polish Academy of Sciences, Warsaw, Poland. Mitochondrial tyrosine transaminating activity during development of **Drosophila melanogaster.**

Tyrosine metabolism in tissues of insects is closely related to the insect development. The development profile of cytoplasmic tyrosine aminotransferase activity reflects all the successive stages of **Drosophila melanogaster** growth (Karlson & Sekeris 1962). The dramatic developmental changes within the insect cells are accompanied by changes in the shape and number of mitochondria (Walker 1966; Tsuyama & Miura 1979) as well as by changes in activity of mitochondrial enzymes (Nowak & Piechowska 1982; Tsuyama & Miura 1979) and oxygen consumption (Guillet 1980).

The aim of the present work was to check whether the rate of tyrosine transamination in **D.melanogaster** mitochondria is dependent on the development stage and whether it is related to changes in the activity of the cytoplasmic enzyme.

MATERIALS AND METHODS: Rate of tyrosine transamination was determined in mitochondria of **D.melanogaster,** wild type, during ten days of its development: from egg to imago, under the following culture conditions: 25°C, medium supplemented with yeast (Mitchell & Mitchell 1968), 12 hr light: 12 hr dark photoperiod. Egg-laying periods of 2 hr were used to synchronize animal development. The mitochondria were isolated from 10% homogenate prepared in the following buffer: 60 mM sucrose, 240 mM mannitol, 0.2 mM EGTA, 2.5% (w/v) bovine serum albumin, 10 mM Tris HCl pH 7.4 (Storey & Bailey 1978). The nuclei and cell debris were removed at 2200g for 5 min, and next washed twice with the same buffer (1/2 and 1/4 volumes of the initial volume of the homogenate). The supernatant and washings were pooled and centrifuged at 7000g for 10 min. The mitochondrial pellet was washed twice as described above. The mitochondria from about 0.5 g of biological material were treated for 1 hr with 100 µl 0.1 M potassium-phosphate buffer pH 7.6, containing 1% Lubrol PX (w/v). The enzyme activity was assayed by the method of Marston & Pogson (1977) at 30°C, and the mitochondrial protein according to Lowry et al. (1951) with bovine serum albumin as a standard. The results are expressed as nanomoles of tyrosine transformed during one minute of the reaction by 1 mg of mitochondrial protein.

RESULTS AND DISCUSSION: The rate of tyrosine transamination in mitochondria, similarly to that of the cytoplasmic enzyme (Karlson & Sekeris 1962), is closely related to the stage of Drosophila development (Figure 1), although the two developmental profiles differ distinctly from each other.

No mitochondrial enzyme activity was found either in embryos or in 24-hr-old larvae. The enzyme activity appeared on the second day and then gradually increased. Its maximum, however, did not appear at the third instar, as it is the case with many Drosophila enzymes, including cytoplasmic tyrosine amino-transferase (Chen 1966; Karlson & Sekeris 1962; Nowak & Piechowska 1982) but after pupa formation, on the 7th day of development; the rate of tyrosine transamination was then as high as 15 nmol/min per 1 mg protein. In older organisms and the emerged adult, the activity slightly decreased. Such a time-course of changes in the mitochondrial tyrosine transaminating activity differs largely from that observed for oxygen consumption (Fourche 1969; Guillet 1980) and cyto-chrome oxidase activity (Chen 1966). The latter ac-tivity increased during the larval stage and decreased in pupae, whereas oxygen consumption was the lowest on the seventh day (Fourche 1969). These observa-tions point to a lack of close relationship in **D.melan-ogaster** mitochondria between the rate of tyrosine transamination and the activity of the respiratory chain, and simultaneously point to the existence of other regulatory mechanism different from the ecdy-sone-dependent mechanism operative in the case of cytoplasmic tyrosine aminotransferase.

References. Chen, P.S. 1966, Adv. Insect Physiol. 3:67,93; Fourche, J. 1969, Ann. Biol. 8:334; Guillet, C. 1980, DIS 55:53; Karlson, P. & C.E. Sekeris 1962, Biochim. Biophys. Acta 63:489; Lowry, O.H., N.J. Rosebrough, A.L. Farr & R.J. Randall 1951, J. Biol. Chem. 193:265; Marston, F.A.O. & Ch.I. Pogson 1977, FEBS Lett. 83:277; Nowak, J. & M.J. Piechowska 1982, DIS 58:117; Tsuyama, S. & K. Miura 1979, Insect Biochem. 9:435; Walker, P.A. 1966, J. Insect Physiol. 12:1009.
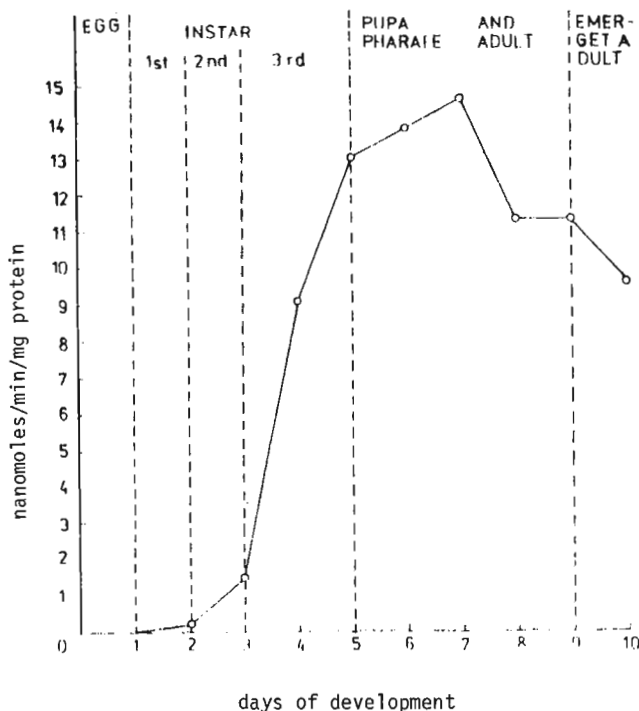
Figure 1. The rate of tyrosine transamination in **D.melano-gaster** mitochondria during the insect development. Each point represents a mean from five experiments.

Rzymkiewicz, D.M. and M.J. Piechowska.
Inst. of Biochemistry and Biophysics, Polish
Acad. of Science, Warsaw, Poland. Effect of
starvation on tyrosine transaminating activity
in mitochondria of **D.melanogaster** larvae.

Generally, the activity of cytosolic and mitochondrial forms of an enzyme are regulated by different mechanisms. It has been found (Nakata et al. 1964) that only cytosolic, hepatic aspartate aminotransferase, but not the mitochondrial, form of the enzyme is affected by the content of protein in the diet, as well as by cortisol treatment, adrenalectomy or starvation. The cortisol has been reported to have little or no effect on induction of the mitochondrial tyrosine aminotransferase from rat liver (Sapico et al. 1974). In contrast to these data, our present experiments have demonstrated that the rate of tyrosine transamination in the mitochondria from **D.melanogaster** larvae becomes altered as the result of starvation.

MATERIALS AND METHODS: **D.melanogaster** larvae reared under conditions described in the accompanying paper (Rzymkiewicz 1987) were subjected to 2 hr lasting starvation in different stage of development. The mitochondria were isolated and solubilized with dubrol Px, as described in the preceding paper (Rzymkiewicz 1987). The rate of tyrosine transamination was determined according to Marston & Pogson (1977) in the suspension of disrupted mitochondria at 30°C. Mitochondrial protein was assayed by the method of Lowry et al. (1951) with bovine serum as a standard. The results are expressed as nanomoles of tyrosine transformed during one minute of the reaction by 1 mg of mitochondrial protein. The Arrhenius plots were constructed from series of four experiments carried out over the temperature range of 5-35°C intervals.

Table 1. Effect of 2-hr starvation on rate of tyrosine transamination in the suspension of disrupted mitochondria from **D.melanogaster** larvae.

Age of larvae (hrs)	V (nmol/min/mg protein)		% of activity
	Control	Starved	
48	0.248	0.374	150.8
72	1.464	2.189	149.5
96	9.133	12.910	141.4

RESULTS AND DISCUSSION: Irrespective of the age of larvae and the level of tyrosine transaminating activity observed in non-starved controls, the rate of tyrosine transamination in the suspension of **D.melanogaster** larval mitochondria was found to be raised as the result of starvation by 40-50% (Table 1). Simultaneously, as it was calculated for 96-hr-old larvae, starvation had no effect on the affinity of enzyme towards tyrosine (not shown). In the mitochondria from **D.melanogaster** larvae, the tyrosine transaminating activity was found to be present in the inner membranes (not shown). It is known that lipid composition of biological membranes is affected by fat content in the diet via induction of acyl chain desaturases (Storch & Schachter 1984; Martin et al. 1984). In starved 96-hr-old larvae, both temperature of the break point in the Arrhenius plot and activation energy above and below this point differed from the values obtained from non-starved ones (Fig. 1).

This indicates that starvation led to alterations in the enzyme microenvironment which resulted in changes in the rate of tyrosine transamination. This suggestion is compatible with several observations concerning other membrane-bound enzymes (McMurchie et al. 1983; Storch & Schachter 1984).

References. Lowry, O.H., N.J. Rosebrough, A.L. Farr & R.J. Randall 1951, J. Biol. Chem. 193:265; Marston, F.A.O. & Ch.I. Pogson 1977, FEBS Lett. 83:277; Martin, F.D., D.A. Wright & J.C. Means 1984, Comp. Biochem. Physiol. 77B:785; McMurchie, E.J., R.A. Gibson, M.Y. Abeywardena & J.S. Charnock 1983, Biochim. Biophys. Acta 727:163; Nakata, Y., T. Suematsu, K. Nakata, K. Matsumoto & Y. Sakamoto 1964, Cancer Res. 24:1689; Rzymkiewicz, D.M. 1987, DIS 66(this issue); Sapico, V., L. Shear & G. Litwack 1974, J. Biol. Chem. 259:2122; Storch, J. & D. Schachter 1984, Biochemistry 23:1165.

Figure 1. Arrhenius plots of tyrosine transaminating activity in mitochondria from 96-hr-old larvae: control (closed circles) and subjected to 2-hr starvation (open circles).

Rzymkiewicz, D.M. and M.J. Piechowska.
Inst. of Biochemistry and Biophysics, Polish
Acad. of Sciences, Warsaw, Poland. Influence
of environmental temperature on the rate of
tyrosine transamination in mitochondria of
Drosophila melanogaster larvae.

In Drosophila larvae an increase of environmental temperature to 37°C (heat shock) leads to development of puffs and changes in gene expression (Ashburner & Bonner 1979). The fact that increased utilisation of oxygen and regression of puffs were observed in the presence of some respiratory chain substrates, among others tyrosine, led to the suggestion that the mitochondrial enzymes including tyrosine aminotransferase were involved in the cell response to shock (Leenders et al. 1974; Sin 1975).

The experiments in the present work were performed to check whether the rate of tyrosine transamination in mitochondria of **Drosophila melanogaster** larvae is dependent on the temperature shock.

MATERIALS AND METHODS: Larvae of **D.melanogaster**, wild type, were reared on Mitchell's medium (Mitchell & Mitchell 1968) supplemented with yeast, at 25°C in a diurnal cycle of alternating 12 hr light: 12 hr dark photoperiod. Egg-laying periods of 4 hr were used to synchronize the animal development. 96-hr-old larvae were used in experiments. The larvae subjected to temperature shocks of 37, 19 or 5°C were kept in a termostat at the appropriate temperature, with free access of air, light and in the presence of growth medium.

The mitochondria were isolated from 10% homogenate prepared in the following buffer: 60 mM sucrose, 240 mM mannitol, 0.2 mM EGTA, 2.5% (w/v) bovine serum albumin, 10 mM Tris HCl pH 7.4 (Storey & Bailey 1978). The nuclei and cell debris were removed at 2200g for 5 min, and next washed twice with the same buffer (1/2 and 1/4 volumes of the initial volume of the homogenate). The supernatant and washings were pooled and centrifuged at 7000g for 10 min. The mitochondrial pellet was washed twice as described above. The mitochondria from about 0.5 g of biological material were treated for 1 hr with 100 µl 0.1 M potassium-phosphate buffer pH 7.6, containing 1% Lubrol PX (w/v).

Mitochondrial acetone powder was prepared after Bernath & Singer (1962) from the mitochondrial fraction. For determination of the enzymatic activity, the mitochondrial powder from 2 g of larvae was suspended in 100 µl of 0.1 m phosphate buffer, pH 7.6.

Rate of tyrosine transamination was assayed by the method of Marston & Pogson (1977) at 30°C, and the mitochondrial protein according to Lowry et al. (1951) with bovine serum albumin as a standard. The results are expressed as nanomoles of tyrosine transformed during one minute of the reaction by 1 mg of mitochondrial protein. The Michaelis-Menten constant for tyrosine was calculated by computer process according to Porter & Trager (1977) and Cornish-Bowden & Eisenthal (1978) from five different substrate concentrations (each with duplication) over the range 0.16 - 2.6 mM. The probability level was established by Student's t test.

RESULTS AND DISCUSSION: An increase in temperature in vivo from 25 to 37°C (heat shock) or lowering to 19 or 5°C caused a decrease in rate of tyrosine transamination (Figures 1 and 2). The rate of enzyme inactivation was dependent both on the extent of changes in temperature and the time of exposure of larvae.

In mitochondria of larvae subjected to heat shock, the fall in tyrosine transamination reached a value of about 70% (Figure 1) after 180 min of exposure. On the lowering of environmental temperature,
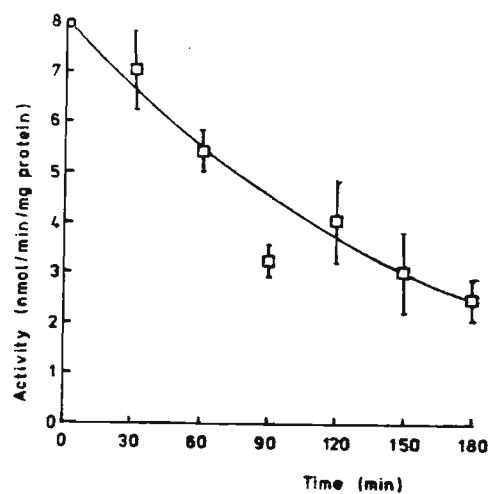
Figure 1. Effect of heat shock on the rate of tyrosine transamination in the suspension of disrupted mitochondria from **D.melanogaster** larvae.

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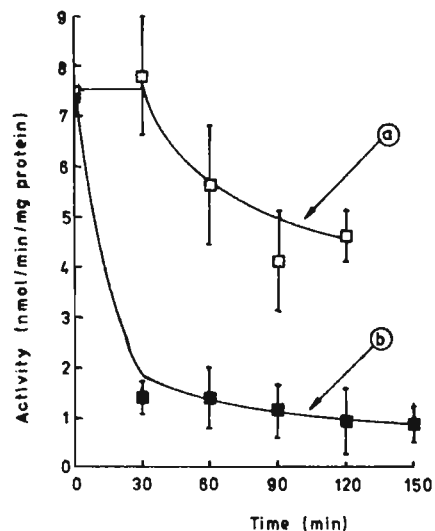
Figure 2. Effect of lowering of environmental temperature on the rate of tyrosine transamination in the suspension of disrupted mitochondria from **D.melanogaster** larvae: acclimated to a temperature of 19°C curvae a, and 5°C curvae b.

after 120 min acclimation of larvae to 19°C, the activity amounted to 60% of initial value and at 5°C the inactivation reached about 86% as early as after 30 min of exposure (Figure 2).

However, the fall in tyrosine transamination observed both at the increased (Fig. 1) and lowered (Fig. 2) environmental temperature, as well as the unchanged K_m value for tyrosine in larvae acclimated to 19°C or subjected to heat shock (not shown) seem to exclude the influence of temperature-dependent changes in membrane composition on conformation of the active site as suggested by McMurchie (McMurchie & Raison 1979). This suggestion is supported by the observation that, in delipidated mitochondria, the enzymatic activity continued to be rather high (not shown), which indicates that the presence of definite membrane phospholipids is not indispensable for membrane-bound tyrosine transaminating activity.

However, it should be noted that the differences, observed in rate of tyrosine transamination in intact mitochondria from the larvae subjected to heat shock or acclimated to 19°C and control ones, disappeared after delipidation of mitochondria. This indicates that conserved structure of mitochondrial membranes and some alterations in their functioning are a prerequisite for the appearance of decreased activity of the mitochondrial enzyme due to changes in environmental temperature. It seems that these alterations in membrane functioning are of the same character both at the raised and lowered temperature.

On the other hand, the decrease in rate of tyrosine transamination observed both at 37°C and lowered temperature seem to exclude the direct relation between the activation of genome and mitochondrial tyrosine transaminating activity as suggested by Leenders et al. (1974).

References. Ashburner, M. & J.J. Bonner 1979, Cell 17:241; Bernath, P. & T.P. Singer 1962, Methods in Enzymology, V.5, Academic Pr, NY pp605; Cornish-Bowden, A. & R. Eisenthal 1978, Biochim. Biophys. Acta 523:268; Leenders, H.J. H.D. Berendes, P.J. Helmsing, J. Derksen & J.F.J.G. Koninkx 1974, Sub-Cell. Biochem. 3:119; Lowry, O.H., N.J. Rosebrough, A.L. Farr & R.J. Randall 1951, J. Biol. Chem. 193:265; Marston, F.A.O. & Ch.I. Pogson 1977, FEBS Lett. 83:277; McMurchie, E.J. & J.K. Raison 1979, Biochim. Biophys. Acta 554:364; Mitchell, H.K. & A. Mitchell 1968, DIS 39:135; Porter, W.R. & W.T. Trager 1977, Biochem. J. 161:293; Sin, Y.T. 1975, Nature 258:159; Storey, K.B. & E. Bailey 1978, Insect Biochem. 8:73.

Serban, N.M. University of Belgrade, Yugoslavia. Differences between compound eyes of two Drosophila species obtained by electron microscopy procedure.

A wide spectrum of fixative mixtures has been used for cytological studies of **D.melanogaster** compound eyes (see, e.g., Waddington & Perry 1960; Shoup 1966; Perry 1968; Hofbauer & Campos-Ortega 1976; Campos-Ortega & Waitz 1978). We tried to fix compound eyes of two Drosophila species (**D.melanogaster** and **D.subobscura**) using the classical scheme: fixation in glutaraldehyde and postfixation in OsO_4, by decreasing the concentration of glutaraldehyde combined with shortening the time of fixation and rinsing in buffers.

The eyes were dissected under the stereomicroscope in the daylight in a drop of 2.5% glutaraldehyde (Sörensen phosphate buffer, pH 7.2, 0.1M) and immediately transferred into a fresh fixative for 1 hr. The eyes were rinsed in the same buffer for 30 min and then postfixed in 1% OsO_4 (phosphate buffer, pH 7.2) for 1 hr. The eyes of the control specimens, dissected under the same conditions, were fixed, according to Campos-Ortega & Waitz (1978), in equal parts of 1% glutaraldehyde and 1% OsO_4 in sodium-cacodylate buffer (pH 7.2) for 30 min, and postfixed in 2% OsO_4 in phosphate buffer (pH 7.2) for 2 hr. After fixation, the material was dehydrated in serial ethanol and embedded in Araldite using propylene oxide. The thick and thin sections were obtained on LKB Ultrotome III. The thick sections were mounted on microscope slides and stained with methylene blue. The thin sections were mounted on copper grids, double-stained in uranyl acetate and lead citrate, and examined on Siemens Elmiscope 101 and Philips EM 201.

Our results of light and electron microscopy show that in all eyes of **D.melanogaster**, fixed in 2.5% glutaraldehyde (Figure 1a, b), the general constitution of ommatidia is radically changed in comparison with the control group (Figure 2a, b). As seen in the cross-sections of ommatidia, their surface is smaller, the cytoplasm of visual cells narrower and rhabdomeres are closely placed to each other, so that the central ommatidial cavity disappears. Such morphological picture of ommatidia of the compound eye of **D.melanogaster** is quite different from the picture obtained after the control fixation, as well as from the cases described in the literature. The compound eyes of **D.subobscura** fixed in two different fixative mixtures (Figure 3a, b and 4a, b) show that the general constitution of ommatidia corresponds to the classical ommatidium with the open rhabdom.

The different results of electron microscopy procedure to the compound eyes of two Drosophila species must be correlated with different intercellular junctions present between the visual cells. Namely, as shown earlier (Serban & Chailley 1984) between these cells in the compound eye of **D.subobscura** in addition to desmosomes and tight junctions, there exist pleated septate junctions which were not observed
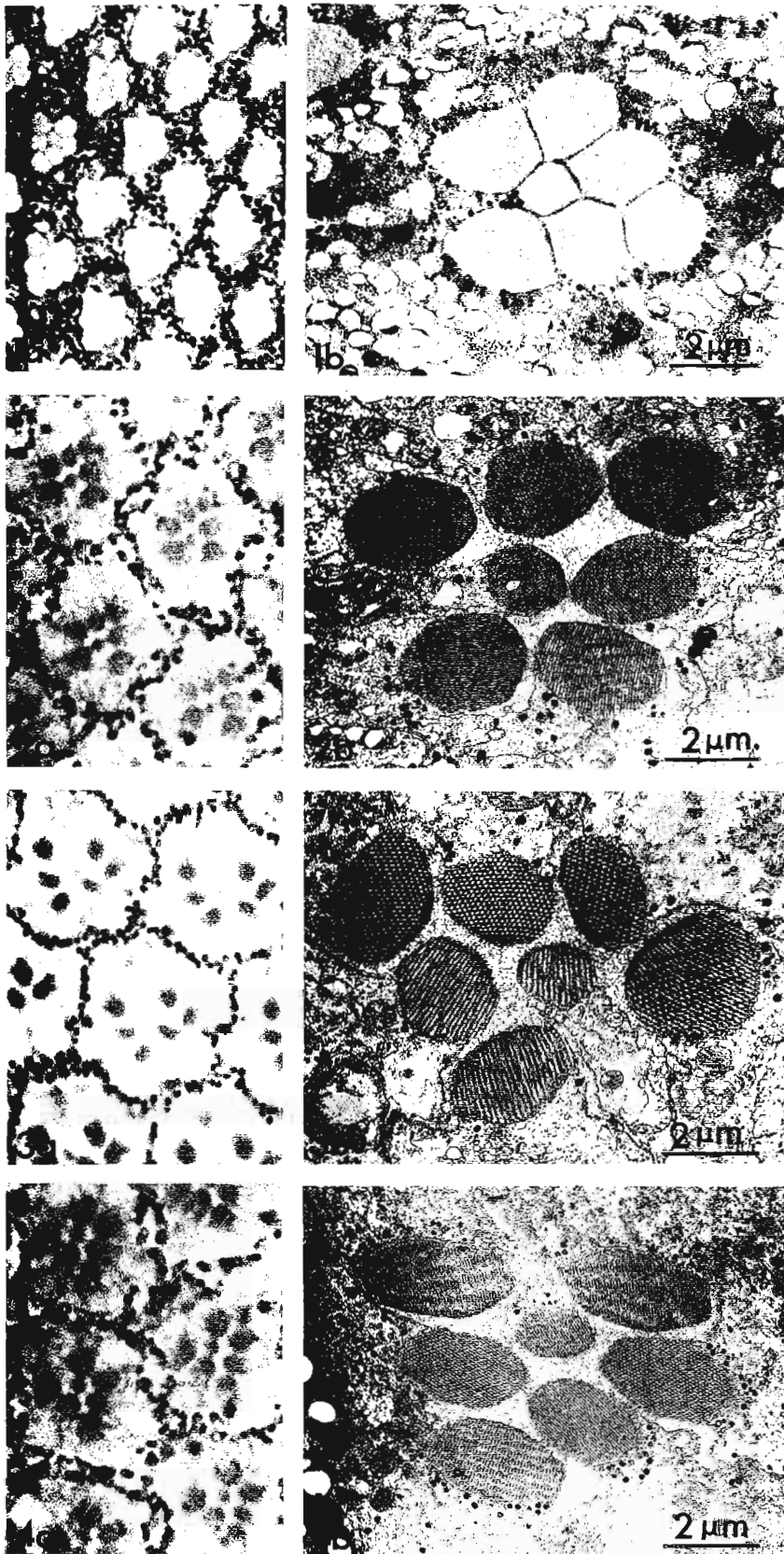
Figure 1. Cross sections through the ommatidia of **D.melanogaster** after fixation in 2.5% glutaraldehyde and postfixation in 1% OsO_4. (a) light microscopy; (b) electron microscopy.

Figure 2. Cross sections through the ommatidia of **D.melanogaster** after control fixation. (a) light microscopy; (b) electron microscopy.

Figure 3. Cross sections through the ommatidia of **D.subobscura** after fixation in 2.5% glutaraldehyde and postfixation in 1% OsO_4. (a) light microscopy; (b) electron microscopy.

Figure 4. Cross sections through the ommatidia of **D.subobscura** after control fixation. (a) light microscopy; (b) electron microscopy.

in the case of **D.melanogaster** (Perry 1968). Since this type of intercellular junction participates in periodical alternative maintaining gradient concentrations in the ommatidial cavity, we suppose that in the compound eye of **D.subobscura** different concentration of glutaraldehyde and different buffers do not cause morphological changes of ommatidium, while in **D.melanogaster** lack of them causes radical changes in the case of fixation in 2.5% glutaraldehyde and phosphate buffer.

References. Campos-Ortega, J.A. & M. Waitz 1978, Wilhelm Roux Arch. Dev. Biol. 184:155-171; Hofbauer, A. & J.A. Campos-Ortega 1976, Wilhelm Roux Arch. Dev. Biol. 170:275-289; Perry, M.M. 1968, J. Morph. 124:227-248; Serban, N.M. & B. Chailley 1984, XVII Int. Cong. Entom. Hamburg p122; Shoup, J.P. 1966, J. Cell Biol. 29:223-249; Waddington, C.H. & M.M. Perry 1960, Proc. Roy. Soc. Lond. B153:155-179.

Serban, N.M. University of Belgrade, Yugoslavia. The presence of virus-like bodies in the compound eye of **D.subobscura.**

Figure. The virus-like bodies (VLB) in the retinula cell of **D.subobscura** surrounded by a membrane (arrows); rh = rhabdomere.

In the last ten years, by using different techniques, the presence of viruses in different laboratory populations of Drosophila was verified. However, as far as we know, the presence of such particles in **D.subobscura** compound eyes has not been described yet. In **D.subobscura** compound eyes prepared for electron microscopic analysis, we observed virus-like bodies.

The eyes were fixed in glutaraldehyde (25 g/l, phosphate buffer, pH 7.2, 0.1 M), postfixed in OsO_4 (10 g/l, phosphate buffer), dehydrated in serial ethanol, embedded in Araldite, cut in thin sections on LKB Ultrotome III, mounted on copper grids, double-stained in uranyl acetate and lead citrate and examined on Siemens Elmiscope 101.

The virus-like bodies were observed in retinula cells, in secondary pigment cells, in different cells of lamina ganglionaris, as well as in the cells of fat tissues surrounding compound eyes. As could be seen from our micrograph, these bodies are of different size, in their inside all of them show a partially paracrystalline arrangement and some of them are surrounded by a membrane. Such a morphological appearance and the size of granulae in their insides lead us to a conclusion that these viruses belong to the group of Picornaviruses in their cytoplasmic morphogenesis. These viruses could be divided in three groups: P, A and C group, the last group of Picornaviruses being found only in tracheal cells (Brun & Plus 1980). Since tracheae are very much present in compound eyes, we may suppose that virus-like bodies observed in different cells of the compound eye of **D.subobscura** belong to C-group of Picornaviruses. As far as we know, the presence of these particles has not been observed so far in the compound eye of any species of Drosophila, or in any tissue of **D.subobscura.**

    References. Brun, G. & N. Plus 1980, in: Genetics and Biology of Drosophila (Ashburner & Wright, eds.), 2nd, 625-702.

Sharma, A.K. and K.S. Gill. Punjab Agricultural University, Ludhiana, India. Distribution of **D.busckii.**

Month-wise distribution of **Drosophila busckii** was studied in natural populations in and around the campus of the Punjab Agricultural University, Ludhiana, India, for two consecutive years, August 1983 to July 1984 and August 1984 to July 1985. The adult flies were collected by trap bait method using fermenting banana and mango pulp. Collections were made at 9:00 A.M. during winter and at 6:30 A.M. during summer season. **D.busckii** in both the years appeared in the beginning of November and disappeared by the end of May. During the first year (1983-84), 1817 **D.busckii** adults, which comprised 29.17% of total Drosophila species, were collected. Out of these, 508 were females and 1309 were males. In the second year (1984-85), 1977 **D.busckii** adults, which comprised 23.38% of total Drosophila species, were collected. Out of these, 25 were males and 1452 females. Thus, in these collections spreading over two years, males of **D.busckii**, on the average, outnumbered the females of this species by a factor of 2.67.

Figure 1. Collection of flies during 1983-84.

Month-wise distribution of **D.busckii** during 1983-84 and 1984-85 is represented in Figures 1 and 2, respectively. The temperature and percent relative humidity prevailing during these months as recorded by the Dept. of Agrometerology of this University are also shown. **D.busckii** was abundant in January 1984 and during this month the average winter temperature was minimum (11.8°C). In 1985, **D.busckii** was abundant in February and during this month, the winter temperature was minimum (11.9°C). Relative humidity during January 1984 and February 1984 was moderate (nearly 62%).
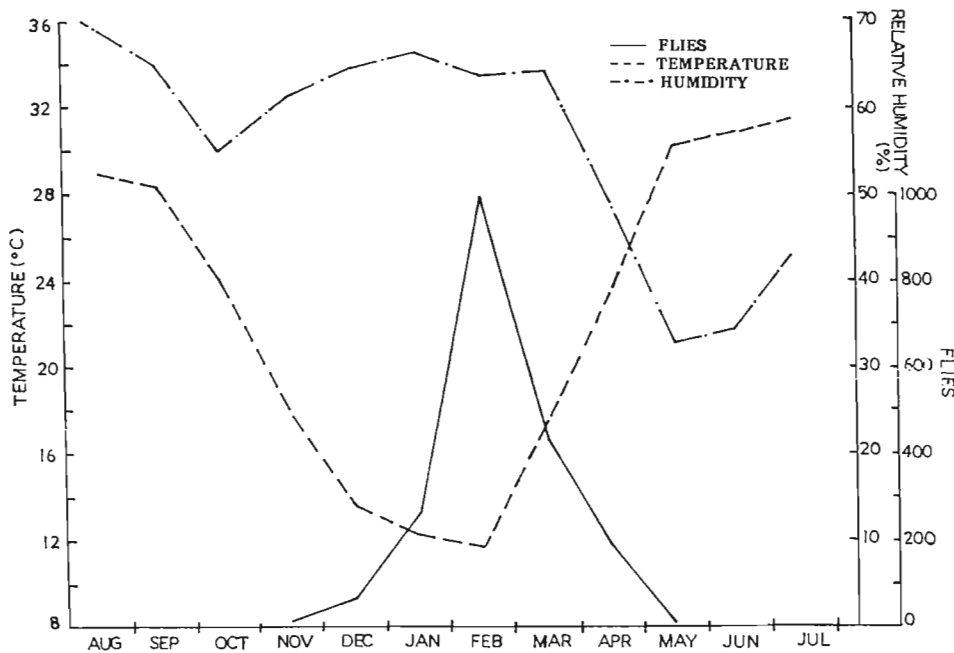
Figure 2. Collection of flies during 1984-85.

Sharp, C.B. and A.J. Hilliker. University of Guelph, Ont., Canada. The k-probit transformation and segregation distortion.

Segregation distortion is revealed by the segregation ratio (k), the proportion of SD progeny among the total number of progeny of a male heterozygous for an SD chromosome. The k statistic is not a valid interval measure for SD, largely because of the striking dependence of the variance of k on the mean of k, even after considering the effects of binomial sampling. Miklos & Smith-White (1971) have proposed that a transformation of k (the k-probit transformation) provides an interval measure of segregation distortion. However, Sharp & Hilliker (1986) have shown that this transformation cannot accurately predict the shape of k frequency distributions that are observed for all SD chromosomes. Therefore, one or more of the assumptions upon which the transformation is based must be in error. Consideration of the assumptions may reveal how empirically sound the model is.

The k-probit transformation (Miklos & Smith-White 1971) makes the following specific assumptions. Among the developing sperm of SD/SD$^+$ males, only those bearing the SD$^+$ chromosome will dysfunction, due to an underlying variable, make (m), the total level of all systems leading to the extinction of SD$^+$-bearing sperm (assumption 1) and m varies according to a normal distribution (assumption 2). If the value of m which a given SD$^+$-sperm encounters exceeds a certain threshold, then that sperm will dysfunction (assumption 3). Further, the threshold level of m is identical for all of the SD$^+$-bearing sperm of a male (assumption 4).

Thus, the k value observed for a given male will depend upon the distance from the threshold of m to the mean of the m distribution, since this will determine what proportion of the SD$^+$-bearing sperm will dysfunction. By setting the mean of the m distribution equal to zero and the standard deviation to one, conventional probit analysis can be used to determine how far the threshold of m is away from the mean of m. This distance is M, the number of standard deviation units plus five. It is assumed that M varies among males according to a normal distribution (assumption 5).

A sixth assumption of the k-probit transformation is that unless different SD stocks differ in the number and magnitude of segregating polygenic modifiers affecting SD, then even though they may have different mean k values they should have the same variance of M.

We will now examine some of these assumptions in more detail. Assumption 3 proposes that there are only two alternative sperm phenotypes: complete ability to function or complete inability to function. Experimental evidence suggests that this is not the case. Tokuyasu, Peacock & Hardy (1977) have described the ultrastructural abnormalities seen in developing SD$^+$-bearing spermatids of SD/SD$^+$ males. Considerable variation exists between the developmental defects seen in different SD$^+$-bearing spermatids within a given cyst. These include: failure to individualize; spermatid heads breaking free; and abnormal chromatin condensation. Some of the SD$^+$-bearing spermatids show very minor abnormalities in chromatin condensation and are otherwise morphologically normal. Some of these "mildly" affected SD$^+$-bearing sperm reach the seminal vesicles and may possess a reduced ability to fertilize. Indeed, Denell & Judd (1969) have shown that if an SD male is mated with females of different genotypes, then the k values can be different and furthermore, that such difference cannot be attributed solely to zygotic viability effects. Thus the ability of at least some of the SD$^+$-bearing sperm to function is dependent upon female genotype. Therefore, there are not simply two states (totally functioning and totally dysfunctioning) for SD$^+$-bearing sperm.

Another unlikely assumption is that the threshold of dysfunction for all SD$^+$-bearing sperm within a male is constant (assumption 4). Hiraizumi & Nakazima (1967) have shown that among the SD$^+$-bearing progeny of an SD male, there is usually an excess of females. Denell & Miklos (1971) have interpreted this result to mean that the threshold of dysfunction of X:SD$^+$-bearing sperm is higher than the threshold of dysfunction for Y:SD$^+$-bearing sperm. If sex chromosome content can affect the threshold of dysfunction for individual sperm, then it appears reasonable to speculate that other chromosomal, developmental and micro-environmental factors could also do so.

The data of Sharp & Hilliker (1986) can be used to directly test assumption 6. They reported k distributions for three different SD chromosomes (each with about 1100 observations). These were SD-5 (k=0.985), R(SD-5)pk cn (k=0.932) and RR(SD-5)lt (k=0.713). The first chromosome is a standard, unrecombined SD chromosome. The latter two were modified by recombination with the b pr lt pk cn chromosome, which shows no indication of segregating modifiers and the genetic background of the 3 types of SD males tested should have been quite uniform with respect to any polygenic modifiers of SD (Sharp & Hilliker 1986). The variances of M used to model the k distributions for these three chromosomes were 0.174, 0.159 and 0.356, respectively. Thus, the variance of M does not appear to be independent of the mean of M in all cases, contrary to assumption 6, as the variance of RR(SD-5)lt is markedly higher than that of the other chromosomes examined.

The k-probit transformation makes a total of six assumptions and we have reviewed evidence which suggests that at least three of these assumptions are not correct. At least one assumption must be incorrect as the model cannot predict the shapes of k distributions for two modified SD chromosomes (Sharp & Hilliker 1986). Therefore, the model cannot be used to provide a valid interval measure for SD and comparisons of the means or variances of M of different SD lines are meaningless.

It should be noted that even if the k-probit transformation model is appropriate, it does not explain Sandler & Hiraizumi's (1960) results which led to their hypothesis of SD states and a Stabilizer of SD gene (which controls mutation from one state to another). Sandler & Hiraizumi (1960) did not propose their model simply because SD lines with lowered mean k values showed an increase in the variance of k. The major observation in support of their hypothesis was that the k value of a particular male appeared to depend somewhat upon the k value of its father, but not upon the k value of its grandfather. Furthermore, an unstable line could be derived by selection from a semistable line, but the reverse result could not be obtained.

The observation that the variance of k is dependent on the mean of k can easily be explained without recourse to either the k-probit transformation or a Stabilizer of SD gene. At very high k values the SD contributed component of the variance of k must approach zero, as no SD^+-bearing sperm are produced. At k values near 0.5, the SD contributed component of the variance of k must also approach zero, since SD is not operating to any appreciable extent.

Because of the number of assumptions and the difficulty of verifying them independently, we consider that it would be problematical to attempt to modify some of the assumptions of the k-probit transformation in the hope of obtaining a better fit between observed and predicted k frequency distributions. Hopefully, analysis of the mechanism of SD will provide the basis for the development of a proper interval measure for SD, as this will allow derivation of the appropriate assumptions.

References: Denell, R.E. & B.H. Judd 1969, Mol. Gen. Genet. 105:262-274; _____ & G.L.G. Miklos 1971, Mol. Gen. Genet. 110:167-177; Hiraizumi, Y. & K. Nakazima 1967, Genetics 55:681-697; Miklos, G.L.G. & S. Smith-White 1971, Genetics 67:305-317; Sandler, L. & Y. Hiraizumi 1960, Genetics 45:1269-1287; Sharp, C.B. & A.J. Hilliker 1986, Can. J. Genet. Cytol. 28:395-400; Tokuyasu, K.T., W.J. Peacock & R.W. Hardy 1977, J. Ultrastruc. Res. 58:96-107.

Shyamala, B.V., P. Meera Rao and H.A. Ranganath. University of Mysore, India. Collection data of Drosophila fauna at four different localities in South India.

Kotagiri & Coonor situated on the Nilgiri hill ranges, which are a part of Western Ghats, is 267 km away from Mysore. Both these places are characterised by a cool and moist climate with an average annual rainfall of 1,524-2,540 mm. Vegetation consists of tropical evergreen rain forests. Charmadi Ghats, located 333 km northwest of Mysore, consist of hilly terrain packed with thick vegetation of evergreen tropical and subtropical forests. Biligirirangana hills, situated to the southeast of Mysore, constitute a discontinuous stretch of hill ranges, varying from 600 to 1800 m in altitude. This chain of hills is isolated from both Western and Eastern Ghats. It receives an average annual rainfall of 1500-2000 mm. Vegetation is made up of evergreen shrubs with dense undergrowth. Collections were made from August to October 1986 at these four localities using fermented banana as bait. In addition to this, net sweeping was also done.

A total of 2014 flies of 23 different species were trapped. Of these, 14 species belong to subgenus **Sophophora**, 8 species to subgenus **Drosophila** and 1 species to subgenus **Dorsilopha**. **D.malerkotliana** and **D.n.nasuta** constituted 52.03% of the total number of flies (Table 1). They are the dominant species occurring abundantly at all localities except at Coonor where almost negligible numbers were found. Wide distribution and occurrence over other species confers to their ecological adaptibility to different environmental conditions and diverse habitats. **D.anomelani, D.mysorensis, D.takahashii** and **D.immigrans** together constituted 30.18% of the total number of flies occurring in moderate numbers at different collection sites. **D.immigrans** were found mainly in two localities, namely, Kotagiri and Coonor; thus, showing its preference for cool and moist climate. In addition to this, **D.immigrans** were found more in nearby domestic areas than in wild. This is similar to the findings of Reddy & Krishnamurthy (1971), Ranganath & Krishnamurthy (1972) and Prakash & Sreerama Reddy (1984). Other species such as **D.rajasekari, D.ananassae, D.melanogaster, D.kikkawai, D.bipectinata, D.parabipectinata, D.nagarholensis, D.truncata, D.hypocausta** and **D.brindavanai** showed a restricted distribution by occurring only at one locality at very low frequencies. While **D.eugracilis, D.coonorensis, D.s.neonasuta, D.formosana, D.nigra** and **D.busckii** occurred at two out of the four localities, but their frequency varied considerably between the two localities.

Table 1. Distribution of different species of **Drosophila** fauna at four different localities in South India.

Species	Biligiri-rangana hills	Kotagiri	Coonor	Charmadi Ghats	Total
SUBGENUS: **SOPHOPHORA**					
D.malerkotliana	178	75	--	248	501
D.eugracilis	10	--	--	8	18
D.takahasii	18	42	48	4	112
D.rajasekari	1	--	--	--	1
D.ananassae	--	--	18	--	18
D.melanogaster	--	--	6	--	6
D.mysorensis	11	52	9	54	126
D.coonorensis	--	44	3	8	55
D.kikkawai	--	--	1	--	1
D.anomelani	52	--	--	199	251
D.nagarholensis	2	--	--	--	2
D.bipectinata	--	--	--	5	5
D.parabipectinata	--	--	--	1	1
D.truncata	--	--	--	15	15
SUBGENUS: **DROSOPHILA**					
D.n.nasuta	322	83	1	141	547
D.s.neonasuta	--	1	--	50	51
D.formosana	28	13	2	7	50
D.immigrans	--	85	26	8	119
D.paraimmigrans	--	--	--	22	22
D.hypocausta	--	--	--	6	6
D.brindavani	--	9	--	--	9
D.nigra	--	--	3	25	28
SUBGENUS: **DORSILOPHA**					
D.busckii	1	62	7	--	70
Total no. of flies	623	466	124	801	2014
Total no. of species	10	10	11	16	23

Though the four collection localities exhibit similarity in habitats with more or less similar macroenvironmental factors, there is a difference with regard to species composition and the number of individuals of different species. Amongst the four, Charmadi Ghats showed the maximum number of flies with more species diversity.

This collection data reveals that most of the species collected are the members of **melanogaster** and **immigrans** species groups of subgenera, **Sophophora** and **Drosophila**, respectively; thus, supporting the opinion of Bock & Wheeler (1972) who proposed that South East Asia is a fertile region for the rapid diversification and speciation of the members of the **immigrans** and the **melanogaster** species group of **Drosophila**.

Acknowledgements: The authors are grateful to Prof. N.B. Krishnamurthy, Chairman, Dept. of Studies in Zoology, for his help and encouragement. They are grateful to Prof. T. Okada for his advice in identification of the flies and to UGC, New Delhi, for financial assistance.

References. Bock, I.R. & M.R. Wheeler 1972, Univ. Texas Publ. 7213:1-102; Prakash, H.S. & G. Sreerama Reddy 1984, J. Bombay Natural Hist. Soc. 81(2):323-345; Ranganath, H.A. & N.B. Krishnamurthy 1972, DIS 49:83; Reddy, G.S. & N.B. Krishnamurthy 1971, DIS 47:116.

Singh, O.P. School of Agricul. Sci. & Rural Dev., North-Eastern Hill University, Medziphema, India. Drosophilidae in North Eastern India: I. A preliminary survey in Nagaland.

Considerable progress has been made in the field of taxonomy and systematics of family Drosophilidae (Diptera) in India. As a result, about two-hundred species belonging to its twenty genera have been reported from different parts of the country to date. However, a vast area of great ecological interest still either awaits exploration or is poorly explored. Particularly, very little is known regarding Drosophilid fauna of North Eastern region of the Indian subcontinent despite its rich and varied floral and faunal heritage. This region includes seven hill states, namely Assam, Arunachal Pradesh, Manipur, Meghalaya, Mizorum, Nagaland and Tripura (Figure 1). Until recently only three states--Assam, Manipur and Meghalaya--have been preliminarily surveyed. The surveys have yielded information regarding several interesting species (Singh 1972; Gupta 1973; Singh & Gupta 1977; Dwivedi & Gupta 1979, 1980; Dwivedi et al. 1979; Gupta & Singh 1979). However, no information is available about the other states of this region so far.

Of late, collections have been made, using fermenting fruits as baits, from three localities having different topography and climatic conditions, in Nagaland. The collection sites include Dimapur (a plain with comparatively warm climate), Medziphema (semi-hilly with a mixed and moderate climate) and Kohima (hilly with cold climate). All three sites come under the Kohima District of Nagaland within a distance of about 75 km.

Table 1. Comparative account of Drosophila species collected from three localities in Nagaland.

Drosophila species collected	No. of specimens at:		
	Dimapur	Medziphema	Kohima
SUBGENUS **Dorsilopha**			
1. **D.busckii**	67	85	137
SUBGENUS **Drosophila**			
2. **D.nasuta**	15	26	72
3. **D.immigrans**	3	7	112
4. **D.lacertosa**	12	45	10
SUBGENUS **Sophophora**			
5. **D.kikkawai**	237	195	25
6. **D.bipectinata**	11	7	0
7. **D.malerkotliana**	22	20	0
8. **D.ananassae**	117	115	28
9. **D.melanogaster**	14	5	0
10. **D.nepalensis**	0	3	12
11. **D.jambulina**	7	0	0
12. Unidentified sp. (1)	3	1	0
13. Unidentified sp. (2)	0	7	1
14. Unidentified sp. (3)	0	5	0

Figure. Map of North-Eastern region of India.

Altogether, 14 different Drosophila species have been collected from these three localities. Of them, 3 species remain unidentified. The identified 11 species of Drosophila belong to its three sub-genera **Dorsilopha, Drosophila** and **Sophophora.** Comparative data indicates that Dimapur and Medziphema with warm and moderate climates, respectively, harbour more species than Kohima with cold climate.

References. Dwivedi, Y.N. & J.P. Gupta 1979, Entomon 4:183-187; _____ & _____ 1980, Proc. Ind. Acad. Sci. (Ani.Sci.) 89:85-89; Dwivedi, Y.N., B.K. Singh & J.P. Gupta 1979, Oriental Insects 13:61-74; Gupta, J.P. 1973, DIS 50:112; _____ & B.K. Singh 1979, Entomon 4:167-172; Singh, A. 1972, DIS 49:78; Singh, B.K. & J.P. Gupta 1977, Proc. Zool. Soc. (Calcutta) 30:31-38.

Sliter, T.J.* University of California, Irvine. A reevaluation of the map location of the ecdysoneless mutation.

The temperature-sensitive (ts) mutation ecdysoneless (ecd) prevents pupariation by interfering with normal ecdysone production at the end of the third larval instar (Garen et al. 1977); ecd also causes a variety of defects in imaginal disc development (Redfern & Bownes 1983), in metamorphosis (Belinski-Deutsch et al. 1983), and in adult fertility (Garen et al. 1977; Audit-Lamour & Busson 1981). It was originally reported that ecd mapped to 3-41 on chromosome arm 3L. This map location was based upon an observed lack of recombination between ecd and the dominant mutation Glued (3-41.0) (Garen et al. 1977). In an attempt to verify the cited map position, ts-lethality in an ecdysoneless stock was mapped by recombination relative to recessive marker mutations. The results indicate a map position very different from the one originally reported.

The ecd st ca stock that was used has been maintained at U.C.-Irvine since before 1979. Lethality at 29°±0.5 was mapped in a homozygous subline that had been made isogenic for all third-chromosomal loci, and that expressed ts-lethality, and the full range of reported ecd phenotypes including nonpupariation, female sterility, pharate adult lethality, and patterning abnormalities in the adult derivatives of the imaginal discs. In a preliminary experiment, lethal factors on the ecd st ca chromosome were mapped relative to recessive marker mutations on the multiply marked rucuca chromosome (Lindsley & Grell 1968). The results indicated the presence of a single lethal factor on the ecd st ca chromosome, located between ru (3-0.0) and h (3-26.5).

To more precisely locate the lethal factor between ru and h, rucuca/ecd st ca females were mated to ruPrica/TM6b males, and ru h+/ruPrica recombinant male progeny were recovered. Thirty-two such males were individually test-crossed to ecd st ca females. Two of the 32 recombinant chromosomes failed to complement ts-lethality in the ecd st ca stock, and therefore represented crossover events between ru and the lethal factor. This indicated an approximate map position of 3-1.7 for the lethal factor.

To determine whether the mapped lethal factor was peculiar to the ecd st ca stock used, ts-lethality was also mapped in an unisogenized ecd st ca stock obtained from the Yale University stock collection. In

Table 1. Survival of T(Y;3)H141; ecd/ecd males at 29.5°.

Third chromosome constitution	Progeny* Recovered at 29.5° females X/X	males X/T(Y;3)H141
ecd st ca/ Bal¶	367	392
ecd st ca/ ecd st ca	0	70
TM6, Ubx[67b]/ TM6b, Tb Hu	48	200

*=Progeny of T(Y;3)H141/X; ecd st ca/TM6,
Ubx[67b] males and ecd st ca/TM6b, Tb Hu females,
reared at 29.5° from the time of egg laying.
¶=Bal: either TM6, Ubx[67b] or TM6b, Tb Hu.

that mapping, 47 ru h+/ruPrica recombinant males were test-crossed to ecd st ca females from the Yale stock. Two of the 47 recombinant chromosomes failed to complement ecd lethality, indicating a map position for the lethal factor of 3-1.1. The lethal factor was therefore not peculiar to the UCI stock of ecd st ca. The two sets of results together indicate an approximate position of 3-1.3 for the lethal factor on the ecd st ca chromosome.

Independent corroboration that the lethal factor carried in the ecd st ca stock lies at the distal tip of chromosome arm 3L was obtained from duplication mapping using the T(Y;3)H141 chromosome, which carries an insertional translocation of salivary gland chromosome region 61A-62E into a fertile Y chromosome (Seattle-La Jolla Drosophila Laboratories, 1971). T(Y;3)H141 rescued lethality at 29.5° in approximately 35% of homozygous ecd males that carried it (Table 1); female sibs that were homozygous for ecd but lacked the translocation chromosome were never recovered as adults. The males that were recovered at 29.5°C were morphologically normal; the translocation therefore rescued not only nonpupariation and pharate adult lethality, but also the nonlethal effects of ecd on imaginal disc development.

The failure of the translocation to rescue completely lethality in ecd homozygotes may have been due to the effects of Y chromosome heterochromatin in close proximity to the translocated ecd+ allele. In subsequent work deletions of ecd have been isolated by generating X-ray induced revertants of the closely linked dominant eye-facet mutation Roughened (3-1.4). These deletions uncover nonpupariation, pharate adult lethality and imaginal disc phenotypes in the UCI and Yale stocks of ecd st ca (Sliter 1986); they also uncover nonpupariation in a stock of ecd st red e ca from the Yale collection (V. Henrich, pers. comm.). The deletions localize ecd to salivary gland chromosome region 62B-D and place it close to the right-hand breakpoint in T(Y;3)H141.

These results taken together strongly indicate that the mutation responsible for the pleiotropic phenotypes typically observed in ecdysoneless stocks lies at approximately 3-1.3. This is far to the left of the map position originally reported for ecd.

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References: Audit-Lamour, C. & D. Busson 1981, J. Insect Physiol. 27:829-837; Belinski-Deutsch, S. et al. 1983, J. Insect Physiol. 29:509-514; Garen, A. et al. 1977, PNAS USA 74:5099-5103; Lindsley, D.L. & E.H. Grell 1968, Carn. Inst. Wash. Publ. 627; Redfern, C.P.F. & M. Bownes 1983, Mol. Gen. Genet. 189:432-440; Seattle-La Jolla Drosophila Laboratories 1971, DIS 47:suppl.; Sliter, T.J. 1986, Doctoral Diss., Univ. Calif-Irvine.

Smith, L. and S. Anderson. University of North Carolina, Greensboro. Thermostability of Drosophila alcohol dehydrogenase before and after exposure to 2-propanol.

Thermostability studies of an enzyme or protein can be used as a means of detecting hidden genetic variation. Amino acid substitutions that do not alter electrophoretic mobility may have an effect on the stability of a protein. In some cases electrophoretic variation may reflect the post-translational modification of a protein rather than genetic variability. The alcohol dehydrogenase enzyme of **Drosophila melanogaster** exhibits both genetic and post-translational variation. We have used a combination of cellulose acetate electrophoresis and heat denaturation studies to address the following questions. (1) Does the thermostability of the enzyme alcohol dehydrogenase produced by different strains of Drosophila uncover such hidden genetic variation? (2) Does exposure to 2-propanol, which affects many properties of alcohol dehydrogenase, alter its thermostability?

Four different strains of flies were analyzed, two homozygous for the Adh-S allele (S-1 and S-2) and two homozygous for the Adh-F allele (F-1 and F-2). Flies that were 5-7 days old were transferred to Instant Drosophila Media (Carolina Biological) prepared with or without 2% 2-propanol in water. After 18 hr of exposure the flies were homogenized and the homogenates electrophoresed on cellulose acetate strips (Gelman Sepraphore III). The homogenizing buffer contained 50 mM Tris-HCL (pH 8), 0.1% Triton X-100, 0.5 mM EDTA, and 1 mM Dithiothreitol. Homogenates were prepared at a ratio of 8 ul of homogenizing buffer per fly for Adh-S strains and 16 ul per fly for Adh-F strains. The difference in the ratio of homogenizing buffer per fly is used to account for the two-fold difference in alcohol dehydrogenase protein found between Adh-F and Adh-S strains. Electrophoresis was performed for 25 min at 200 volts in a Gelman Sepratex chamber using a 25 mM Tris-192 mM Glycine buffer (pH 8.5). After electrophoresis the cellulose acetate strips were floated on a petri dish in a 40°C water bath. The control group strips were heated for 0, 2.5., 5, and 7.5 min while the 2-propanol group was heated for 0, 2.5, 5, 7.5, and 10 min. All strips were stained for 15 min on a 1% agarose staining gel containing 5% 2-propanol, 1.5 mg/ml nicotinamide adenine dinucleotide, 1.0 mg/ml nitroblue tetrazolium, and 0.5 mg/ml phenazine methosulfonate in Tris-Glycine (pH 8.5). The strips were fixed in 5% acetic acid and rinsed in distilled water.

Figure 1 demonstrates that 18 hr exposure to 2-propanol is sufficient to cause a modification of electrophoretic mobility for both the ADH-S and ADH-F proteins. The proteins modified by exposure to 2-propanol migrate further towards the anode than do the control proteins. Figures 2-5 show the thermostability profile for the alcohol dehydrogenase protein in control flies. For all four strains 5 min exposure to 40°C significantly reduces the amount of alcohol dehydrogenase protein and by 7.5 min all the enzyme has been denatured. Figures 6-9 show the thermostability profile after 2-propanol exposure. In addition to altering electrophoretic mobility such exposure increases the heat stability of the protein, i.e., > 10 min is needed to denature all the enzyme.

The alcohol dehydrogenase proteins from the four strains examined are similar in their heat denaturation response. However, the protein from strain F-1 is slightly more heat stable than the other three. It may represent an Adh-Fr strain (heat resistant) as has been described by Sampsell (1977) and Wilks et al. (1980). These results are also similar to those obtained for purified alcohol dehydrogenase (Anderson et al. 1980). Exposure to 2-propanol, which causes a post-translational modification of the protein, also causes an increase in the heat stability. This was again especially true for stain F-1, which retains significant activity after 10 min exposure to 40°C. It is possible that the post-translational modification of alcohol dehydrogenase and the altered properties reflected by the enzyme

+

S1   S1   F1   F1

2-PROPANOL CONTROL 2-PROPANOL CONTROL
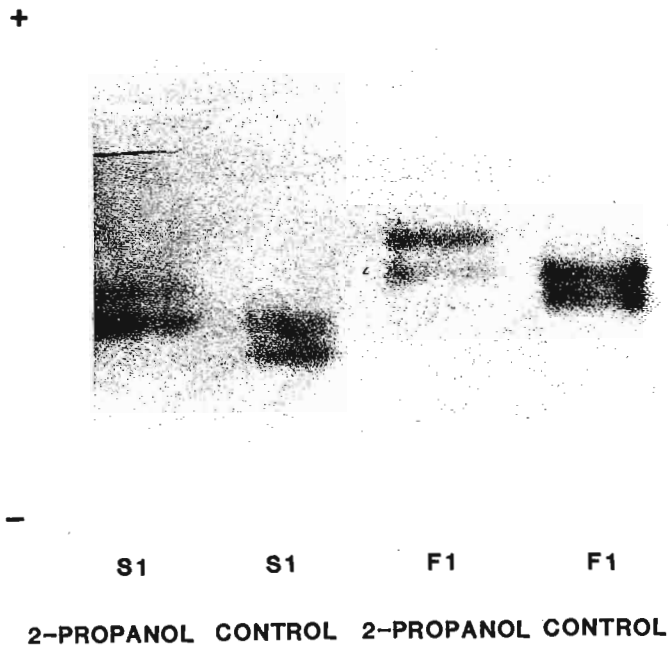
Figure 1. Cellulose acetate electrophoresis of alcohol dehydrogenase from 2-propanol treated and control flies. Strain S1 is an Adh-S strain and F1 an Adh-F strain.

CONTROL

FIG. 2-5

0′   2.5′   5′   7.5′

S1   F1   S2   F2   S1   F1   S2   F2   S1   F1   S2   F2   S1   F1   S2   F2

2-PROPANOL

FIG. 6-9

0′   2.5′   7.5′   10′

S1   F1   S2   F2   S1   F1   S2   F2   S1   F1   S2   F2   S1   F1   S2   F2
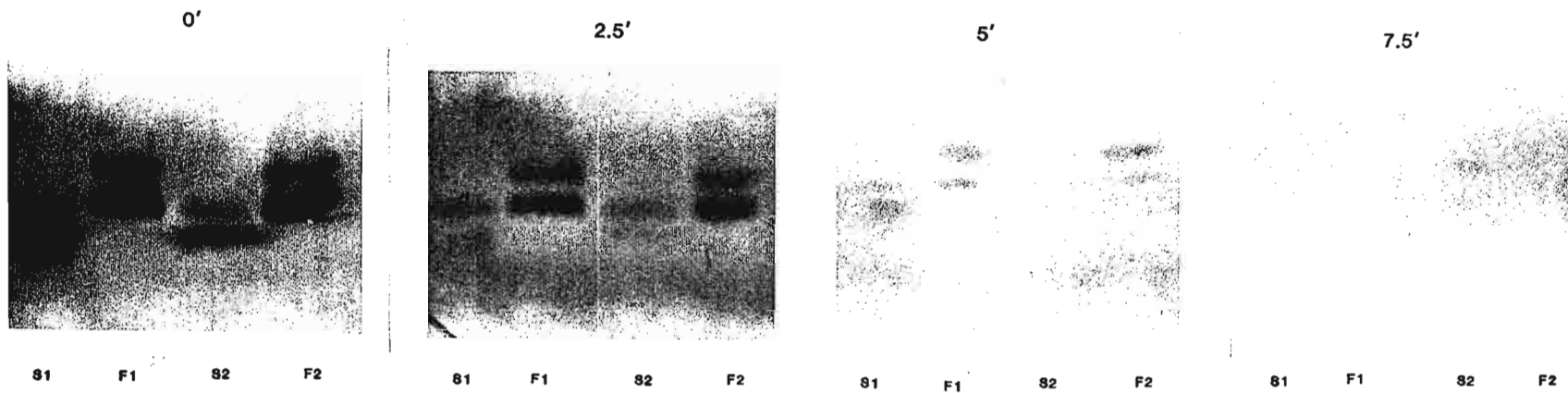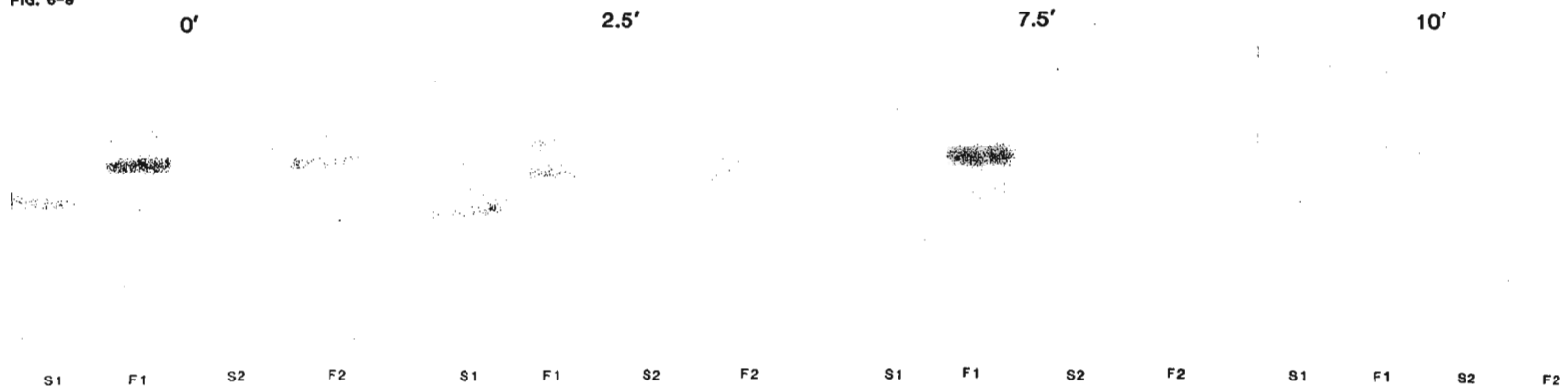
Figure 2 - 9. Thermostability of alcohol dehydrogenase from control (Fig. 2-5) and 2-propanol treated flies (Fig. 6-9). Heat treatment of the cellulose acetate strips was for 0, 2.5., 5, and 7.5 min for control homogenates and 0, 2.5., 7.5, and 10 min for homogenates from flies exposed to 2-propanol.

could be an adaptation to environments in which alcohol content and high temperatures are significant components.

References: Anderson, S., M. Santos & J. McDonald 1980, DIS 55:13-14; Sampsell, B. 1977, Biochem. Genet. 15:971-988; Wilks, A.V., J.B. Gibson, J.G. Oakeshott & G.K. Chambers 1980, Aust. J. Biol. Sci. 33:575-585.

Søndergaard, L. and D. Kolbak. University of Copenhagen, Denmark. More precise map position of the semidominant suppressor of black Su(b)$^{80\ell}$ and a new allele, Su(b)^{DK7}.

The semidominant suppressor of black Su(b)$^{80\ell}$ has previously been mapped close to the r-locus (Pedersen 1982). Using rudimentary mutants at the opposite, extreme ends of the r-locus (r^{X917} and r$^{MAHI 13A}$, respectively), we have mapped Su(b)$^{80\ell}$ to a region between 10 to 19.5 x 10^{-5} map units from the distal end of the r-locus (Carlson 1971). This is the transition region between the ATCase and CPSase defining domains of the locus and also the region in which the largest intron in the r-locus is found (Freund et al. 1986). Considering that ATCase and DHOase are normal and CPSase only slightly reduced in Su(b)$^{80\ell}$ (Kolbak 1986), and that this mutant does not exhibit a rudimentary phenotype, the possibility exists that Su(b) is defining a gene, the function of which is not related to the rudimentary locus (i.e., a gene within a gene, as exemplified by the Gart locus [Heinikoff 1986]). In a search for EMS induced suppressor of black mutants, six recessive mutants allelic to su(b)^{31} (Sherald 1981) were found. Independently, a spontaneous dominant Su(b) called Su(b)^{DK7} (Kolbak 1986) was found, indicating that EMS does not induce mutants with the dominant suppressor of black phenotype.

References: Carlson, A.P. 1971, Genet. Res. 17:53-81; Freund, J.N., W. Vergis, P. Schedl & B.P. Jarry 1986, J. Mol. Biol. 189:25-36; Kolbak, D. 1986, Thesis, Univ. of Copenhagen; Pedersen, M.B. 1982, Carlsberg Res. Commun. 43:391-400; Sherald, A.F. 1981, Mol. Gen. Genet. 183:102-106.

Søndergaard, L. and K. Sick. University of Copenhagen, Denmark. Evidence of geoneutrality in **Drosophila melanogaster** larvae, with respect to pupation site preference.

Pupation height, i.e., the distance from the surface of the medium to the site of pupation in culture vials, has been used as an expression for an ascending tendency (or general locomotion activity) of wandering third instar Drosophila larvae (Markow 1979). As pointed out by this author, ordinary culture conditions only allow larvae to go up when they leave the medium to pupate, and since they may in fact respond to a number of environmental factors within the vials, the merit of pupation height for judging the intensity of geotactic behavior is uncertain.

To get an impression of the usefulness of pupation height as a measure of the ascending tendency, we took advantage of the fact that when one hundred or more larvae are feeding on our yeast-agar medium, this becomes fluid before the larvae reach third instar. We placed cultures (**D.melanogaster**; Oregon wildtype) at a slant of 45°. When the larvae are about to pupate the medium is fluid and its surface horizontal and at an angle of 45° to the side of the vials. The vials were kept in darkness at 25°C and were photographed when all (or nearly all) larvae had pupated. Figure 1 shows that pupation height is clearly of no significance with respect to pupation site preference, whereas the distance from the plug is decisive. This indicates larval response to some atmospheric gradient such as humidity or CO_2/O_2 tension.

Figure 1. Pupation location in a vial which has been kept at a slant of 45° during larval and early pupal development.
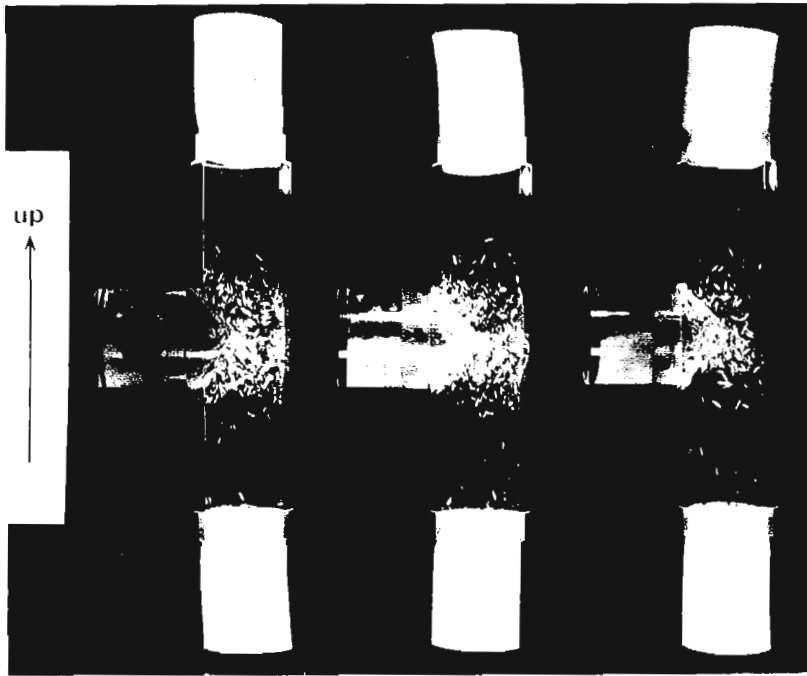
Figure 2. Pupation location in vertical tubes. Wandering 3' instar larvae have entered the tubes from the detachable food cups half way up the cylinders.

In order to make it possible for the wandering larvae to move both up and down after having left the medium for pupation, we used especially designed plexiglass split vials that are open at both ends and which have a detachable cup at the middle of the cylinder for the medium. New polyether-foam plugs were inserted to the same depth at both ends to ensure equal ventilation of the two arms. Before being attached to the experimental vial the cup with yeast-agar medium was exposed to a number of egg-laying **D.melanogaster** females (Oregon wildtype) in an ordinary split vial for 24 hr. The cultures were set up with the cylinder in a vertical position. A partition kept the fluid medium within the cup, but still allowed the larvae to move freely into the vertical cylinder. They were kept under the same conditions and photographed at the same stage as described above.

Even without applying statistics to the results, it is obvious from Figure 2 that there is no indication of an ascending or descending tendency in larval locomotion before pupation. We, therefore, conclude that third instar larvae of our strain are geoneutral, when wandering before pupation.

References: Markow, T.A. 1979, Behav. Genet. 9:209-217.

Stark, W.S. and J.M. Eidel. University of Missouri, Columbia. Ultraviolet phototaxis of Drosophila in the Hirsch-Hadler maze.

The phototactic behavior of the fly can be quite complex. On the one hand, there is fast phototactic choice in a simple Y-shaped arena, the concentration of this laboratory's approach (Stark et al. 1976; Hu & Stark 1977, 1980; Miller et al. 1981; Johnson et al. 1982). In this approach, Drosophila are highly positive in their phototaxis, and each receptor type in the compound eye (R1-6, R7 and R8*) mediates positive phototaxis at its own intensity range of function. Many different experimental paradigms have been used to study phototaxis in many different laboratories, but one of the most striking differences involves the behavior of flies in the Hirsch-Hadler maze (see Stark et al. 1981 and above references for a more thorough bibliography). This apparatus, which has been used to partition flies for genetic selection on the basis of phototactic ability, shows unselected flies to be photoneutral (having neither positive nor negative phototactic preference) or slightly photonegative.

At all but the dimmest light levels, flies were highly attracted to ultraviolet (UV) light in our fast phototaxis experiments (Hu & Stark 1977, 1980); comparisons among strains with vs without specific receptor types (genetic dissection) showed that this attraction was mediated by R7, a UV receptor. Because spectral manipulations were absent from most studies using the Hirsch-Hadler maze, we had initiated a study using calibrated blue stimuli in the Hirsch-Hadler maze (Stark et al. 1981). In the present pilot study, we extend the earlier work using UV light.

Specifically, a 10 outcome Hirsch-Hadler maze was constructed from UV transmitting Plexiglas (Rohm & Haas). The half transmission cut off of 1/2 inch of UV transmitting Plexiglas is at 310 nm while the half cut off of the standard Plexiglas is around 355 nm (determined on a Gilford update of a Beckman DU spectrophotometer). A bank of 6 blacklight (UV) 15 W fluorescent tubes (2 were Westinghouse F15T8/BL and 4 were General Electric F15T8-BLB) were mounted about 8 inches above the maze. These lights transmit above 300 nm, peaking around 350 nm and having numerous sharp peaks; the Westinghouse

lights have a little more light at visible wave-lengths. Calibrating such broad band light is difficult. We measured about 15 log quanta/cm^2 x s incident on the maze (and presumably slightly less inside the choice points). This is fairly intense relative to the levels used in our earlier work. We used and EG & G HUV 4000 B photodiode for the calibration (see Miller et al. 1981). White eyed (cn bw) flies were given overnight to make their choices.

The accompanying figure plots the experimental and binomial distribution of flies in our experiment using 194 flies in 6 runs. Outcome 10 is toward the light. The figure shows that the flies have a slight negative phototaxis in the UV. The difference in the distributions is significant beyond the 0.01 level (Kolmogorov-Smirnov nonparametric test, Siegel 1956). If this intensity of light had been used in our fast phototactic arena, flies would have been very positively phototactic. Instead, they are slightly negatively phototactic in the Hirsch-Hadler maze, even to UV light. These data corroborate the earlier suggestion that phototactic behavior depends substantively on the task involved. Further studies should take advantage of selective receptor mutants and control of spectral stimuli in order to determine the receptor specific mediation of phototactic behavior in the Hirsch-Hadler maze.

References: Hu, K.G. & W.S. Stark 1977, J. Comp. Physiol. 121:289-305; _____ & _____ 1980, J. Comp. Physiol. 135:85-95; Johnson, M.A., K.L. Frayer & W.S. Stark 1982, J. Insect Physiol. 28:233-242; Miller, G.V., K.N. Hansen & W.S. Stark 1981, J. Insect Physiol. 27:813-819; Siegel, S. 1956, Nonparametric statistics for the behavioral sciences, McGraw Hill, New York; Stark, W.S., K.G. Hu & R.B. Srygley 1981, DIS 56:131-132; _____, A.M. Ivanyshyn & K.G. Hu 1976, Naturwissenschaften 63:513-518.

Stark, W.S., C.R. Hartman, R. Sapp, S.D. Carlson,* P. Claude* and A. Bhattacharyya.* University of Missouri, Columbia; *University of Wisconsin, Madison. Vitamin A replacement therapy in Drosophila.

There has been a recent upsurge of interest in vitamin A metabolism relative visual to function in the Dipteran fly. Experimental strategies have involved mutants of **Drosophila melanogaster** (e.g., ninaD) which have impaired vitamin A metabolism (Giovannucci & Stephens 1986). Furthermore, flies (including Drosophila) and several other insect species have been shown to utilize 3-hydroxy-retinal rather than retinal as the visual pigment's chromophore (Vogt 1984; Vogt & Kirschfeld 1984; Goldsmith et al. 1985, 1986; Tanimura et al. 1986; Seki et al. 1986). The blowfly Calliphora was found (Schwemer 1983, 1984) to have no trans to cis isomerase for its chromophore but rather photoisomerization could be effected by a blue or violet light to restore the native visual pigment's precursor.

Those background studies impelled us to continue this laboratory's earlier work (Stark 1977) on dietary vitamin A supplementation in Drosophila. In particular, it was reported earlier (Stark 1977) that visual sensitivity (assayed by the electroretinogram, ERG) in vitamin A reared flies could be restored by feeding adult flies carrot juice. One shortcoming of this earlier work was that no direct measurements of visual pigment were made. Here we report the restoration of rhodopsin levels in Drosophila eyes as determined by microspectrophotometry (MSP, Stark & Johnson 1980; cf. Stark et al. 1985). Further we determined number of P face particles (putative rhodopsin molecules) using transmission electron microscopy of platinum freeze fracture replicas (e.g., Harris et al. 1977; Stark & Carlson 1983).

The accompanying graph depicts the recovery of rhodopsin as measured using MSP. The methodology for determining visual pigment levels from R1-6 receptors of the eyes of living Drosophila (Stark & Johnson 1980) and its application to measured altered rhodopsin levels (recently, Stark et al. 1985) are presented in detail elsewhere. In the present study male white-eyed (cn bw) flies were reared from egg to adult on Sang's medium (deficient in vitamin A, see Stark et al. 1977). Adults were then isolated in vials

"VITAMIN A REPLACEMENT"

ABSORBANCE DIFFERENCE

DAYS OF VITAMIN A REPLACEMENT

with nothing to consume except for carrot juice (soaked in a Kimwipe) for vitamin A "replacement therapy." Plotted is absorbance difference, a measurement of the relative visual pigment level (see Stark et al. 1985), as a function of days of replacement. For comparison, a control level was measured from white-eyed flies reared on our normal medium with yellow corn meal. That diet supplies adequate vitamin A to Drosophila (Stark 1977; Stark et al. 1977). As a precaution we supplemented that "normal" diet with beta-carotene (Miller et al. 1984). Basically, the graph shows that our vitamin A replacement therapy is completely effective within one week.

In order to fully verify this finding, we freeze fractured Drosophila heads (from animals which had been maintained on carrot juice for 6 days) for transmission electron microscopy (see Stark & Carlson 1983 for methods). We found that such flies had 4308 ± 254 (SE based on 6 different measurements) P face particles per square micron in the rhabdomeric microvilli. This figure can be compared with the data for vitamin A deprived Drosophila (1030 ± 31) vs for control, i.e., vitamin A replete, Drosophila (4214 ± 163) (Harris et al. 1977). Most of the particles are thought to be rhodopsin molecules. Basically, our therapy brings the membrane particle count up to the control level by the end of a week of replacement.

It is important to emphasize that the adult, with its fully formed photoreceptors, can be fed vitamin A and can make visual pigment from it. In this context, it is useful to know that carrot juice, a convenient source of vitamin A, is effective. We expect that in the near future there will be a considerable number of papers on vitamin A metabolism in Drosophila. Yet to be answered with certainty are questions concerning the relative effectivenesses of different forms of vitamin A based on their convertability to the visual pigment chromophore.

References: Giovannucci, D.R. & R.S. Stephenson 1986, Invest. Ophth. Vis. Sci. Suppl. 27:191; Goldsmith, T.H., G.D. Bernard, B.C. Marks & I. Tangoren 1985, Invest. Ophth. Vis. Sci. Suppl. 26:293; _____, B.C. Marks & G.D. Bernard 1986, Vision Res. 26:1763-1769; Harris, W.A., D.F. Ready, E.D. Lipson, A.J. Hudspeth & W.S. Stark 1977, Nature (Lond.) 266:648-650; Miller, G.V., K.A. Itoku, A.B. Fleischer & W.S. Stark 1984, J. Comp. Physiol. A. 154:297-305; Schwemer, J. 1983, Biophys. Struct. Mech. 9:287-298; _____ 1984, J. Comp. Physiol. A. 154:535-547; Seki, T., S. Fujishita, M. Ito, N. Matsuoka, C. Kobayashi & K. Tsukida 1986, Vision Res. 26:255-258; Stark, W.S. 1977, DIS 52:;47; _____ & S.D. Carlson 1983, Cell Tiss. Res. 233:305-317; _____, A.M. Ivanyshyn & R.M. Greenberg 1977, J. Comp. Physiol. 121:289-305; _____ & M.A. Johnson 1980, J. Comp. Physiol. A. 140:275-286; _____, K.D. Walker & J.M. Eidel 1985, Curr. Eye Res. 4:1059-1075; Tanimura, T., K. Isono & Y. Tsukahara 1986, Photochem. Photobiol. 43:225-228; Vogt, K. 1984, Z. Naturforsch. 39c:196-197; _____ & K. Kirschfeld 1984, Naturwissen. 71:S.211-212.

Starmer, W.T. and J.S.F. Barker.* Syracuse University, Syracuse, New York; *University of New England, Armidale, Australia. The effects of larval density on the development of D.buzzatii in Opuntia stricta tissue.

It is well known that density is an important factor affecting larval development, viability and subsequent adult size. Mangan's (1982) studies on cactophilic Drosophila suggest that there may be nonlinear effects of larval density on these characters and that these effects are dependent on food quality and competitive interactions. We were particularly interested in the existence of an optimal density for larvae in the natural habitat and if an Allee effect (Allee 1931) were present. We have made preliminary studies on the effects of larval density on the growth of D.buzzatii in decaying Opuntia stricta cladodes. The experiments were conducted by seeding increasing numbers (1, 2, 8, 32 and 128) of 0-1 hour old, axenic, first instar larvae onto 5 gram pieces of O.stricta cladode tissue (previously homogenized, autoclaved and inoculated with the cactus yeasts Pichia cactophila and Candida sonorensis). The tests were run in 30 ml shell vials at 25°C with the above larval densities replicated 128, 64, 32, 8 and 4 times, respectively. Time from egg to adult emergence in days, adult thorax length in mm and percent emergence were observed. One of the 8 32-larvae vials was discarded due to contamination by mold.

Table 1. The effect of larval density (per 5 grams of **Opuntia stricta** tissue) on percent emergence, development time and thorax size.

No. of larvae	No. of replicates	Percent emergence	Females Time Days±SD	Females Size mm±SD	Males Time Days±SD	Males Size mm±SD
1	128	82.8	15.14±0.49	1.17±0.008	15.55±1.02	1.08±0.008
2	64	92.2	15.24±0.06	1.18±0.008	15.63±0.71	1.08±0.008
8	32	80.9	17.14±1.50	1.12±0.009	17.33±1.33	1.05±0.008
32	8	41.1	22.96±2.02	1.11±0.007	23.05±2.49	1.03±0.007
128	4	2.9	27.67±2.60	0.96±0.015	28.33±1.97	0.90±0.013
r^2 for V vs D		0.90	0.86*	0.96*	0.89*	0.99*
r^2 for SD of V vs D			0.71	0.88*	0.32	0.78*

*=significant $P < 0.05$;   V vs D = Variable vs Density

Table 1 lists the percent emergence, time (days) and adult thorax length in mm. It is apparent that percent emergence and adult size decrease and development time increases as density increases. Inspection of Table 1 shows 2 larvae per vial to have the greatest viability (92.2%) but this does not differ significantly ($P > 0.05$) from either 1 larva or 8 larvae per vial treatments. Almost all of the variation in the three variables is due to linear effects of density and there is no significant quadratic or cubic component. We also note that the variation (SD) in development time and thorax size increases as a function of density but that the r^2 is only significant for size (both sexes). We thus conclude that one to eight larvae per 5 grams is optimal under these conditions and that there is no evidence for an Allee effect in **D.buzzatii**.

References: Allee, W.C. 1931, Animal Aggregations: A Study in General Sociology, Univ. of Chicago Press; Mangan, R.L. 1982, "Adaptations to competition in cactus breeding Drosophila," in: Ecological Genetics and Evolution: The cactus-yeast-Drosophila model system, Barker & Starmer (eds.), Academic Press Australia, Sydney, pp257-272.

Stenveld, H. and R. Bijlsma. University of Groningen, Haren, Netherlands. Differences in male mating success in **D.melanogaster.**

Since the results of Prout (1971a, b) and Bundgaard & Christiansen (1972), it has become clear that also the adult component of selection, besides viability selection, is an important component of the total selection during the life cycle of **D.melanogaster.**

In this respect especially factors concerning the mating process, such as mating preference and mating success, seem to be of significance (for a review, see Spiess 1982). It seems, therefore, worthwhile to study different aspects of the mating process. In this paper we want to present some data on male mating success when an excess of females is available. For the experiments we made use of the same fourth chromosome recessive mutant system as used by Bundgaard & Christiansen (1972). This system behaves as a one locus-two alleles system and three visually different genotypes can be distinguished: homozygous **cubitus interruptus shaven-naked** (cisv), homozygous **sparkling poliert** (spa) and the **heterozygote** (het) which is wildtype (for a description of the mutants, see Lindsley & Grell 1967).

The tests were started by bringing together in one vial (without etherization) 4 virgin females of the same genotype with one virgin male. All flies used were 5 days old. The flies were left together for 1 hr. Thereafter, the male was, again without etherization, transferred to a new vial with 4 virgin females, of the same genotype as before, and after one more hour this procedure was repeated. During the three periods the flies were observed and the number of matings that occurred was noted down. This was done for all 9 possible male and female genotype combinations and each combination was replicated five times. The experiments were performed at 22°C.

Table 1 shows the total number of matings observed for each male-female combination during the three 1 hr periods. In most cases the number of matings observed per male in any one period was three or less, as might have been expected from a copulation duration time around 15-25 min in **D.melanogaster.** Only in one case four matings were observed in the same vial within 1 hr. This indicates that the number of virgins was not limiting. A contingency χ^2 for the distribution of the number of matings of the different combinations over the three periods turned out to be not significant: nor for the different combinations within each male genotype (spa: $\chi^2_4 = 2.06$, $0.75>p>0.50$; het: $\chi^2_4 = 5.68$, $0.25>p>0.10$, and cisv: $\chi^2_4 = 4.93$, $0.50>p>0.25$) nor between the different male genotypes ($\chi^2_4 = 1.55$, $0.90>p>0.75$). Therefore, all data can be lumped together. The data show a strong decrease in the number of matings performed during the successive periods. This decrease, approximately 30% from one period to the next, is highly significant ($\chi^2_4 = 39.19$, $p<0.001$). Nevertheless, the data show that the males are quite capable of performing a number of matings within a relatively short period and can even exceed six successive matings within three hr (some **het x spa** combinations).

Table 1. Total number of matings performed by the different male genotypes during the three 1 hr periods.

male X female	1st period	2nd period	3rd period
spa X spa	9	9	1
X het	10	7	4
X cisv	7	5	2
total spa males	26	21	7
het X spa	15	12	3
X het	8	2	5
X cisv	12	11	6
total het males	35	25	14
cisv X spa	7	2	0
X het	7	3	4
X cisv	8	7	3
total cisv males	22	12	7
TOTAL ALL MALES	83	58	28

Table 2. Total number of matings observed over the whole three hr period for the different genotype combinations.

FEMALES		MALES : spa	: het	: cisv	: total
	spa	19	30	9	58
	het	21	15	14	50
	cisv	14	29	18	61
	total	54	74	41	

Although the different genotype combinations showed no differences with respect to the relative decrease in the number of matings during the successive periods, substantial differences in the total number of matings were observed. Table 2 shows these total numbers summed for each genotype combination over the whole three hr period. First of all, a contingency χ^2 for the interaction between males and females turned out to be not significant ($\chi_4^2 = 9.15$, $0.10 > p > 0.05$). Therefore, male and female totals may be treated separately. For the females there are no significant differences between the genotypes with respect to the number of copulations ($\chi_2^2 = 1.67$, $0.95 > p > 0.90$), although the heterozygous females appear to be somewhat less receptive than the other genotypes. The males, on the other hand, show significant differences between the genotypes. The heterozygous males have performed significantly more matings than both homozygous genotypes ($\chi_2^2 = 14.28$, $0.01 > p > 0.005$). The heterozygous males are particularly successful with respect to their own genotype. This may indicate that the heterozygous females are more reluctant than the other female genotypes. Using the terminology of the model evolved by Van den Berg (1986), the fact that males are more successful and females more reluctant leads to the conclusion that the heterozygous genotype is the sexually most vigorous of the three. Under the assumption that the homozygous lines were to some degree inbred, this higher sexual vigor is most probably the result of a general hybrid vigor which is often observed when two inbred strains are crossed. The phenomenon that hybrid males are sexually the most successful has also been observed by Kosuda (1983). In conclusion, we can say that the differences in male mating success form a significant part of the sexual selection component observed by Bundgaard & Christiansen (1972) for this mutant system.

References: Bundgaard, J. & F.B. Christiansen 1972, Genetics 71:439-460; Lindsley, D.L. & E.H. Grell 1967, Carnegie Inst. Publ. 627; Kosuda, K. 1983, Experientia 39:100-101; Prout, T. 1971a, Genetics 68:127-149; _____ 1971b, Genetics 68:151-167; Spiess, E.B. 1982, Am. Nat. 119:675-693; Van den Berg, M.J. 1986, Am. Nat. 127:796:808.

Syomin, B.V. and N.G. Schuppe. Inst. of General Genetics, Moscow, USSR. Difference in intracellular localization of retrotransposon reverse transcription intermediates.

A lot of experimental data are consistent with a model that retrotransposons -- mobile genetic elements resembling structurally the proviral forms of the retroviruses -- use for their transposition reverse transcription pathway (Boeke et al. 1985; Arkhipova et al. 1986). Reverse transcription intermediates -- hybrid molecules consisting of single stranded DNA of retrotransposons and suitable poly(A)+RNA -- were isolated from **Drosophila melanogaster** cultured cells and the steps of their formation were studied (Arkhipova et al. 1984; 1986). Virus-like particles possessing reverse transcriptase activity (Schiba & Saigo 1983) and containing RNA of retrotransposon copia were were detected in cultured Drosophila cells and such particles may be artificially induced in yeast cells transformed by Ty1- containing plasmids (Garfinkel et al. 1985; Mellor et al. 1985).

In our preceding paper (Syomin & Schuppe, in press), we have described the intracellular distribution of reverse transcription intermediates of retrotransposon mdg1 in different subcellular fraction. The study reported herein describes that intermediates of various retrotransposons are distributed between subcellular fraction unlikely.

The 67j25D cultured **D.melanogaster** cells were divided on three fractions. The nuclei were isolated according to the method of Mayrand & Pederson (1983). After centrifugation of the postnuclear supernatant at 15,000 g for 20 min, the pellet fraction was named membrane, and the supernatant fraction

Figure 1. Southern blot analysis of reverse transcription intermediates of mdg1 and mdg3. 1 = membrane; 2 = cytoplasmic; 3 = nuclear fractions.

was named cytoplasmic. The nucleic acids were extracted from all fractions, fractionated on agarose gels, transferred onto nitrocellulose filters and analysed by hybridization with plasmids, containing full sequences of different retrotransposons (for details of the methods, see Arkhipova et al. 1984).

To obtain the distribution of intermediates in the same cells, one filter was subsequently hybridized with plasmids containing different retrotransposons. First of all, data obtained show that reverse transcription intermediates can be detected in all fractions of choice (Figure 1). Hybrid molecules of mdg1 correspond to full-sized molecules of retrotransposons. DNAs in such hybrids are represented by molecules with one or two LTRs. The main part of mdg1 intermediates is localized in membrane fraction. When copia-containing plasmid was used as a probe for detection of intermediates, the state of their distribution between subcellular fractions was just the same as for mdg1 (data not shown). However, mdg3 intermediates show distribution distinct from mdg1 and copia retrotransposons. In this case, the main part of intermediates were detected in cytoplasmic fraction. Moreover, the intermediates of mdg3 are represented not only by full-sized molecules, but also several bands of hybridization were detected.

The differences in the distribution of intermediates discussed above have respect to membrane and cytoplasmic fractions. As to nuclear fraction, in all cases various retrotransposons have the same distribution. One band corresponds to full-sized molecules, and the second -- about 4 k.b. long -- represents according to our preliminary data the circular extrachromosomal DNA of retrotransposons.

We cannot now explain our data uniformly. It is only evident that peculiarities of intracellular distribution of retrotransposon reverse transcription intermediates reflect peculiarities of retrotransposition cycles of various retrotransposons in the cells. It may also be that retrotransposons can be differently distributed in different cell lines and our data reflect in this case the distribution of certain transposons in cell lines.

References: Arkhipova, I.R., T.V. Gorelova, Y.V. Ilyin & N.G. Schuppe 1984, NAR 12:7533-7548; _____, A.M. Mazo, V.A. Cherkasova, T.V. Gorelova, N.G. Schuppe & Y.V. Ilyin 1986, Cell 44:555-563; Boeke, J.D., D.J. Garfinkel, C.A. Styles & G.R. Fink 1985, Cell 40:491-500; Garfinkel, D.J., J.D. Boeke & G.R. Fink 1985, Cell 42:507-517; Mayrand, S. & T. Pederson 1983, Mol. Cell. Biol. 3:161-171; Mellor, J. S.M. Fulton, M.J. Dobson, W. Wilson, S.M. Kingsman & A.J. Kingsman 1985, Nature 313:243-246; Shiba, T. & K. Saigo 1983, Nature 302:119-124.
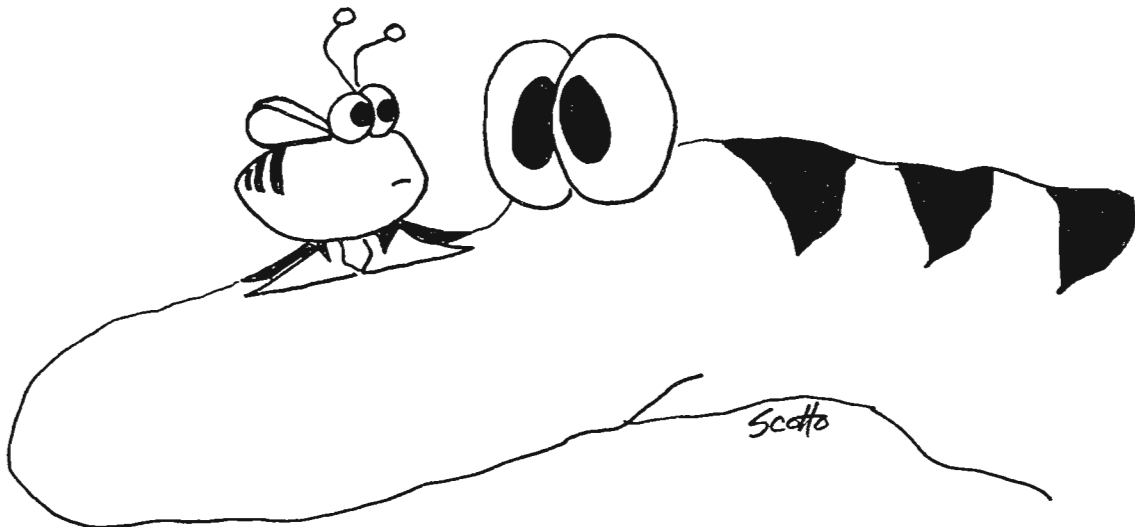
Tarin, J.J. and C. Najera. Universidad de
Valencia, Spain. Alcohol tolerance. I. Effect
of alcohol on longevity of different populations
of **D.melanogaster.**

In order to research the ethanol tolerance in adult
flies of **D.melanogaster** from different populations,
four collections were made in different natural
habitats: the first one in a cellar in Cheste
(Valencia), the second and the third ones at 500 and
1000 m from the cellar and the fourth one in a
pinewood in La Canada (situated at 25 km from the cellar). Other three populations from cellar, vineyard
(at 4 km from the cellar) and pinewood, but maintained in laboratory conditions for five years were studied.

Twenty-two isofemale lines were tested for each one of the seven populations.

Longevity throughout time (from 5 hr to 12 days) was studied by placing the flies in vials containing
filter paper with a water solution of 3% sucrose and 2, 5, 7, 10, 12 or 15% ethanol. For each isofemale line
of each population, two replicate vials were prepared containing 10 flies per vial for each sex.

In Figure 1, the evolution of the LC50 (lethal concentration killing 50% of the flies) throughout the
time can be observed in each population.

By means of a Student-Newman-Keuls multiple range test, differences among the means of the
populations can be observed only on the second and the third day, although statistically they are not
significant. In natural populations the order of tolerance was pinewood > cellar > 1000 m from cellar > 500
m from cellar. In laboratory populations the order of tolerance was cellar > vineyard > pinewood, being
the populations from laboratory more tolerant to ethanol than the natural ones.

The lack of statistically significant differences can be attributed to the big differences found among
the strains within populations, mainly in the natural pinewood population where some strains are twice or
more times more tolerant than others.

A curious fact found was that tolerance of females was higher from 5 hr to 7 days, taking place an
inversion from this moment in the tolerance of the sexes, the males being more tolerant.
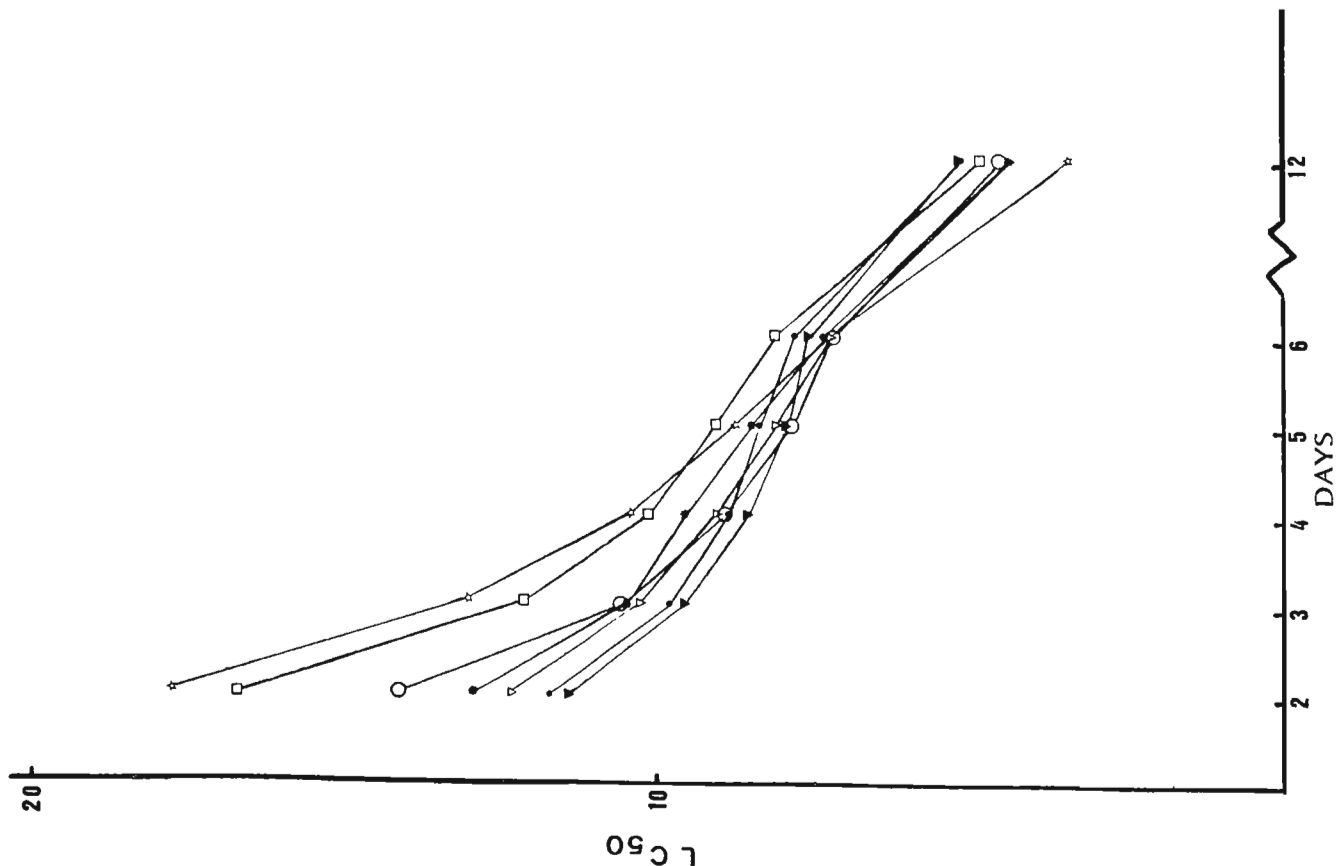
Figure 1. Evolution of the LC50 throughout the time. ∇ = cellar population. ▼ = population 500 m from cellar.
● = population 1000 m from cellar. ＊ = pinewood population. ★ = laboratory cellar population. ◻ =
laboratory vineyard population. ○ = laboratory pinewood population.

Tarin, J.J. and C. Najera. Universidad de
Valencia, Spain. Alcohol tolerance. II. Effect
of alcohol on viability of different populations
of D.melanogaster.

In order to prove the influence of alcohol on the
viability of the same seven populations of
D.melanogaster described in the previous work, four
levels of alcohol concentration (0, 5, 10 and 15%)
were used. The same 22 isofemale lines were tested
in each population. One hundred eggs were placed
in each vial and a total of five replicas were made for each strain and each alcohol concentration.

In Figure 1, the cocients between the mean viabilities at the different alcohol concentrations and
the mean viability at 0% are represented.

A cline in the tolerance between the three closest natural populations (cellar, 500 m, 1000 m) can
be observed and the pinewood natural population has the highest mean viabilities except at 10% where the
viability of cellar and pinewood was the same.

The laboratory populations have lower mean viabilities than the natural ones at 5%, the vineyard
being the most tolerant at 5% and the pinewood at 10%. Nevertheless, differences among populations are
not very high.

By means of a three-way ANOVA made for each population, it can be observed that strains affect
viability in all populations the same as alcohol concentration. Moreover, the sex has no significance except
in the laboratory cellar population. The effect of interaction between strains and alcohol concentration
is always significant and the only population where the interaction of alcohol concentration-sex was
significant was the natural pinewood one, due to the fact that males are more tolerant to ethanol than
females. The interaction of strains-sex is never significant.
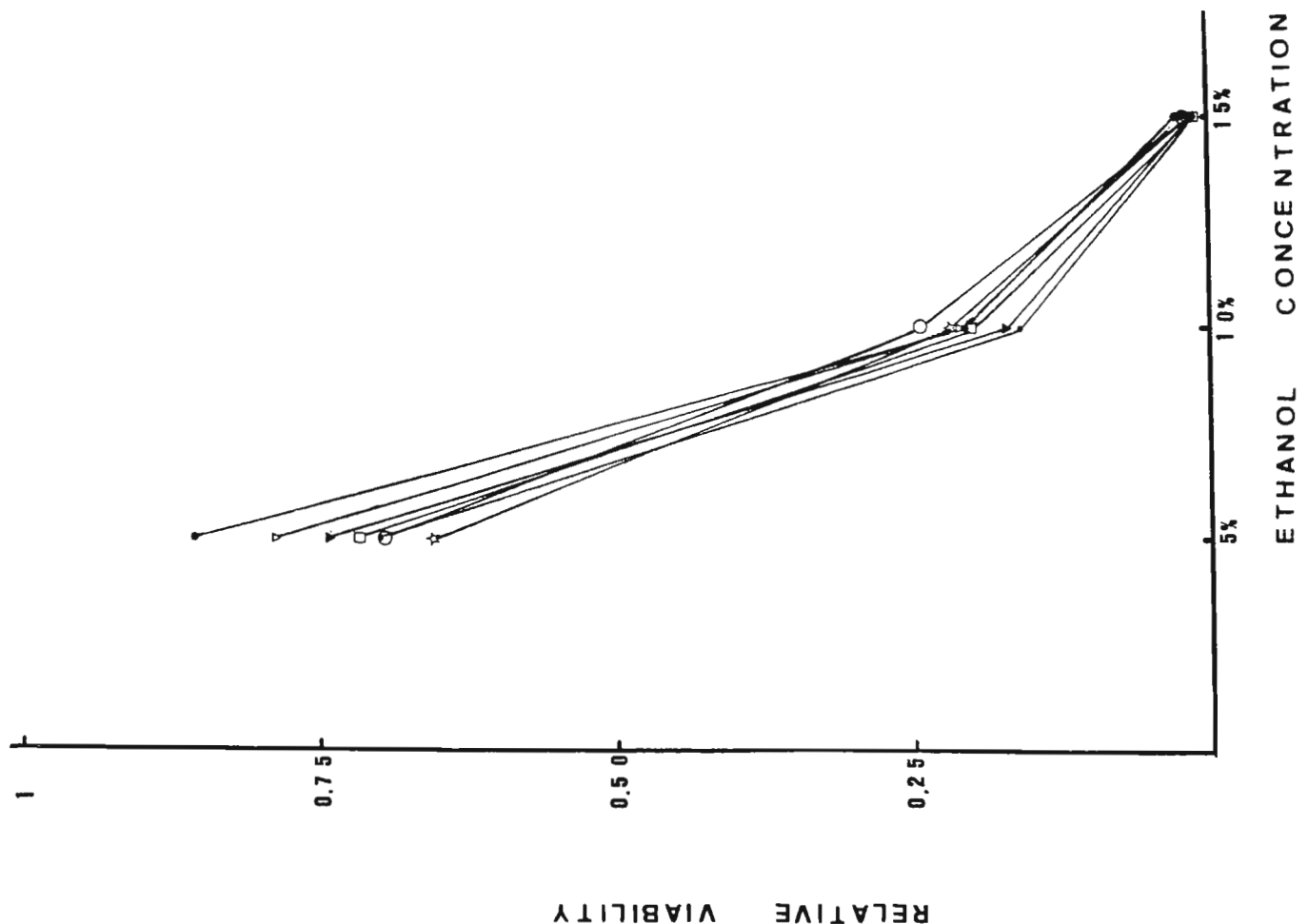
Figure 1. Relative viabilities at different ethanol concentrations. ∇ = cellar population. ▼ = population 500
m from cellar. ● = population 1000 m from cellar. ＊ = pinewood population. ⭐ = laboratory cellar
population. ◘ = laboratory vineyard population. 0 = laboratory pinewood population.

Thompson, V., M. Mayfield and K. Venkatachalam.
Roosevelt University, Chicago, Illinois. New host
record for the spittle maggot **Cladochaeta inversa.**
Larvae of the drosophilid genus **Cladochaeta** inhabit
nymphal spittle masses of the spittlebug genus **Clas-
toptera** (Homoptera). Both genera are predominantly
neotropical but several clastopteran species and the
fly **Cladochaeta inversa** occur as far north as Quebec.
To date **C.inversa** has been reported from just two host spittlebugs, **Clastoptera obtusa** (the alder
spittlebug) in the eastern United States, and **C.lineaticollis** in California (review in Ashburner 1981). We
have found **C.inversa** larvae and pupae in spittles of a third species, **Clastoptera proteus** (the dogwood
spittlebug), at three Chicago area sites, in each case on the leaves and stems of shrubby dogwoods (**Cornus
spp.**).

At the Little Red School House Nature Center (Cook Co., Ill., June 20, 1986), 10 of 368 **C.proteus**
nymphs examined were associated with **C.inversa** larvae and three with **C.inversa** pupae, an infestation rate
of 3.5%. At the Sand Ridge Nature Center (Cook Co., Ill., June 26, 1986), four larvae and three pupae were
found among 326 **C.proteus** nymphs, an infestation rate of 2.1%. At Bridgeman, Michigan (July 5, 1986),
four pupae were found among 27 **C.proteus** nymphs. The larvae and pupae were always found within the
spittle mass proper (the larvae often directly on the spittlebug nymph), with one larva or pupa per spittle.

At the Bridgeman site an American hop hornbeam tree (**Ostrya virginiana**) about five meters from
the **C.proteus** infested dogwood shrubs harbored a population of **C.obtusa.** Almost every **C.obtusa** spittle
had associated **C.inversa** pupae, in many cases several (two larvae were also observed). Most of these
pupae lay close to but outside the spittle mass proper. Specific identities of the flies and spittlebugs from
all three sites were confirmed by rearing adult flies from pupae and adult spittlebugs from late instar
nymphs.

These observations stand in contrast to Baerg's 1920 report that **C.inversa** was absent from
C.proteus spittles while common in nearby **C.obtusa** spittles (locality of observations unspecified).
Evidently **C.obtusa** is a preferred host, but **C.proteus** is an acceptable alternative. There is also a single
record of a **C.inversa**-like larva associated with the red cedar spittlebug **C.arborina** (Kuenzi & Coppel
1985). Close observation of other **Clastoptera** species may disclose an even broader host range for
C.inversa.

References: Ashburner, M. 1981, in: Genetics and Biology of Drosophila (Ashburner, Carson & Thompson, eds.),
Academic Press, NY, 3a:395-429; Baerg, W.J. 1920, Entom. News 31:20-21; Kuenzi, F.M. & H.C. Coppel 1985, Trans.
Wis. Acad. Sci. Arts Lett. 73:144-153.

Umbetova, G.H. and I.F. Zhimulev. Institute of
Cytology & Genetics, Novosibirsk, Moscow, USSR.
Structure and transcriptional activity of the
pompon-like X chromosomes of l(3)tl mutant of
Drosophila melanogaster.
It is known that male X chromosome in the l(3)tl
mutant of **Drosophila melanogaster** is short, without
clear banding pattern and looks like a structureless
mass (Kobel & Breugel 1968; Zhimulev et al. 1976).
Similar chromosomes appear in polytene cells of Scia-
rids after microsporidian infection (Pavan & Basile
1966), in normal development of **Phryne cincta** (Wolf
1957) and in many other cases. This kind of structure was called "pompon"-like chromosomes and according
to the opinion of Roberts et al. (1967), the whole chromosome represents a generalized puff. However,
extra ^{3}H-uridine incorporation was not found in "pompons" (Zhimulev et al. 1976; Wolf & Sokoloff 1976;
Lakhotia & Mishra 1982). It is not known what happens with individual chromonemas of the X polytene
chromosome when it turns into "pompon". It was shown that some stability of longitudinal arrangement
of the "pompon"-like chromosomes still retains because some clear cut bands, probably 9A1-4 or 11A6-9
in "pompons" appearing after long-term cultivaton of the salivary glands in imago abdomen, are visible
(Zhimulev & Belyaeva 1976). A set of late replication regions characteristic for the normal X chromosome
can be distinguished in "pompons" as well (Lakhotia & Mishra 1982).

To get new information on the "pompon"-like chromosomes of the l(3)tl mutant, the following
experiments were performed: To estimate transcriptional activity of the "pompon"-like chromosomes, the
method of fluorescent antibodies against DNA:RNA hybrids was used. DNA on squashed preparations of
the salivary gland chromosomes was denatured, reannealed, incubated with goat antihybrid serum and
stained with fluorescein labelled rabbit IG as described by Vlassova et al. (1985). The method permits
localizing both RNAs, nascent and already synthesized in situ. In Figure 1 showing two nuclei with
"pompon"-like chromosomes, it is seen that intensity of the autosome fluorescence is high; however, that
of the "pompons" is dull. It is rather evident that there is no extra synthesis of RNA in this type of
chromosome.
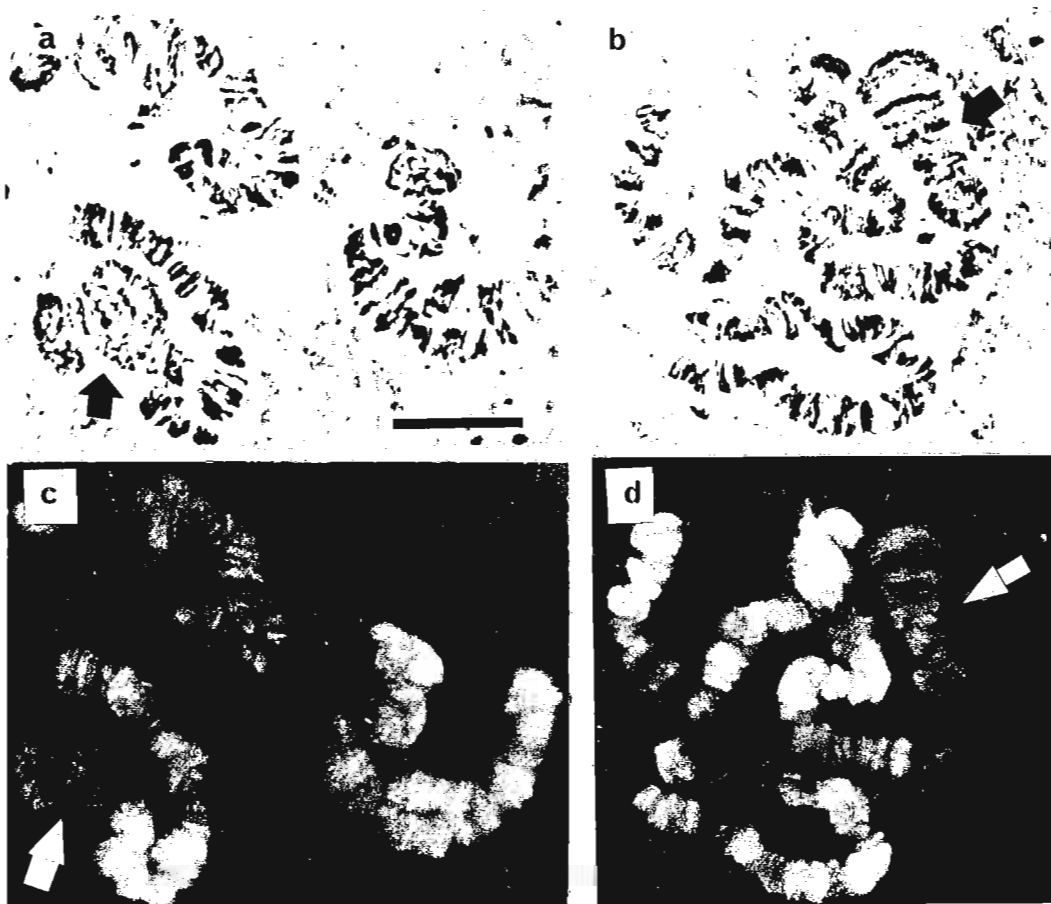
Figure 1. Fluorescence of the chromosome sets after administratioan of squashed preparations with labelled antibodies against DNA:RNA hybrids. Arrows indicate "pompon"-like X chromosomes. The same nuclei are shown under phase control (a,b) and in fluorescence microscope (c,d). The bar represents 10 micra.

To get information on organization of the chromosome fibrils in the "pompons", a DNA clone with known localization was used. The DmP202 genomic clone was obtained as a result of chromosome walking. It is located by in situ hybridization on distal end of normal X chromosome in the 2B3-5 region (Belyaeva et al. 1987). In situ hybridization with "pompon"-like X chromosomes reveals several types of localization: on the very tip of "pompon" (Figure 2d), on the tip but with disperged labelling (Fig. 2f). Fig. 2e gives an example of several stripes of labelling. As a whole, the results show that during "pompon" formation irregular mixing of chromosome fibers does not occur.

To explain the phenomenon of "pompon"-like chromosomes, we can suggest that during their formation complete (chromomere to chromomere) conjugation of the individual chromatides is distributred without changes of transcriptional activity. As a result, only some chromomeres can contact to homologous chromomeres of neighbouring chromatides, and the whole polytene chromosome turns into a net with partial conjugation of chromomeres (Figure 3). Probably, the bigger the chromomeres, the stronger their conjugation; that is why in "pompon" some bands are still visible (Zhimulev & Belyaeva 1976). Some longitudinal order is still maintained; therefore, in situ hybridization of definite DNA fragment gives rise to a strip of labelling. Pattern of labelling is easier for interpretation if we allow that the net of chromatides is represented by flat ribbon. When the chromomere conjugation is distri-
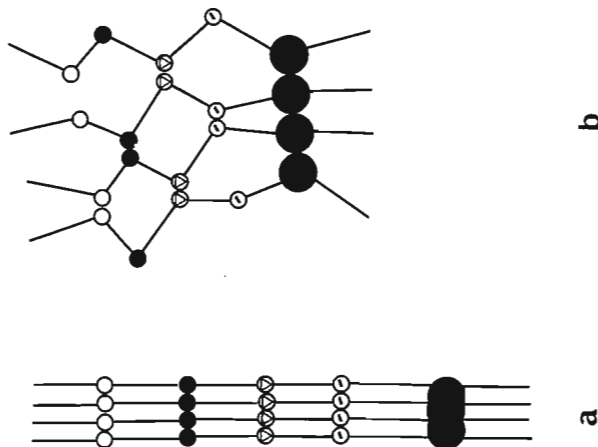
Figure 3. Schematic representation of the individual chromonemas in normal (a) and "pompon"-like (b) chromosomes.

buted, the whole chromosome becomes shorter. Shortening of the "pompons" is strengthened for some other reasons, because all autosomes in the l(3)tl mutant are very short as well (Zhimulev et al. 1976).

References: Belyaeva, E.S. et al. 1987, Chromosoma 95:295; Kobel, H.R. & F.M.A. van Breugel 1968, Genetica 38:305; Lakhotia, S.C. & A. Mishra 1982, Ind. J. Exp. Biol. 20:643; Pavan, C. & R. Basile 1966, Science 151:1556; Roberts, P.A. et al. 1967, Exp. Cell Res. 47:408; Vlassova, I.E. et al. 1985, Chromosoma 91:251; Wolf, E.B. 1957, Chromosoma 8:396; _____ & S. Sokoloff 1976, in: Chromosomes Today 5:91; Zhimulev, I.F. & E.S. Belyaeva 1976, Tsitologia (USSR) 18:5; _____ et al. 1976, Chromosoma 55:121.

Figure 2. In situ hybridization of the DmP202 from the X chromosome 2B3-5 puff to "pompon"-like X chromosomes of l(3)tl (arrows). Bars representing 10 micra in "a" and "c" are common for "b", "f" and "d", "e", respectively.

Van Breugel, F.M.A. and B.W. van Zijll Langhout. University of Leiden, Netherlands. Determination of aldehydeoxidase activity in amputated legs of Drosophila: information about the genotype of living flies.

To establish enzymatic defects in Drosophila, one usually has to kill the animal. It is possible, however, to get information about the living flies from a single amputated leg. When amputation is properly done, the fly will stay alive and may reproduce afterwards. Amputation of the legs was carried out with a pair of fine scissors (Albert Heiss H-4240) using a binocular while the anesthesized fly was kept in position with a fine-pointed forceps. The amputated legs were fixed for 15 min in ice-cold insect salt solution containing 1% glutaraldehyde as a fixative. Subsequently the fixative was removed by a 15 min wash in normal ice-cold insect salt solution and the legs were then stained 1 hr for the presence of aldehyde oxidase activity in the following mixture: 5 ml 0.2 M Tris-HCl buffer pH 8.0, 2.5 mg dimethylaminobenzaldehyde (DMAB), 2.5 mg nitrobluetetrazolium (NBT) and 0.12 ml of a fresh 2 mg/ml solution of phenazinemethosulphate (PMS).

In the cross-sectioned femora of the fore-mid-or hind leg a dark plue precipitate of reduced NBT was observed on the wound surface of wild type legs. No colour developed in an incubation medium lacking the substrate DMAB. It is known that both aldehyde oxidase (AO) and pyridoxal oxidase (PO) are able to oxidize DMAB. To determine which of these enzymes caused the reduction of NBT in the femora, we tested the mutant strains Aldox^{n1} sbd (lacking AO-activity), se lpo (lacking PO-activity) and y v ma-1 (lacking both AO- and PO-activity) with the respective AO- and PO-specific substrates heptaldehyde and 2,4,5 trimethoxy benzaldehyde (TMB) (Cypher et al. 1982). The results (Table 1) show that in the femora only AO-activity is present. The AO-activity seems to be mainly located in the connective tissues to which the muscles are attached. A more detailed analysis is needed, however.

Table 1. Enzyme activities in cross-sectioned femora of **D.melanogaster** with various substrates.

	DMAB (AO; PO)	Heptaldehyde (AO)	2,4,5 TMB (PO)
Wild type	+	+	-
Aldox^{n1} sbd	-	-	-
se lpo	+	+	-
y v ma-1	-	-	-

Amputation in the coxal, tibial or tarsal area of legs gave the same results as femoral cuts. However, various joints (as for example between the coxa and femur, the femura and tibia, or between the tibia and first tarsal element) showed some traces of NBT staining. This even occurred in the aldehyde oxidase negative mutants and suggests the local presence of another NBT reducing substance or enzyme system. This means that for a search of mutants of the AO-controlling system, amputation in the mid of the coxa, femur or tibia of any of the legs is required. Although we have not tried amputated legs for the diagnosis of other enzyme defects, we infer that more biochemical mutants can be traced with this method provided that some histochemical staining method for the enzyme in question is available and on the condition that the gene in question is expressed in the tissues of the legs.

References: Cypher, J.J., H.L. Tedesco, J.B. Courtright & A.K. Kumaran 1982, Bioch. Genet. 20:315-332.

Van Delden, W. and A. Kamping. University of Groningen, Haren, Netherlands. Differential resistance of Adh genotypes to starvation.

Survival of alcohol dehydrogenase (Adh) genotypes in **Drosophila melanogaster** in the presence of toxic concentrations of alcohols is positively correlated with their in vitro ADH activities (reviewed in Van Delden 1982). Evidence for balancing selection at the Adh locus in polymorphic populations in the absence of alcohols was obtained by Van Delden et al. (1978). It thus appears that ADH plays a metabolic role apart from the detoxification of alcohols. This conclusion is confirmed by the results obtained from Adh null mutants. Strains homozygous for Adh null mutants can easily be maintained under regular laboratory conditions. Populations polymorphic for Adh null alleles and Adh positive alleles (either Adh^F or Adh^S), however, show a gradual decrease in Adh null frequency in the course of time (Van Delden & Kamping 1981). The latter finding points to a selective disadvantage of genotypes lacking ADH activity even in the absence of alcohols.

In an earlier report (Van Delden & Kamping 1986), we have shown that the Adh^{FF} homozygote (FF) has a higher resistance to starvation than the Adh^{SS} homozygote (SS). This leads us to perform an experiment where survival of adults was recorded over time on agar medium. For this purpose four homozygous strains were compared: FF, SS, O(F) and O(S). The first two strains were derived from the Groningen population and were homozygous for the Adh^F and Adh^S alleles, respectively. The O(F) and O(S) strains were both homozygous for the Adh^{n1} null allele (Grell et al. 1968) which had been introduced into the background of the FF and SS strains, respectively. These null strains were identical with their Adh positive counterparts except for the Adh region. Before the starvation test the flies were kept on regular
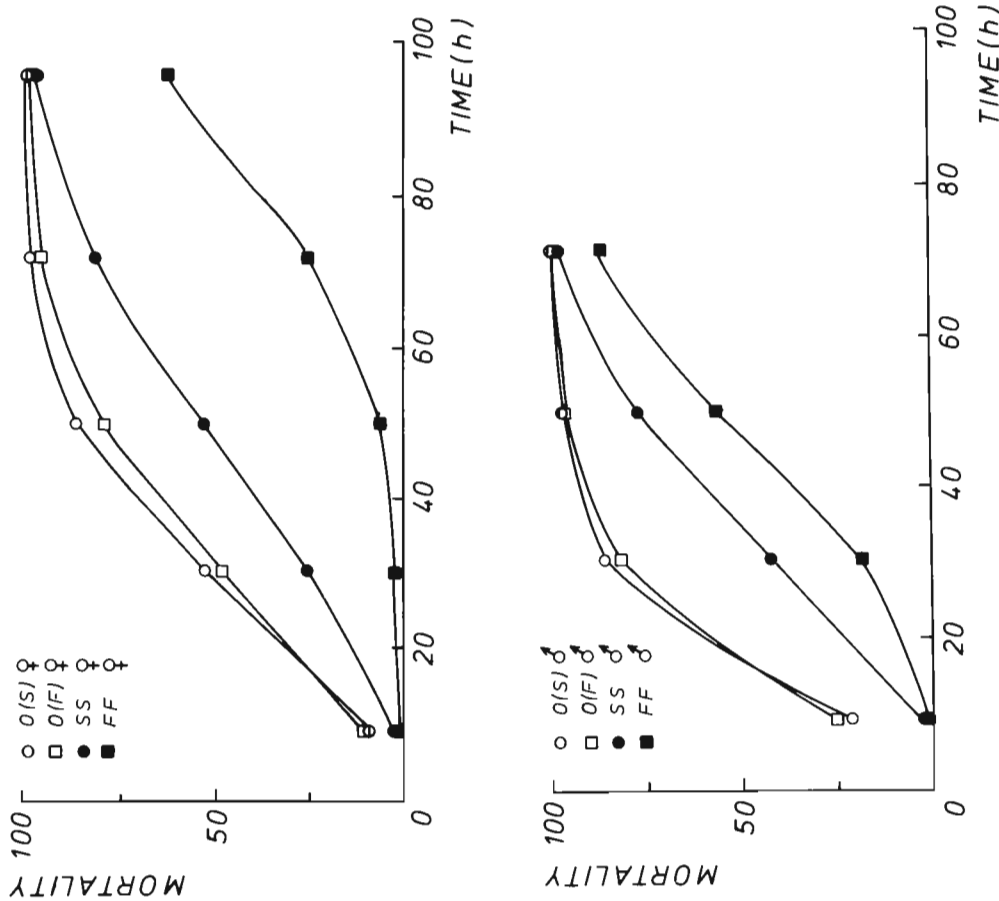
Figure 1. Cumulative mortality percentages of flies in a starvation test.

food. At the start of the test the flies were 6-7 days old. They were put in vials without etherizing (20 flies per vial). The vials contained 9 ml agar (18 g agar in 1 liter water); the number of replicas per sex and per strain was five.

Mortalities were recorded at intervals, the results are shown in Figure 1. It appears that females are more resistant to starvation than males. The FF strains survive significantly longer than the SS strains, while the O(F) and O(S) strains are less resistant to starvation than the ADH positive strains. The results emphasize the importance of ADH under conditions with-

out alcohol stress, though the exact nature of the phenomenon is still unclear.

References: Grell, E.H., K.B. Jacobson & J.B. Murphy 1968, Ann. NY Acad Sci 151:441-455; Van Delden, W. 1982, Evol. Biol. 15:187-222; _____, A.C. Boerema & A. Kamping 1978, Genetics 90:161-191; _____ & A. Kamping 1981, DIS 56:149-150; _____ & _____ 1986, DIS 63:134-135.

Van den Berg, M.J. Biological Centre, Haren, Netherlands. White noise inhibits **Drosophila melanogaster** copulation success.

Since the first reports concerning Drosophila courtship sounds (Shorey 1962; Bennet-Clark & Ewing 1967), many others have appeared. Mostly, it was concluded that all Drosophila species studied had their own specific song. These differences are mostly thought to be important for species recognition (e.g., Ewing & Bennet-Clark 1968). In a few cases, it has been proven that **D.melanogaster** females copulate more readily when hearing artificial **D.melanogaster**-like pulse song than when hearing **D.simulans**-like pulse song (Bennet-Clark & Ewing 1969; Kyriacou & Hall 1982). The songs of **D.melanogaster** and **D.simulans**, however, are rather easy to describe in terms of a few parameters so that an apparatus may be built to simulate their song (Beukema et al. 1986). However, Drosophila songs are sometimes more complex, e.g., the structure of a burst of **D.mauritiana** pulse song is rather complex as is the pulse song of **D.teissieri** (Cowling & Burnet 1981).

In trying to prove that the specific songs of some other species of the **melanogaster** subgroup are indeed important for species recognition, I soon realized that with more complex sounds one could easily neglect some of the relevant (unknown) details of the song structure. Therefore, with complex sounds, it is better to repeat natural songs for females and deaf-mute males, when testing for a possible mate recognition function of these sounds.

However, when natural sound is recorded, white noise will also be recorded due to unavoidable technical reasons. In my case the signal to noise ratio could very well be as low as 7 to 1 (when courtship sound had a low volume). If such a sound had been played back at a volume of 102 dB, the noise volume would have been 85 dB. The problem is, does such a strong noise influence the copulation behavior of Drosophila?

Table 1. Mean of the cumulative number of copulations after a given time and s.e.m. (given between brackets).

	signal	signal+noise	signal+noise (after noise)	noise	analysis of variance#
15 min	1.58(0.56)	0.25(0.13)	0.42(0.19)	0.00(0.00)	7.42
30 min	3.75(0.61)	0.67(0.33)	0.75(0.35)	0.00(0.00)	23.45*
45 min	4.92(0.53)	1.00(0.41)	1.08(0.43)	0.00(0.00)	26.39*
60 min	5.67(0.51)	1.33(0.40)	1.33(0.40)	0.17(0.11)	29.26*

#=chi square value (df=3) using a Kruskall-Wallis analysis of variance.
*=p<0.001. Means sharing the same line are not significantly different (Mann-Whitney U test; alfa=0.05).

To answer this, experiments were done with **D.melanogaster** using 10 deaf-mute males (the antenna's were clipped off and the wings were glued together) and 10 females. These were put into a mass-mating chamber and the flies were constantly observed for one hour for copulations while background sound was played. Four types of sounds were played (12 replications per type). These were: (1) artificial **D.melanogaster** courtship song at 102 dB; (2) white noise (from 30 Hz to 500 Hz) at 85 dB; (3) a mixture of 1 and 2; (4) 15 min white noise and later one hour a mixture of (1) and (2). This last type was to see if it was possible to adapt the females to noise before the real courtship sound was played.

The results are given in Table 1. It is obvious that courtship sound mixed with noise gives more copulations than noise alone. However, it is also obvious that white noise inhibits the number of copulations very strongly. Thus, if one wants to use natural courtship sounds to check for mate recognition, one must filter out the noise. Technically, this is only possible using modern digital techniques.

References: Bennet-Clark, H.C. & A.W. Ewing 1967, Nature 215:669-671; ___ & ___ 1969, Anim. Behav. 17:755-759; Beukema, W.J., T.W. Nyboer & M.J. van den Berg 1986, DIS 63:138-139; Cowling, D.E. & B. Burnet 1981, Anim. Behav. 29:924-935; Ewing, A.W. & H.C. Bennet-Clark 1968, Behav. 31:288-301; Kyriacou, C.P. & J.C. Hall 1982, Anim. Behav. 30:794-801; Shorey, H.H. 1962, Science 137:677-678.

Vaysse, G. and M. Corbiere. Universite Paul Sabatier, Toulouse, France. Influence of the serotonin level, modified by an alimentary chronical treatment, on the learning performance of **Drosophila melanogaster.**

As for many other animals, it is possible to modulate neurotransmitters level by alimentary track in Drosophila. In a previous work (Marenco et al. 1984), we have shown that it is possible to reduce the learning performance by chronical treatment: either with para-chlorophenylalanine (p-CPA) added to the synthetic chemically nutritive medium (S), or by a depletion of tryptophan in the same type of medium, during the whole larval instars. Later on, the same results were obtained when those treatments were carried out during the first week of the imago life. In all the cases, the decrease of the performance was correlated with a lower level of serotonin (5-HT).

In another work (Galissie et al. 1986), we have shown that for chronical treatments, variations were obtained in a few days, but in spite of a permanent treatment, the amount of 5-HT returned to the normal level. In case of the addition of a serotonin synthesis inhibitor (p-CPA at .6g/l) or in case of addition of the immediate serotonin precursor (5-hydroxytryptophan -5-HTP- at .6g/l), the amount of 5-HT regularly decreased (in the first case) or increased (in the second case) until the fourth or fifth day, then it returned to the normal level for the seventh day. All these experiments were performed with seven day old imago males. The experimental groups were fed all the time with chemically defined medium, and for the treated groups the treatment was maintained until the seventh day when they were frozen in liquid nitrogen at about 4 p.m.

Table 1. Analysis of variance of data. Mean of inhibitions for 4 trials.

Source	DF	Mean square	F value	
Between subjects	65	706.57765		
A(Groups)	2	85.82197	4.355001	p<.025
Suj.Within groups	63	620.75568		
Within subjects	462	434.875		
B(Trials)	7	175.64962	44.06840	p<.001
Inter. A*B	14	8.11742401	1.01828	n.s.
Inter. B*Suj(wg)	441	251.107956		n.s.

The present work joins both approaches. Imago males that have been fed with a synthetic medium after the pupal stage, then treated from the third to the sixth day (to obtain a best effect) by p-CPA (.6g/l) or by 5-HTP (.6g/l), are individually tested at the seventh day. The paradigm of learning is inspired from Medioni et al. (1978), but it is a pavlovian conditioning in this case. Every two minutes, the legs of a walking fly arrived in contact with a sugared solution which was followed immediately by a new contact with a quinin chloride solution. This sugared solution is chosen after a previous determination of the individual behavioral threshold for sucrose. The quinin chloride concentration is the same for all the flies: $M.10^{-4}$.
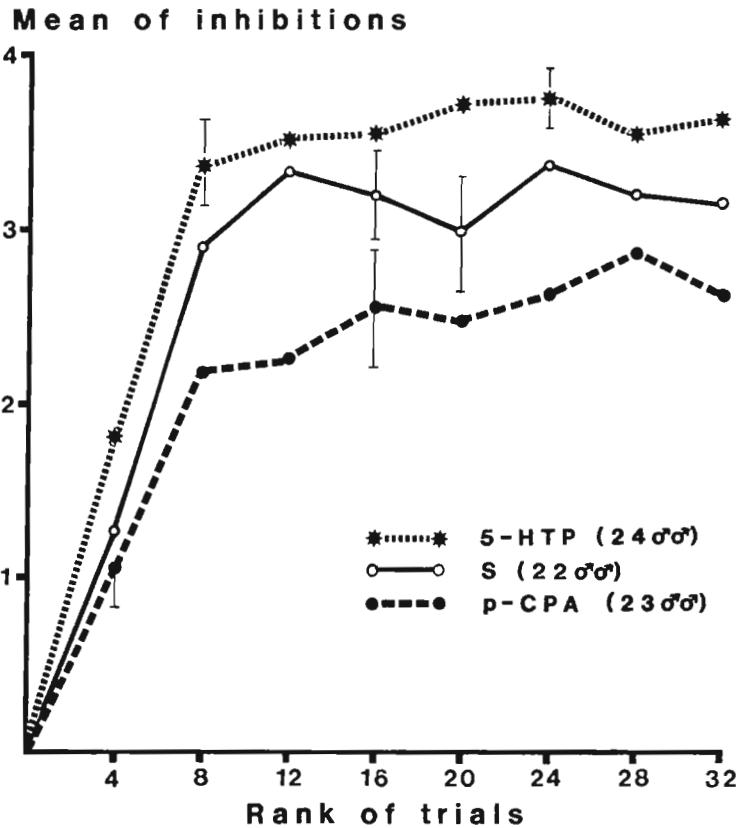
Mean of inhibitions

Our experimental protocol is as follows: (1) after 16±4 hr of starvation, the behavioral sucrose-threshold of each male is estimated using a psycho-physic method by increasing concentration solutions; (2) the sucrose concentration for the learning procedure is determined (4 to 64 times more than the behavioral threshold); (3) 32 trials, with a 2 min inter-trial-interval, are then performed. For each trial, the occurrence and the intensity of the proboscis extrusion (tarsal reflex) are registered in reference to a behavioral scale.

We only have reported here the number of inhibitions (of the tarsal reflex) for 69 blind tested flies. Figure 1 gives the results for three groups (realized after opening the anonymity). If the mean of inhibitions (for groups of 4 trials) is increasing for the 3 types of flies, the variation is statistically different for each group (Table 1 represents an ANOVA with 22 flies by group, necessitating a random elimination of 2 flies in the 5-HTP group and one fly in the p-CPA group). As can be seen on Figure 1, when the level of 5-HT is increased, the performance of learning is the same. On the contrary, when the level of this neurotransmitter is decreased by an addition of a serotonin synthesis inhibitor, the performance of learning is lowered. These results concerning the effect of p-CPA on the tarsal reflex inhibition with a pavlovian conditioning are similar to those obtained with an operant conditioning. This suggests a modulatory effect of serotonin on different processes of inhibitory learning.

References: Galissie, M., G. Abravanel & G. Vaysse 1986, DIS 63:53-54; Marenco, M.J., M. Galissie & G. Vaysse 1984, DIS 60:221; Medioni, J., N. Cadieu & G. Vaysse 1978, C.R. Soc. Biol. 172:961-967.
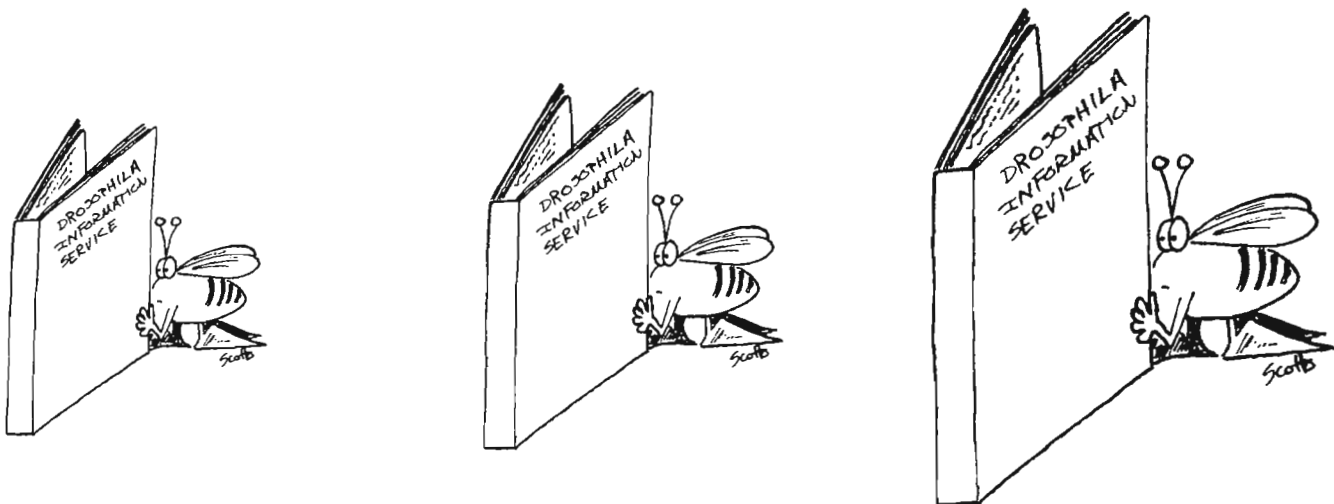
Vinson, C.R. and P.N. Adler. University of Virginia, Charlottesville. A Minute genetic background alters the cell-autonomous phenotype of the tricorner (trc) locus.

The zygotic lethal and somatic viable locus, trc (Gubb & Garcia-Bellido 1982; Ferrus 1976), has been examined as a suitable gratuitous cell marker for chromosome arm 3L. Our interest was to use trc to mark mitotic clones of tissue polarity loci (Gubb & Garcia-Bellido 1982). The cuticular cellular phenotype of trc is a rossette-like set of hairs instead of a single well elaborated hair as is typically seen on the notum and wing. Large trc clones (greater than 100 cells) often have a patch of cells in the center of the clone that do not elaborate a rossette of hairs, a phenomenon even more pronounced if the trc clone is in a Minute genetic background. The borders of the trc mitotic clone clearly delineate between the rossette-like trc cells and neighboring wild type cells.

We have generated mitotic clones of trc in both wild type and Minute genetic backgrounds and examined them to determine if the polarity of surrounding wild type hairs is disrupted. The results of these experiments were unexpected. trc mitotic clones in a wild type background were completely cell-autonomous (i.e., no effects were seen on wild type cells surrounding the trc clones) in over 95% (n=46) of the clones examined, thus suggesting that trc can be used as a gratuitous cell marker of mitotic clones. The two mitotic clones that had a non cell-autonomous phenotype showed a disruption of the polarity of hairs proximal to the mitotic clone (see Figure 1). Using a Minute genetic background (M(3)i[55]), over 50% (n=29) of the mitotic clones had a non cell-autonomous phenotype. Again, only wild type cells proximal to the clone exhibited a disruption of hair polarity (see Figure 1).

We draw three conclusions from these results. One, trc is a useful gratuitous cell marker for mitotic clones when using a wild type genetic background. Two, trc has a non cell-autonomous phenotype; proper trc function is required for the correct polarity orientation of hairs proximal to the trc mitotic clone, especially in a Minute genetic background. Three, the most general conclusion is a note of caution in interpreting mitotic clone data when using the Minute procedure. The phenotype of mitotic clones as determined using the Minute procedure is not necessarily what would be obtained if one used a wild type genetic background. This result suggests that in at least some cases, differential cell growth rates are important in controlling morphogenesis.

References: Gubb, D. & A. Garcia-Bellido 1982, J. Embryol. Exp. Morph. 68:37-57; Ferrus, A. 1976, PhD Thesis, Univ. de Madrid, p42.
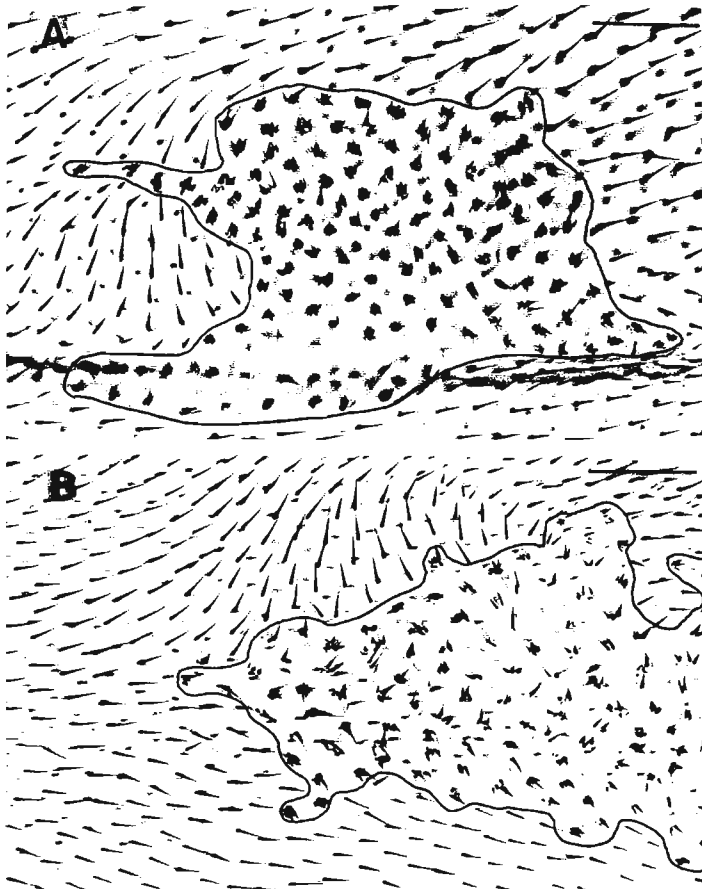
Figure 1. (A) Photomicrographs of a trc mitotic clone in a wild type genetic background induced 2-3 days after egg laying with 1500R gamma rays. Note the disruption of hair polarity proximal to the mitotic clone. (B) Photomicrograph of a trc mitotic clone induced in a Minute (M(3)i[55]) genetic background induced 5-6 days after egg laying with 1500R gamma rays. Again, note the disruption of hair polarity proximal to the mitotic clone. The scale bar = 50 microns.

Vlassova,I.E., I.F. Zhimulev, E.S. Belyaeva and V.F. Semeshin. Inst. of Cytology & Genetics, Novosibirsk, USSR. The effect of DRB and α-amanitine on the RNA synthesis in **Drosophila melanogaster** polytene chromosomes.

In this paper, we report about some peculiarities of inhibition of RNA synthesis in **Drosophila melanogaster** polytene chromosomes by 5,6-dichloro-1-β-D-ribofuranosyl benzimidasole (DRB) and α-amanitine.

Salivary glands were incubated for 30 min or more (up to 8 hr) in Drosophila cell culture medium (Poluektova et al. 1980) with 25 or 50 μg/ml of DRB or 50 μg/ml α-amanitine (both from Calbiochem, USA) or with both of them in these concentrations. Then they were transferred into the same medium with inhibitors, and ^{3}H-uridine (GosNiiChim Photo Project, Moscow) and exposed for 9 days. Some slides were prepared for immunofluorescence localization of DNA:RNA hybrids (see Vlassova et al. 1985). As a control, squashes from salivary glands, incubated in medium without inhibitors were used. Stages of larval development were determined using puffing stages (PS) of Ashburner (1972).

Prominent inhibition effect of DRB was observed after 2 hr of treatment, but even after 8 hr of treatment, there were some bright fluorescent regions (Figure 1). Fluorescence of 93D (Fig. 1c', d') was always seen and quite often 87B; sometimes, after prolonged incubation, heat-shock puffs appeared and we observed fluorescence of 87A and 87C instead of 87B (Fig. 1c'). In some nuclei 43E, 46D, 47B, 47D, 50C, 51F, 53E, 53F, 84EF, 86F, 88D, 91D, 91F, 92A, 94C, 94EF regions showed fluorescence. In control glands, DNA:RNA hybrids were detected in the most decondensed chromosome regions (Fig. 1a-d).

Data on DRB inhibition of ^{3}H-uridine incorporation were taken from analysis of chromosomes from larvae at PS1, PS4-5 and O-h prepupae with known puffing patterns.

At PS1, when ecdysone-induced puffs are not developed, after 8 hr of DRB treatment, we always observed labelling of 93D and 87B regions, and often 23F and 4F. Besides that, there were diffuse weak labelling along chromosomes and strong labelling of nucleolus. In control glands, we observed strong labelling of chromosomes.

After 2 hr DRB treatment of O-h prepupae salivary glands, the most intensive labelling regions were 93D (Fig. 2a) and 71C-E (Fig. 2b); labelling was also found in some other regions: large puffs 50CD, 50F, 63E, small puffs 30EF, 49EF, 91CD, 94BC; weak labelling along chromosomes was also present.
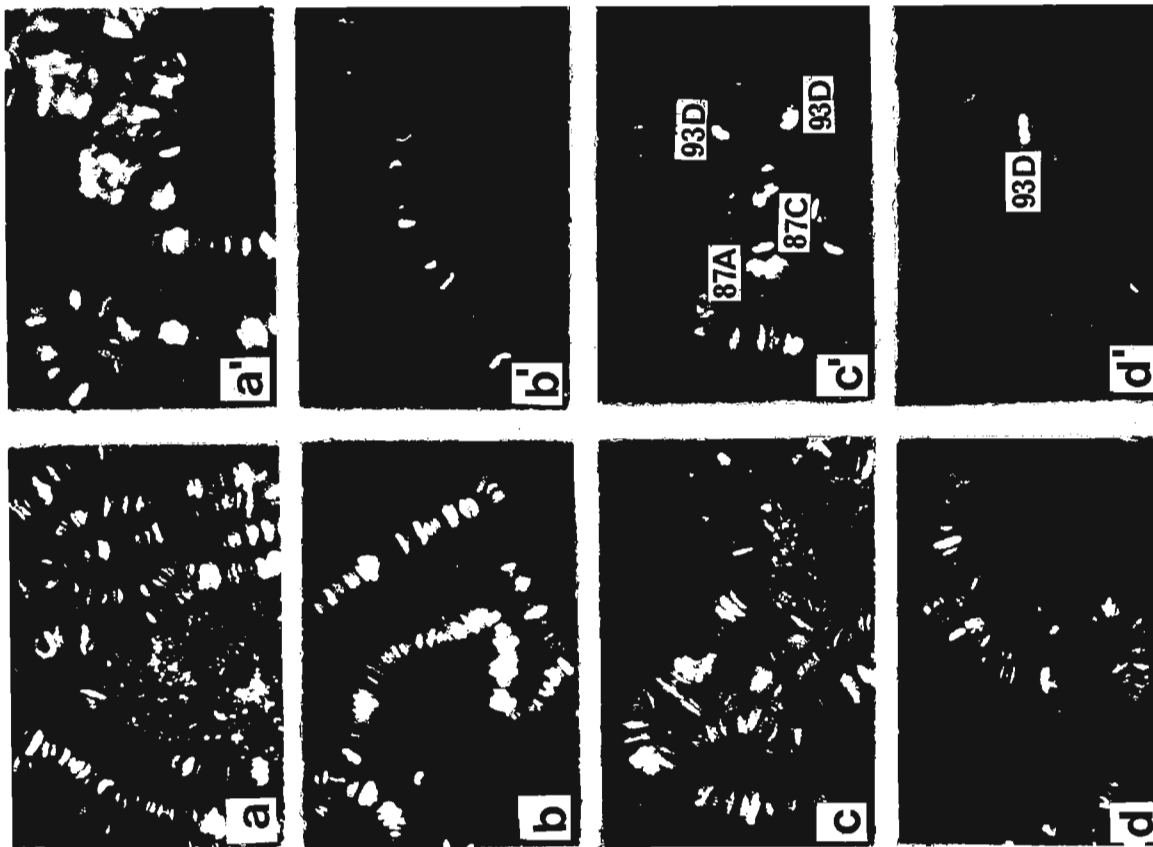
Figure 1. Immunofluorescent localization of DNA:RNA hybrids on polytene chromosomes in control salivary gland (a-d) and after DRB treatment (a'-d'). 1 (a,a'), 2 (b,b'), 4 (c,c') and 8 (d,d') hr of incubation.
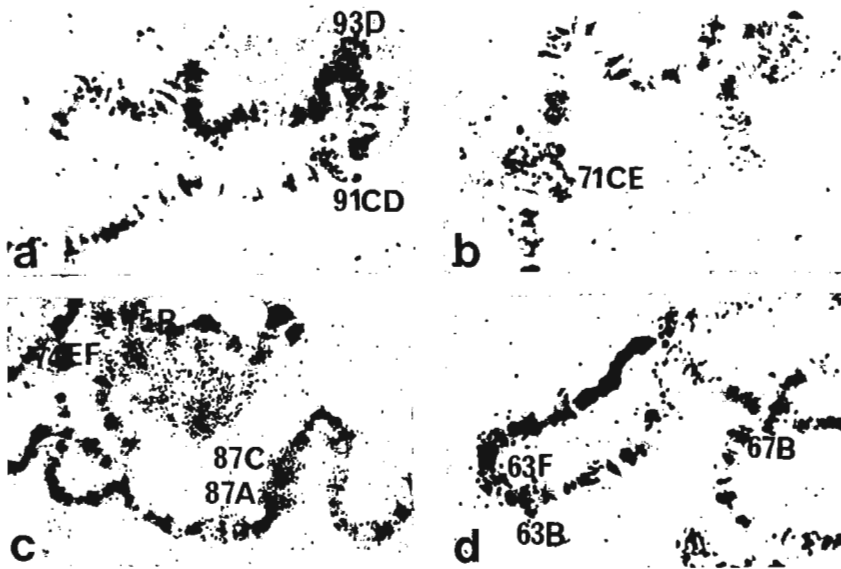
Figure 2. ^{3}H-uridine incorporation in polytene chromosomes after 2 hr treatment with DRB. a,b = salivary glands were taken from O-h prepupae. c,d = PS4-5 larvae.
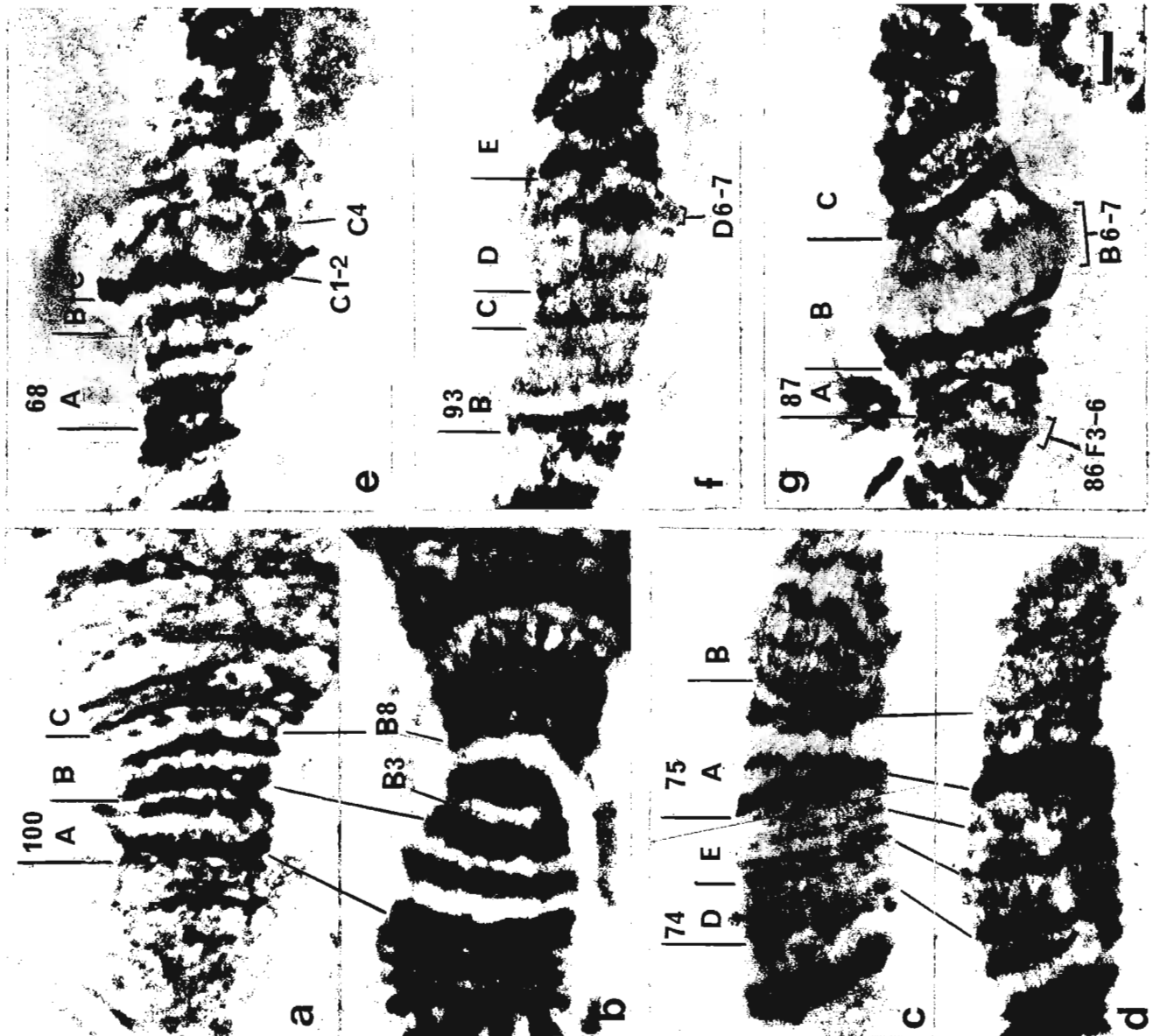
Figure 3. EM mapping of polytene chromosomes at PS1 in control (a,c) and after 2 hr DRB treatment (b, d-g).

Table 1. Effect of 2 hr incubation with alpha-amanitine (50 μg/ml) on ^{3}H-uridine incorporation in salivary gland nuclei of **Drosophila melanogaster.**

No. of larvae	No. of nuclei	% of heavy labelling nuclei		% of nuclei, labelling at level of back-ground or some higher
		continuous labelling	labelling of some regions	
alpha-amanitine:				
1	88	14.8	5.7	79.5
2	120	25.0	20.0	55.0
3	186	--	--	100.0
4	205	--	--	100.0
5	179	--	--	100.0
6	122	2.5	1.0	96.5
7	290	17.2	0.7	82.1
8	180	--	--	100.0
9	310	22.6	--	77.4
Control:				
1	235	88.0	12.0	--
2	238	68.0	32.0	--
3	263	8.7	91.3	--
4	106	33.7	66.3	--
5	191	92.1	7.9	--
6	126	44.4	55.6	--

Patterns of ^{3}H-uridine incorporation in nuclei shown in Fig. 4.
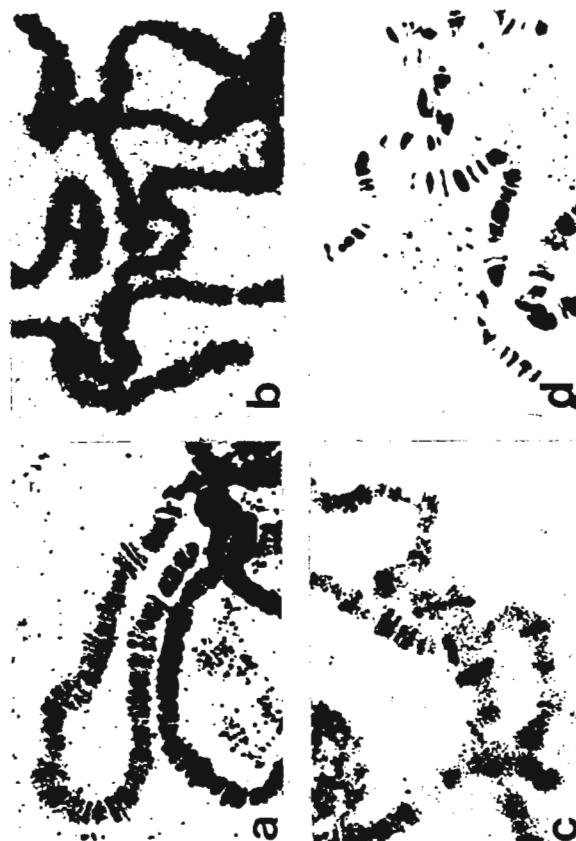
Figure 4. Patterns of ^{3}H-uridine incorporation in nuclei, found in the same salivary gland after alpha-amanitine treatment for 2 hr (a-c) and simultaneous DRB and alpha-amanitine treatment for 2 hr (d). a = labelling at the level of background or some higher; b = continous labelling; c = labelling of some regions.

At PS4-5 after 2 hr of DRB treatment (Fig. 2c,d) accidentally accompanied by slight heat shock, we observed labelling of specific for these PS puffs such as 74EF, 75B, 63F, 50CD as well as heat shock puffs: 87A, 87C, 63B, 67B, 93D. Weak labelling was also seen in puffs: 29F, 39C, 56E, 71CE, 72D, 72EF, 85F, 88D, 88E, 90C, 92B, 96E, 100DE.

It is interesting that after 2 hr of inhibitor's treatment, there was not obvious PS shift during salivary gland incubation. Also we found the simultaneous labelling of PS-specific and heat-shock puffs, which is not the case in the normal conditions (Evgen'ev et al. 1985).

EM analysis of some regions after prolonged DRB treatment was also performed (Figure 3). We did not observe compactization of interband material: after RNA synthesis inhibition, we did not find noticeable changes of banding pattern of 100A-C region of 3R chromosome (Fig. 3b). However, some loose bands become more compact: for example, the 100 B8 band (Fig. 3b). The same situation was observed for 74-75 region (Fig. 3c,d). PS1 specific puff 68C after 2 hr of DRB treatment preserved some transcriptional activity and under EM looked like a well developed puff (Fig. 3e). After the prolonged DRB treatment, we observed heat shock puff 93D, and the appearance of puffs in the regions 86F3-6 and 87B6-7 (Fig. 3f,g).

The mechanisms of RNA synthesis inhibition by DRB is still unknown. Egyhazi et al. (1982) suppose the inhibitory effect on initiation, others (Tamm et al. 1979) on premature termination of transcription of Hn RNA. From our data it is seen that longterm DRB treatment does not inhibit synthesis of all RNAs. Some regions of fluorescence and ^{3}H-uridine incorporation are tRNA loci, which transcription is under control of RNA-polymerase III: 49EF, 56E, 72EF, 84EF, 87B, 90BC (Kubli 1980). But in the majority of large puffs, there is RNA-polymerase II (Jamrich et al. 1977) and their transcription must be inhibited by DRB. However, in our experiments after longterm DRB treatment we even find initiation of RNA synthesis in heat-shock puffs.

Data on α-amanitine influence on ^{3}H-uridine incorporation in polytene chromosomes are presented in Table 1 and Figure 4. This toxin greatly reduced ^{3}H-uridine incorporation in the most nuclei (55-100%); there was both slight labelling along all chromosomes at the level of background or some higher (Fig. 4a). However, it is in agreement with other authors (Beermann 1971; Holt & Kuijper 1972); some cells appeared

to be resistant to the toxin (0-45% in different salivary glands) and incorporated ^{3}H-uridine along the whole chromosome: "continuous" labelling (Fig. 4b) or in some regions (Fig. 4c). In control, there were no nuclei with weak labelling at the level of background (Table 1).

The level of ^{3}H-uridine incorporation in nucleolus was the same in control and after α-amanitine treatment.

Simultaneous treatment with DRB and α-amanitine produced strong inhibiting effect. Labelling along chromosomes was at the level of background (Fig. 4d). There were no nuclei with considerable ^{3}H-uridine incorporation in comparison with control. Labelling of nucleolus is lower than in control, which may be due to the simultaneous effects of high concentration of DRB and α-amanitine.

Therefore, we demonstrated that neither DRB nor α-amanitine alone do not completely inhibit transcription. The increase of inhibition effects of simultaneous action of DRB and α-amanitine may indirectly be witnesses for existence of DRB-resistant, but α-amanitine-sensitive, RNA fraction. However, even with both inhibitors acting together, there is weak ^{3}H-uridine incorporation along chromosomes, thus implying the absence of complete inhibition of transcription in puffs.

Acknowledgements: We are thankful to Dr. V.N. Bolshakov for help preparing and critical reading of the manuscript.

References: Ashburner, M. 1972, in: Result and Problem in Cell Differentiation (W. Beermann, ed.), Berlin, Heidelberg, New York: Springer, v4:101-151; Beermann, W. 1971, Chromosoma 34:152-167; Egyhazi, E., A. Pigon & J. Rydlander 1982, Eur. J. Biochem. 122:445-451; Evgen'ev, M.B., O.L. Zatsepina, V.T. Kakpakov & I.E. Vlassova 1985, Molecular Biology (USSR) 19:483-488; Holt, Th.K.H. & A.M.C. Kuijpers 1972, Chromosoma 37:423-432; Jamrich, M., A.L. Greenleaf & E.K.F. Bautz 1977, PNAS USA 74:2079-2083; Kublie, E. 1980, in: Genetic maps (S.J.O. Brien, ed.), NY: Cold Spring Harb. Lab, v1:195; Poluektova, E.V., V.G. Mitrafanov & V.T. Kakpakov 1980, Ontogenez (USSR) 11:175-180; Tamm, J. & T. Kikuchi 1979, PNAS USA 76:5750-5754; Vlassova, I.E., G.H. Umbetova, V.H. Zimmermann, C. Alonso, E.S. Belyaeva & I.F. Zhimulev 1985, Chromosoma 91:251-258.

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Cytological analysis of deficiency 16-3-35 at the base of the X chromosome of **D.melanogaster.**

In their major review of the cytogenetics of the proximal region of the X chromosome, Schalet & Lefevre (1976) refined and supplemented earlier maps of divisions 19 and 20. They also drew attention to unresolved issues and to remaining ambiguities such as the proximal-distal orientation of a number of lethal complementation groups. These authors examined 26 chromosomal deficiencies both genetically and cytologically and pointed out that only one discrepancy was encountered between the cytological and genetic extents of these deficiency-bearing chromosomes. This particular case was Df(1)16-3-35 (19D2,3-19E6-7), whose proximal cytological breakpoint was not consistent with its predicted proximal genetic breakpoint which should have been near 19E4,5. Genetically, Df(1)16-3-35 (19D2,3-19E6-7) was found to complement Df(1)A118 (19E4,5-19E8) and thus one might have expected Df(1)16-3-35/Df(1)A118 heterozygotes to be at least deficient for band 19E5 and to be lethal, since the common overlap should delete the R-9-28 lethal complementation group. However, Df(1)16-3-35/Df(1)A118 heterozygotes are fully viable, as documented by these authors and verified by us. In view of these results, Schalet & Lefevre (1976) entertained the possibility that a minute section of the base of the X chromosome (namely band 19E5) may well have been translocated to another part of the genome.

Owing to our extensive interest in the neurological genes which are found in this region of the X chromosome (Miklos et al. 1987) as well as to the interesting structural properties of this part of the X (Schalet & Lefevre 1976; Miklos et al. 1984), we reexamined a number of deficiencies genetically and cytologically with particular emphasis on Df(1)16-3-35. The cytological breakpoints of those examined, with one exception, are in agreement with Schalet & Lefevre's (1976) previous studies. We find, however, that Df(1)16-3-35 does not extend as far proximally as previously thought and thus its genetic and cytological attributes now become congruent. Figure 1 illustrates the base of the X chromosome from a Df(1)16-3-35/Canton-S heterozygote. On the control homologue (top), we have illustrated bands 19D1,2,3 (long lines) and 19E1,2,3,4, (short lines). The Df(1)16-3-35 chromosome (bottom), however, only carries 19D1,2 and 19E4. Thus, Df(1)16-3-35 has its distal breakpoint near 19D2,3 and its proximal breakpoint near 19E3,4. These cytological results, thus, clarify the ambiguity which Schalet & Lefevre (1976) encountered and to which they drew attention.

It is important that this discrepancy between the cytological and genetic attributes of this deficiency has now been resolved, since both Df(1)16-3-35 and Df(1)A118 have breakpoints near the **shakingB** locus (Miklos et al. 1987) where chromosomal walks are in progress (Davies et al. 1987) and where firm breakpoint assignments are essential for correct molecular analyses.
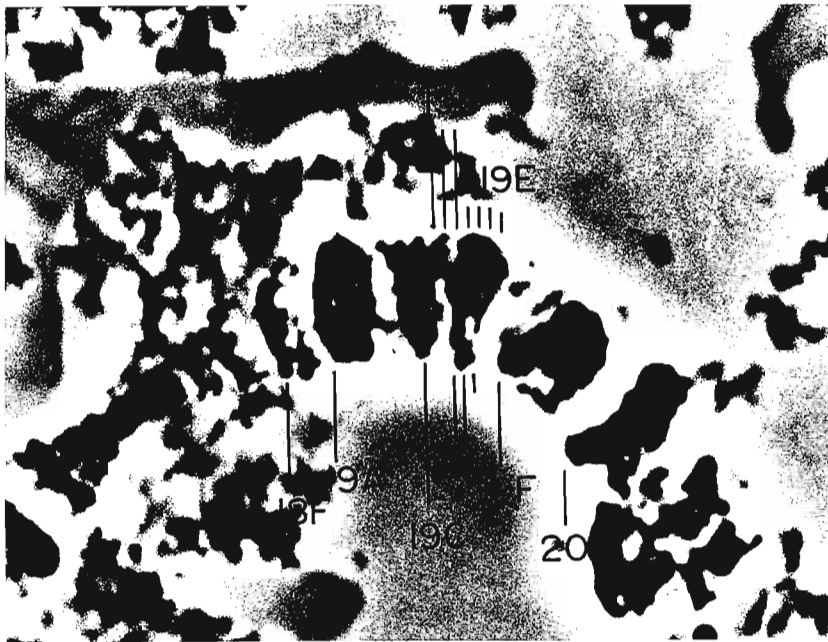
Figure.

References: Davies, J., V. Pirrotta & G.L.G. Miklos 1987, J. Neurogenetics 4:123; Miklos, G.L.G., M.J. Healy, P. Pain, A.J. Howells & R.J. Russell 1984, Chromosoma 89:218; ――, L.E. Kelly, P.E. Coombe, C. Leeds & G. Lefevre 1987, J. Neurogenetics 4:1; Schalet, A.P. & G. Lefevre 1976, in: Genetics and Biology of Drosophila (Ashburner & Novitski, eds.), Acad. Pr. 1b:848.

TECHNICAL NOTES

Abdelhay, E., T.M. Castro and H. Rocha. Inst. de Biofisica Carlos Chagas Filho, UFRJ, Rio de Janeiro, Brasil. A method to isolate segments of **D.melanogaster** embryos in great quantities.

In order to isolate specific segments from Drosophila embryos, in amounts that permit biochemical analysis of morphogens distribution, we developed a method to cut hundreds of embryo segments at the same time.

Glass slides mounted as in Figure 1 under an entomological microscope are used to position hundreds of dechorionated eggs with 0 to 4 hr of development, in a parallel distribution, all with the same relative position (A to P). Pressing carefully slide 4 (see Fig. 2), embryos are restricted to a narrow channel which is filled with Tissue-Tek OCT compound 4583 (Miles Scientific) and quickly immersed in liquid nitrogen. Slide 4 is pulled apart and the resulting frozen block containing the embryos is separated with the aid of chilled tweezers. The blocks can be stored one week at -20°C.
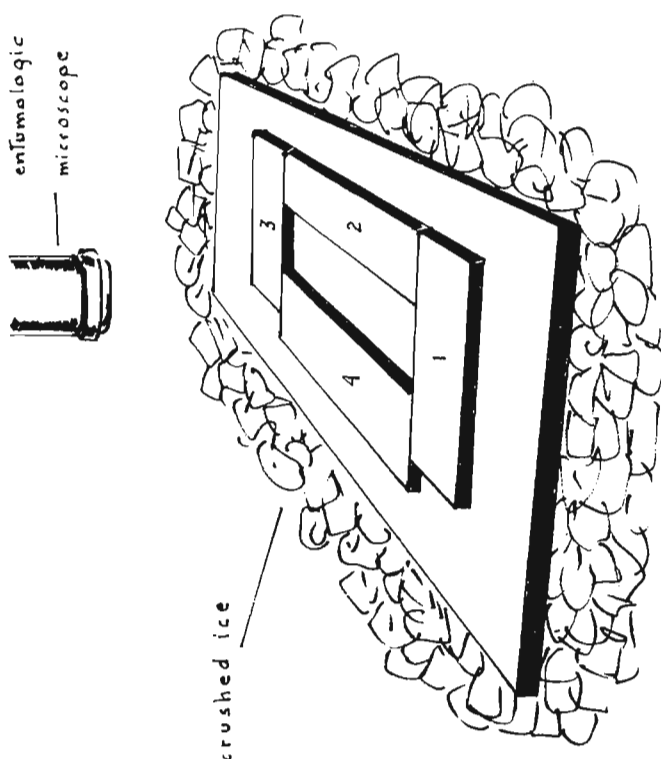
Figure 1. Schematic drawing of the mount.

Figure 2. Close-up of some **Drosophila melanogaster** embryos positioned.

Blocks are sliced in a microtome in several widths (10 µ to 80 µ). As the medium is totally inert, the slices containing segments or subsegments can be used directly for macromolecular or ionic analysis.

Chou, Tze-Bin, I. Mims, M. Belanich, Z. Zachar and P.M. Bingham. State University of New York, Stony Brook. Procedural improvements in injections for P-mediated germline gene transfer.

Germline gene transfer is now widely carried out by the microinjection procedure of Germeraad (1975) as modified by Spradling & Rubin (1982) and Karess & Rubin (1984) to capitalize on the high, germline-specific rate of transposition of the P element transposon (Bingham et al. 1982).

We have made modifications in several details of the procedure for injection of DNA that improve consistency and reliability. First, control of ambient relative humidity during dechorionation is crucial. (Dechorionations are done at temperatures from 21° to 23°C by rolling embryos on a sheet of double stick tape as originally described.) Very low relative humidity (less than 20%) produces very low survivals to hatching (as low as a few percent in extreme cases). Very high relative humidity is optimal and either of two strategies is comparably effective: (1) Dechorionation can be done in a confined area humidified to 80% or higher with one of the inexpensive, domestic ultrasonic humidifiers widely available. (2) In larger, hard-to-humidify areas slides bearing embryos are placed directly in the water aerosol from an ultrasonic humidifer for several seconds every two to three minutes throughout the process of dechorionating a strip of ca. 20 embryos on the slide. After the set of embryos has been dechorionated they are allowed to desiccate without further hydration. This method requires limited practice to master, but is quite reliable and is now in common use in our laboratory.

High survivals require careful control of the extent of preinjection desiccation of embryos. The optimal extent of desiccation is slight and is easily determined by a small number of trials. The large effects of relative humidity on survival may result from the difficulty of controlling extent and uniformity of desiccation under conditions of very rapid desiccation at low relative humidities.

We further note in this regard, that conventional chemical dechorionation procedures (50% commercial bleach, 0.7% NaCl, 0.1% triton X-100; 30-90 second room temperature treatment followed by extensive washing in 0.7% NaCl, 0.1% triton) produce acceptable survivals to hatching (ca. 30%) largely independently of ambient relative humidity. However, in our hands, chemical dechorionation produces tenfold to one hundredfold lower rates of G_0 rosy[+] expression and of successful gene transfer than mechanical dechorionation. The reason for this reduced efficiency is obscure; however, selective killing of the age group of embryos optimal for DNA uptake is a viable hypothesis.

Second, a thin strip (ca. 0.5 mm) of rubber cement (Carter's brand) has lower toxicity than some other materials widely used for mounting embryos for injection. Strips are conveniently produced by rapidly pulling a microscope slide through a very narrow flow of thinned (commercial thinner) rubber cement streaming from the tip of pasteur pipette or glass rod. Slides should be cured a minimum of about two hr at room temperature before use.

Third, mineral oil seems to be less toxic than the more expensive and difficult to obtain halocarbon oils as covering medium for embryos. We use conventional commercial preparations suitable for human ingestion and supplement coverage (one to three hr after beginning postinjection incubation to hatching at 18°C) with mineral oil equilibrated at 18°C. This supplementation is necessary to prevent the less viscous mineral oil from settling and uncovering the embryos.

The following are the rates of survivals to hatching (followed by total numbers of injected embryos) from all gene transfer procedures carried out in our laboratory in the last several months using these procedural improvements: 39%(827), 30%(401), 50%(425), 37%(413), 41%(403), 36%(417), 38%(770), 43%(409), 32%(800), 43%(396), 41%(469), 33%(600), 27%(614), 29%(358), 36%(389), 37%(682), 39%(770). Survival of hatched larvae to adulthood is consistently 40-50%; fertility among surviving adults is commonly 70-85%; frequencies of G_O expression of the rosy[+] marker on conventional plasmid vectors (injected as ca. 200 micrograms per milliliter) among surviving adults is commonly 40-60% and the fraction of fertile adults producing stably transformed progeny is commonly 10-25% (injecting conventional plasmid gene transfer constructions at 200 micrograms per milliliter and P element helper at 100 micrograms per milliliter).

References: Bingham, P.M., M.G. Kidwell & G.M. Rubin 1982, Cell 29:995-1004; Germeraad, S. 1975, Genetics 80:534-535; Karess, R.E. & G.M. Rubin 1984, Cell 38:135-146; Spradling, A.C. & G.M. Rubin 1982, Science 218:341-347.

Gorczyca, M. and J.C. Hall. Brandeis University, Waltham, Massachusetts. The INSECTAVOX, an integrated device for recording and amplifying courtship songs.

Since the late 1960's investigators of courtship song have usually used a particle velocity sensitive device, termed a ribbon microphone, to pick up the faint acoustic signal made by the male's wing vibrations (e.g., Schilcher 1976; Cowling & Burnet 1981). Bennet-Clark (1984) discovered a new type of song recording device by modifying an electret condenser microphone (see below). This alteration made the electret especially sensitive to particle velocity, which is the major component of sound produced by wing beating. The design also gave a higher signal to noise ratio than the ribbon microphone, was lower in cost, smaller in size, had decreased sensitivity to 60 Hz pick-up, and was not nearly as fragile. We have followed Bennet-Clark's lead and have designed plus constructed an integrated, easily portable unit containing the modified electret microphone, a low-noise power supply with ample current, noise filters, and a relatively cool light source. This INSECTAVOX also involves a microphone-containing compartment that is somewhat insulated from extraneous environmental noise; the flies, in a small plastic chamber set on top of the microphone (see below), are readily visible without a dissecting microscope.

Enclosure: To dampen environmental noise, the INSECTAVOX was constructed from 1/4 inch or thicker plates (Figure 1). The microphone compartment also contains a lining of open cell foam to reduce acoustic reflections from the inner walls. Flocking lines the surface areas of the lid/box interface, and the latch design ensures that the doors to the microphone and battery compartment seal tightly. Directly above the mating chamber is a glass lens which allows a magnified view and is thick and dense enough to limit sound penetration. The metal enclosure also reduces stray 60 Hz electronic noise.

The enclosure was separated into three compartments to isolate both electronic and acoustic noises (Figures 1 and 3). Transformers often produce an audible hum as well as electronic noise and magnetic fluxes which can interfere with the microphone. For these reasons, the transformer (of toroidal design) was situated far from the microphone and had two walls interposed. The electronic compartment was also shielded from the transformer.

Electronic circuit (also see Figures 2 and 3): The condenser microphone (Radio Shack #270-090) is very simple in design and has few components. To alter the microphone such that it is sensitive to particle velocity, the back side of the electret was opened to air. This required removing the printed circuit board and back plate and resoldering the FET input to the fixed electrode (cf. Bennet-Clark 1984). Power to the microphone is supplied by a 9 volt battery. Battery operation is necessary to take full advantage of the low noise of the preamplifier integrated circuit (LM387, National Semiconductor). Standard regulated DC power supplies have noise levels in the hundreds of microvolts which would obscure any fly-produced signal. The microphone output passes through a large capacitor to eliminate DC voltage. An attenuator is supplied at this point in case larger species of flies (with much louder songs) are to be recorded. This will prevent saturation of the input stage of the amplifier. The preamplifier circuit is a standard configuration designed to integrate the input signal and adjust its phase. The output of the preamp (powered with single-sided supply voltage) is passed through a capacitor to eliminate DC voltage before it encounters, at the next stage, the operational amplifiers, which have split supply voltages. At this point, the circuit branches in two directions: to the audio output and to the instrument output (e.g., for recording on magnetic tape; see legend to Figure 4). A buffer amp (TL062, Texas Instruments) is situated before the audio amplifer, as it was empirically determined that, without it, volume adjustment affected the instrument's output. A low-pass filter after the buffer stage prevents oscillation. The audio amplifier (TL081, Texas Instruments) input is controlled by a potentiomenter that serves as a volume control. The output is designed for high impedance headphones.
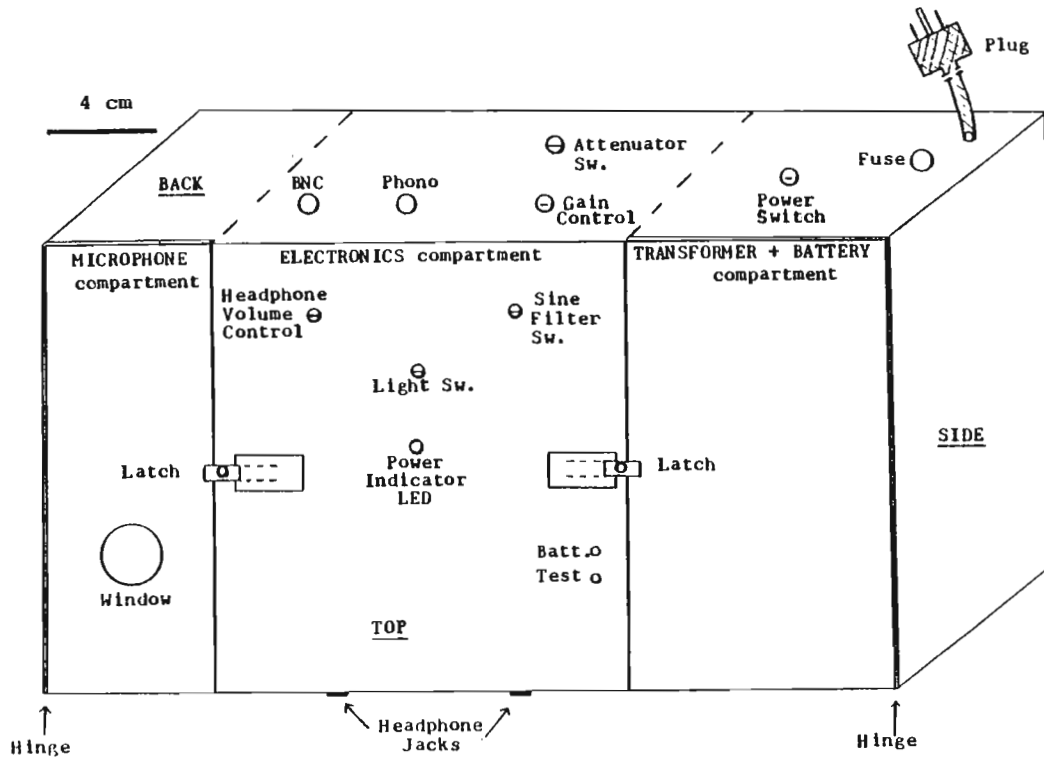
Figure 1. INSECTAVOX enclosure and external components. View is from above and behind. Switches are represented by 'sw'.
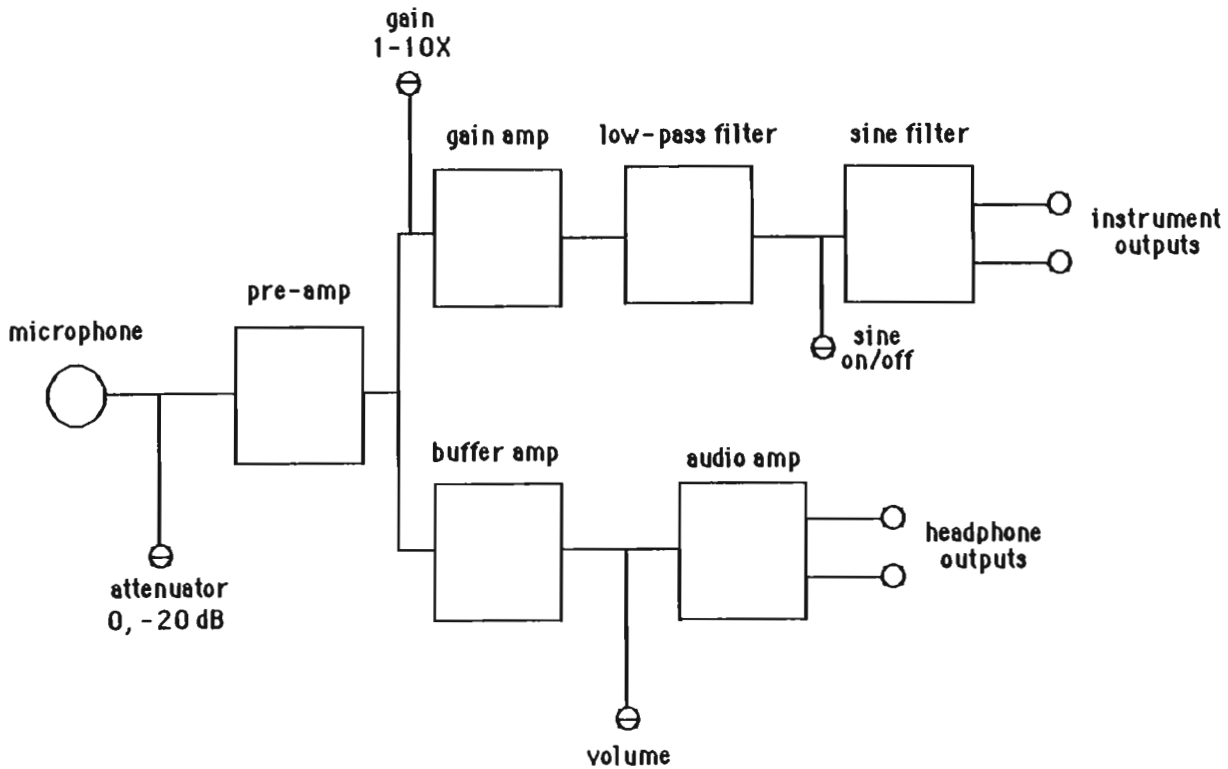
Figure 2. Block diagram of amplifier circuit. Loud signals from the microphone can be attenuated before reaching the high gain, low noise preamplifier. Audio signal output is adjustable through the volume control, whereas instrument output is controlled via the 1-10X gain adjust terminal. Filters attenuate excessive high frequency room noise and sine song, respectively.
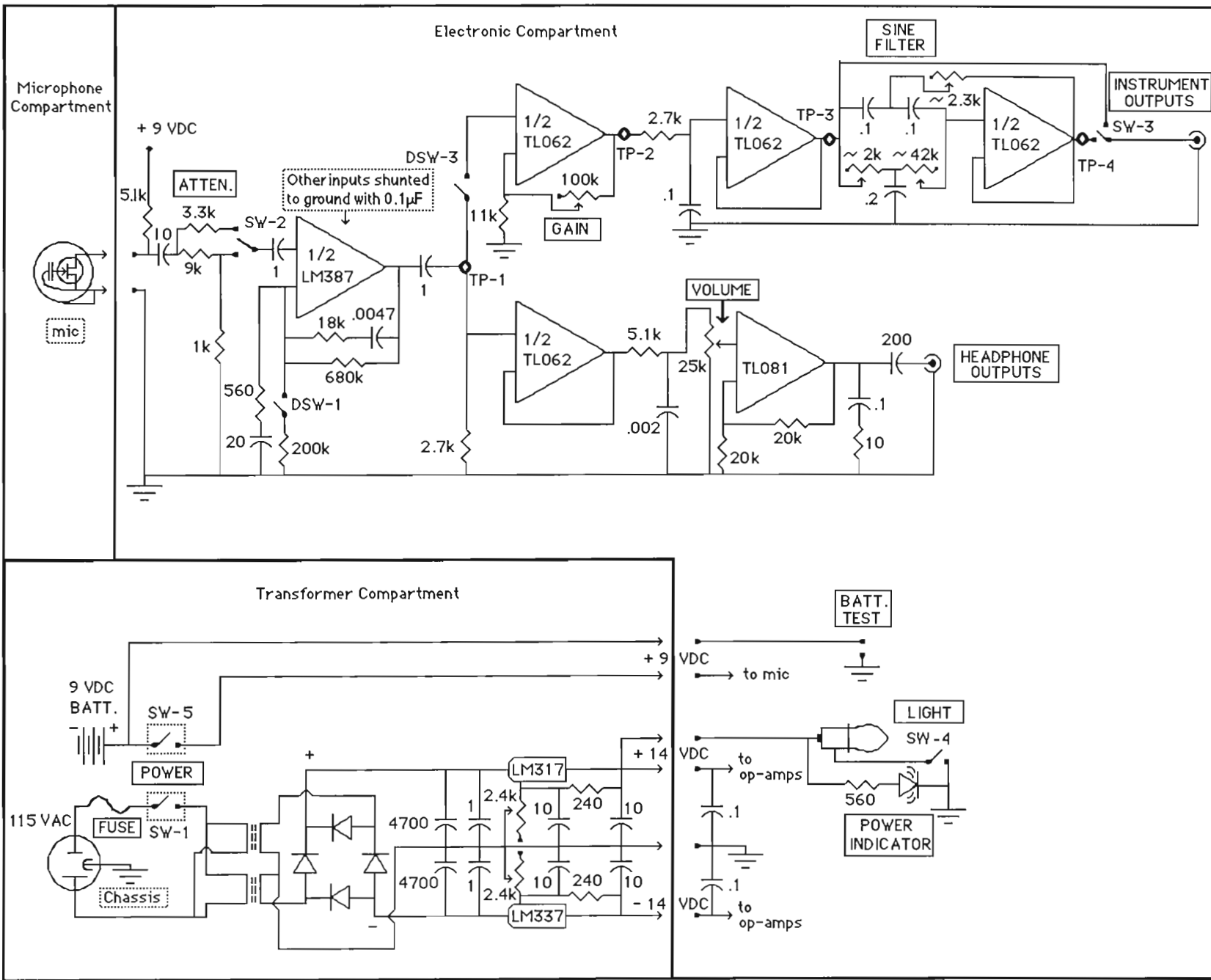
Figure 3. Circuit diagram of amplifier and power supply. Darkened rectangles represent features with external components accessible to the user. All resistances are in ohms and capacitances in microfarads. Internal dip-switches (DSW) are used for testing purposes; test points are designated TP.

pulse   song   sine   song
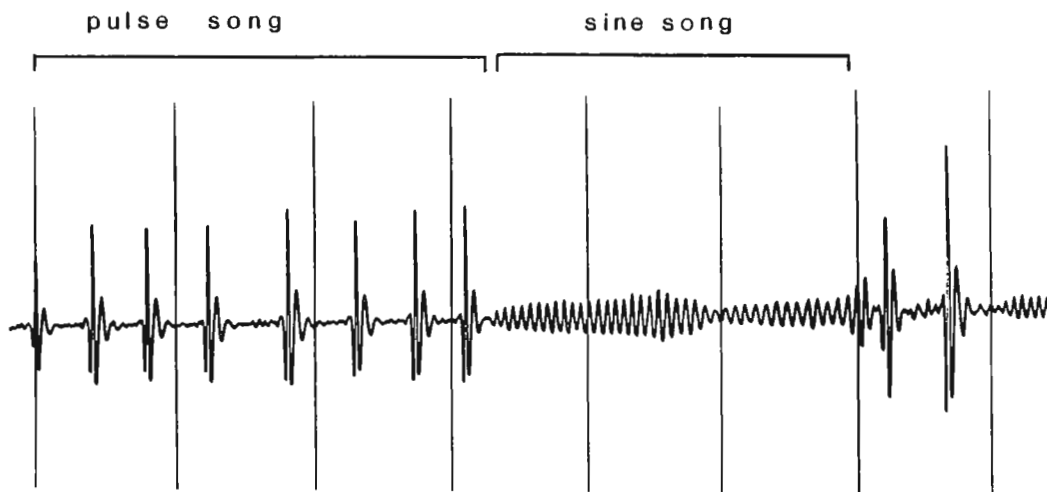
Figure 4. Oscillogram of ca. 0.7 s of courtship song from a **D.melanogaster** male. Output of the INSECTAVOX fed a reel-to-reel tape deck (but cassette recorders, too, perform well for this purpose); the song was converted to a visual record by feeding the tape deck's playback into a Datagraph 5-144 oscillograph (cf. Kyriacou & Hall 1985, 1986). Sine song, when concurrent with pulse song (right-hand part of trace), is always of lower amplitude. Distance between horizontal timing marks indicates 100 ms.

Instrument output is fed through an adjustable gain stage and then through a low pass and notch filter. The cutoff frequency for the low-pass was chosen to eliminate high frequency room sounds but not to decrease "pulse song" amplitude (see below, Figure 4). A notch filter was included to decrease the amplitude of the "sine song" (Figure 4), since it was thought that the normal amplitude of this song component might interfere with computer analyses that can be designed to determine various parameters involving the tone pulses (such as "interpulse intervals"; see, for example, Kyriacou & Hall 1985, 1986). Since the frequency of the sine song varies from 140-190 Hz, a low Q filter was employed.

The internal light source greatly improved observation of courting animals. Unlike bulky and noisy external light sources, it permitted convenient viewing through dissecting scopes; but because of the magnifying lens these were generally not needed. The lucite rod through which the light is transmitted apparently filters out most of the infra-red component. Temperature increases measured at the microphone surface were less than 1°C/0.5 h.

Results: The microphone and the amplifier performed admirably, in recordings during which male-female pairs were put into a cylindrical plastic chamber (height, 2.5 mm; inside diameter of the chamber itself, 9 mm); the top and bottom of the cylinder are covered with Nitex (nylon) mesh. The softest pulse trains from these recordings appeared larger in amplitude than intrinsic (electrical) background noise. In general, signal strengths -- 100-500 millivolts (mv) -- far exceeded this noise, which was 10 mv or less (Figure 4). Most of the troublesome noise encountered on the recordings was extrinsic (acoustic); it emanated from the flies and the environment. The former resulted from non-courting activities: walking, grooming, falling from the top of the chamber, and occasionally from the female beating her wings. In a relatively quiet room, the environmental noise (the main source of which was the building's heating/cooling system) from an unfiltered circuit was generally less than 50 mv. Depending on the exact frequencies of noise, filters can reduce this to about 10-15 mv (a 0.1 dB, 5th order Chebyshev with f_0=140 Hz was used for the recording displayed in Figure 4).

References: Bennet-Clark, H.C. 1984, J. Exp. Biol. 108:459; Cowling, D.W. & B. Burnet 1981, Anim. Behav. 29:924; Kyriacou, C.P. & J.H. Hall 1985, Nature 314:171; ——— 1986, Science 232:494; Schilcher, F.v. 1976, Anim. Behav. 24:18.

Hendriks, P.J. Catholic University Nijmegen, Netherlands. A new method of preparing squash preparations of Drosophila testes.

In elucidating the function of the Y-chromosome in spermatogenesis in **Drosophila hydei**, it is necessary to prepare squash preparations of Drosophila testes and to perform immunocytochemical procedures with these preparations. Also, these preparations are used in developing an electron microscopic postembedding labeling procedure.

In our laboratory a new method of preparing squash preparations of testes of **Drosophila hydei** was developed. This new method allows the preparation of microscopic slides featuring good preservation of cytological structure, little loss of testis-material and the possibility of performing immunocytochemical staining procedures. Also, with this procedure any fixative such as glutaraldehyde or formaldehyde may be used. Conventional procedures offered either a satisfactory preservation of cytological structure combined with a great loss of testis-material, or little loss of material combined with unsatisfactory preservation of cytological structure. Furthermore, fixation procedures involving coagulating fixatives such as acetic acid, methanol, ethanol or aceton were normally used.

In our new method, poly-L-lysine coated slides are used together with 100 μm thick teflon foil instead of conventional glass coverslips. Microscopic slides are coated with poly-L-lysine by applying 3-4 μl of a 1 mg/ml solutio of poly-L-lysine MW 80,000-120,000 (Sigma Chemicals Inc.). This drop of solution is then covered with a glass coverslip and left to react with the slide surface at room temperature, for at least 15 min. This method is more economical than methods described in the literature, in which slides or coverslips are immersed in poly-L-lysine solution for at least 30 min.

Immediately before use, the coverslip is removed with a scalpel, any remaining poly-L-lysine solution is left to dry in the air. Subsequently, Drosophila testes are isolated in preparation buffer and carefully squashed in a drop of fixative on the coated slide, not using a glass coverslip, but a small piece of 100 μm teflon foil (Yellow Springs Instruments Co.). The teflon foil is floated off on the surface of the fixative in which the slide is subsequently immersed. This obviates the freezing of the preparation in liquid nitrogen which is done in conventional procedures in order to remove the coverslip, and which causes damage to ultrastructural preservation.

Furthermore, 0.1% Triton-X-100 was included in the fixative and in all washing buffers, in order to lower the surface tension of these solutions, since it was observed that major loss of material occurs during immersion of the preparations in these solutions. An additional advantage of the application of Triton-X-100 is an improved penetration of antibodies.

References: Huang, W.M., S.J. Gibson, P. Facer, J. Gu & J.M. Polak 1983, Histochemistry 77:257-259; Lorber, P. 1972, Stain Technology 47:1972; Mazia, D., G. Schatten & W. Sale 1975, J. Cell Biol. 66:198-200.

Hostert, E.E., R.D. Seager and V.S. Berg. University of Northern Iowa, Cedar Falls. A thermotaxis apparatus with controlled water vapor pressure for Drosophila.

We describe a device to study thermotactic behavior in Drosophila through the use of a temperature/humidity gradient. The gradient is established and maintained through the use of a thermal gradient bar (Barbour & Racine 1967). This is an aluminum bar (110 cm long, 48.5 cm wide, 0.6 cm thick) with one end submerged in a cold antifreeze reservoir kept at -2°C and the other end submerged in a hot water reservoir kept at 34°C. A chamber with insulated sides and covered with clear plexiglass rests on the bar. The clear plexiglass top allows light from four 30 W cool white fluorescent light bulbs to enter the chamber.

The thermotaxis apparatus (60 cm long, 10 cm wide, 1 cm high) rests on the thermal gradient bar. Temperatures inside the thermotaxis apparatus range from 16°C to 29°C. The apparatus (resting directly on the thermal gradient bar) has no bottom, and is divided into six equal compartments, each 10 cm long and 10 cm wide. These compartments can be separated from each other by partitions which can be raised or lowered from the top of the apparatus. Each compartment has an individually hinged lid made of layers of fine wire and nylon mesh. The small spaces between the lids and the sliding doors are filled with cotton to prevent the flies from escaping.

Thermocouples (30 gauge) are placed at the ends of the apparatus and near the edges of each compartment, measuring a temperature range across each compartment. The thermocouples are shielded by aluminum foil from direct radiation from the lights above and to the gradient bar below. The temperature difference between the bottom of the apparatus (the bar) and the top is less than 1°C.

Perttunen & coworkers (Perttunen & Erkkila 1952; Perttunen & Salmi 1956; Perttunen & Ahonen 1956) and Prince & Parsons (1977) have shown that flies move in response to humidity differences. In an attempt to minimize this response we have designed our apparatus to maintain a constant water vapor

pressure. To do this, air which has been bubbled through water is continually pumped into a larger plexiglass box (90 cm x 20 cm x 15 cm high) placed over and surrounding the thermotaxis apparatus. The air is directed into six hoses which enter the back of the box and force all incoming air to come into contact with the gradient bar. This, along with the low flow rate, allows the air temperatures to quickly equilibrate with those generated by the gradient bar. The top of the thermotaxis apparatus itself is made of fine wire and mesh; this allows free air circulation into the apparatus. Since air of constant humidity is continually being added, the relative humidity within the apparatus stays between 25 to 40%. The temperature of the coldest part of the apparatus is above the dew point of the air being added.

An important point to consider when discussing thermotaxis (and when discussing temperature effects in general) is that warmer air is not only at a higher temperature, but also has greater drying power (a larger vapor pressure deficit) than cooler air. Vapor pressure deficit is the difference between the saturation vapor pressure, which is the total amount of water vapor that air of a given temperature and pressure could hold at saturation, and the actual vapor pressure. As the temperature increases, the saturation vapor pressure also increases, approximately doubling for each 10°C increase in temperature. Thus, if the actual vapor pressure remains constant, the vapor pressure deficit will increase as temperature increases (Fritschen & Gay 1979).

Controlling the vapor pressure helps to reduce wide fluctuations in vapor pressure deficit, but does not prevent a gradient of vapor pressure deficit from becoming established along with the temperature gradient. Thus flies in our and other (e.g., Prince & Parsons 1977) apparatuses may be responding to both a temperature and a humidity (a vapor pressure deficit and/or vapor pressure) gradient. To remove the vapor pressure deficit gradient one would have to set up a relative humidity gradient which was appropriately coordinated with the temperature gradient, so that the warmer air had a higher relative humidity. Since the flies, and thus the air, must circulate freely in these apparatuses, it is difficult to do this. It should be emphasized that flies in the wild also potentially respond to both temperature and humidity; thus our apparatus approximates the natural situation.

We introduce an equal number of flies into each compartment of the apparatus with the doors down (other methods of introduction are possible). Males and females are tested separately. The flies are three to five days old when tested since younger flies are more resistant to desiccation (Perttunen & Ahonen 1956; Ringo & Wood 1984). After the flies are allowed time to recover from transfer, the doors are raised and the flies are allowed to move in response to the gradient. After two hours the partitions between the chambers are lowered separating the flies as to their thermal preferences. If desired, the flies in each chamber can be removed by anesthetizing them with CO_2 and retested to check for repeatability or kept for breeding.

In order to control for possible behavioral differences due to differing physiological states, the flies are kept in a controlled temperature (20°C) and humidity (35%) incubator set on a 12 hour light/12 hour dark cycle. Flies are tested at the same time each day and thus are at the same point in the light/dark cycle when tested.

Acknowledgements: We thank P.D. Whitson for his help with this work.

References: Barbour, M.G. & C.H. Racine 1967, Ecology 48:861-863; Fritschen, L.J. & L.W. Gay 1979, in: Environmental Instrumentation, Springer-Verlag, NY, pp119-163; Perttunen, V. & U. Ahonen 1956, Suomen Hyonteistieteellinen Aikakauskirja 22:63-71; _____ & H. Erkkila 1952, Nature 169:78; _____ & H. Salmi 1956, Suomen Hyonteistieteellinen Aikakauskirja 22:36-45; Prince, G.J. & P.A. Parsons 1977, Aust. J. Zool. 25:285-290; Ringo, J. & D. Wood 1984, J. Heredity 75:181-184.

Jäger, R.F. and K.F. Fischbach. Institut für Biologie III, Univ. Freiburg, FR Germany. Some improvements of the Heisenberg-Böhl method for mass histology of Drosophila heads.

Obviously, the use of internal markers in genetic experiments with Drosophila is more tedious than the use of external ones. This is the main reason why most known mutants of Drosophila have visible external defects. Therefore, the Heisenberg-Böhl method (1979) which allows simultaneous handling and sectioning of up to 20 flies for paraffin histology marks a major breakthrough in Drosophila genetics. Our work with anatomical brain mutants depends heavily on it. The method is indispensable for quick screens of large and small numbers of brains, i.e., after mutagenesis, for housekeeping of mutant stocks, for complementation tests, for the evaluation of crosses, etc.

We recently extended its use to cryostat sections (Fischbach & Technau 1984). Here we list our standard protocol for paraffin histology and add some very simple procedures which allow the choice between frontal, horizontal and sagittal sections. Especially horizontal sections are a need for all who work with the visual system of flies.

FIRST DAY: (1) **Threading:** Up to 20 anaesthetized flies are carefully threaded by their necks into the collar (see Figure 1 for a blueprint of a collar). It is helpful to fix the collar with a double sided tape. Use a stereomicroscope. Please note that the orientation of flies in the collar restricts the possible planes

of sectioning (Figure 2). Adding of one or two white eyed flies facilitates identification of individual flies after sectioning. (2) **Fixate** in freshly made **Carnoy solution** for 4 hr: 60 ml ethanol, 30 ml chloroform, 10 ml acidic acid. (3) **Transfer** 3 times into 100% ethanol (technical): 3 x 30 min. (4) **Transfer** into 100% ethanol (dried): 30 min. (5) **Transfer** into methylbenzoate: overnight. Steps 1 to 5 at room temperature.

    SECOND DAY: (1) **Transfer** into an already prepared 1:1 mixture of methylbenzoate and paraffin for 1.5 hr. Choose a paraffin with a low melting point <65°C. (2) **Transfer** 4-5 times into pure paraffin, 1 hr each, <65°C. (3) **Embed into paraffin:** Put collar at the bottom of an aluminium trough (see Figure 2). The orientation of the collar in the trough determines the possible planes of sectioning (see Fig. 2). Fill trough with melted paraffin. Put filled trough into the refrigerator or on a cooling plate. (4) **Blocking:** Moderately heat a block of aluminium of adjusted size, put on top of the trough and cool it. (5) **Breaking:** Remove unnecessary paraffin. Break collar off (see Figures 3a, 4a). Heads should remain neatly aligned in the paraffin on the aluminium block. Bodies still stick in collar.

    THIRD DAY OR EVENING OF SECOND (see Figures 3, 4): (1) **Trim** the paraffin **parallel** to the row of heads on both sides. (2) **Cut 7** μm sections. If trimming was alright, you should get a nice band of sections. (3) **Transfer** band of sections onto a slide coated with water. Notice that the red eye pigment

A   **B**
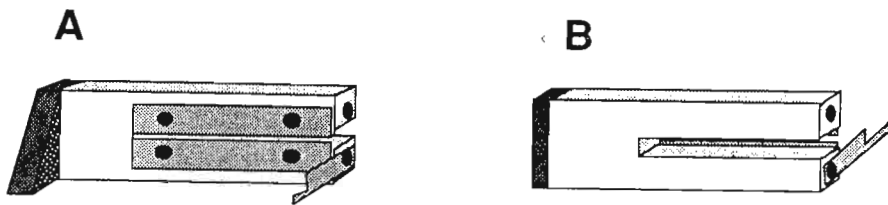
Figure 1. The fly collar. A: front view. B: back view. C: blueprint.

C
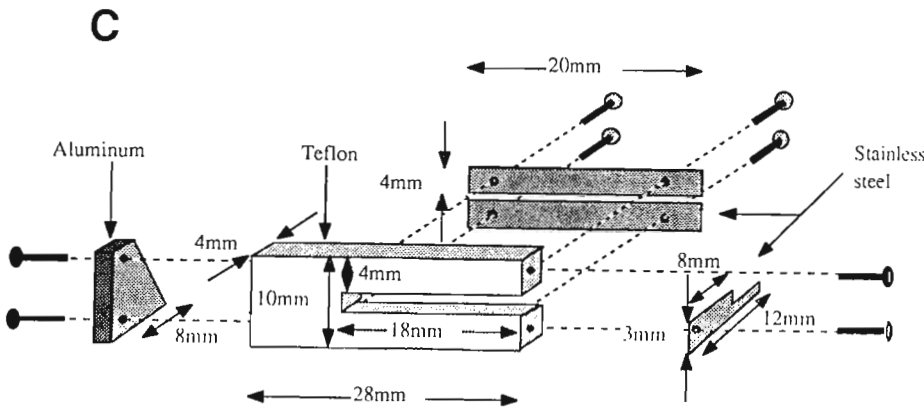
Figure 2. Embedding. Orientation of collar in trough and of flies in collar for frontal (A), horizontal (B), and sagittal sections (C).
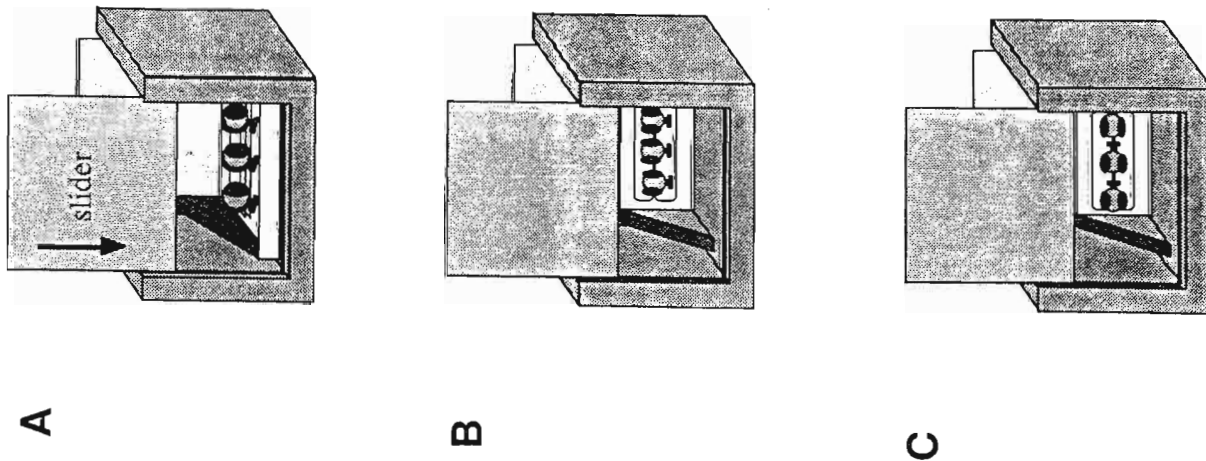
A   **B**   **C**
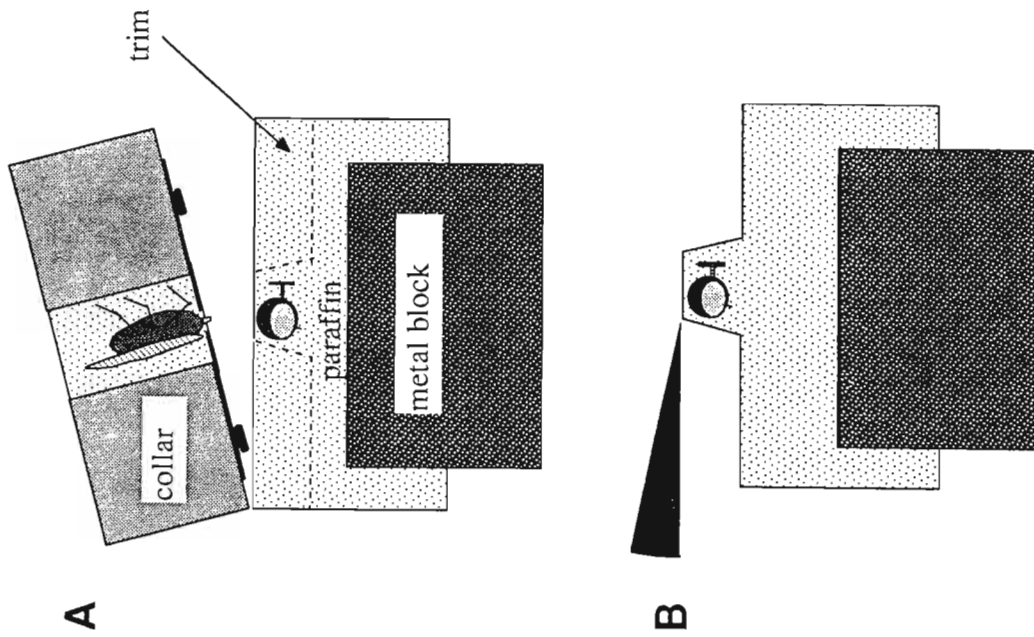
Figure 3. Blocking and breaking (A) of a collar embedded for frontal sectioning (B).
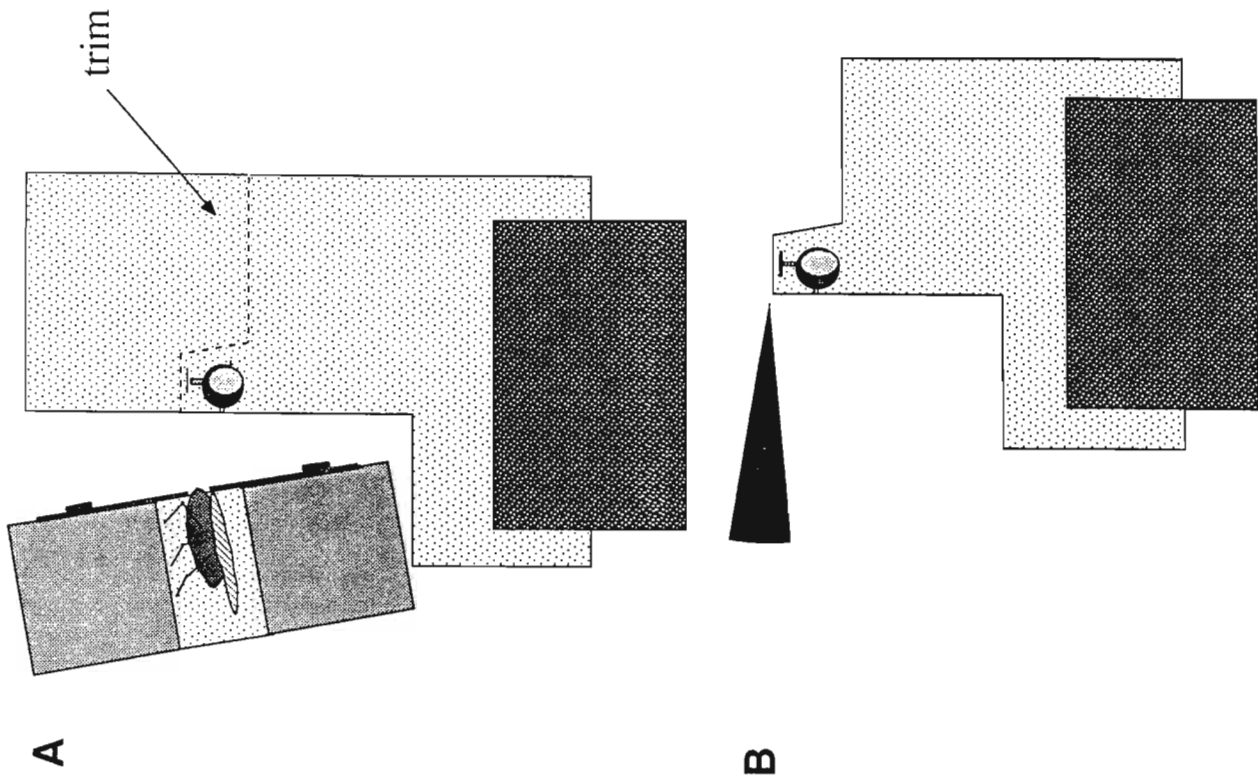
Figure 4. Blocking and breaking (A) of a collar embedded for horizontal sectioning (B). After embedding for sagittal sections, these procedures are identical.

Figure 5. Sector of a slide with horizontal sections through the heads of several **Drosophila melanogaster.** The flies had been prepared as described in Figs. 2b, 4. Sections were stained with the Holmes-Blest method. Serial sections of single fly heads are aligned in columns.

dissolves in the water and distributes itself evenly over the sections. Dry slides below 50°C. (4) **Remove** paraffin by xylol; 2x10 min. After this step, you may branch to a silver staining procedure (e.g., the Holmes-Blest method; Blest 1961), or: (5) **Cover** slides with DePeX. (6) **Use** the fluorescence of the red eye pigment for viewing the sections under the fluorescence microscope. A result is shown in Figure 5 which represents horizontal sections through the heads of Drosophila.

sents horizontal sections through the heads of Drosophila.

References: Blest, A.D. 1961, Q. J. Micr. Sci. 102:413-417; Fischbach, K.F. & G. Technau 1983, Dev. Biol. 104:219-239; Heisenberg, M. & K. Böhl 1979, Z. Naturforsch. 34:143-147.

Piven, V.N. and V.A. Rizhikov. Biophysics Dept., Kazakh State Univ., Timiriazeva 46, Alma Ata, 480121 USSR. A simple device for cold immobilization of Drosophila.
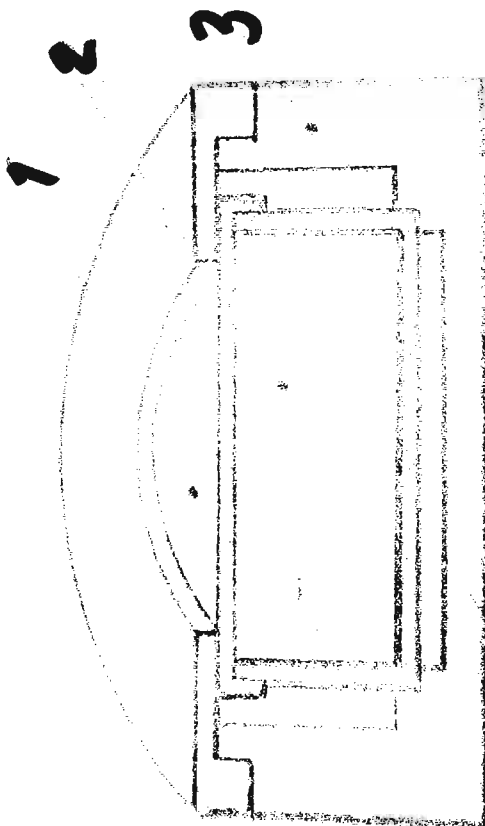
Anesthesia methods with diethyl ether or carbon dioxide usually used have some disadvantages. So ether treatments decrease fly viability and reproductive capacity. Diethyl ether vapours are also toxic for the operator. To anesthetize with carbon dioxide for a long period of time, it is necessary to establish a continuous flow of the gas through a vessel with flies. A high concentration of carbon dioxide in the working room is also undesirable. So a simple device for cold immobilization of flies and larvae is proposed. A metallic box filled with ice is placed in a thermo-isolating case. The box and the case are easily disassembled (Figure 1). Flies after a brief carbon dioxide anesthetization or cooling in a refrigerator are placed on the upper plate of the metallic box. Larvae can be placed directly onto the plate. With the help of the device, it is possible to manipulate the flies or larvae on a working table for a long period of time.

Figure 1. 1 = metallic box; 2 = ice; 3 = disassembled thermoisolating case.
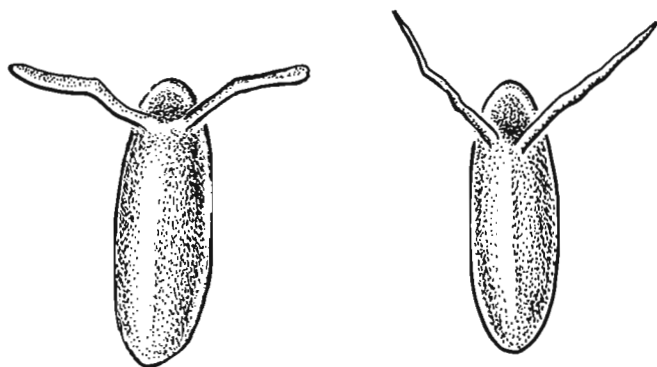
Figure 1. Eggs of **D.melanogaster** (left) and **D.simulans.** Note the thicker, flatter filaments of **D.melanogaster.**

Ringo, J., K. Chess and H. Dowse. University of Maine, Orono. Distinguishing the eggs of **D.melanogaster** and **D.simulans.**

Here we report characters of the eggs of **D.melanogaster** and **D.simulans** which allow them to be distinguished quickly and reliably. This may be useful for behavioral, ecological, and taxonomic studies. Sameoto & Miller (1966) observed that the egg filaments of **D.simulans** are slightly longer than those of **D.melanogaster.** In addition, the egg filaments of **D.melanogaster** have a distinctive club-like shape while those of **D.simulans** are markedly straighter and narrower (Figure 1).

To check the reliability of egg morphology as a character to distinguish the two species, we allowed females of both species to oviposit together on watch glasses. There was considerable mixing of eggs, although **D.melanogaster** had a stronger tendency to lay near the edges than its sibling species. Four hundred eggs identified as each species were collected and placed in lots of 50 on moist tissue in culture vials. Adults developing from these eggs were collected until virtually all eclosed, male imagoes being scored using genitalia (Sturtevant 1920). One out of the 164 males (0.6%) in the **D.melanogaster** vials was **D.simulans,** and 4/185 (2.2%) males in the **D.simulans** vials were **D.melanogaster.**

References: Sameoto, D.D. & R.S. Miller 1966, Ecology 47:695-704; Sturtevant, A.H. 1920, Genetics 5:488-500.

Silva, F.J. and J.L. Mensua. Universidad de Valencia, Spain. A rapid method for quantifying isoxanthopterin in **Drosophila melanogaster.**

The biosynthesis of pteridines in **Drosophila melanogaster** seems to be controlled by a great number of genes. Mutants at these genes have been characterized by measuring their amounts of pteridines in the eyes and other parts of the body. Several investigators have normally used paper chromatography or thin layer chromatography to study these mutants (Taira 1961; Ferre et al. 1986; etc.). One of the quantified pteridines is isoxanthopterin, a violet fluorescent compound mainly accumulated in the male bodies (testes).

Since two-dimensional thin layer chromatography (TLC) plus fluorescence is a slow and not completely accurate method for the quantitation of isoxanthopterin, we have tried to develop a method that would permit the quantitation of isoxanthopterin using its violet fluorescence but without using the TLC procedure. This method would be faster and would avoid degradations due to the short period of time between the extraction of the compound and its measurement. Since the excitation and emission wavelength of isoxanthopterin (excitation at 350 nm and emission at 408 nm) is rather different from that of other pteridines, we tested the measurement of this compound in a homogenization of flies. To determine the interference of other fluorescent pteridines (mainly blue fluorescent pteridines), we used the strain marron-like (mal) as a control, since this mutant prevents the synthesis of isoxanthopterin and leads to accumulation of some blue fluorescent pteridines.

The experiment was carried out with male and female bodies and with heads. The extracts contained two headless male bodies, two headless female bodies and twenty heads, respectively, in two milliliters of distilled water.

The results (Table 1) showed that the maximum interference of other pteridines in male bodies was less than 1% of the amount of wild type male bodies, since the mutant mal, that accumulates more blue fluorescent compounds in the bodies than wild type, presented only 1% of the wild type fluorescence. The amount of isoxanthopterin in female bodies was low, as expected.

In heads, the amount of isoxanthopterin seems to be very low and the relative interference of other pteridines is comparatively high, since the strain mal (a mutant without detectable levels of isoxanthopterin) presented 90% of the fluorescence of the wild type strain.

In Table 2 are shown the amounts of isoxanthopterin in headless male bodies of various double mutant strains. Note that the amount of isoxanthopterin in the strain bo lix is nearly 0, indicating that the effect of other compounds in the fluorescence is probalby lower than 1%. The standard deviations in these quantitations (3 or 4 repetitions for strain) are very low.

Table 1. Quantitation of isoxanthopterin in the strains Oregon R and marron-like.

		Fluorescence	ng IXP/fly[3]	%Or-R
Or-R	male bodies[1]	57.5 ± 2.0	198.4 ± 6.9	100.0
	female bodies[1]	1.8 ± 0.1	6.2 ± 0.4	3.1
	heads[2]	5.7 ± 0.3	2.0 ± 0.1	1.0
mal	male bodies	0.6 ± 0.0	2.1 ± 0.0	1.0
	female bodies	0.6 ± 0.0	2.1 ± 0.0	1.0
	heads	5.1 ± 0.2	1.8 ± 0.1	0.9

1=two headless bodies in 2 ml distilled water; 2=twenty heads in 2 ml distilled water; 3=ng isoxanthopterin/body or head, respectively.

Table 2. Quantitation of isoxanthopterin in headless male bodies of various mutant strains.

Strain	ng IXP/body	%Or-R
bo lix	1.0 ± 0.0	0.5
rb pr	5.5 ± 1.8	2.8
lix se	10.6 ± 0.6	5.4
ltd ca	11.5 ± 0.2	5.9
bo se	13.1 ± 0.4	6.7
lix dke	16.6 ± 0.6	8.5
g dke	17.8 ± 0.6	9.1
g rs[2]	50.8 ± 3.9	26
cn rs[2]	105.5 ± 7.8	54
sf st	123.0 ± 3.9	63
pn dke	179.7 ± 2.0	92

References: Ferre, J., F.J. Silva, M.D. Real & J.L. Mensua 1986, Biochem. Genet. 24:545-569; Taira, T. 1961, Jap. J. Genet. 36:18-31.

Stearns, S.C., T. Diggelmann, M. Gebhardt, H. Bachmann and R. Wechsler.* Zoology Inst., Basel, Switzerland; *-Birsfelden, Switzerland. A device for collecting flies of precisely determined post-hatching age.

In the course of work on the evolution of reaction norms for age and size at hatching, it became desirable to measure age at hatching with the same precision that one can measure dry weight. Measuring age is always labor-intensive when done by hand, especially when sample sizes are as large as they must be for work on the quantitative genetics of reaction norms. Therefore, we developed a device resembling a fraction collector that automatically collects all flies that hatch in pre-determined and adjustable intervals and holds them for later analysis.

DESCRIPTION OF THE DEVICE (see Figure 1). Vials containing larvae and pupae are placed upside down in a bridge that travels over a tray of upright vials. At pre-set intervals CO_2 is sprayed into the vials for 30 sec, any hatched flies fall into the upright vials below, the bridge is advanced to the next row of upright vials, and a plate that travels with the bridge advances to seal the flies into the vials. The collection intervals in our version can be set at 2, 4, 6, 8, 10 or 12 hr, and the device will hold 42 vials of hatching flies.
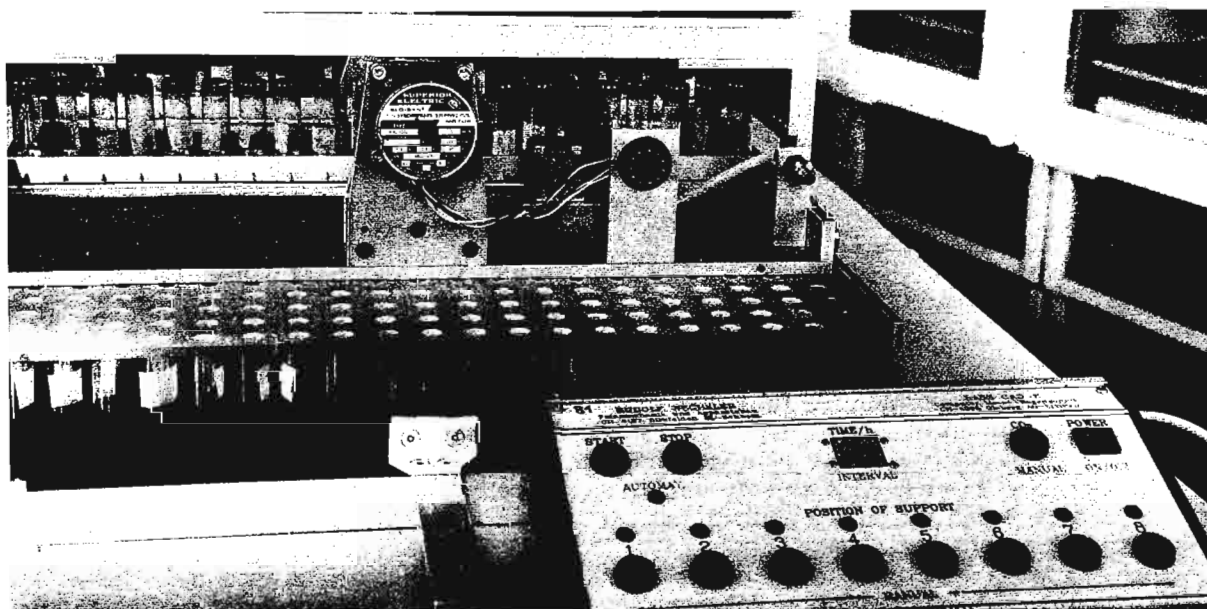
Figure 1. Photograph of the control panel (lower right) and the main body of the device. Labeled collection vials stand in rows below the rigid plastic plate on which the vials containing larvae and pupae travel. The collection vials are set into a tray that is removed from the device to collect the flies.

Table 1. Effects of device/chamber, food, and line on age at hatching (days) in **D.melanogaster**.

Device	Food	Line	N	Females Mean	SE	N	Males Mean	SE
1			30	9.24	0.11	29	9.38	0.12
2			30	9.06	0.10	30	9.31	0.10
	n		30	8.72	0.06	30	8.90	0.06
	o		30	9.58	0.08	29	9.80	0.09
		MA4	20	9.18	0.13	20	9.55	0.17
		MB1	20	9.21	0.10	20	9.35	0.08
		MG2	20	9.05	0.16	19	9.11	0.14
	n	MA4	10	8.69	0.05	10	8.91	0.04
	n	MB1	10	8.96	0.09	10	9.16	0.10
	n	MG2	10	8.50	0.13	10	8.64	0.06
	o	MA4	10	9.67	0.12	10	10.19	0.16
	o	MB1	10	9.46	0.15	10	9.55	0.10
	o	MG2	10	9.60	0.13	9	9.64	0.13

Table 2. ANOVA for effects on age at hatching of females.

Source	SS	df	MS	F	P
Machine	0.507	1	0.507	3.93	0.053
Food	11.173	1	11.173	86.55	0.000***
Line	0.285	2	0.142	1.10	0.340
M x F	0.024	1	0.024	0.19	0.666
M x L	0.009	2	0.005	0.04	0.966
F x L	0.995	2	0.498	3.85	0.028*
M x F x L	0.603	2	0.301	2.34	0.108
Error	6.197	48	0.129		

Table 3. ANOVA for effects on age at hatching of males.

Source	SS	df	MS	F	P
Machine	0.071	1	0.071	0.76	0.388
Food	11.811	1	11.811	127.06	0.000***
Line	1.877	2	0.938	10.09	0.000***
M x F	0.044	1	0.044	0.48	0.493
M x L	0.843	2	0.422	4.53	0.016*
F x L	1.888	2	0.944	10.15	0.000***
M x F x L	0.574	2	0.287	3.09	0.055
Error	4.369	47	0.093		

Table 4. Effects of device/chamber, food, and line on dry weight at hatching (μg) in **D.melanogaster**.

De-vice	Food	Line	N	Females Mean	SE	N	Males Mean	SE
1			30	269.61	9.29	29	215.79	7.21
2			30	256.14	24.63	30	213.06	4.86
	n		30	277.02	13.07	30	227.31	5.55
	o		30	248.73	22.61	29	201.05	5.64
		MA4	20	210.36	33.21	20	191.50	5.95
		MB1	20	306.20	8.24	20	230.23	7.70
		MG2	20	272.07	13.17	19	221.85	5.47

Table 5. ANOVA for effects on dry weight at hatching of females.

Source	SS	df	MS	F	P
Machine	2723.0	1	2723.0	0.30	0.586
Food	12006.3	1	12006.3	1.32	0.256
Line	94387.4	2	47193.7	5.20	0.009***
M x F	4113.9	1	4113.9	0.45	0.504
M x L	48060.9	2	24030.5	2.65	0.08
F x L	3556.6	2	1778.3	0.20	0.82
M x F x L	4879.2	2	2439.6	0.27	0.77
Error	435646.8	48	9076.0		

Table 6. ANOVA for effects on dry weight at hatching of males.

Source	SS	df	MS	F	P
Machine	110.1	1	110.1	0.15	0.697
Food	10172.2	1	10172.2	14.22	0.000***
Line	16554.9	2	8277.4	11.57	0.000***
M x F	617.3	1	617.3	0.86	0.358
M x L	545.4	2	272.7	0.38	0.685
F x L	809.7	2	404.9	0.57	0.572
M x F x L	360.8	2	180.4	0.25	0.778
Error	33623.7	47	715.4		

The device can be controlled either manually or automatically. The flies are unloaded by sliding the receiver tray out, manually pushing a button to spray CO_2 to anaesthetize one row of flies at a time, and capping each row as it emerges from the machine.

TECHNICAL DATA: Weight (ca. 23 kg); Volts (220/24); Watts (22); Gas (CO_2); Pressure (0.5 bar); L x W x H (375 x 490 x 200 mm).

This version was built to fit our growth chambers. Mr. Wechsler can build such devices to fit any chamber. The constraints on the design are: (1) the size of the vials, (2) the width of the growth chamber, which together determine how many vials will fit in one row, and (3) the depth of the growth chamber, which determines how many vials will fit in a column and thereby the number of possible sampling intervals. Our chambers are 21 vials wide and 16 vials deep. Since 8 collection intervals are more than enough to get through a weekend, the device actually has two bridges that each hold 21 vials traveling over 8 collection columns.

The technical challenge in building such a device is to keep the traveling bridge absolutely flat while preserving a tolerance of less than 0.1 mm across the collection tray so that the flies remain sealed in their vials, to ensure that this tolerance will be held during years of service, and to find a material to hold the collection vials that is both stiff and slippery. The use of a microprocessor, the special materials, and the fine work needed to deliver the same amount of CO_2 to each vial automatically do increase the cost of the device, but in our experience it functions precisely and reliably.

Figure 2. Age and dry weight at eclosion of female and male **D.mercatorum** in two different culture media (new and old). The lines connect the means from the same treatment, genotype and sex obtained by replication in two collecting devices which were located in two separate climate chambers. The four encircled clusters represent all combinations of food media and sex which had the largest effects on both age and dryweight. For example, the upper left cluster contains the points obtained from females in the new medium. The line with code numbers 5 and 21, for example, connects the means obtained in the two devices from one of the three isofemale stocks (the smaller code number always represents data from device number 1).

AN EXPERIMENT TO TEST IT. To illustrate the type of experiment where the device is a considerable help, we report here a pilot experiment done to compare the effects of two food media and of position in two such devices, each placed in a different climate chamber, on age and size at hatching in **D.melanogaster**. The **new** medium is described by Backhaus, Sulkowski & Schlote (1984); it was cooked for 2-3 min. The **old** medium was 100 g cornmeal, 60 g sucrose, 15 g dried yeast and 13.8 g agar made up to 1 liter with distilled water; it was cooked for 1 hr.

Three isofemale lines collected from the Basel area in the fall of 1986 were tested on each medium. After an egg-laying period of 5 hr, 5 replicate vials were prepared for each line, food treatment, and device/chamber (5 vials x two food treatments x 2 device treatments x 3 lines = 60 vials). 12 eggs were placed into each vial, which contained 2 ml (±5%) of medium. The vials were mounted in the devices in the climate chambers at 25°C and 80% relative humidity. As the flies hatched, they were collected at 8 hr intervals, dried at 70°C for 3 hr, and weighed on a Mettler microbalance to 0.01 mg.

The analysis was done on the replicate means for age and weight and for males and females. First the means, then the ANOVAs are presented. Figure 2 summarizes all effects.

From this analysis, we can draw the following conclusions: (1) The device functions reliably and can be used to detect differences in mean age at hatching that are as small as 0.5 days in this type of design. In less complicated designs, smaller differences would be detectable. The device saves labor and results in data in which time is precisely measured. (2) Pure machine effects were never significant, although for age at hatching of females they were almost significant (P=0.053). Machine effects probably express differences between climate chambers as much as they express differences between machines. (3) The difference in food media had a large and significant effect on age at hatching of both females and males and on dry weight of males. Both sexes hatch about 1 day sooner on the new media. (4) Differences among lines were expressed in males for age and weight but in females for weight only. (5) Interaction effects were significant only for the interaction between food and lines in determining age (both sexes) and for the interaction of machine and line in determining age of males. There were no interaction effects detected for the determination of weight for either sex.

Anyone interested in ordering a similar device should contact Mr. Wechsler directly (Gartenstrasse 5), including in the letter details on the dimensions of the vials to be used, the permissable limits on the overall dimensions of the device, and any other specifications thought necessary. We suggest that you request a written offer and an estimated delivery date from him.

References: Backhaus, B., E. Sulkowski & F.W. Schlote 1984, DIS 60:210-212.

Whiting, J.H.Jr., J.L. Farmer and D.E. Jeffery. Brigham Young University, Provo, Utah. Improved in situ hybridization and detection of biotin-labeled **D.melanogaster** DNA probes hybridized to **D.virilis** salivary gland chromosomes.

We have modified and improved the procedure described in Pliley, Farmer & Jeffery (1986). We can now detect hybridization of probes which previously had given us negative results. Probes which have been reported to hybridize weakly to multiple sites on **D.melanogaster** chromosomes can now be seen to hybridize to apparently homologous multiple sites on **D.virilis** chromosomes.

Hybridization is now reliable enough that we routinely photograph chromosomes before and after hybridization. Since hybridization always leads to deterioration of morphology, the before and after pictures allow us to identify the site of hybridization with much greater precision. Before hybridization, the chromosomes are photographed with 95% ethanol under the coverslip. After hybridization, they are photographed with water under the coverslip. Ethanol bleaches the stained probes.

We have not changed the procedure for preparing chromosomes.

PREPARATION OF PROBES FOR HYBRIDIZATION: The sensitivity of the procedure is greatly improved if the labeled probe is not purified by gel exclusion chromatography. Apparently a large amount of probe is lost on the column.

The probe is labeled with BRL (Bethesda Research Lab.) biotin-11-dUTP and the BRL nick translation kit using the protocol on the biotin-11-dUTP data sheet. The reaction is stopped with BRL stop buffer and a 10 min incubation at 65°C.

To the 55 µl of nick-translation reaction mixture is added 17 µl of sonicated salmon testes DNA (12 µg/µl), 12 µl of 5% blue dextran solution (a convenient dye marker), 0.28 ml TE (10 mM TRIS-HCl, pH 7.5; 1 mM EDTA) and 40 µl 20% sodium acetate (pH 5.4). The DNA is precipitated with 1 ml of 95% ethanol. The tube is chilled in an ice-water bath for 10 min. The tube is centrifuged at 10,000 x g for 10 min. The supernatant is carefully removed with an aspirator and the alcohol is evaporated in a hood or vacuum. The pellet is dissolved in 117 µl of water. If the DNA is not to be denatured immediately, store the solution in the freezer until needed.

DENATURATION OF DNA AND ADDITION OF HYBRIDIZATION REAGENTS: The probe is denatured in a boiling water bath (94-96° in Provo) for 5 min and plunged into an ice bath. The denatured probe is never allowed to rise above 0° until transferred to the slides. The following is added to the probe solution: 40 µl 20XSSC (SSC is 0.15 M NaCl, 0.015 M sodium citrate, pH 7.0), 4 µl 50X Denhardt's solution (Denhardt 1966), and 40 µl 10% dextran sulfate (5,000 MW, which works as well as 500,000 MW). The probe is divided among as many tubes as desired and stored at -20°C until needed. Probes may be thawed and refrozen at least four times with little change in hybridization. They are stable indefinitely.

We have not been able to detect hybridization when we omit both dextran sulfate and Denhardt's solution.

DENATURATION OF CHROMOSOMES: The chromosomes are denatured in 0.07 N NaOH for 3.5 min at 37°C. The slides are immediately washed in 70% ethanol for 4 min and then in 95% ethanol for 4 min. The slides are dried in air at room temperature and used immediately for hybridization.

HYBRIDIZATION: About 12 µl of probe solution are placed on each slide, directly over the chromosomes, and covered with an acid-washed coverslip. (Coverslips are prepared shortly before use by dipping them in 0.05 N HCl, distilled water, and acetone, allowing them to dry in air.) The edges of the coverslip are sealed by dripping long strands of rubber cement around the edges. The slides are placed in a sealed plastic box with a towel saturated in 2XSSC. The box is floated in 58.5° water bath for 6 hr. Hybridization can be detected after three hr, but the staining is not as intense.

The rubber cement is carefully removed, taking care to move the coverslip as little as possible. The coverslip is removed by dipping the slide briefly in a beaker of 2XSSC. The slides are washed twice in 2XSSC at 53.5° for 20 min each and once in SSC at room temperature. You may proceed directly to the next step, or you may leave the slides in SSC at 4°. They can be stored this way for at least 3 days.

DETECTION OF HYBRIDIZED PROBE: We use the BRL BluGene kit for detecting biotinylated DNA. We have found it to be much more sensitive than the previous detection kits. The slides are washed in BRL BluGene Buffer 1 (0.1 M TRIS-HCl, pH 7.5, 0.15 M NaCl) supplemented with 1% BSA for one hr. The slides are drained but not allowed to dry. About 100-200 µl of diluted streptavidin-poly(AP) conjugate (2 µl of BRL stock solution in 1.5 ml of BRL BluGene Buffer 1) is placed directly over the chromosomes. A coverslip is placed over the chromosomes and the slides are placed in a sealed plastic box containing a towel saturated in the same buffer. The box is left at room temperature for 2 hr.

The coverslip is removed by dipping the slide in a beaker of BRL BluGene Buffer 1. The slides are washed twice in about 25 ml of the same buffer at room temperature for 10 min each and once in about 25 ml of BRL BluGene Buffer 3 (0.1 M TRIS-HCL, pH 9.5, 0.1 M NaCl, 0.05 M MgCl$_2$) at room temperature for 10 min. The slides are drained but not allowed to dry. About 100-200 µl of dye solution (3.3 µl NBT

solution in 0.75 ml BRL BluGene Buffer 3, mix gently, add 2.5 µl BCIP solution) is placed directly over the chromosomes. A coverslip is placed over the dye solution and the slides are placed in a sealed plastic box containing a towel saturated in the same buffer. The box is incubated at room temperature, in the dark, overnight.

     The coverslip is removed by dipping the slide in a beaker of distilled water. The slides are washed in distilled water for up to 3 hr. The chromosomes are photographed with water under the coverslip. The slides are dried in air and stored in the dark at 4°. The quality of the slides has been maintained (in our dry climate) for many months this way. Slides have also been stored in the dark at room temperature with little or no loss of quality.

     SPECIAL NOTE: A detailed, step-by-step description of this procedure, prepared for our laboratory, is available on request from J.L. Farmer.

     References: Denhardt, D.T. 1966, Biochem. Biophys. Res. Com. 23:641-646; Pliley, M.D., J.L. Farmer & D.E. Jeffery 1986, DIS 63:147-149.

Woodruff, R.C., J.N. Thompson Jr.*, A.A. Szekely and J.S. Gunn. Bowling Green State University, Bowling Green, Ohio; *University of Oklahoma, Norman. Characterization of Drosophila lines for transposable elements by Southern blot analysis with biotinylated-DNA probes.

We have used the following protocol with biotin to identify P and I elements in Drosophila. This protocol, which should work for the identification of other cloned Drosophila DNA sequences, comes from a mixture of trial and error, the literature, a New England BioLabs Workshop, the Bethesda Research Laboratories, Davis et al. (1986), Maniatis et al. (1982), and our friends, including M. Ashburner, R. Brodberg, D. Deters, S. Easteal, C. Langley, R. Lyman, E. Montgomery, J. Phillips, S. Popoff, B. Slatko, E. Strobels, E. Underwood, and M. Young. This protocol is written in detail as an aid to individuals (such as ourselves) that have little molecular biology experience. If one is interested only in the biotin technique, skip to item number seven.

     1. Drosophila Genomic DNA Extraction: Grind 100 adult flies (mixed sex) in one ml of grinding buffer. The grinding buffer is made up of four parts Drosophila homogenizing buffer (0.1 M NaCl, 0.2 M sucrose, 0.01 M EDTA, 0.03 M Tris-HCl, pH 8.0), one part phage lysis buffer (0.25 M EDTA, 2.5% sodium dodecyl sulfate-SDS, and 0.5 M Tris-HCl, pH 9.2), and 1/100 volumes of diethylpyrocarbonate (DEPC). The final concentration of the grinding buffer is 0.08 M NaCl, 0.16 M sucrose, 0.058 M EDTA, 0.024 M Tris-HCl, pH 8.0, 0.1 M Tris-HCl, pH 9.2, and 0.5% SDS. Use fresh grinding buffer from stocks and grind flies in a glass grinder, minimizing foam (keep all of these grinds on ice). Rinse the grinder with 0.4 ml of grinding buffer and filter through sterile cheesecloth or fine-pore nylon mesh into sterile microfuge tubes. Place cheesecloth or nylon directly into top of tubes during filtration. We find that more material is collected using nylon, but sometimes the filtrate inhibits DNA digestions. If you use cheesecloth, it absorbs liquid and you will need to press solution out of the cloth with a pipette tip; this method can give variable volumes of filtrate. Try the filtration steps with nylon and cheesecloth, and then decide which method is best for you.

     Invert tube once and incubate at 70°C for 30 min to precipitate proteins (do not vortex genomic DNA). Measure the volume in the microfuge tube with disposable 1 ml pipette (or graduated microfuge tubes). Add 8 M K-acetate to final concentration of 1 M (volume/8; for example, for a one ml sample, add 0.125 ml of 8 M K-acetate). Mix by inverting tube about four times and incubate on ice for approximately 30 min or longer. Spin in cold microfuge for 10 min, and transfer supernatant to new microfuge tube. This is done by pouring off the supernatant into the new tube and removing the rest of the liquid next to the pellet with a pipetter (with grinds of 100 or so flies, we do not attempt to remove the liquid from around the pellet). Discard the pellet.

     Again, measure the volume with a disposable 1 ml pipette. Add one or more volumes of isopropanol to the tube (may need to separate the supernatant into two or more tubes dependent on volume collected). Mix by inverting repeatedly and incubate for 2 min. Spin for 10 min and pour off the supernatant (occasionally there is a fat-like glob that comes off in the supernatant). Be careful; the pellet may be loose here and in subsequent steps. Wash pellet by suspending in two or more volumes of cold 70% ethanol. Spin for 1-2 min and discard supernatant. Resuspend in 1 ml of 70% ethanol and pour off supernatant.

     Air dry the pellet at room temperature in vacuum (we use a vacuum oven) for about 5-10 min. Do not dry too long or it will be difficult to resuspend the pellet. We usually dry until liquid drops on the side

of tube are not visible. Dissolve the pellet in 60 ul of TE (pH 7.4). The amount of TE placed into the tube depends on the number and sex of the flies that are ground at the beginning of the protocol; differences in TE amounts are to compensate for differences in sizes of male and female flies. If all flies are females, use 1 ul of TE per fly that was ground. If all flies are males, or if a mixture of males and females, use 0.5 ul of TE per fly that was ground. (Note that we use 60 ul instead of 50 ul in this protocol to increase the recovery of DNA and because we use 20 ul samples per well during electrophoresis.) Also keep in mind that you must allow for any splitting of the solution into separate tubes at the above isopropanol step. Finally, to help dissolve the pellet you can heat the mixture at 37°C for five min. However, even with heating it is sometimes difficult to get the pellet into solution. You now have 100 flies worth of DNA in 60 ul of TE. The DNA can be stored at 4°C.

R E A G E N T S:

Drosophila homogenizing buffer (100 ml): 2.0 ml 5 M NaCl, 20.0 ml 1 M sucrose, 2.0 ml 0.5 M EDTA, 3.0 ml 1 M
    Tris-HCl (pH 8.0), 73 ml water (always use double distilled or the equivalent)

Phage lysis buffer (100 ml): 50.0 ml 0.5 M EDTA, 25.0 ml 10% (w/v) SDS, 25.0 ml 2 M Tris-HCl (pH 9.2)
    (heat to get SDS into sol.)

Grinding buffer (10.1 ml): 4 parts Drosophila homogenizing buffer = 8 ml, 1 part phage lysis buffer = 2 ml,
    add 1/100 vol. of diethylpyrocarbonate (DEPC) just before use = 0.1 ml

8 M K-acetate (100 ml): 78.56 g K-acetate. Fill to 100 ml with water

5 M NaCl (1 liter): 292.2 g NaCl. Fill to 1 liter with water.

1 M sucrose (100 ml): 34.23 g sucrose. Fill to 100 ml with water.

0.5 M EDTA, pH 8.0 (1000 ml): 186.1 g EDTA, Fill to 800 ml with water. Stir and add about 20 g NaOH pellets
    to adjust pH to 8.0. Fill to 1 liter with water.

1 M Tris-HCl, pH 8.0 (1000 ml): 121.1 g Tris base. Fill to 800 ml with water. Adjust pH by adding
    concentrated HCL (about 42 ml).

1 M Tris-HCl, pH 7.4 (1000 ml): As above using more HCl.

2 M Tris-HCl, pH 9.2 (1000 ml): 242.2 g Tris base. Fill to 800 ml with water. Adjust pH by adding concentrated HCl.
    Fill to 1 liter with water.

TE, pH 7.4 (100 ml): 1 ml 1 M Tris (pH 7.4), 0.2 ml 0.5 M EDTA, 98.8 ml water.

2. Restriction Endonuclease (RE) Digestion of Genomic DNA: Use standard digestion protocol with appropriate buffer, etc. The following is an examle using Hind III. To a microfuge tube add, in order, 6 ul water, 8 ul 10X buffer, and the 60 ul of DNA collected in the above procedure (use a fresh, sterile pipette tip for each addition). Mix by flicking with finger and add 6 ul of Hind-III enzyme (60 units). Mix again by flicking and spin in microfuge for 5 sec to bring drops down from side of the tube. Digest at 37°C for five hr or longer (can check the progress of digestion by running a sample on an agarose mini-gel).

Add 8 ul of a 1 mg/ml stock of RNAse (gives approximately 100 ug per ml), and incubate at 37°C for 30 min. Add 3.7 ul of 5 M NaCl and then 240 ul of isopropanol to each tube. Store at -20°C overnight or longer.

REAGENTS: Restriction Endonuclease: 1 unit of activity = amount of enzyme that will cut 1 ug of lambda DNA at all specific sites in 1 hr at 37°C. It is best to use 2-3 units of RE per ug of DNA; the 60 units of RE used above is probably overkill. Can incubate up to 4 hr or more if reaction is free of DNAse and exonuclease. Do not leave RE out of freezer longer than necessary; keep it on ice. Use fresh, sterile pipette tip every time you dispense RE.

3. Isolation of Digested Genomic DNA: Remove the digested DNA from -20°C and spin in cold microfuge for 15 min. Pour off isopropanol and dry the DNA pellet in a vacuum at room temperature for 5-10 min. Resuspend the DNA in 60 ul of TE (pH 7.4) (the DNA is often hard to get back into solution; try flicking the tube and pipetting the solution up and down gently in a sterile pipette tip). You now have 100 flies worth of DNA, cut by Hind-III, in 60 ul of TE. Can be stored in a refrigerator at 4°C.

4. Electrophoresis of Digested Genomic DNA: We use 100 ml of 1% agarose in a 12 x 16 cm Ann Arbor gel with a 12 tooth comb. The 1% agarose mixture is placed on a stirrer-hot plate or in a microwave oven until the solution is clear (be careful and watch the mixture while it is heating, because it can evaporate and become greater than 1%). There should be no particulate agarose left after heating.

Let the 100 ml agarose solution cool to 55-60°C and add 10 ul of ethidium bromide (from a 10 mg/ml stock) and stir. CAUTION: ethidium bromide is a carcinogen -- use gloves. When the solution reaches 50-55°C, with a Pasteur pipette place a bead of agarose at the edge of the tape on the gel bed. Let the bead harden to seal the tape and slowly pour the rest of the agarose into the gel bed that contains a comb. (If you use a gel bed with four side walls, you do not need to place a bead of agarose in the corners of the gel bed.) The Ann Arbor unit holds the entire 100 ml of agarose. Remove any bubbles with a pipette tip before the agarose cools. The teeth of the comb should be close to the bottom of the gel bed, but not touching the bottom, and the unit should be level. Let the agarose cool for at least 30 min until it becomes

clear. Gently remove the comb and remove the tape from the ends of the gel bed. Place the gel bed and agarose gel into the electrophoresis unit.

Add 35 ul of ethidium bromide to 350 ml of 1X Tris-borate (TBE) buffer, and place the 350 ml of buffer into the electrophoresis unit. Since the ethidium bromide is light sensitive, place it into the buffer just before use. Make sure that the buffer is slightly above the surface of the agarose gel.

Heat the DNA for 5 min at 65°C to make it single stranded, and spin for 5 sec to get the drops on the side to the bottom of the tube. Load 20 ul of the DNA, plus 5 ul of 5X loading dye, into each well of the agarose gel. Use a molecular weight standard such as Hind-III cut lambda DNA or biotin-labeled Hind-III cut lambda DNA. If possible, do not use the two outside wells, or place the standard in one of the outside wells. Connect the electrodes with the negative at the DNA end; the DNA is negatively charged and will migrate toward the positive pole. Run the gel at the appropriate voltage and time. For example, with the Ann Arbor unit we usually run it at 25-30 volts overnight, at room temperature, for 17-18 hr. (Keep an eye on the dye front.) The gel can also be run at 100 volts for 2-3 hr.

After electrophoresis, remove the gel bed carefully and view with UV. It is not necessary to stain the gel after electrophoresis, because ethidium bromide is in the buffer. Do not let the gel dry out; keep it wet with water. Photograph the gel on the UV box, either in the gel bed or removed from the gel bed. If you remove it from the gel bed, there is an increased chance that it will be damaged (broken). Run Southern blot or store for a few hr in buffer. It may also be possible to store the gel wrapped in plastic in a refrigerator at 4°C. We have not done the latter procedure.

R E A G E N T S:

5X Tris-borate (TBE) buffer (1 liter):   54 g Tris base, 20 ml 0.05 M EDTA (pH 8.0), 27.5 g boric acid.
     Fill to 1 liter with water.
1X TBE (350 ml):   70 ml 5X TBE, 280 ml water
5X Loading dye: 1.25% bromophenol blue, 40% (w/v) sucrose
1% agarose (100 ml):   1 g agarose, 20 ml 5X Tris-borate buffer (TBE), 80 ml water

5. Southern Blot of Genomic DNA: After electrophoresis, depurinate the DNA on the agarose gel in 0.25 M HCl for 10 min with gentle agitation on a shaker at room temperature. This depurination and many subsequent steps in this protocol are performed in plastic sandwich boxes that are just bigger than the gel (or nitrocellulose) and hold at least 900 ml. Use only extra clean, sterile boxes.

Pour off the HCl and rinse the gel briefly in about 200 ml of water. Pour off the water and denature the DNA by soaking the gel in 250 ml of solution A (1.5 M NaCl and 0.5 M NaOH) for 15 min. Agitate the solution in the plastic box periodically by hand. Pour off the solution and replace with 250 ml of fresh solution A and continue periodic shaking for 15 more min. This procedure cleaves at depurinated sites and denatures the DNA into single strands. Rinse briefly in about 200 ml of water. To return the gel to neutral pH, remove the water and wash twice with 250 ml of solution B (1 M NH$_4$OAc and 0.02 M NaOH) for 15 min each at room temperature. (Shake periodically by hand.)

One can now run a single or double Southern blot. For a single Southern blot, use solution B as the buffer and to wet the 3MM wick and nitrocellulose. Use a standard protocol as shown on page 385 of Maniatis et al. (1982) or page 64 of Davis et al. (1986). Mark the nitrocellulose with a couple of small holes in the upper left corner for later orientation of DNA lanes. With the Ann Arbor gel, use 15 x 17 cm blotting paper and 15 x 35 cm wick. In addition, place Parafilm around the edges of the 3MM filters so that buffer will only be absorbed through the gel. Make sure there are no air bubbles between the gel and the nitrocellulose; push the bubbles to one side with a pipette. Run the blot overnight (for at least 16 hr). Replace the wet paper towels once or twice during the run.

After the blot is finished, remove the weight, glass, paper towels, 3MM Whatman papers, and discard the emaciated gel (you can place the gel on a UV box to see if all of the DNA has been transferred; there will still be some in the gel). Rinse the nitrocellulose for 2 min in 200 ml of 6X SSC in a plastic sandwich box, and air dry the nitrocellulose between 3MM filter paper for a few min at room temperature in the dark. Heat the nitrocellulose between filter paper in a vacuum oven at 70°C for 1 hr. It is not necessary to heat any longer or at a higher temperature. In fact, this will make the nitrocellulose brittle. The nitrocellulose filter can be stored between 3MM paper at room temperature in the dark.

R E A G E N T S:

0.25 M HCl (1 liter):   21.5 ml of concentrated HCl, 978.5 ml of water
10 M NaOH (500 ml):   200 g NaOH in 450 ml of water. Then bring vol to 500 ml (is hot).
Solution A (1 liter):   300 ml 5 M NaCl, 50 ml 10 M NaOH, 650 ml water.
     "   OR   :   87.55 g NaCl, 20 g NaOH. Fill 1 liter water.
Solution B (1 liter):   100 ml 10 M NH$_4$OAc, 2 ml 10 M NaOH, 898 ml water.
     "   OR   :   77.08 g NH$_4$OAc, 0.8 g NaOH. Fill 1 liter water.
10 M NH$_4$OAc (500 ml):   385.4 g of NH$_4$OAc with 150 ml water. Then bring vol to 500 ml.
20X SSC (1 liter):   175.3 g NaCl, 88.2 g sodium citrate, 800 ml water.
     Adjust to pH 7.0 with 10 N NaOH; adjust to 1 liter.
6X SSC (200 ml):   60 ml 20X SSC, 140 ml water.

6. Isolation of Plasmid DNA: Streak out the bacterial culture on to appropriate antibiotic plates and grow overnight at 37°C. Inoculate 10 ml of liquid antibiotic medium with a single colony from the overnight plates and place in a shaker at 37°C overnight. Inoculate 500 ml of liquid antibiotic medium with 4 ml of the overnight culture after removing 5 ml of antibiotic medium as a blank for spectrophotometric assay. Place the 500 ml in an orbital shaker at 37°C at 200 rpm. After 4 hr, check the OD every half hr until OD_{600} = 5-6; then add 3.5 ml of chloramphenicol in ethanol (34 mg/ml). Leave in orbital shaker overnight (approximately 16 hr).

Put equal amounts of the chloramphenicol culture into four sterile 250 ml plastic centrifuge bottles. (Be sure not to fill the bottles more than 3/4 full or they may leak during centrifugation.) Place bottles into JA-14 rotor, pellet at 5000 rpm for 5 min at 4°C, and discard the supernatant. Resuspend the cells in 20 ml of GTE buffer and transfer to a 500 ml flask. Add 2 ml of a 40 mg/ml lysozyme/GTE solution (prepared fresh). Swirl to mix and incubate at room temperature for 10 min and on ice for 5 min. Add 40 ml of lytic mix slowly by running it down the side of the flask. Roll or swirl the flask gently. (Vigorous mixing will shear the DNA.) The solution will turn clear and moderately viscous as the cells lyse. Incubate on ice for 10 min. Add 20 ml of ice cold 3M/5M potassium acetate (see Reagents for 3M/5M potassium acetate). Swirl gently to mix and incubate on ice for 15 min, swirling occasionally.

Put the solution evenly into six 30 ml cortex tubes and pellet at 12,500 rpm for 20 min at 4°C using a Beckman JA-20 rotor. Wipe any solid material on the sides of the tubes above the liquid or on the surface of the liquid with a cotton swab or a Kimwipe. (This solid is unwanted protein and cellular debris.) Carefully pour the supernatant into a 250 ml graduated cylinder and add 0.6X the volume in the cylinder of isopropanol. Can stop and save at -20°C overnight if desired.

Centrifuge the isopropanol/DNA solution at 8500 rpm for 10 min at 4°C and discard the supernatant. Wash the pellet with 35 ml of 70% ethanol and repellet as above. Discard the supernatant and drain the tubes upside down for 10 min. (Wipe away any excess liquid at the lip.) Resuspend the pellets in 7.7 ml of TE and add 8.8 g CsCl. Mix well, but do not vortex. Transfer this solution in equal volumes to two polyallomer quick-seal tubes with a Pasteur pipette. Fill the remainder of the tubes with the CsCl/ethidium bromide balancing solution. The tubes should be filled all the way to the neck so there will be no air space, or the tube may collapse during ultracentrifugation. The tubes should then be balanced to within 0.05 g of each other. Seal the tubes with quick sealer. (Tubes may be stored in the dark at room temperature for several days.)

Place tubes in a Beckman VTi65 vertical rotor and run in ultracentrifuge at 45,000 rpm at 20°C for at least 16 hr. (Assembly hints about VTi65 rotor. Place blue spacers on tubes and put into rotor. Put grease on screws and tighten them with a torque wrench to 120 ft/lbs.) Remove tubes from ultracentrifuge and place in a clamp. The band (or lower band if two are present) visible under long wave UV light will contain the plasmid. Cut the knob off the top of the tubes with a razor blade to vent them. Place an 18 or 20 gauge needle, bevel side up, at a slight angle through the tube at the bottom of the band, and let the band drip out into a glass tube. (If the needle clogs, create pressure in the quick-seal tube by squeezing a Pasteur pipette bulb over the opening of the tube.) Extract the ethidium bromide from the plasmid solution with an equal volume of NaCl saturated isopropanol. Repeat this procedure 4-6 times, discarding the upper pink layer each time. The last few times no pink may be visible, but the interface will still be visible. Load the plasmid solution into dialysis tubing that has been boiled in TE. Dialyze for at least three hr, changing the TE at least once. The plasmid solution can be stored at 4°C at this point.

Concentrate the plasmid by adding 0.1 times the volume of the plasmid solution of 3M sodium acetate (pH 6.0) and 2 times the volume of the solution of ethanol. Chill at -20°C for at least 20 min or can chill overnight. Centrifuge at 10K rpm at 4°C for 30 min, and resuspend the pellet in 1 ml of TE. To determine the concentration of the DNA, take a 1:100 dilution of the plasmid in water (10 ul in 1 ml) and read at 260 nm (U V) (1 OD = approximately 50 ug/ml), and then at 280 nm. DNA of good purity should have a ratio of 260 nm/280 nm equal to about 2. Store DNA at -20°C.

REAGENTS:

<u>3M sodium acetate, pH 6.0 (100 ml)</u>: 40.8 g sodium acetate·3HOH. Fill to 80 ml with water. Adjust pH with glacial acetic acid and fill to 100 ml with water.

<u>TE, pH 8.0 (500 ml)</u>: 7.5 ml of 1 M Tris (pH 8.0), 1 ml of 0.5 M EDTA. Fill to 500 ml water.

<u>GTE (150 ml)</u>: 3.12 ml 1 M Tris (pH 8.0), 2.50 ml of 0.5 M EDTA, 1.12 g glucose. Fill to 150 ml water.

<u>3M/5M potassium acetate (100 ml)</u>: 60 ml 5 M potassium acetate, 11.5 ml glacial acetic acid, 28.5 ml water.

<u>Lytic mix (100 ml)</u>: 0.8 g NaOH, 1 g SDS, Fill to 100 ml water (Prepare fresh every 2 weeks).

<u>CsCl/ethidium bromide bal sol (100 ml)</u>: 98.2 g CsCl, 2.73 ml of 10 mg/ml ethidium bromide. Fill to 100 ml TE (Store in dark bottle at 4°C).

<u>NaCl saturated isopropanol (100 ml)</u>: 80 ml isopropanol, 10 ml of 5 M NaCl, 10 ml of 50 mM Tris/1 mM EDTA made by the following: 0.5 ml of 1 M Tris (pH 8.0), 0.02 ml of 0.5 M EDTA. Fill to 10 ml water.

7. Nick Translation of DNA Probes (for example, P or I Elements) with dUTP-Biotin: We use the BRL Nick Translation Kit, plus biotin-11-dUTP (BRL). To a microfuge tube add 292 ul of water, 40 ul of BRL Sol A4 (contains dGTP, dATP, dCTP, and no dTTP), 8 ul (8 ug) of DNA probe (for example, pπ25.7 WC), and 20 ul of biotin-11-dUTP (the BRL Nick Translation Kit protocol we have seen indicates that one tube is labeled as Sol A4, but they are not; make sure you use the tube that contains dGTP, dATP, dCTP, and no dTTP, since there are other tubes in the kit that contain other combinations of nucleotides). Mix by flicking and add 40 ul of DNAse/Pol 1 (solution C, BRL). Mix by flicking and spin in a microfuge for 10 sec. Place at 15°C for 90 min, remove from water bath, and spin in microfuge for 10 sec. Add 40 ul of the BRL stop buffer and 10 ul of 5% SDS. Mix by flicking, add 45 ul of 5M NaCl, and mix again. Split the solution into approximately equal parts in two microfuge tubes. Add 1 ml of cold 95% ethanol to each tube and mix well by flicking or vortex gently. Place the tubes at -20°C overnight. The next day spin the tubes in a cold microfuge for 20 min, drain off the ethanol without disturbing the DNA pellet (the pellet is often invisible), and dry the pellet in a vacuum at room temperature for about 10 min (do not dry the pellet too much). After drying, add 160 ul of TE (pH 7.4) to each tube, and store the tubes at 4°C (before storage the DNA can be heated to 37°C for 5 min and mixed to assist in dissolving the pellet). You now have two tubes, each with 4 ug of biotin-labeled DNA probe in 160 ul of TE (a total of 8 ug of DNA in 320 ul TE). This may not sound like a lot of labeled DNA, but as we will discuss later the DNA can be used as a probe a number of times.

REAGENTS:

5% SDS (100 ml): 5 g SDS. Fill to 100 ml water (avoid breathing SDS dust).

8. Prehybridization and Hybridization of Digested Genomic DNA with Biotin-Labeled DNA Probes: We also do these reactions in plastic boxes, and the nitrocellulose filter is left in the same box throughout this and subsequent steps. By this method there is less chance of tearing the filter, and you do not have to touch the filter. The problem with using plastic boxes instead of plastic heat-sealable bags is that more liquid must be used in the boxes. However, we find that the nonspecific background on the nitrocellulose is less intense if we use boxes, and the boxes are easier to handle than having to seal and reseal the plastic bags (or remove the filters from one bag to another). It is also a good idea in this step to include a test strip of lambda DNA, which will be probed with biotin-labeled lambda DNA. This is a control to make sure that the subsequent visualization reaction works.

Soak nitrocellulose filters from the Southern blots in about 50 ml of 2X SSC until they are uniformly hydrated. Then prehybridize the filter for 4 hr at 42°C, in a shaker water bath, in 40 ml of the following prehybridizing solution:

　　　50% formamide (deionized) - 20 ml; 5X SSC - 10 ml (of 20X SSC); 5X Denhart's solution - 4 ml
　　　(of 50X Denhart's); 25 mM sodium phosphate (pH 6.5) - 1 ml (of 1M sodium phosphate, pH 6.5);
　　　0.5 mg/ml Salmon sperm DNA (freshly denatured for 10 min in boiling water and then on ice for
　　　5 min) - 2 ml (of 10 mg/ml Sal. sperm DNA); water - 3 ml.

Note that we do not use 0.5% SDS or 5% dextran sulfate in the prehybridization. In addition, BRL recommend the use of 20-100 ul of prehybridization solution per cm^2 of filter -- this would give about 20 ml of solution for the 12 x 16 cm filter we use. However, we believe it is best to use twice this much solution to be sure that the filter is completely prehybridized. In addition, we have found that a good way to keep the solution in the plastic box at 42°C is to place the box in a shaking water bath and place a weight on top of the lid to keep the solution below the water line (shake the box at a low rate).

After the 4 hr of prehybridization, pour off the solution and replace it with 20 ml of the following hybridization solution:

　　　45% formamide (deionized) - 9 ml; 5X SSC - 5 ml (of 20X SSC); 1X Denhart's solution - 0.4 ml
　　　(of 50X Denhart's); 20 mM sodium phosphate (pH 6.5) - 0.4 ml (of 1 M sodium phosphate); 0.2
　　　mg/ml Salmon sperm DNA (freshly denatured for 10 min in boiling water and 5 min ice) - 0.4 ml
　　　(of 10 mg/ml Sal. DNA); water - 4.8 ml; pπ25.7 WC biotin-labeled DNA probe (freshly boil for
　　　10 min and then place on ice for 5 min) - 320 ul (8 ug) in TE.

Place the hybridization solution and the nitrocellulose filter at 42°C for 20 hr in a shaking water bath (as before). Note that we do not use 0.5% SDS in the hybridization solution as suggested by BRL. In addition, be sure to use sheared Salmon sperm DNA, and do not denature biotin-labeled DNA probes with NaOH (will get cleavage of biotin from nucleotides).

REAGENTS:

50X Denhart's Solution (500 ml): 5 g Ficoll, 5 g BSA, 5 g polyvinylpyrrolidone. Fill to 500 ml water.
　　　　Filter and store at -20°C.

1M sodium phosphate, pH 6.5 (1 liter): 137.99 g NaH_2PO_4 (monobasic). Fill to 1 liter water; adjust pH
　　　　to 6.5 by adding NaOH.

9. Posthybridization Washes: Save the hybridization solution from the above step by pouring it into a vial and storing it at -20°C. We have reused the same hybridization solution up to 6 times over a six-month period. Wash the nitrocellulose filter twice in 250 ml of Wash Solution 1 (2X SSC and 0.1% SDS) for 3 min each at room temperature on a shaker. Remove Wash Solution I and wash twice with 250 ml of Wash Solution II (0.2X SSC and 0.1% SDS) for 3 min each on a shaker at room temperature. Remove Wash Solution II and wash twice in 250 ml of Wash Solution III (0.16X SSC and 0.1% SDS) for 15 min each on shaker at 65°C (use preheated Wash Solution III). For this step, we place a shaker into a 65°C incubator; it is important to keep the temperature at 65°C. Remove solution and briefly rinse the filter with 250 ml of 2X SSC at room temperature.

REAGENTS:

Wash Sol. I (500 ml):   50 ml 20X SSC, 10 ml 5% SDS, 440 ml water.
Wash Sol. II (500 ml): 5 ml 20X SSC, 10 ml 5% SDS, 485 ml water.
Wash Sol. III (500 ml): 4 ml 20X SSC, 10 ml 5% SDS, 486 ml water.

10. Nitrocellulose filter blocking: Remove the 2X SSC and wash for 1 min in 200 ml of BRL buffer 1 (0.1 M Tris-HCl, pH 7.5 and 0.15 M NaCl) at room temperature with gentle shaking by hand. Remove buffer and incubate filter for 1 hr at 65°C in 200 ml of prewarmed BRL buffer 2 (3% bovine serum albumin-BSA in BRL buffer 1) on a shaker. The BSA may begin to precipitate out at 65°C; this does not seem to affect the blocking. Decant and rinse the filter in 250 ml of BRL buffer 1 at room temperature for 1 min. You may dry the filter now at room temperature and continue this protocol later. However, we have found that you get a clearer background if you go directly to the next step without letting the filter dry.

REAGENTS:

BRL buffer 1 (1 liter): 100 ml 1 M Tris-HCl (pH 7.5), 30 ml 5 M NaCl, 870 ml water.
BRL buffer 2 (1 liter): 30 g BSA. Fill to 1 liter with BRL buffer 1 (store at 4°C).

11. Detection and Visualization of Genomic DNA Hybridized to Biotin-Labeled DNA Probe: Biotin-labeled DNA probe is detected using the BRL BluGENE nonradioactive nucleic acid detection system. Incubate the filter for 10 min in 25 ml of streptavidin-alkaline phosphatase (SA-AP) solution with gentle agitation on a shaker at room temperature. (Should use about 7 ml of SA-AP solution per 100 cm^2 of filter.) Decant and wash twice with 850 ml of BRL buffer 1 for 15 min each at room temperature. Decant and wash for 10 min in 200 ml of BRL buffer 3 (0.1 M Tris-HCl, pH 9.5, 0.1 M NaCl and 50 mM $MgCl_2$) at room temperature. (Use gentle agitation on all of these steps.)

To visualize the DNA bands, incubate the filter in approximately 20 ml of dye solution (nitroblue tetrazolium --NBT, 5-bromo-4-chloro-3-indolylphosphate --BCIP in dimethylformamide, and BRL buffer 3) on a shaker at room temperature for up to 3 hr. Cover the plastic box completely with foil; the dye solution is light sensitive. The dye solution should be freshly prepared prior to use and should be kept in the dark. BRL recommends that the color development should go overnight. However, incubations longer than 3 hr may result in increased background color. Note that the color usually comes up in only 5-10 min. In addition, be aware that the color shows up better on one side of the filter. The first time we got a filter upside down in a box, we thought that we had a poor dye reaction, until someone looked at the filter through the bottom of the box.

Decant the dye solution and wash filter in 25-50 ml BRL wash buffer (20 mM Tris-HCl, pH 7.5 and 0.5 mM Na_2EDTA). Filters should then be stored dry in reduced light (in a drawer) between 3MM filter paper. We do not dry the filter in a vacuum oven.

REAGENTS:

Streptavidin-alkaline phosphatase (SA-AP) (25 ml): 25 ul SA-AP (BRL), 25 ml BRL buffer 1.
BRL buffer 3 (1 liter): 100 ml 1 M Tris-HCl (pH 9.5), 20 ml 5 M NaCl, 50 ml 1 M $MgCl_2$, 830 ml water.
Dye solution (~ 20 ml): 88 ul NBT, 66.6 ul of BCIP, 20 ml of BRL buffer 3 (dimethylformamaide in the BCIP
     is harmful and should be used in a hood; it is also combustible).

12. Results: See Figure 1 for a photograph of **Drosophila melanogaster** genomic DNA digested with Hind-III and probed with biotin-labeled P-element DNA. The **D.simulans** lane is a control that is known not to contain P elements (the one visible band is the 17C region of the X chromosome that is included in the pπ25.7 WC probe). It is best to photograph the filters wetted with water. It might be useful to note that a better estimation of the number of intact P elements present in a strain can be made from Apa 1 digested genomic DNA; Apa 1 does not cut within intact P elements (I. Boussey & S. Easteal, pers. comm.). We have not yet made a comparison of this biotin technique for Southerns with P^{32} labeled DNA probes. We suspect that the latter technique is more sensitive.
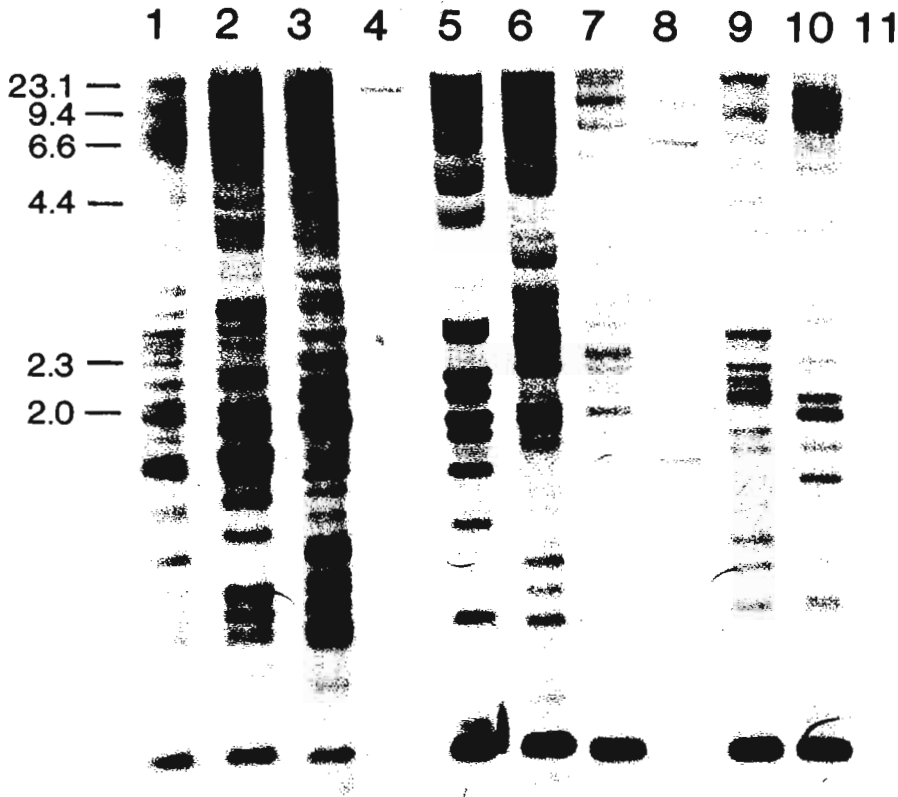
Figure 1. Hind-III digest of genomic DNA from ten **D.melanogaster** lines (lanes 1-10) and one **D.simulans** line (lane 11). These digests were probed with biotin-labeled pπ25.7 WC DNA. Lines 1, 2 and 3 are Q lines (based on gonadal dysgenesis and interactions with sn^W) that contain presumed defective P elements. Lines 1 and 2 (CT106 and CT119) were collected by P. Parsons in May 1980 at the Chateau Tahbilk winery, Victoria, Australia, whereas line 3 (TW3) was collected in Varna, New York, by B. Wallace in 1977. Lines 4 and 8 are M lines, with line 4 being devoid of P elements and line 8 containing defective P elements (line 4 is W11D, which was collected by B. Wallace in Oxford, North Carolina, in 1966 and line 8 is Canton-S). Lines 5, 6, 7, 9 and 10 are P lines that contain intact P elements. Line 5 (WV80A) was collected in Waterville, Ohio, by RCW in 1980, line 6 (BG80A) was collected in Bowling Green, Ohio, by RCW in 1980, line 7 (W8D) was collected in the Okefenokee Swamp, Georgia, by B. Wallace in 1966, line 9 (OK1) was collected in Oklahoma City, Oklahoma, by JNT in 1973, and line 10 (π2) was collected in Madison, Wisconsin, in 1975 by W. Engels. The **D.simulans** line (lane 11) was C135.20 from Cambridge, England. The detailed data on the molecular biology and mutagenesis associated with these lines will be published in the future.

References: Davis, L.G., M.D. Dibner & J.F. Battey 1986, Basic Methods in Molecular Biology, Elsevier, NY; Maniatis, T., E.F. Fritsch & J. Sambrook 1982, Molecular Cloning: A Laboratory Manual, Cold Spring Harbor Lab., Cold Spring Harbor, NY.
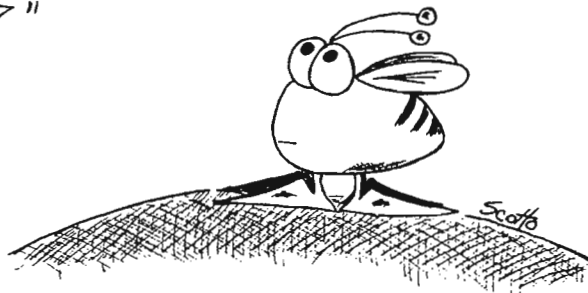
Morrison, W.J. Shippensburg University, Shippensburg, Pennsylvania. Demonstration of the heat shock response by means of ADH activity in a transformed line of **Drosophila melanogaster.**

For the past three years my undergraduate genetics students have conducted a simple experiment that demonstrates both the heat shock response and an enzyme deficiency. The experiment employs a construct in which the coding region of the alcohol dehydrogenase gene (Adh) has been joined to the promotor region of the hs70 heat shock protein gene (Bonner et al. 1984). This heat shock-sensitive Adh gene has been introduced into the genome of a strain homozygous for a null Adh allele by germline transformation at 61C on chromosome 3. When such flies, identified as Adh^{hs61c}, are maintained at 25°C and are heat shocked by exposure to 37°C for one hour followed by a recovery period of 24 hr, they synthesize Adh in nearly all tissues (Bonner et al. 1984). Heat shocked Adh^{hs61c} flies are compared in ADH activity to wild type, null strain, and non-heat shocked Adh^{hs61c} flies by a simple, indirect test. The basis of the test is that flies possessing ADH activity convert pentynol to a toxic compound that causes paralysis and eventual death.

Five to ten adult flies of each strain/treatment category are placed in empty shell vials (25 mm x 95 mm) plugged with cotton. In a fume hood, two drops of 5% (v/v) pentynol (1-pentyne-3-ol; Pfalz & Bauer, Inc., 172 East Aurora St., Waterbury, CT 06708) are absorbed into a square (2 cm x 2 cm) of thick filter paper (Schleicher & Scheull, grade 470), which is inserted into a shell vial containing the flies to be tested. The vial then is immediately sealed with Parafilm, and the student closely observes the condition of the flies while noting the elapsed time. The wild type and the heat shocked Adh^{hs61c} flies begin to exhibit paralysis after about five minutes, whereas neither the null mutant nor the non-heat shocked Adh^{hs61c} flies show any early response to the pentynol. (However, after several minutes, all flies succumb to this treatment.)

The experiment may be done conveniently and effectively as a lecture demonstration by inserting the pentynol-moistened paper squares into vials containing flies whose silhouettes are projected onto a screen with an overhead projector. This experiment sparks the interest of students by vividly indicating a heretofore unseen difference in the flies. Furthermore, it involves the student with a product of recombinant DNA technology.

References: Bonner, J.J. et al. 1984, Cell 37:979-991; O'Donnell, J. 1975, Genetics 79:73-83.

SUBMITTED STOCK LISTS - **all species**

AMHERST COLLEGE. Virginia White, Webster Ctr for Biol. Sciences, Amherst, MA 01002.

Chromosome 1

Basc	C(1)RM, cm ct^6 sn^3 & FM6 ♀/FM6 ♂ Triploid		
C(1)DX, y f/cm ct^6 sn^3 oc	C(1)RM, g^{53d} sd & FM6 ♀/FM6 ♂ Triploid		
C(1)DX, y f/cm ct^6 sn^4 oc ptg	C(1)RM, ras^{79f19} m & FM6 ♀/FM6 ♂(see notes) Triploid		
C(1)DX, y f/cm ct^6 sn^{36a}	C(1)RM, y w & FM6 ♀/FM6 ♂ Triploid		
C(1)DX, y f/ct^6 oc	cm		
C(1)DX, y f/ct^n oc	cm ct^6		
C(1)DX, y f/ec rb^{64f14} cv (see notes)	cm ct^6 sn^2		
C(1)DX, y f/oc	cm ct^6 sn^3		
C(1)DX, y f/oc ptg	cn ct^6 sn^4	g^{53d} sd	w
C(1)DX, y f/sn	cm ct^6 sn^{34e}	g^w	w^a
C(1)DX, y f/sn^2 oc ptg^3	ec	mg^{64b11} f^{36a} (see notes)	wy^2
C(1)DX, y f/sn^3 oc	FM6/y w^{sp}	ras dy	wy^2 g
C(1)DX, y f/sn^4 oc ptg^3	g	sd	wy^2 g^2
C(1)DX, y f/sn^{36a}	g sd	sn^2	wy^2 g^3
C(1)DX, y f/w sn^5	g^3 sd	sn^3	wy^2 g^{53d}
C(1)DX, y f/y^2 sn^3 ras^4 m	g^{50e}	sn^3 g^{53d}	wy^2 g^{53d} sd
C(1)DX, y w/FM6	g^{53d}	sn^4	y ct^6 ras^2 f

Chromosome 2

;al cl b c sp^2/SM5
;al cl nub sca^2 sp^2
;al nub lt stw^3 sca^2 sp^2
;b Bl vg bw/In(2L)Cy In(2R)Cy, Cy cn^2 bw^{45a} or ^{45a} sp^2
;Bl L^2/SM5
;cl
;cn bw (not tested for lethals)
In(2L)Cy In(2R)Cy, dp^{lvl} Bl lt^3 cn^2 L^4 sp^2/
     In(2LR) bw^{Vl}, ds^{33K} bw^{Vl}
;In(2L)t In(2R)Cy, Roi cn^2 bw^{45a} or^{45a} sp^2/
     In(2R)vg^U, vg^U
In(2R)vg^U, S^X Sp vg^U/SM5
;In(2R)vg^U, Sp vg^U If/SM5
;In(2R)vg^U, vg^U If/SM5
;lt stw^3
;net^{38j} b^{38j} cn^{38j} bw^{38j}
;Sp BL L^2/SM5
;vg^{51h25}

Multiple Chromosomes

g^{53d};vg
;bw;h
bw;h st
;In(2L)Cy In(2R)Cy, Cy cn^2 Sp^2/In(2LR)bw^{Vl} ds^{33k} dp b bw^{Vl};In(3LR)Dcxf D/In(3R)Mo, Sb sr
;In(2L)Cy In(2R)Cy, Cy cn^2 bw^{45a} or^{45a} sp^2; TM3, Sb Ser; T(2;3) bw;h (see notes)
;net or ^{45a} sp^2 ;ru bv
;SM5;TM1;T(2;3)gl^{63d}, gl^{63d} (see notes)
;;TM2;T(2;3)ap^{Xa}, ap^{Xa}
;vg^{51h25};se^{50k}

Chromosome 3

;;bar-3
;;cu gl^3
;;cu ss^{ax}
;;h th st p^p cu sr e^s
;;In(3L)D, D^3/In(3LR)TM3, y$^+$ ri p^p sep Sb bx^{34e} e^s Ser
;;In(3L)D, D^3/TM1
;;In(3L)D, D^3 Sb/In(3L)P In(3R)P
;;ru h st cu sr e^s ca
;;ru st ss ca
;;se ss Su(ss)^3
;;se^{50K}
;;ss bx Su(ss)^3
;;ss gl^3
;;ss^{ax}
;;ss^{ax} gl^3

Chromosome 4

;;;ey^2

;vg^{51h25} bw;se^{50k} e
;vg^{51h25} sp^2;se^{50k}
;vg^{51h25};se^{50k};spa^{pol}

NOTE 1. rb^{64d14}, ruby^{64d14} 1-7.5. Found by P.T. Ives in one sperm of an Oregon-R ♂ that had been irradiated with 1 kr X-ray. Phenotype like rb.

NOTE 2. g^{64b11}, garnet^{64b11}. 1-44.4. Found by P.T. Ives in one sperm of an Oregon-R ♂ that had been irradiated with 1 kr X-rays. Phenotype like g.

NOTE 3. sca^2 is the allele described as sca^{65131} in DIS 48:16.

NOTE 4. T(2:3) gl^{63d29}. Found by P.T. Ives in one sperm of an Oregon-R ♂ that had been irradiated with 1 kr X-rays. It is lethal free and its phenotype is white with yellowish center. A.S. Robinson (Robinson & Curtis 1972, Canadian J. of Genetics and Cytology 14:129-137) puts its breaks at 47B and 91A, the latter being near gl and suggesting that the gl phenotype is a position affect mutation. The T and the mutant have not been observed to separate by crossing over.

NOTE 5. T(2:3) bw h found by P.T. Ives after irradiation of a bw;h ♂ with 1 kr X-rays. Not studied cytologically and not tested for the amount of crossing over between the T and the markers.

NOTE 6. ras^{79f19}, raspberry^{79f19}. 1-32.2. Found by P.T. Ives in one sperm from a ♂ that was heterozygous for Massachusetts and Tennessee chromosomes. Phenotype like ras^4. ♀♀ either sterile or produce few developing eggs at 25°C.

Northern Illinois University. Jack Bennett Laboratory, Dept. of Biol. Sciences, DeKalb, IL 60115-2861.

     I have produced and maintain a set of co-isogenic stocks which share more than 50 generations of backcrossing to an Oregon-R inbred line sib mated in this laboratory for more than 200 generations. All lines are designated as ORI (Oregon-R Inbred) followed by a standard designation of the allele recovered through all of the backcrosses and for which the line is named. For example, ORI-cn bw, is a line produced by combining ORI-cn and ORI-bw to give a white phenotype, homozygous line isogenic with the other ORI lines at all other loci. The available lines are:

wild-type: **D.melangoaster**

white	white-buffy	brown-Dominant	cinnabar, brown-Dominant
white-honey	white-coffee	cinnabar	scarlet, brown-Dominant
white-coral	white-Brownex	scarlet	cinnabar, sepia
white-eosin	wild type allele of white	sepia	scarlet, sepia
white-apricot	from Canton-S	cinnabar, brown	yellow
white-apricot 2	brown	brown, scarlet	

University of Maryland. Jerry A. Coyne, Zoology Dept., College Park, Maryland 20742.

The following **D.simulans** stocks are available on request; I have also sent them to the National Drosophila Species Resource Center in Bowling Green, Ohio.

Chromosome 1 stocks	Chromosome 2 stocks	Chromosome 3 stocks
w^{xD1}	nt	st
w^a	pm	e
m	stw	ry^{i83}
f^2	Lhr	"outspread"
g	b^2	st e
y w	"dimpled"	"outspread" st
y w f^2	nt pm	"outspread" st e
lz^s \$/XXY y w ⚢	nt b py sd pm	"orange-3"
+ \$/XXY y w ⚢	Cn	"purple"
y w m f^2 \$/XXY y w ⚢		
y^2		

Chromosome 1 stocks (continued):
- y^2
- v
- y w m f^2
- vmg

Translocations
T(Y;2)3, +/nt b
T(Y;3)1, Ubx^m/cutsy ca^2

Multichromosomal

nt; st	f^2; nt pm; st e	f^2; nt pm
nt; e	nt pm; st e	f^2; st e
	"outspread;"	"dimpled"

Yale University. Dept. of Biology, New Haven, CT 06511. Submitted by Gregory M. Fitzgerald

Drosophila melanogaster:

Chromosome 1

0 / C(1)RM, y pn v / $Y^SX \cdot Y^L$, In(1)EN, y B	1(1)jn11 ct v / Binsinscy, y w sn B
amx / In(1)d1-49, m^2 g^4	1(1)jn12 ct v / Binsinscy
B	1(1)ogre^{1jn13} cm v / Binsinscy
C(1)A, y / FM7c	1(1)jn14 ct v / Binsinscy
C(1)A, y / y cho 1(1)ogre^{vcb8} sn^3	1(1)LEAK cm v / Binsinscy
C(1)A, y / y w 1(1)ogre^{vcb8}	1(1)ogre^{1j555} cm ct^6 sn^3 / Binsinscy
cho sn^3 Df(1)lz$^{10-5}$, 70d30 / FM6	1(1)ogre^{1j555} cm v / Binsinscy
cho sn Zw^n / C(1)DX, y f	1(1)ogre^{11523} cm v / Binsinscy
cm ct^6 sn^3	1(1)ogre^{vcb8} / C(1)A, y
cm ct^6 sn^3 / FM6	1(1)ogre$^{vcb8-8J}$ / C(1)A, y
cmot^1	1(1)ts-480 / FM6
ct^n oc / FM1, y^{31d} sc^8 w^a lz^s B	1(1)ts-1126 f^{36a} / FM6
Df(1)HA32 / Binsinscy	1(1)ts-5697
Df(1)HA32 / FM7c	1(1)ts-UC13
Df(1)JC70 / FM7c	lz^{rfg} / C(1)DX, y w f
Df(1)mal^3 y^2 ct^6 f / y$^+$Y mal^{106} / y v f mal^2	M(1)Bld / C(1)M3, y^2 / y^{2}Y 67g19.1
Df(1)mal^{10} sc^8 B / In(1)d1-49, v sn^{X2} mal^2 / y$^+$Y mal^{106}	M(1)o^{Sp} / FM6
Df(1)w$^{258-42}$, y / In(1)d1-49, y Hw m^2 g^4	M(1)o^{Sp} Ace$^+$ / FM6
f^{36a} car	mal / C(1)DX, y f
FM6 / FM7c	mei-41^{A1} f74:10/ C(1)DX, y f bb$^-$
g shi^{ts1} f / FM6	peb v
Hw^{49c} / FM1, y^{31d} sc^8 w^a lz^s B	ras^2
In(1)d1-49, Df(1)ct^{J4} f / FM6	rg
In(1)d1-49, Df(1)ct^{J6} f / FM6	rst^3
In(1)d1-49, m^2 g^4 / FM6	shi^{ts1} / FM6
In(1)w^{vc} / In(1)d1-49, y w lz^s / sc^{8Y}, y$^+$	shi^{ts1} / C(1)A, y
In(1)y^{3PL} sc^{8R}, y$^-$ ac$^-$ sc^8	sol^{KS58}
KS74-VIII / C(1)M3, y^2	sol^{KS58} / C(1)M3, y^2
KS201-XVIII / C(1)M3, y^2	spl / C(1)A, y
KS221-XVI^{10} / C(1)M3, y^2	swb^{62b12}
1(1)d.deg.X-34 w^a / Binsn	tko / FM6

Chrom. 1 (continued)

un Bx^2 / In(1)AM, ptg
vs
w
w sn^3
w spl
w^a mw mit
w^a N^{60g11} / FM7c
w^{bl}
w^e
y
y cho cv mit / FM6
y cho cv sn^3
y cho cv sn^3 / C(1)M3, y^2
y cho cv v f · y$^+$
y cho 1(1)ogre^{vcb8} sn^3 / FM6
y cho 1(1)ogre^{vcb8} v g f / FM6
y cho 1(1)ts-1126 f^{36a} / FM6
y cho peb
y cho rux^2 sn^3 / FM7a
y cho sn^3
y cho sn^3 1(1)d.deg. X-55 / FM6
y cho vs / C(1)A, y
y cv 1(1)ts-2366
y Df(1)bb^{1158} / FM1 / y$^+$Y
y 1(1)EN2 w spl sn bb / Binsc, oc ptg
y 1(1)EN8 B / Binsc, oc ptg

Chromosome 2

ad
Adn^{n1} Got^{lo}
al dp b pr c px sp
arch chl / SM5
ast ho cl
ast^4 ch cl
ast^4 dp cl
b pr sdh^2 bw^{V32g} / CyO
Bl / CyO
Bl / In(2L&2R)Cy
Bl / SM1, dp^{lvI} Cy L^4
Bl L^2 / SM5, al Cy 1t^v cn^2 sp^2
Bl L^{2+R1} / SM5
Bl L^{2+R2} / SM5
Bl L^{2+R3} / SM5
Bl L^{2+R4} / SM5
Bl L^{2+R5} / SM5
Bl L^{2+R6} / SM5
Bl L^{2+R7} / SM5
Bl L^{2+R8} / SM5
Bl L^{2+R9} / SM5
Bl L^{2+R10} / SM5
Bl L^{2+R11} / SM5
Bl L^{2+R12} / SM5
Bl L^{2+R13} / SM5
Bl L^{2+R14} / SM5
Bl L^{2+R15} / SM5
Bl dke c / SM1, Cy
blo / SM1, dp^{lvI} Cy L^4
C(2)Novitski
c / SM1, Bl L^2
c px nw^D sp

y mei-9 f^{36a} / y$^+$Y
y mei-9 car · y$^+$ Dp(1;1)sc^{V1} / C(1)DX, y f bb$^-$
y mei-9 mei-41 [5] / C(1)DX, y f bb$^-$ / y$^+$Y
y mei-9 mei-41 / FM7c / y$^+$Y
y mei-41 / C(1)DX, y f
y mei-41 / FM6 y$^+$Y
y v f mal^{bz}
y w
y w f^{36a} 1(1)ts-726 / C(1)DX, y f / y$^+$Y
y w 1(1)d.norm-13 sn^3 / Binsn
y w 1(1)ogre^{ljn13} cm v / Binsinscy
y w 1(1)ogre^{vcb8} / FM6
y w mei-41 / C(1)DX, y f bb$^-$ / y$^+$Y
y w mei-41 f^{36a} / C(1)DX, y f / y$^+$Y
y w spl sn 1(1)EN1 / Binsc, oc ptg
y w spl sn 1(1)EN3 bb / Binsc, oc ptg
y w spl sn 1(1)EN4 bb / Binsc, oc ptg
y w spl sn 1(1)EN5 / Binsc, oc ptg
y w spl sn 1(1)EN6 bb / Binsc, oc ptg
y w spl sn 1(1)EN7 / Binsc, oc ptg
y w spl sn 1(1)EN11 bb / Binsc, oc ptg
y$^+$ mei-4 f^{36a} [4] / C(1)DX, y f bb$^-$ / B^{S}Y
y$^+$ mei-41 f^{36a} [4] / C(1)DX, y f bb$^-$ / B^{S}Y
y^2 sc w^a ec
y^2 w^a spl sn^3 / C(1)DX, y w f
z w^{11e}

c wt px
chl
cl
cn sdh^3 bw / CyO
cn sdh^4 bw / CyO
cn sdh^8 bw / CyO
Df(2L)J69^L-H56^R, y$^+$ / CyO
dke c
dl / CyO^{DTS}
dl^2 / CyO^{DTS}
en^{01}
ex ds S^x / SM1, al^2 Cy cn^2 sp^2
α-Gdh
Got2^o dp Adh^{n1} Got^{lo} / SM1, Cy dp^{lvI} L^4
Got2^o dp b pr px sp
Got2^o dp Got1^{Crimea} / SM1, dp^{lvI} Cy L^4
Got2^o dp Got1^{Crimea} / SM5
Got2^o dp Got1^{lo} / SM1, Cy dp^{lvI} L^4
Got2^J cl cn
Got2^{nNC1} / SM1, Cy
Got2^{nNC3} / SM1, Cy
Got2^{nr40058b} / SM1, Cy Bl L
Got2^{nr40058c} / SM1, Cy Bl L
In(2L)Cy, In(2R)Cy, al^2 Cy Bl cn^2 L^4 sp^2 /
     In(2R)NS, S Sp ab^2 ap^4 px sp
In(2L)Cy, In(2R)Cy, Cy dp^{lvI} pr Bl cn^2 L^4 sp^2 /
     In(2R)NS, S Sp mr
In(2L)Cy, In(2R)Cy, S^2 Cy dp^{lvI} cn^2 bw sp^2 /
     In(2R)NS, ab^2 j mr
In(2L)Cy, In(2R)Cy, S^2 ls Cy pr Bl cn^2 L^4 bw sp^2 /
     In(2R)NS, dp^{olv} Sp cn mr
In(2LR)Cy, al^2 E(S) cn^2 sp

Chrom. 2 (contin.)

In(2LR)Gla, cn / al^1 Cy pr Bl cn^2 c vg sp^2
lm / In(2LR)Cy, Cy S^2 dp^{lvI} E(S)
M(2)H^{S5} / SM5
M(2)I^2 / In(2LR)bw^{V1}, ds^{33k} bw^{V1}
M(2)S7 / SM5
pr cn ix / SM5
puf
rdo
Roi / CyO

Chromosome 3

Acph-1^{n11b}
Aph^n Acph^n
Aph^n Acph-1^{n5}
bar-3
C(3L)RM (P3), ri / C(3R)RM (P3), sr
cand
cand / TM3, Sb Ser y$^+$ ri p^P sep bx^{34e} e
cand / TM6, ss^{P88} bx^{34e} Ubx^{P15} e
Cha^{12} / MRS
Cha^{ts1} / TM6
D / Gl
D^3 Ly
D^3 st ca / Ly st ca
Df(3R)Cha1 / MKRS
Df(3R)Cha1 / TM6
Df(3R)Cha1 / TM6B, Hu e Tb ca
Df(3R)e^{H5} / TM3
Df(3R)126c / MKRS
Df(3R)sr-gl / TM3, Sb Ser
Dl^3 / In(3R)C, e
e^{11}
e^s cand / TM6
ecd^1 st ca
eyg
fz^R st ca / TM3, Ser
gl
gl^2
gl^2 e^4
gl^3
gl^3 cand / TM1, Me ri sbd^1
gl^3 cand / TM6
Gl / TM3, (Sb) Ser
Gl red e / MRS
Gl red e / TM3, Ser
Gl Sb / LVM, In(3L)P In(3R)P l(3)LVML l(3)LVMR
Gl st / TM3, Sb Ser
Gl st cand / TM3, Sb Ser
Gl st cn red e / MRS
Gl st Ki p^P / TM3
Gl^{P1-3} fz red e / TM2, red e
Gl^{P1-3} fz red e / TM3, Ser
Gl^{P1-3} fz st ca / TM3, Ser
Gl^{P7-1} fz st ca / TM3, Ser
Gl^{P7-1} st / TM3, Sb Ser
Gl^{P15-C} st red e / TM3, Ser
Gl^{P32-2} fz st red e (ca) / TM3, Ser
Gl^{+R2} / TM3
Gl^{+R3} / TM3
Gl^{+R9} / TM3

SM1 / In(2LR)O2, ds^W sp^2
S^R / bw^{V1} ds^{33k}
sca
Sco / CyO
shv ho
Sp Bl N-2G / SM5, al^2 Cy lt^V sp^2
Sp vg^U If / SM5
std / SM5
vg^U If / SM5

Gl^{+R17} / TM3
Gl^{+R18} / TM3
Gl^{+R22} / TM3
Gl^{+R26} / TM3
Gl^{+R27} / TM3
Gl^{+R33} / TM3
Gl^{+R34} / TM3
Gl^{+R50} / TM3
Gl^{+R57} st / TM3, Ser
Gl^{+R70} / TM3
Gl^{+R72} / TM3
Gl^{+R73} / TM3
Gl^{+R77} / TM3
Gl^{+R80} / TM3
Gl^{+R86} / TM3
Gl^{+R102} / TM3
Gl^{+R114} / TM3
Gl^{+R160} / TM3
h st cu red e
h st cu sr e^s ca
In(3LR)DcxF, ru h D Sb e^s / In(3L)P In(3R)C (Me) e l(3)e
kar^2 Df(3R)126c / MKRS, kar ry^2 Sb
kar^2 Df(3R)126d / MKRS
kar^2 Df(3R)126d / MRS
kar^2 l(3)S8 / MRS
Ki p^P / TM1, ro^{831}
l(3)IX-11 mwh e / TM1
l(3)1803R e / TM1
l(3)Ace^{J19} / MRS
l(3)Ace^{J50} / MRS
l(3)Ace^{J50} red e / MRS
l(3)Ace^{m15} / MRS
l(3)Ace^{m38} / MRS
l(3)Ace^{m40} / MKRS
l(3)Ace^{m40} / MRS
l(3)Ace^{m40} / TM3, Sb Ser
l(3)Ace^{m40} / TM6B, Hu e Tb ca
l(3)C8 / TM3, Sb Ser
l(3)f^{26} mwh e / TM1
l(3)g30R mwh red e / TM1
M(3)h^{S37} / TM6
M(3)i^{55} / LVM
M(3)i^{55} / TM1
mah
MRS / TM6B, Hu e Tb ca
mwh jv hyl / TM3, Ser
Pr^K Dr / Payne
Pr^L Bd^G / TM1, Me
red

Chrom. 3 (contin.)

red Cha^{ts1}
red Cha^{ts2} / TM6
red Cha^{ts2} / TM6B, Hu e Tb ca
red Df(3R)sr-gl / Ubx^{130}, red
red gl^3
red gl^3 / Ubx^{130}, red
ro
ro Pd ca In(3R)C, l(3)a
ro cand
ro cand / TM6B, Hu e Tb ca
ro ra ca
ru h th st cu sr e^S ca
ry^{42}
Sb / Ubx^{101}
Sb cand / TM6
Sb H Df(3R)e^{F3} / TM2

Sb Ubx / LVM
se
se ss k e^S ro
st ca
Su(G1)77 [1] h th st cu st e^S ca / TM3
Su(G1)102 [4] h th st cu sr e^S ca / TM3
Su(G1)160 [4] h th st cu sr e^S ca / TM3
th st cp in ri p^p
TM1 / LVM
TM1 / TM3, Ser
TM1 / TM6
TM3, Sb Ser / TM6B, Hu e Tb ca
TM3 / TM8
tx
Ubx e^4 / In(3L&3R)P, ca (Dfd)
wo

Chromosome 4

C(4)RM(P1), ci ey^R · gvl sv^n
ci^D / ey^D

ci ey^A
spa^{pol}

Chromosomes 1,2

C(1)DX, y / In(1)d1-49, y; Sco / In(2LR)Cy
C(1)M3, y^2 / e(S)^X; al S ast ho / SM1, Cy
C(1)M3, y^2 / FM7a; Sco / CyO
C(1)M3, y^2 / l(1)ogre^{vcb8}; CyO / +
C(1)M3, y^2 / l(1)ogre^{vcb8}; Sco / CyO
C(1)M3, y^2 / M(1)Bld / y^{2}Y 67g19.1; pal / Cyo
C(1)RM, y bb / FM7c; Sco / CyO
C(1)RM / Y^S · X · Y^L, In(1)EN, y Sco / In(2L&2R)Cy
Df(1)N^{r69h9} / C(1)DX, y w f; SM1, Cy Dp(1;2)w^{+51b7} / +
Df(1)RC40 / C(1)DX, y w f; Dp(1;2)A1125 / +
Df(1)w$^{258-45}$ / Dp(1;2)w^{170h31}
FM6 / FM7c; Sco / CyO

peb v; B1 L^2 / SM5
peb v; dke c
y w; Dp(2;1)sc^{19} M(2)z / SM5
y w; In(2LR)Cy, Roi / Df(2L)Gdh
y w; In(2LR)Cy, Roi / Gdh^{n5} spd^{fg}
y w / y$^+$Y; Df(2R)XTE-11 / CyO
y w / y$^+$Y; Df(2R)XTE-18 / CyO
w^a N^{60g11} / C(1)DX, y w f; SM1 , Cy Dp(1;2)w^{+51b7} / +
y w^a Df(1)N^{5419} / C(1)DX, y w f; SM1, Cy Dp(1;2)w^{+51b7} / +
y w^a N$^{264-103}$ / C(1)DX, y w f; SM1, Cy Dp(1;2)w^{+51b7} / +
y w^a N^{60g11} / C(1)DX, y w f; SM1, Cy Dp(1;2)w^{+51b7} / +

Chromosomes 1,3

B170^D / R83^P, y
B170^D / J132^P, y
C(1)A, y / FM6; Sb / TM2
C(1)A, y / FM7c; Sb / TM6
C(1)A, y / In(1)d1-49, Df(1)ct^{J6} f; Dp(1;3)sn^{13a1} / TM2
C(1)A, y / l(1)ogre^{1jn13} cm v; Dp(1;3)sn^{13a1} / (TM2) / +
C(1)DX, y w f / Df(1)HA32; Dp(1;3)sn^{13a1} / Ki
C(1)DX, y w f / FM6; Ki / TM6
C(1)DX, y^2 / In(1)d1-49, y; Sb / TM2
C(1)M3 / +; gl^3
C(1)M3 / spl; E(spl) / TM2
C(1)M3, y^2 / In(1)d1-49, m^2 g^4 mit; Acph-1^{n11b}
C(1)RM / FM6; Sb / TM2
C(1)RM, y^2 / FM7; Sb / TM2
In(1)d1-49, Df(1)ct^{J4} f / C(1)DX, y w f; Dp(1;3)sn^{13a1} / (Ki)
Df(1)w-ec^{64d}, cm ct^6 sn^3 / C(1)DX, y w f; Dp(1;3)w^{+67k27} / +
Dp(3;1)115 / FM6; Df(3R)115 e^S cand / TM1
Dp(3;1)Ace$^+$ / C(1)DX, y^2; Df(3R)126d / TM6
Dp(3;1)Ace$^+$ / C(1)DX, y^2; 1(3)Ace^{m38} / TM2
Dp(3;1)Ace$^+$ / C(1)DX, y^2; 1(3)Ace^{m115} / TM6
Dp(3;1)Ace$^+$ M(1)o^{Sp} / FM6; Df(3R)126d / TM6
Dp(3;1)Ace$^+$ M(1)o^{Sp} / FM6; + / TM6
Dp(3;1)Ace$^+$ M(1)o^{Sp} / FM6; 1(3)Ace^{j50} / TM6B, Hu e Tb ca
Dp(3;1)Ace$^+$ M(1)o^{Sp} / FM6; 1(3)Ace^{m15} / TM6B, Hu e Tb ca

Dp(3;1)Ace$^+$ M(1)o^{Sp} / FM6; 1(3)Ace^{m38} / TM2
Dp(3;1)Ace$^+$ M(1)o^{Sp} / FM6; 1(3)Ace^{m115} / TM6B, Hu e Tb ca
Dp(3;1)O5, D / C(1)DX, y f
Dp(3;1)O5, D / C(1)M3, y^2; Cha^{12} gl^3 / MRS
Dp(3;1)O5, D / C(1)M3, y^2; gl^3
Dp(3;1)O5, D / FM6; gl^3 cand / TM6
Dp(3;1)O5, M(1)o^{Sp} / FM6; gl^3
Dp(3;1)ry$^+$ / C(1)DX, y^2; Df(3R)126d / TM6
Dp(3;1)ry$^+$ / C(1)DX, y^2; 1(3)Ace^{m15} / TM6
Dp(3;1)ry$^+$ / C(1)DX y^2; 1(3)Ace^{m38} / TM6
Dp(3;1)ry^{35XR} / C(1)M3; sr
f^{36a} car; Df(3R)126d / TM6
FM6 / +; MRS / TM6
FM6 / FM7c; Sb / TM2
FM6 / FM7c; TM3, Sb Ser / TM6B, Hu e Tb ca
J132^D / R91^P, y f B^S
1(1)ogre^{1j555} cm ct^6 sn^3 / C(1)A, y; Dp(1;3)sn^{13a1} / +
1(1)ogre^{11523} cm ct^6 sn^3 / C(1)A, y; Dp(1;3)sn^{13a1} / +
P9 / P5
R(1;3)O5, (f) / C(1)M3, y^2; Cha^{11} / MRS
R(1;3)O5, f / C(1)M3, y^2; red Cha^{11} / MRS
R(1;3)O5, f / C(1)M3, y^2; sr
S / In(2LR)Cy, Cy E(S); K-pn
w; gl^3

Chrom. 1,3 (contin.)

w / C(1)M3, y^2; ro
w^a mw mit; gl^3
y; Dp(1;3)sc^{J4} flr / TM1
y; Dp(1;3)sc^{J4}, y^+ sc^{J4} M(3)i^{55} / TM1, Me mwh
y; In(3LR)C269, mwh / TM1
y; Sb / TM6
y; jv ca^{nd} / TM3, Sb Ser
y cho cv mit / FM3; gl^3
y cho cv sn^3; ro
y cho cv sn^3 / FM7; ro / TM6
y cho mit / FM3; gl^3
y cho sn^3; gl^3
y cho sn^3; Df(3R)126d / TM3
y cho sn^3; kar^2 p^P Df(3R)126d / TM3
y cho sn^3 / FM3; gl^3
y cho sn^3 l(1)d.deg.X-55 / FM6; l(3)Ace^{m38} / TM2
y $comt^1$; Acph-1^{n11b}
y Hk^{1P}; Acph-1^{n11b}

Chromosomes 1,4

y A16-129 / FM7a / y^+Y; spa^{pol}
y mei-9 bb^1 / C(1)DX, y f bb^- / y^+Y; spa^{pol}
y mei-41 / C(1)DX, y f bb^- / y^+Y; spa^{pol}

Chromosomes 2,4

dke c Bl / SM5; spa^{pol}
Roi / Cyo; spa^{pol}
Sco / CyO; spa^{pol}

Chromosomes 3,4

e^{11}; spa^{pol}
e^{11} / TM3, Sb Ser; spa^{pol}

Chromosomes 1,2,4

y / y^+Y; eff; spa^{pol}

Translocations

lz^{50e} T(1;2)v^{65b} / C(1)DX, y w f
T(X;Y)G24, y^+ f · y^+ / A(1)XB, y w^{m4} / 0
T(3;2)ry^{+w70hR} (87C-88C) Dp(1;2)w^{+70h} In(2L)22F-31A; MKRS
Sb Ubx / T(2;3)ap^{Xa}
T(3;4)A12 / ci^D
T(1;2)Bld M(1)o^{Sp} / FM7c
C(1)DX, y f / T(1;2)sn^{+72d}, f car / bw^D
T(Y;2)A165
T(Y;2)D70
T(Y;2)J59
T(Y;2)L23
T(Y;2)L107
T(Y;2)L110
T(Y;2)H52
T(Y;2)H69
T(Y;2)H142
T(1;3)OR60 / TM2 / dsx^D Sb e
T(Y;3)A13
T(Y;3)A23
T(Y;3)A32
T(Y;3)A60

y mit; l(3)Ace^{m35} / MRS
y mit; l(3)Ace^{m115} / TM6
y mit; red Df(3R)Cha / MRS
y w; Acph-1^{n11b}
y w; Dp(1;3)w^{+67k27} / TM3, Sb Ser
y w; Gl / TM3, Sb Ser
y w $comt^1$; Acph-1^{n11b}
y w Df(1)$Sx1^{10ra}$ sn / C(1)A, y; Dp(1;3)sn^{13a1} se / +
y w l(1)$ogre^{1jn13}$ cm v / In(1)dl-49, Df(1)ct^{J6} f;
　　Dp(1;3)sn^{13a1} / (TM2) / +
y w l(1)$ogre^{1jn13}$ cm v mit / FM6; Acph-1^{n11b}
y w l(1)$ogre^{vcb8}$ cm v B / FM6; Sb / TM2
y w l(1)$ogre^{vcb8}$ cm v mit / TM6; Acph-1^{n11b}
y w sn^3 / C(1)DX, y f; Df(3R)126d / TM2
y w sn^3; l(3)Ace^{j50} / TM6
y w sn^3; Sb / TM2
y w^a mit; Cha^{12} / MRS
y w^a mit; l(3)ace^{m115} / TM6

Chromosomes 2,3

al dp b Bl c px sp / In(2L)Cy, Cy; In(3L)D, D / Payne
bw; st
Dp(3;2)ry^+ Ace^+ / +; Df(3R)ry / MKRS
Drl / CyO; sbd^2 Mc / TM6
eff; mah / TM2, Ubx^{130}
Pu^2 / CyO; In(3LR)HR33
Sco / CyO; TM3, Sb Ser / TM6B, Hu e Tb ca

Chromosomes 1,2,3,

M(1)o^{Sp} / C(1)M3, y^2; CyO / +;
　　Dp(1;3)A59 Acph-1^{n11b} / Acph-1^{n11b}
nj^3 / C(1)M3, y^2; bw; st
nj^{19} / C(1)M3, y^2; bw; st
nj^{94} / C(1)M3, y^2; bw; st
tfd-1; tfd-2; DcxF tfd-3 / h tfd-3
Dp(1;3)$Acph^+$ / C(1)M3, y^2; pal / CyO; Acph-1^{n11b}

T(Y;3)A117
T(Y;3)A121
T(Y;3)A169
T(Y;3)B71
T(Y;3)B93
T(Y;3)B99
T(Y;3)B152
T(Y;3)B162
T(Y;3)B170
T(Y;3)B172
T(Y;3)B197
T(Y;3)B204
T(Y;3)B207
T(Y;3)B223
T(Y;3)B226
T(Y;3)B233
T(Y;3)B240
T(Y;3)D100
T(Y;3)D221
T(Y;3)D226
T(Y;3)G73

Translocations (contin.)

T(Y;3)G75	T(Y;3)J112	T(Y;3)R87
T(Y;3)G145	T(Y;3)J121	T(Y;3)R91
T(Y;3)H135	T(Y;3)J132	C(1)M3, y^2 bb / $Y^S X \cdot Y^L$, In(1)EN, y; TM6;
T(Y;3)H156	T(Y;3)J151	T(Y;3)H156^L-J132^R Df(3L)70c, B^S B^S
T(Y;3)H173	T(Y;3)R13	T(2;3)D^4 / CyO In(3LR)HR33
T(Y;3)J111	T(Y;3)R83	y; T(3;4)A12 Gl Sb Ubx / LVM

Wild Stocks - **D.melanogaster**

Ace^{+5}	Crimea	Lausanne-S	Sevelen
Canton-S	Hikone	Oregon-R	Swedish-b
Cockaponsett	Idaho Falls-38	Samarkand	Urbana-S
			Waginengen

NEW MUTANTS

Report of I.D. Alexandrov and M.V. Alexandrova. Research Inst. of Medical Radiology, Obninsk, USSR.

Genetics and cytogenetics of the vestigial mutations induced by gamma-rays, ^{252}Cf and fission neutrons.

The following list contains information on the 110 out of 166 transmissible vestigial mutations which were recovered in various experiments (see Alexandrov, this issue: Research Note, for details) designed for estimating the relative proportion of gene (VV^g) versus chromosome (LV, VV^{ch}) mutations at the locus in question after irradiation of the different post-meiotic germ cells of the wild type (D-32, D-18) or c(3)G males (see the last column list) by low- or high-LET radiation under pre-treatment, for some experiments, with radiomodifiers used (the fifth column). The vestigial mutations were named (first column) by the accepted alphanumeric code. The second, third and fourth columns are respectively giving information on the phenotype, pattern of complementation with nw83b27 as well as cytology for each mutation.

Taking account of the pleiotropic action of the viable mutations at the locus in question, all the major traits affected (wings, halteres, postscutellars and fertility) were borne in mind for phenotypic description of the mutant selected as well as its complementation pattern when mutant under study compounded with nw83b27 which was used on account of its unique phenotype and complementary features (see list below for details). Briefly, both males and females nw83b27 show fully reduced wings and halteres, good viability and fertility as well as normal postscutellars. When compounded to the pseudoalleles (vg, s, np) or some homoalleles, the nw83b27 is found to produce a phenotype more normal than that of either allele under study.

As seen, 34 transmissible and viable in homozygote vestigial mutations as well as 11 non-viable ones, but with separable lethal phenotype (so-called "twin" vestigial mutants), have a normal 49 DE region 2R chromosome (the putative location of the gene in question) whereas 63 out of 109 mutations preserved proved to be associated with either chromosome aberrations among which inversions found to be predominant changes (35 out of 63 rearrangements studied) in the wild type as well as c(3)G genome. Also, the "twin" vestigial mutants arise more frequently (9 out of 12) after action of photons than neutron irradiation and this picture is analogous to that for the "twin" mutants at the black locus (Alexandrov 1986; Alexandrov & Alexandrova 1986). Further, among 45 non-lethal and unassociated with the visible rearrangements vestigial mutations 27 (60%) are sterile in homozygote, and 20 of them are extreme nw mutants which, as with other sterile vg mutations, weakly or not all complement with nw83b27; that is why all these can be accepted as microdeletions. On the other hand, some vg mutations associated with chromosome aberrations (e.g., 72a1, 78b4, 78j3, 81l18, etc.) complement with nw83b27, showing that such vg mutants appear to be due to "position effects" rather than radiation-induced lesions within the locus in question. Finally, the 17 different vestigial mutants are fertile and free from any rearrangements detected by the methods employed. Therefore, they can be accepted as true gene mutations (VV^g). A share of VV^g in the spectrum of all transmissible vestigial mutations induced by radiations studied was found to be one fifth as much as that among the transmissible black mutations (Alexandrov & Alexandrova 1986). This distinction must have been conditioned by unique features of the fine structure of the loci themselves. On the other hand, it can be that the frequency of the black VV^g is overstated at the expense of microdeletions unrecognized at this locus for lack of interallelic complementation -- generally speaking, being of particular concern for the most exact definition of VV^g from microdeletion.

References: Alexandrov, I.D. 1986, DIS 63:19-21; ――― & M.V. Alexandrova 1986, DIS 63:159-161; Lindsley, D.L. & E.H. Grell 1968, Carnegie Inst. Wash. Publ. 627.

Table 1. Notes: * See Lindsley & Grell 1968; phenotype: wings (wg), halteres (h: + wild type, - lack, ± intermediate), postscutellars (ps: + wild type, - lack, ± intermediate), fertility (f: + good, - sterile); s^C or np^C - crumpled and blistered wings in the complementary mutants;   ** C - caffeine and AD - actinomycin-D; *** MS - mature sperm, L, M, and E - late, middle, and early spermatids (Sd), respectively; @ The "twin" vestigial mutations with independent lethal separated by crossing over; # - lost mutants.

Designation of mutation	Phenotype* under 25°C wg h ps f	Complementation with nw83b27 under 25°C wg h ps	Cytology	Modifier*** used, radiation, dose	Genotype, germ cells irradiated
67d1	lethal	as 83b27	T(2;3)49C2-D2;93E-F1	γ-rays, 40 Gy	D-32,MS***
67d2	vg@- - -	vg - +	Normal	"	D-18,E Sd
71k2#	nw - - +	+ + +	Normal	"	D-18,L Sd
72a1	lethal	+ + +	In(2R)44C2-5;49D2-E1	"	D-18,MS
74b1	lethal	s^C + +	In(2LR)37F-38A1;49D2-E1	C + γ-rays, 40 Gy	D-32,L Sd
74b2#	nw - - +			"	D-32,M Sd
74c1	nw - - -	vg - +	Normal	"	D-32,MS
74c4	nw - - -	as 83b27	In(2LR)22A5-B1;49D2-E	"	D-32,M Sd
74c5	s@ ± + +	s^C ± +	Normal	γ-rays, 40 Gy	D-32,L Sd
74c6	nw@- - -	nw - +	Normal	"	D-32,MS
74c7	nw@- - -	nw - +	Normal	"	D-32,MS
76d1	vg@- - -	s^C ± +	Normal	C + γ-rays, 40 Gy	c(3)G,MS
76d2	vg - -	s^C ± +	T(2,Y)58B+Dp(2R)58B-D	"	c(3)G,MS
76f	vg - - +	s^C + +	Normal	γ-rays, 40 Gy	c(3)G,MS
76i1	lethal	as 83b27	T(2;3)49D2-E1;84E2-3	AD+γ-rays, 40 Gy	D-32,MS
76i2	vg - - +	np^C + +	Normal	"	D-32,MS
76j1	lethal	np ± +	Tp(2)49D;60B;60A;50F;49E;60C	"	D-32,MS
76k2	lethal	np^C ± +	Normal	"	D-32,MS
77a4	np + + -	np^C ± +	Normal	"	D-32,M Sd
77d1	lethal	as 83b27	In(2LR)25C-D;49D2-E1	0.85MeV n,20 Gy	D-32,MS
77d2	s - -	s ± +	Tp(2R)49B4;49B5-12;49D2-E	0.35MeV n,10 Gy	D-32,MS
78a1	no -	+ ± +	In(2R)41D-E;49D3-E1	γ-rays, 40 Gy	c(3)G,MS
78a2	vg - - -	as 83b27	In(2R)49D3-E;56E	"	c(3)G,M Sd
78b1	nw - - -	s^C - +	Normal	"	c(3)G,M Sd
78b2	nw - - +	s^C ± +	Normal	"	c(3)G,MS
78b3	vg - - +	np^C + +	Normal	"	c(3)G,MS
78b4	lethal	+ + +	T(2,3)49D2-3;49E7-F1;80C	NaF + γ-rays, 40 Gy	D-32,MS
78f2	vg - - -	s^C ± +	Normal	"	D-32,M Sd
78j1	nw - - -	+ ± +	Tp(2R)49D2-3;49D7-E1;50C1-6	γ-rays, 40 Gy	c(3)G,L Sd
78j3	lethal	+ + +	In(2R)41D2-E1;49D2-E1	"	c(3)G,M Sd
78k2	vg - - +	+^C + +	Normal	NaF + γ-rays, 40 Gy	D-32,MS
78k3	lethal	as 83b27	In(2R)49D2-E1;59D4-8	"	D-32,M Sd
79a	lethal	as 83b27	In(2LR)34B2-C1;49B12-C3 + Df(2R)49D1-E2	0.35MeV n,20 Gy	D-32,MS
79b1	vg - - +	s^C + +	Normal	γ-rays, 40 Gy	D-32,MS
79b3	lethal	as 83b27	T(2,3)49C2-3;94A2-3 + Df(2R)49C4-49E2	"	D-32,MS
79b4	lethal	as 83b27	In(2R)41C-D;49D2-E1	0.85MeV n,10 Gy	D-32,MS
79b6	lethal	as 83b27	Tp(2R)49D2;50C9-14;49E1	0.35MeV n,10 Gy	D-32,MS
79d2	lethal	as 83b27	Df(2R)49C2-D2;49D7-E1	0.85MeV n,10 Gy	D-32,MS
79d3	lethal	s^C ± +	In(2R)41A±;49D2-E2	0.35MeV n,20 Gy	D-32,MS
79d4	nw@- - -	as 83b27	In(2R)41E;49D2-E1	0.85MeV n,10 Gy + γ-rays, 10 Gy	D-32,MS
79d5	np + + +	+ + +	Normal	"	D-32,MS
79d6	nw - - -	s^C ± +	Normal	0.85MeV n,10 Gy	D-32,MS
79d7	nw - - -	as 83b27	In(2R)41D-E;49D3-F1	"	D-32,MS
79d8	lethal	as 83b27	Df(2R)49C4-D1;49D7-E1	"	D-32,MS
79f1	lethal	as 83b27	Tp(2LR)36D;53F;49E;41A;54A;41A	NaF + γ-rays, 40 Gy	D-32,M Sd
79f2	nw - - -	- - +	In(2R)49D2-E1;49E7-F13	"	D-32,M Sd
79h1	nw - - -	vg - +	Normal	γ-rays, 40 Gy	D-32,E Sd
79h4	lethal	as 83b27	In(2LR)24D;49D1-E1	"	c(3)G,M Sd
79h5	vg - - -	s^C ± +	In(2R)49D2-E;50A2-3	"	c(3)G,MS
79h6	nw - - -	as 83b27	In(2R)41E-F;49D2-E1 + In(2R)42B2-3;57F-58A1	"	c(3)G,E Sd
79h7	nw - - -	as 83b27	In(2R)49D2-7;49F10-13	"	c(3)G,E Sd

ID				Rearrangement	Treatment	Marker
8011	lethal		as 83b27	Df(2R)49D2-3;49E7-F1	0.7MeV n,10 Gy	D-32,MS
8012	nw - - -	s^C ± +		Normal	"	D-32,MS
81a	lethal		as 83b27	T(2,3)49D2-F1;64B2-12	γ-rays, 40 Gy	c(3)G,E Sd
81a-s	s^2 ± ± -	+ + +		Normal	γ-rays, 60 Gy	D-32,MS
81b1	lethal		as 83b27	In(2R)48C4-D1;49D2-E	γ-rays, 40 Gy	c(3)G,M Sd
81b2#	nw - - +	nw + +		Normal	"	c(3)G,L Sd
81c13	vg - - +	s^C + +		Normal	"	D-32,M Sd
81c18	nw - - -	s^C - +		Normal	"	D-32,M Sd
81c28	nw - - -		as 83b27	In(2R)41D-E;49D3-E7	"	D-32,L Sd
81c41d	lethal	vg - +		In(2R)49C2;49F14	"	D-32,L Sd
81f	vg@- - +	np^C + +		Normal	0.1MeV n,10 Gy	D-32,MS
81k1	nw - - -	s^C - +		Normal	γ-rays, 10 Gy	D-32,MS
81l18	vg - - -	+ + +		In(2LR)36C4-D1;49D2-F1	γ-rays, 20 Gy	D-32,MS
81l24	vg@- - +	np^C ± +		Normal	γ-rays, 10 Gy	D-32,MS
81l26	nw - - -	s^C - +		Normal	γ-rays, 20 Gy	D-32,MS
82c13	vg@- - +	+^{C} + +		Normal	^{252}Cf, 14 Gy	D-32,MS
82c14	nw - - -		as 83b27	In(2LR)36C-D;49D2-E	^{252}Cf, 20 Gy	D-32,MS
82c61	lethal		as 83b27	In(2LR)24E2-F1;49D2-E7	^{252}Cf, 14 Gy	D-32,L Sd
83b	lethal		as 83b27	Df(2R)49B2-3;49E7-F1	γ-rays, 40 Gy	D-32,M Sd
83b22	nw - - -	s^C ± +		Normal	"	D-32,E Sd
83b24	nw - - -	s^C ± +		Normal	"	D-32,M Sd
83b27	nw - + +			Normal	"	D-32,M Sd
83b39	nw - - -	s - +		In(2R)49D2-E;51D2-6	"	D-32,L Sd
83c	np - ± -	+ + +		T(2,3)49D2-E;65F6-66A	"	D-32,MS
83c3	nw - - -		as 83b27	In(2R)41C-D;49D2-E1	"	D-32,L Sd
83c5	nw - - -	vg - +		Normal	"	D-32,L Sd
83c7	nw - - -	s + +		Normal	"	D-32,M Sd
83c24	nw - - -	s ± +		Normal	"	D-32,MS
83c31	lethal		as 83b27	Df(2R)49C3-D2;49F15-50A1	"	D-32,MS
83c42	s - - -	s^C ± +		Normal	"	D-32,L Sd
83c43	nw - - -		as 83b27	In(2R)43C2-3;49D2-E	"	D-32,MS
83c45	vg@- - -	vg - +		Normal	"	D-32,M Sd
83d	nw - - -	s^C ± +		Normal	^{252}Cf, 14 Gy	D-32,MS
83d4	nw - - -		as 83b27	In(2R)48E2-F1;49D2-E1	^{252}Cf, 28 Gy	D-32,MS
83f15	lethal		as 83b27	Df(2R)49C3-D1;49E7-F1	γ-rays, 40 Gy	D-32,MS
83f36	lethal		as 83b27	Df(2R)49C3-D1;49E7-F1	"	D-32,MS
83f38	lethal	+ + +		In(2R)49D±;59D-E	"	D-32,M Sd
83f58	lethal		as 83b27	Df(2R)49B12-C1;49D7-E1	"	D-32,M Sd
83f-XD	nw - - -	♀nw - + / ♂4		In(2R)48E2-F1;49D2-E	X-rays, 40 Gy	c(3)G,MS
8312a	lethal		as 83b27	Df(2R)49D2-3;49D7-E1	0.7MeV n,10 Gy	D-32,MS
8312b	lethal		as 83b27	In(2R)41D-E;49D2-E1	"	D-32,MS
831-N	lethal		as 83b27	Df(2R)49C3-D1;50B9-C1	"	D-32,MS
831-s	lethal	+ + +		Normal	"	D-32,MS
84f	nw@- - -	nw - +		Normal	X-rays, 40 Gy	c(3)G,M Sd
84f65	lethal		as 83b27	Df(2R)49B11-C1;49D7-E1	"	c(3)G,MS
84f51	nw - - -		as 83b27	In(2R)44F2-45A1;49D2-E1	"	c(3)G,M Sd
84hXD	nw - - -		as 83b27	In(2R)41B-C;49D2-E1	γ-rays, 40 Gy	c(3)G,E Sd
84h49	lethal		as 83b27	Df(2R)49B2-5;49F10-14	X-rays, 20 Gy	c(3)G,L Sd
84hXC	lethal	+ + +		In(2R)48E6-F1;49D2-E1	γ-rays, 40 Gy	c(3)G,M Sd
85b	np + + +	+ + +		Normal	0.85MeV n,5 Gy	c(3)G,MS
85c	lethal		as 83b27	In(2R)41B-C;49F1 + Df(2R)49B12-C1;49E2-F1	0.85MeV n,10 Gy	c(3)G,MS
85d1	nw - - -	nw - +		Normal	0.85MeV n,15 Gy	c(3)G,MS
85d2	nw - - -	vg - +		T(2,3)49D2-E;84F4-6	"	c(3)G,MS
85e2	nw - - -		as 83b27	Tp(2R)41B;49E;55F	γ-rays, 40 Gy	c(3)G,MS
85e3	nw - - -	vg - +		Normal	γ-rays, 20 Gy	c(3)G,MS
85e4	nw - - -	nw - +		Normal	"	c(3)G,MS
85f1	lethal		as 83b27	Df(2R)49B11-C1;49E7-F1	γ-rays, 20 Gy	c(3)G,MS
85f2	lethal		as 83b27	Df(2R)49B11-C1;49E7-F1	γ-rays, 10 Gy	c(3)G,MS
85f3	vg@- - +	+^{C} + +		Normal	"	c(3)G,MS

Report of C.N.L. Bogahawatta* and W.E. Ratnayake.
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Six new mutants of **Drosophila ananassae** from Sri Lanka.

A study was carried out to isolate morphological mutants of **Drosophila ananassae** in Sri Lanka to estimate the mutation load. Eighteen mutants of spontaneous origin were isolated, and we report here six new mutants of **Drosophila ananassae** in these populations. These six new mutants are demi wing (dw), net wing (nt), semi wing (sw), ballooned semi wing (bsw), reduced eye (re), and Curled wing (Cu). Three of the mutants are recessive autosomal, two are recessive sex-linked while the other is autosomal dominant. They are described below.

(1) **dw : demi wing.** The wing is reduced to about half the normal length while the breadth remains more or less the same (length of normal wing = 2.33 mm ± 0.143 mm, n=10; length of mutant wing = 1.293 mm ± 0.063 mm, n=10). Main veins are visible with L4 interrupted but cross veins are indistinct. The mutant females showed low viability and low fertility.

The F_2 results (Table 1) show that they do not fit an expected 3:1 ratio, showing more wild type flies than mutant types. This may have been due to the fact that the mutant females were weak and showed less viability, and, hence, all mutant flies may not have emerged from the pupal cases. However, when the results of all experimental crosses are considered, it can be concluded that the demi wing (dw) mutant is due to a recessive autosomal gene.

(2) **nt : net wing.** The wings have a network of extra veins apart from the normal five longitudinal veins and four cross veins. This mutant shows viable expressivity and full penetrance.

The F_2 results (Table 1) show a fit for the 3:1 ratio from one parental cross only. The departure from the expected ratio at p = 0.05 level in the reciprocal parental cross may be due to misidentification. However, both crosses taken together approximate to a 3:1 ratio. This mutant is, therefore, concluded to be due to a recessive autosomal gene.

(3) **re : reduced eye.** The eye is smaller in size than the normal and is bar like. Expression is variable. The F_2 results of the parental crosses (Table 2) show that they fit a 3:1 ratio which is expected for the segregation of a recessive autosomal gene.

The back cross of the F_1 females crossed to reduced eye males did not give a 1:1 ratio as expected for a recessive gene ($X^2_{(1)}$ = 10.00; p < 0.01). From a consideration of the results of the experimental crosses, it can be concluded that reduced eye mutant is a recessive autosomal gene with lowered penetrance and variable expressivity.

(4) **sw : semi wing.** The wing size in which the length and the breadth varies from about half to two-thirds of the normal wing (length of normal wing = 2.33 mm ± 0.14 mm, n=10; length of mutant wing = 1.65 mm ± 0.13 mm, n=10) (breadth of normal wing = 1.85 mm ± 0.05 mm, n=10; breadth of mutant wing = 1.32 mm ± 0.14 mm, n=10).

Table 1. Results of the F_2 progeny of five new mutants isolated from D.ananassae in Sri Lanka.

| | wild-type | | mutant-type | | Total no. flies | | x^2 |
Mutant and Cross	♀♀	♂♂	♀♀	♂♂	normal	mutant	Values
(1) **demi wing**							
(a) dw/dw ♂♂ x +/+ ♀♀	551	457	146	134	1008	280	7.30*
(b) dw/dw ♂♂ x +/+ ♀♀	136	132	22	28	268	50	14.60*
(2) **net wing**							
(a) nt/nt ♂♂ x +/+ ♀♀	129	121	35	26	250	61	4.81*
(b) nt/nt ♂♂ x +/+ ♀♀	124	114	19	28	238	47	11.00*
(3) **reduced eye**							
(a) re/re ♂♂ x +/+ ♀♀	466	346	134	107	812	241	2.51
(b) re/re ♂♂ x +/+ ♀♀	588	541	196	178	1129	374	0.02
(4) **semi wing**							
(a) sw/y ♂♂ x +/+ ♀♀	419	181	--	126	600	126	25.61**
(b) sw/sw ♂♂ x +/y ♀♀	70	63	45	51	133	96	6.72*
(5) **ballooned semi wing**							
(a) bsw/y ♂♂ x +/+ ♀♀	312	165	--	98	477	98	19.79**
(b) bsw/bsw ♂♂ x +/y ♀♀	68	51	34	40	119	74	13.86*

Table 2. Results of the Curled wing mutant from **D.ananassae** in Sri Lanka.
* = significant at p>0.05; ** = significant at p>0.01.

| | wild-type | | mutant-type | | Total no. flies | | x^2 |
Mutant and Cross	♀♀	♂♂	♀♀	♂♂	normal	mutant	Values
(6) **Curled wing**							
(a) Cu/+ ♂♂ x +/+ ♀♀	321	257	290	246	578	536	1.58
(b) Cu/+ ♀♀ x +/+ ♂♂	253	241	217	174	494	391	11.99*
(c) Cu/+ ♂♂ x Cu/+ ♀♀	204	219	487	433	423	920	2.03

Figure 1.
  dw: demi wing

Figure 2.
  nt: net wing

Figure 3.
  re: reduced eye

Figure 4.
  sw: semi wing

Figure 5.
  bsw: ballooned
       semi wing

Figure 6.
  Cu: Curled wing

The F_2 results of the reciprocal parental crosses (Table 1) show that this is probably due to a recessive sex-linked mutation although there is significant deviation from the expected ratio of 2:1:1 (2 wild ♀♀ : 1 wild ♂♂ : 1 mutant ♂♂), but fit a 1:1:1:1 (1 wild ♀♀ : 1 wild ♀♀ : 1 mutant ♂♂ : 1 mutant ♂♂). Perhaps the mutant shows incomplete penetrance and poor viability, which would have produced the significant departure from expected ratios.

This mutant is clearly different from the miniature wing described by Moriwaki & Tobari (1975). This semi wing (sw) mutant resembles the demi wing (dw) mutant described above, but semi wing is recessive sex-linked while demi wing (dw) is recessive autosomal.

(5) **bsw : ballooned semi wing.** The ballooned semi wing (bsw) arose from the sw culture. This is very similar to the semi wing (sw) mutant, except for the presence of a bubble (like a balloon) in the middle of the wing, and also in that the wing venation is slightly irregular. The wing length and breadth are also slightly smaller than those of the semi wing (sw).

The F_2 results of both crosses (Table 1) show that they do not fit the expected ratios of 2:1:1 (2 wild ♀♀ : 1 wild ♂♂ : 1 wild ♂♂) and 1:1:1:1 (1 wild ♀♀ : 1 wild ♀♀ : 1 mutant ♂♂ : 1 mutant ♂♂). This may have been due to incompete penetrance and poor viability.

(6) **Cu : Curled wing.** About two thirds of the wings of this mutant are curled upward. Some wings are variably Curled in which about half of the wings are Curled upward. The F_1 results of both crosses show that this is a dominant autosomal mutation. The results of the intercross of the heterozygotes show a 2:1 ratio (Table 2). This would mean that the homozygous mutant flies die, that is, the mutant gene acts as a recessive lethal gene. However, a few flies with crumpled wings do appear which when outcrossed produce Curled wing mutants.

Mutation frequency. The mutation frequency of the Sri Lankan population was calculated from the formula given by VandeHay in 1961 (modified from Spencer). The mutation frequency of these populations is 0.28 per individuals compared to Spencer's value (obtained also for **D.ananassae** from a Pacific Island) of about 0.63 per individual (VandeHay 1964). The frequency obtained by us is seen to be lower.

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References. Moriwaki, D. 1938, Jpn. J. Genet. 14(1&2):1-22; Moriwaki, D. & Y. Tobari 1975, in: Handbook of Genetics (King, ed.), v.3 on **D.ananassae**; VandeHay, R.C. 1964, Ann. Entom. Soc. Amer. 57:488-495.

Report of J. Gausz, H. Gyurkovics and J. Szabad.
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Ki^S, a new cell marker mutation.

Ki^S is an EMS-induced dominant mutation. Ki^S (like Ki^1) is homozygous viable with slightly reduced viability. In Ki^S heterozygotes, all bristles (including those on the terminalias) are strongly reduced and twisted without fluting. In Ki^S homozygotes, most of the micro and a few of the macrochetae are missing; however, sockets develop normally. Phenotype of the Ki^S/Ki^1 heterozygotes is similar to that of the Ki^S/Ki^S ones; hence, Ki^S is allelic to Ki^1. Ki^S is located on proximal 3R (Lindsley & Grell 1968; Garcia-Bellido 1972). Salivary chromosomes of the $Ki^S/+$ larvae appear normal.

Ki^S is an excellent tool for clonal analysis. It is very easy to identify Ki^+ (wild-type) clones on $Ki^S/+$ background (see Figure). Size of these clones is identical to those seen in the case of the best cell marker mutations (like f^{36a}, Pr and Bsb). In contrast to Ki^S, it is difficult to detect (especially small) wild-type clones on the $Ki^1/+$ background, where most of the clones consists of only one bristle per clone over the disc-derived structures (Szabad & Bryant 1982).

References. Garcia-Gellido, A. 1972, Mol. Gen. Genet. 115:54; Lindsley, D.L. & E.H. Grell 1968, Genetic variations of **Drosophila melanogaster**, Carn. Inst. Wash. Publ. No. 628; Szabad, J. & P.J. Bryant 1982, Devel. Biol. 93:240.

Report of F. Mestres and G. Pegueroles.
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New information on the af mutant of **D.subobscura.**

The tarsal fusion and other abnormalities which characterize the af mutant were described previously (Mestres 1985). The linkage analysis shows that the af mutation is located in the U chromosome. Furthermore, the cytological study of the mutant strain proved that it was homokaryotypic for the following chromosomal arrangements: O_{3+4}, U_{st}, E_{st}, J_1, A_{st}.

   Missing legs was another trait analyzed in mutant individuals. In most of the 92 individuals with five legs, the missing leg is a posterior one. Most of the 40 flies with only four legs have two legs on each side; they seldom have three legs on one side and only one on the other. Only 3 individuals with three legs and 2 with two legs were observed. On the other hand, three individuals with extra articles, always in the posterior legs, were detected.

   It seems probable that af mutant, under certain conditions, produces alterations of the normal pattern of leg development. All mutant individuals have fused articles, and a smaller number of them is found as the number of legs decreases: individuals with six legs are more frequently found than individuals with five legs, and so on. When some legs are missing, they are most probably the posterior ones. The forward pair of legs is the more conserved one. The individuals with only two or three legs have the forward pair.

   References. Mestres, F. 1985, DIS 61:214-215.

Report of C. Najera and M.L. Aparisi.
Dept. of Genetics, Faculty of Biology, University of Valencia, Spain.

Different eye colour mutants, not previously described, from natural populations of **D.melanogaster.**

ches:chestnut (2:101.8±3.0). Eye colour dark chestnut. 90% wildtype red pigment and 81% wildtype brown pigment. RK2.

gra:grape (3:34.7±1.3). Eye colour ruby. 57% wildtype red pigment and 92% wildtype brown pigment. RK1.

sb:spoony-brown (3:38.3±0.6). Eye colour spoony brown darkening with age. 60% wildtype red pigment and 42% wildtype brown pigment. RK1.

pur:purplish-ruby (3:39.5±0.2) Eye colour purplish ruby. 75% wildtype red pigment and 122% wildtype brown pigment. RK1.

alm:almond (3:25.6±1.7) Eye colour light brownish, darkening with age. 62% wildtype red pigment and 114% wildtype brown pigment. RK2.

jew:jewel (2:107.5±1.0) Eye colour ruby. 47% wildtype red pigment and 39% wildtype brown pigment. RK1.

ifa:irregular facet (3:23.4±0.4). Eye colour like wild darkening with age, irregular facets. 53% wildtype red pigment and 70% wildtype brown pigment. RK2.

dr:dark-red (2:11.1±1.6) Eye colour dark red, garnet in aged flies. 67% wildtype red pigment and 97% wildtype brown pigment. RK3.

dch:dark-chestnut (3:25.7±1.4). Eye colour dark chestnut. 93% wildtype red pigment and 148% wildtype brown pigment. RK3.

man:mandarin (2:54.3±2.0). Eye colour bright red, almost orange. 66% wildtype red pigment and 18% wildtype brown pigment. RK1.

Report of G. Pegueroles.
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List of mutants obtained after sib mating in a population
of **Drosophila subobscura** from Eureka (California).

   From isofemale lines, some sib-matings were carried out and the following spontaneously arisen mutants -- to be confirmed later on (that is why they have not been given any name) -- were obtained. The numbers are those from the original strains.

Number of the strain	Morphological Features	Number of the strain	Morphological Features
15	Right leg of the first pair absent.	17	Posterior right mesothorax absent, right wing absent.
38	Open wings, bent downwards slightly.	4	Wings unfolded and bent downwards.
10	Wings extended and bent upwards.	126	Brown eggs; females showing this character are semi-sterile.
28	Reduced or absent eyes. Character very variable. Low fertility.		

Report of Y. Pérez-Chiesa, C.N. Cintrón, A. Rodríguez, A.M. Lugo and L. Díaz.
University of Puerto Rico, Rio Piedras, Puerto Rico, USNA.

Several lethals were isolated during a sex-linked recessive lethal test performed for detecting the mutagencity of fagaronine in Drosophila. The mutants obtained with fagaronine were balanced with FM6 and three of them, 1(1)CP7, 1(1)CP8 and 1(1)CP9, were localized through a 4-point test as follows: 1/FM6 females were crossed to y cv v f males; their F_1 1/y cv v f daughters were also crossed to y cv v f males. Only the F_2 male progeny was scored. Cultures were grown in banana medium at 25°C.

1(1)CP7 - 1-14.1 The lethal was localized between **cv** and **v**. There were 554 recombinants in this region out of 2,358 males scored. Of these 542 were **cv +** and only 12 were **+ v**. According to the standard map the distance between **cv** and **v** is 19.3. Thus, the distance between **cv** and the lethal is: (12/554) (19.3) = 0.42, and the locus of 1(1)CP7 is 14.1 (13.7 plus 0.4).

1(1)CP8 - 1-22.1 The lethal was localized between **cv** and **v**. There were 497 recombinants in this area out of 2066 males scored, of which 282 were **cv +** and 215 were **+ cv**. Therefore, the distance between **cv** and the lethal is: (215/497) (19.3) = 8.35 and the locus for the lethal is 22.1 (13.7 + 8.7).

1(1)CP9 - 1-0.1 The lethal was localized between **y** and **cv**. There were 430 recombinants in this area, out of 2724 males scored, of which 426 were **y +** and only 4 were **+ cv**. According to the standard map, the distance between **y** and **cv** is 13.7. Thus, the distance between **y** and CP9 is: (4/430) (13.7) = 0.13.

References: Perez-Chiesa, Y. & C.N. Cintron 1986, Mutation Res. 173:193-196; Strickberger, M.W. 1962, Experiments in Genetics with Drosophila, John Wiley & Sons, Inc., New York.

Report of P. Roberts.
Dept. of Zoology, Oregon State University, Corvallis, Oregon 97331.

Drosophila gibberosa is a large, cytologically favorable species that is an apparent soil pupater yet can be easily cultured on standard medium (Roberts & MacPhail 1985). Using the X-linked mutant, yellow, we have, by means of X-irradiation, generated an attached-X chromosome. This rearrangement, C(1)RM,y should be useful in accumulating additional mutants and in mapping the X.

References: Roberts, P.A. & L. MacPhail 1985, Chromosoma 92:55-68.

Report of M-T. Yamamoto.
Dept. of Cell Genetics, National Institute of Genetics, Mishima, Shizuoka 411, Japan.

The following translocations were constructed by X-ray irradiation to Oregon-R males. The screening method employed here was essentially the same as that of Roberts (1972). Newly emerged adult male flies were irradiated with 4000-4500 R of X-rays and the treated males were then crossed to ci ey[R] virgin females. These were then transferred to new culture bottles every second day for three times before being discarded. The cultures were kept at 18°C throughout the life cycle in order to maximize the penetrance of the ci phenotype. F_1 males exhibiting the ci phenotype were then individually crossed to C(1)DX,y f; bw; e; ci ey[R] females. Male progeny from this cross were then backcrossed to females of the marker strain and flies of the F_3 generation were checked for segregation characteristics of a translocation heterozygote. All T(2;4) translocations were then balanced over CyO and T(3;4) balanced over TM3.

All stocks except those marked with an * have been transferred to Drosophila Stock Center, National Institute of Genetics, Mishima, because of my temporary absence from the Institute. Stocks marked with an * were lost prior to the transfer, and those designated as (h.v.) are homozygous viable translocations.

References: Roberts, P.A. 1972, Genetics 71:401-415.

T(2;4) TRANSLOCATIONS

T(2;4)Y2/CyO; ci ey^R	35D3-4;101F
*T(2;4)Y7/CyO; ci ey^R	50C14-D1;102C-D (wing margin depleted)
*T(2;4)Y22/CyO; ci ey^R	41;101F
T(2;4)Y34/CyO; ci ey^R	36C-D;101F
T(2;4)Y40/CyO; ci ey^R	56B-C;101F
T(2;4)Y47/CyO; ci ey^R	31D-E;101F
*T(2;4)Y64/CyO; ci ey^R	35C;101F
T(2;4)Y76/CyO; ci ey^R	40-41;101F (h.v.)
T(2;4)Y86/CyO; ci ey^R	21E1-2;101F
T(2;4)Y92/CyO; ci ey^R	57B4-6;101F
*T(2;4)Y100/CyO; ci ey^R	21D;101F
T(2;4)Y106/CyO; ci ey^R	55A;101F (h.v.)
T(2;4)Y109/CyO; ci ey^R	40-41;59B;101F
*T(2;4)Y141/CyO; ci ey^R	40-41;101F
T(2;4)Y142/CyO; ci ey^R	52E;102C (h.v.)
T(2;4)Y164/CyO; ci ey^R	33A;101F
T(2;4)Y185/CyO; ci ey^R	52D;101F
T(2;4)Y209/CyO; ci ey^R	36C-D;101F (h.v.)
T(2;4)Y220/CyO; ci ey^R	56F6-8;101F
T(2;4)Y231/CyO; ci ey^R	52F;102D
T(2;4)Y241/CyO; ci ey^R	54D-E;101F
T(2;4)Y296/CyO; ci ey^R	
T(2;4)Y308/CyO; ci ey^R	49F10-15;101F
*T(2;4)Y316/CyO; ci ey^R	
*T(2;4)Y318/CyO; ci ey^R	40-41;101F
T(2;4)Y325/CyO; ci ey^R	34E-F;101F (h.v.)
T(2;4)Y344/CyO; ci ey^R	59F;101F
T(2;4)Y375/CyO; ci ey^R	60F5;101F
T(2;4)Y376/CyO; ci ey^R	33C;101F
T(2;4)Y423-A/CyO; ci ey^R	21B3-5;101F
T(2;4)Y465/CyO; ci ey^R	52D9;101F
T(2;4)Y476/CyO; ci ey^R	29D;101F
T(2;4)Y492,In(2LR)/CyO; ci ey^R	30A7-9;56F8-9;101F
T(2;4)Y496/CyO; ci ey^R	22A1-2;101F
T(2;4)Y517/CyO; ci ey^R	54F;101F
T(2;4)B III17/CyO; ci ey^R	40-41;101F
T(2;4)K10/CyO; ci ey^R	57F3-6;101F
T(2;4)K12/CyO; ci ey^R	49B;101F
*T(2;4)K13/CyO; ci ey^R	25A3-4;101F
T(2;4)K22/CyO; ci ey^R	56F12-15;101F (eye colour brownish)
T(2;4)K44/CyO; ci ey^R	
T(2;4)K53/CyO; ci ey^R	53C;101F
T(2;4)K55/CyO; ci ey^R	27E;101F
T(2;4)K86/CyO; ci ey^R	42A;101F (h.v.)
T(2;4)K89/CyO; ci ey^R	52F7-9;101F (h.v.)
T(2;4)K92/CyO; ci ey^R	47E;101F
T(2;4)K109/CyO; ci ey^R	50C1-2;101F
T(2;4)K419/CyO; ci ey^R	35F;101F

T(3;4) TRANSLOCATIONS

T(3;4)Y9/TM3; ci ey^R	65B4;101F
T(3;4)Y13/TM3; ci ey^R	80-81;101F
T(3;4)Y25,In(3R)/TM3; ci ey^R	90C;98C;101F
T(3;4)Y37/TM3; ci ey^R	80-81;101F
T(3;4)Y57/TM3; ci ey^R	75F;102C
*T(3;4)Y61/TM3; ci ey^R	92A;101F
T(3;4)Y63/TM3; ci ey^R	80-81;101F (h.v.)
T(3;4)Y71/TM3; ci ey^R	81F;101F
T(3;4)Y78/TM3; ci ey^R	100F;102C14
T(3;4)Y81/TM3; ci ey^R	64E;101F (eye deformed)
T(3;4)Y104/TM3; ci ey^R	96A1-4;101F
T(3;4)Y121/TM3; ci ey^R	80-81;101F
*T(3;4)Y140/TM3; ci ey^R	96A20-25;101F
T(3;4)Y144/TM3; ci ey^R	88C;101F
T(3;4)Y151/TM3; ci ey^R	78B;101F
T(3;4)Y154/TM3; ci ey^R	72C;101F
T(3;4)Y161/TM3; ci ey^R	99E;101F
T(3;4)Y168/TM3; ci ey^R	76B1;102D
T(3;4)Y175/TM3; ci ey^R	91D;101F
T(3;4)Y177/TM3; ci ey^R	77B;101F
*T(3;4)Y183/TM3; ci ey^R	68D;101F
T(3;4)Y210/TM3; ci ey^R	67E;101F (h.v.)
T(3;4)Y226/TM3; ci ey^R	63D;101F (h.v.)
T(3;4)Y249/TM3; ci ey^R	80-81;101F
T(3;4)Y252/TM3; ci ey^R	80-81;101F (h.v.)
T(3;4)Y255/TM3; ci ey^R	94B;101F
T(3;4)Y262/TM3; ci ey^R	71F;101F (h.v.)
T(3;4)Y285/TM3; ci ey^R	96D-E;101F (h.v.)
T(3;4)Y291/TM3; ci ey^R	80-81;101F (h.v.)
T(3;4)Y320/TM3; ci ey^R	88D;101F (h.v.)
T(3;4)Y391/TM3; ci ey^R	98F11-12;101F
T(3;4)Y403/TM3; ci ey^R	67E3-4;101F
T(3;4)Y425/TM3; ci ey^R	83A;101F (h.v.)
T(3;4)Y434/TM3; ci ey^R	80-81;101F
T(3;4)Y446/TM3; ci ey^R	70D;101F
T(3;4)Y449/TM3; ci ey^R	88E4-6;101F
T(3;4)Y477/TM3; ci ey^R	75C;101F
T(3;4)Y494/TM3; ci ey^R	98B;101F
T(3;4)495/TM3; ci ey^R	70C;101F (h.v.)
T(3;4)Y512,In(3R)/TM3; ci ey^R	94B-C;96E;101F
T(3;4)BII 8/TM3; ci ey^R	77A;101F
T(3;4)BII13/TM3; ci ey^R	93E;101F
T(3;4)K5/TM3; ci ey^R	
T(3;4)K11/TM3; ci ey^R	(h.v.)
*T(3;4)K19/TM3; ci ey^R	
*T(3;4)K30/TM3; ci ey^R	
T(3;4)K19/TM3; ci ey^R	94D;101F
T(3;4)K30/TM3; ci ey^R	
T(3;4)K31/TM3; ci ey^R	79F;101F
T(3;4)K85/TM3; ci ey^R	64C;101F
T(3;4)K87/TM3; ci ey^R	87D;101F
T(3;4)K95/TM3; ci ey^R	
T(3;4)K104/TM3; ci ey^R	(h.v.)
T(3;4)K108/TM3; ci ey^R	
T(3;4)K116/TM3; ci ey^R	(h.v.)
T(3;4)K117/TM3; ci ey^R	(h.v.)

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Genetic loci in the 9E-10B region in the **Drosophila melanogaster** X chromosome.

The following series of mutations mapped in the 9E-10B region were used: (1) 56 EMS induced mutations of different kinds (lethal, semilethal, delaying development, male and female sterility, hypomorphic lethals and vermilion). (2) 2 X-ray induced lethals (G3 and G6), (3) X-ray induced v mutations (P and B series in Table 1). All three groups of the mutations were obtained in this work. (4) Representatives of 1 to 21 loci (Geer et al. 1983), ny (Lindsley & Grell 1968). (5) sesB1 and sesB2, stress sensitive (Homyk et al. 1980). (6) l(1)M1 (Gateff, unpubl.). (7) l(1)HM21^{cs} (Mayoh & Suzuki 1973). (8) tu(1)Sz^{ts}, tumors in larvae (Rizki & Rizki 1980). (9) l(1)EM15 and l(1)EM16 (Lefevre, unpubl.). (10) Numerous mutations cited in Zhimulev et al. (1981).

All mutations were mapped in different subsections of the 9E-10B region using a set of chromosome rearrangements known for this region (Zhimulev et al. 1981, 1982). The mutations falling into the same cytological interval were tested for allelism by pairwise crosses between all members of this group.

The complementation analysis has shown that mutations obtained by different authors, and called by them differently, frequently fall into the same complementation groups. Mutations sesB1 and sesB2, for instance, appeared to be alleles to the l(1)S12 known before (Lefevre 1971), the v26 lethal (Geer et al. 1983) appeared to be allele of dsh (Lindsley & Grell 1968), and lethals of 18 locus (Geer et al. 1983) are alleles to the l(1)L4 (Lefevre 1971) as well as to the fs(1)M43$^{14-31}$ mutation of female sterility (Mohler 1977), and so on.

In the majority of cases (although not always), we tried to follow a nomenclature of earlier description. Loci described in the paper by Geer et al. (1983) and conditionally called by them 1 to 21 we propose to call according to the author's initials and number of corresponding locus, if it is not an allele of some locus known before, e.g., the locus 20 we call l(1)GLM20. Mutations of locus 3 appeared to be allele to fliG and locus 4 mutations are alleles to l(1)BP3, so these two are called as fliG and l(1)BP3, respectively.

Analysis of data of Table 1 shows that 32 loci are revealed in the 9E-10B region, including two new ones found in this study: the fs(2)BP2, a locus of female sterility and l(1)EM16.

Numerous v mutations give the only complementation group.

We failed to confirm Rizki & Rizki's (1980) conclusion that tu(1)Sz^{ts} is overlapped by Dp(1 → 2)v^{+63i}. These crosses were repeated thrice with simultaneous cytological control of the duplication and the results led us to the conclusion that the locus is mapped between proximal breakpoint of Dp(1 → 2)v^{+63i} and distal break of Df(1)N71, i.e., in the 10B1-3 region.
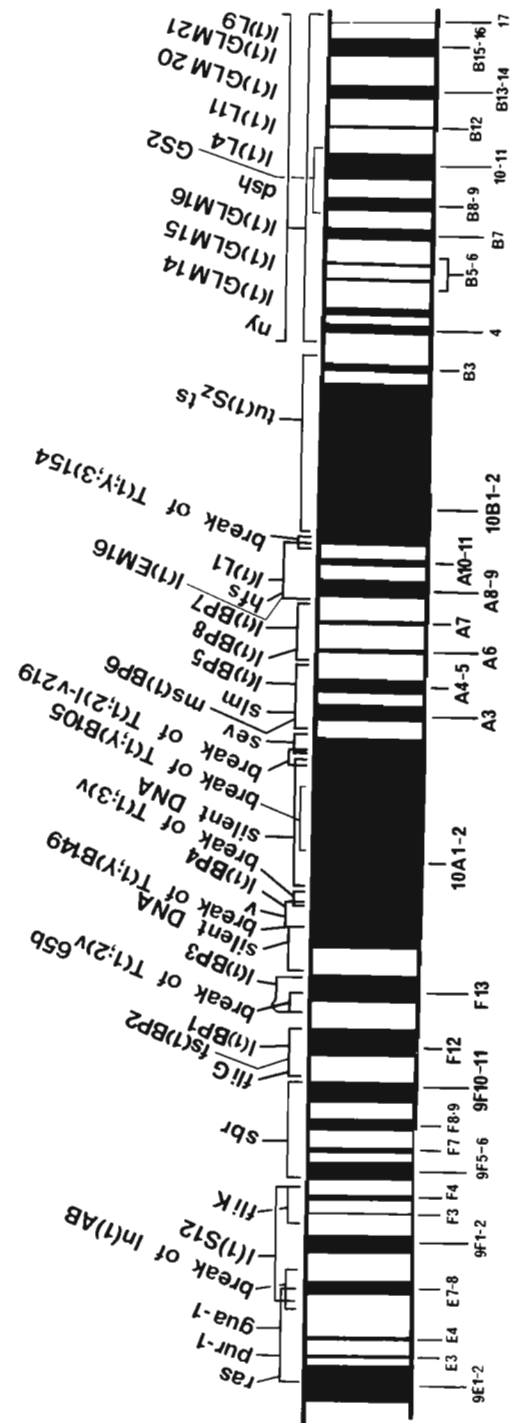
Figure 1. Cytogenetic map of the 9E-10B region of the **Drosophila melanogaster** X chromosome. Positions of gua1 and pur1 are taken from Johnson et al. (1979). GS2 locus was mapped according to data of in situ hybridization of glutamine synthetase DNA clone (Cagesse et al. 1986).

Summary cytogenetic map of the 9E-10B region is shown in Figure 1. Several types of band-gene relationships can be established in this region.

    1. Oligogene bands 10A1-2 and 9F12. The 10A1-2 band contains three genes: v, l(1)BP4 and sev and two zones of silent DNA comprising about 70% of recombinational length of the band (Zhimulev et al. 1981).

    The 9F12 contains three genes: fliG, fs(1)BP2, and l(1)BP1. In contrast to the 10A1-2 band, 9F12 does not contain a great amount of "silent" DNA since the genetic distance occupied by these genes is only 0.11 m.u., i.e., about 42 kb according to calculations.

    2. The 9F13 band contains the l(1)BP3 gene and some nonessential sequence broken by T(1;2)v^{+65b} translocation (Fig. 1).

    3. In the 10A3-5 and 10A8-11 intervals, it has been found that the number of genes exceeds slightly that of bands (Fig. 1): each pair of bands include three genes.

    4. In the region of two finest bands, 10A6 and 10A7, two genes were found; thus, this interval is the only case of 1 gene: 1 band relationship.

    5. The 10B1-3 interval is especially interesting. It includes the very large band 10B1-2 and the fine band 10B3. In spite of saturation of this region performed by Geer with coworkers (Geer et al. 1983), no loci in it has been revealed. Specification of the tu(1)Sz^{ts} locus location allowed it to be placed in this two band region. It is clear that at least one of the bands (10B1-2 or 10B3) consists fully of "silent" DNA. This conclusion, however, can be drawn with reservations, since the 10B1-17 is not well saturated. One may say the same about the 9E1-2 to 9F10-11 region, where we do not analyze band:gene relationship.

    Other problems of band-gene relationships are discussed in the review by Zhimulev & Belyaeva (1985).

References: Arking, R. 1971, Genetics 80:519-537; Bridges, C.B. 1938, DIS 9:121; Caggese, C. et al. 1986, Mol. Gen. Genet. 204:208-213; Geer, B.W. et al. 1983, J. Exp. Zool. 225:107-118; Harris, W.A. et al. 1976, J. Physiol. 256:415-439; Homyk, T. & D.E. Sheppard 1977, Genetics 87:95-104; _____ et al. 1980, Mol. Gen. Genet. 177:553-565; Johnson, M.M. et al. 1979, Mol. Gen. Genet. 174:287-292; Lefevre, G. Jr. 1969, Genetics 63:589-600; _____ 1971, Genetics 67:497-513; _____ & K.B. Wiedenheft 1974, DIS 51:83; Lindsley, D.L. & E.H. Grell 1968, Genetics Variations of **Drosophila melanogaster**, Carn. Inst. Wash. Publ. 627; Mayoh, H. & D.T. Suzuki 1973, Can. J. Genet. Cytol. 15:237-254; Mohler, J.D. 1977, Genetics 85:259-272; _____ & A. Carroll 1984, DIS 60:236-241; Nash, D. & F.C. Janca 1983, Genetics 105:957-968; _____ et al. 1981, Can. J. Genet. Cytol. 2:411-423; Rizki, T.M. & R.M. Rizki 1980, W. Roux's Archives 189:197-206; Zhimulev, I.F. & E.S. Belyaeva 1985, Biol. Zentralbl. 104:633-640; _____ et al. 1981, Chromosoma 82:25-40; _____ et al. 1982, DIS 58:210-214.

Table 1. Loci and mutations in the 9E1-2 to 10B17 region of **Drosophila melanogaster** X-chromosome.
Ref. = references at end of table. * = hs, heat temperature sensitive (29°C), cs = cold temperature sensitive (18°C). ** = absence of references means data of present paper. *** = probably sx of Bridges (1938). **** = according to photograph of M.J. Fahmi (pers. comm.).

| Loci | Alleles | | Ref. |
	Phenotype	Name	
ras	eyes color dark ruby	1,2,3,4,ras^V	1
	lethal	RC1,S27,EA140	2
gua-1 pur-1	auxotrophic mutants apparently involved in purine metabolism, fail to complement with ras-lethals	gua-1,pur-1,pur-2	2, 3
l(1)S12	lethal	l(1)S12	4
	cs,hs* semilethal (viability: at 18 and 29°C is 02-%, at 25°C - 10%), sensitive to mechanic stress	sesB1,sesB2	5; this paper
fli K	hs semilethal (viability: at 18 and 25°C is 25%, at 29°C - 0-2%), flightless	fli K	5; this paper
	lethal	l(1)HM25	6
		l(1)EM15	**
sbr	visible, small bristles	sbr	1
	hs lethal	l(1)ts403	7
	lethal	K2,K3,K4,K5,K6,K7,K11,24/45A,HM424	6
		v1,v14,v19,v107	8
	semilethal (viability: 11%), male sterility, female sterility against exposing lethals	msv7	8; this paper
fli G***	hs,cs semilethal (at 25°C viability is 20-60%, at 18° and 29°C - 1-2%), flightless, male fertility reduced, wings often apart and with additional veins	1,2,B186,dp224	5, 6, 9
		F403,F417	

fs(1)BP2	female sterility	F58,F129,F456,F469	
l(1)BP1	lethal	Q54,EA86	1, 4, 6
		G98,G101,171,dpS42	6
		v126, v348	8
		F412, F419	
	semilethal (4.4% of viability)	191	6
	visibile, absence of humeral bristles against exposing deficiencies	bir 336	6
l(1)BP3	lethal	163,167,183,dpS22	6
		v5,v40,v103,v172,v205,v207,v363	8
	semilethal (6% of viability)	F423	
v	vermilion eyes	1,2,48a,36f,0f,51c	1
		ESB,E37,E57,E63,E70,E76,E78,E82,E84, E107,E110,E118,E119,E124,E146,E154, E158,E160,E184,E195,dpG1,G57,G64,G73, G90,G100,G117,G118,G119,G121,G126, AM1,BN,DK,NK,NN,OS,tm1,tm2,2B-27,2B-37, 2B54,2B-157,2B-160,2B-162,2B-165,2B-195, 2B-206,2B-207,2B-236,2B-237,J9,J25,J27, J29,J28,dpZ1,dpZ2,dpZ7,dpG1,M4,M8,05, 06,74k,61j,s16,F364,s15-1,B1,F303,F308.	6
		F1,F2,F3,F4,F5,F6,F7,F8,F9,P30,P31,P32, P33,P34,P35,P36,F10,F11,F12,F13,F14,F15, F16,F17,B84,B85,B86,B126,B127,B150,B152, B153,B154	
l(1)BP4	lethal	E115,E128,E143,E147, dpG1,G50,G99,G130,J20	6
		v88,v301,v318,v370	8
		L68,allelism with others not tested	10
		F305,F404,F407,F411	
	hs semilethals showing csk**** phenotype (1) against exposing deficiencies and lethals	166,162,IE109,G92	6
		F436,F57,F69,F97,F101,F124,F128	
sev	absence of seventh eye rhabdomer, unsensitivity to UV-light	LY3	11
ms(1)BP6	male sterility	dpS53	6
		msv3,msv6,msv13,msv16,msv101, msv102,msv105,msv111	8
		F3, F5	
slm	small slim body, semilethal (30-100% of normal viability), delay in fly eclosion	1	1
		G94,G102,G138,G136,E148,E149	6
		F118,F150,F424,F427,F433	
l(1)BP5	lethal	L12	4
		G62,G67,G93,G95,G105,E112,E114,E120 G139,169,187,193,dp025,dpZ4,dpS445, J21,E54,G76,164	6
		v201,v202,v361,v372	8
		F409, F437	
	cs lethal	csHM21	12
	hs hypomorphic lethal (viability of homozygous ♀♀ ♀♀ and ♂♂ is almost normal; viability of hemizygous ♀♀ ♀♀ at 18 and 25°C - 20% at 29°C - 1%)	F230	
	hs hypomorphic lethal viability of homozygous ♀♀ ♀♀ at 18°C - 30%; at 25°C - 5%; at 29°C - 0%; viability of hemizygous ♀♀ ♀♀ is zero at three temperature	F439	
l(1)BP8	lethal	Q66	1, 4
		E62,E72,G97,153,174,194,HM4,HM26, HM445,dpZ3,dp05,dp024	6
		v145	8
		F431	

	cs hypomorphic lethal. Viability of homozygous ♀♀ and ♂♂ is normal at three temperatures; viability of hemizygous ♀♀: at 18°C - 0%; at 25°C - 30%; at 29°C - 100%; female sterility in homozygotes.	F99	
	hs hypomorphic lethal. Viability of homozygous ♀♀ and ♂♂ is normal at three temperatures; viability of hemizygous ♀♀: at 18°C - 100%; at 25°C - 5%; at 29°C - 0%.	F59	
1(1)BP7	lethal	L8	4
		G52,G65,E66,E67,E142,TE108,170	6
		v153	8
		F440, F100	
	lethal induced in MR^{h12} system	D41	
	hs semilethal	F60	
	hs hypomorphic lethal (viability of homozygous ♀♀ at 18°C - 30%, at 25°C - 60%, at 29°C - 0%; viability of hemizygous ♀♀ at 18°C - 15%, at 25 and 29°C - 0%).	F432	
	cs hypomorphic lethal (at three temperatures, viability of homozygous ♀♀ and ♂♂ is normal; viability of hemizygous ♀♀ at 18°C - 0%, at 25°C - 5%, at 29°C - 50%.	F313	
1(1)L1	lethal	L1	4
		v24,v68,v152	8
	lethal induced in MR^{h12} system	D40	4
hfs	haplo female sterile	no mutations	4, 13, 14
1(1)EM16	lethal	EM16	
tu(1)Sz^{ts}	pseudotumor formation at 26-29°C	tu(1)Sz^{ts}	15
ny	more than 90% of flies have notches on both margins of wings at 18°C and less than 40% at 25°C and 30°C	ny	1; this paper
1(1)GLM14	lethal	v7	8
1(1)GLM15	lethal	v16,v17,v18,v22,v64,v149,v212	8
1(1)GLM16	lethal	v4	8
dsh	Toracic hairs deranged, female sterile, males poor fertile	dsh	1, 6
	lethal	v26	8
1(1)L4	lethal	L4	4
		v12,v20,v48,v82,v109,v148,v223	8
	semilethal (9% of viability), male sterility	msv1,msN1	8
	hypomorphic lethal, female sterile, male fertility reduced	fs(1)M43$^{14-31}$	6, 16, 17
1(1)L11	lethal	L11, v55, v59, M1, G3, G6	4, 8; this paper
1(1)GLM20	lethal	v21	8
1(1)L9	lethal, allelism with others was not tested		4
1(1)GLM21	lethal	v73	8
	semilethal (viability - 30%), male sterility against exposing lethals	msv12, msv22	8

Table 1 References: **(1)** Lindsley & Grell 1968; **(2)** Nash et al. 1981; **(3)** Johnson et al. 1979; **(4)** Lefevre 1971; **(5)** Homyk et al. 1980; **(6)** Zhimulev et al. 1982; **(7)** Arking 1975; **(8)** Geer et al. 1983; **(9)** Homyk & Sheppard 1977; **(10)** Lefevre & Widenheft 1974; **(11)** Harris et al. 1976; **(12)** Mayoh & Suzuki 1973; **(13)** Lefevre 1969; **(14)** Zhimulev et al. 1981; **(15)** Rizki & Rizki 1980; **(16)** Mohler 1977; **(17)** Mohler & Carroll 1984.

BIBLIOGRAPHY ON **Drosophila hydei**

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This bibliography covers a vast deal of the literature on **D.hydei** of this century up to the middle of 1986. For the period before 1976, the list of references is more extensive than the ten year old list given by Hess (1976) in his review of the genetics of **D.hydei**. His work only contained about 40 entries. Unnecessary to say that some papers may have been overlooked, in particular those lacking the key word **D.hydei** in the title. I would like to apologize to the reader for eventual incorrect omissions.

ALANEN, M. 1985. Identification of a heat shock locus by whole mount electron microscopy of polytene chromosomes. Hereditas 103:39-46.

ALONSO, C. 1971. The effects of gibberellic acid upon developmental processes in **D.hydei**. Entomol. Exp. Appl. 14:73-82.

───. 1971. The effect of gibberellic acid on the development of **D.hydei**. DIS 46:135.

───. 1972. In situ hybridization of RNA synthesized in larval salivary glands of **D.hydei** under in vitro conditions. DIS 48:143.

───. 1972. The influence of molting hormone on RNA synthesis in isolated polytene nuclei of Drosophila. Dev. Biol. 28:372-381.

───. 1973. RNA metabolism during metamorphosis of **D.hydei**. J. Insect Physiol. 19:2135-2142.

─── & H.D. BERENDES. 1975. The locations of 5 S ribosomal RNA genes in **D.hydei**. Chromosoma 51:347-356.

───, P.J. HELMSING & H.D. BERENDES. 1974. A comparative study of in situ hybridization procedures using CRNA applied to **D.hydei** salivary gland chromosomes. Exp. Cell Res. 85:383-390.

ANANIEV, E.V. & V. BARSKY. 1982. A photographic map of **D.hydei** polytene chromosomes. Chromosoma 87:239-245.

BAUMAN, J.G.J., J. WIEGANT, P. VAN DUYN, N.H. LUBSEN, P.J.A. SONDERMEYER, W. HENNIG & E. KUBLI. 1981. Rapid and high resolution detection of in situ hybridisation to polytene chromosomes using fluorochrome-labeled RNA. Chromosoma 84:1-18.

BECK, H. 1972. The phenotype of X^{bb+}/O males in **D.hydei**. DIS 49:76.

───. 1973. Some observations on Y-chromosomal bobbed mutants in **D.hydei**. DIS 50:94.

───. 1974. An enhancer of bobbed in **D.hydei**. IVth Eur. Dros. Res. Conf., Umea, Sweden.

───. 1975. Zur Wirkung eines autosomales Gens auf den bobbed-Phänotyp bei **D.hydei**. Arch. Genetik. 48:69.

───. 1976. High frequency of Y-chromosomal marker loss in irradiated spermatocytes of **D.hydei**. Vth Eur. Dros. Res. Conf., Louvain-La-Neuve, Belgium.

───. 1976. New compound (I) chromosomes and the production of large quantities of X/O males in **D.hydei**. Genet. Res. 26:313-317.

───. 1976. High frequency of Y-chromosomal marker loss in irradiated spermatocytes of **D.hydei** (Abstr.). Vth Eur. Dros. Res. Conf., Louvain-La-Neuve, Belgium.

───. 1977. Etude des mutations bobbed (bb) et Minute (M) de **D.hydei**. Condensation of Thesis no. 1812, 32 pp, Univ. Geneva, Fac. Science.

───. 1978. The Y-chromosome of **D.hydei**: localization and isolation of genes by means of Y-fragments (Abstr.). Arch. Genet. 51:109.

───. 1981. Unstable magnification at the bobbed locus of **D.hydei**. VIIth Eur. Dros. Res. Conf., Oulu, Finland.

─── & F.M.A. VAN BREUGEL. 1975. Minute-type mutants in **D.hydei**. Arch. Genet. 48:128-147.

───, ─── & Z. SRDIC. 1979. A marked ring-Y chromosome in **D.hydei** with a new type of white variegation. Chromosoma 71:1-14.

───, ─── & B.J.M. VAN ZIJLL-LANGHOUT. 1985. Cloning of telomeric DNA sequences of **D.hydei** with a modified microdissection and microcloning procedure. Experientia 41:794-795.

─── & Z. SRDIC. 1978. Differential coloring of X and Y heterochromatin in mitosic chromosomes of **D.hydei** (Abstr.). Arch. Genet. 51:110.

─── & ───. 1979. Heterochromatin in mitotic chromosomes of **D.hydei**. Genetica 50:1-10.

BELEW, K. & T. BRADY. 1978. Changes in nonhistone chromosomal proteins correlated with induction of puff II-48C in **D.hydei** polytene salivary gland nuclei. Tex. J. Sci. 30:81-94.

─── & ───. 1978. Changes in nonhistone chromosomal proteins correlated with induction of puff II-48C in **D.hydei** polytene salivary gland nuclei (Abstr.). N.M. Acad. Sci. Bull. 18:35-36.

─── & ───. 1979. Tyrosine amino transferase - immuno - precipitation of a newly synthesized heat shock protein from **D.hydei** (Abstr.). J. Cell Biol. 83:170a.

─── & ───. 1981. Changes in phenol-soluble nuclear proteins correlated with puff induction in **D.hydei**. Cell Differ. 10:229-235.

─── & ───. 1981. Induction of tyrosine amino transferase by pyridoxine in **D.hydei**. Chromosoma 82:99-106.

─── & ───. 1981. Tyrosine amino transferase from **D.hydei** salivary glands: characterization, purification and antibody production. Insect Biochem. 11:239-245.

BERENDES, H.D. 1961. Observaties over de structuur van speekselklier-chromosomen van **D.hydei**. Genen en Phaenen 6:70-75.
———. 1963. The salivary gland chromosomes of **D.hydei Sturtevant**. Chromosoma 14:195-206.
———. 1963. Functional structures in cytoplasm and chromosomes of **D.hydei** salivary gland cells. Genetics Today 1:113.
———. 1963. Asynapsis in the salivary gland chromosomes of **D.hydei**. Genetica 33:288-300.
———. 1965. Salivary gland function and chromosomal puffing patterns in **D.hydei**. Chromosoma 17:35-77.
———. 1965. The induction of changes in chromosomal activity in different polytene types of cell in **D.hydei**. Dev. Biol. 11:371-384.
———. 1965. Gene homologies in different species of the repleta group of the genus D. I. Gene activity after temperature shocks. Genen en Phaenen 10:32-41.
———. 1965. Functional structures in the salivary glands of **D.hydei**. Arch. Nèerl. Zool. XVI:404-405.
———. 1966. The effect of temperature shocks in some related species of the genus Drosophila. DIS 41:168-169.
———. 1966. Gene activities in the Malpighian tubules of **D.hydei** at different developmental stages. J. Exp. Zool. 162:209-217.
———. 1966. Differential replication of male and female X-chromosomes in Drosophila. Chromosoma 20:32-43.
———. 1967. Aminoacid acid incorporation into giant chromosomes of **D.hydei**. DIS 42:102.
———. 1967. The hormone ecdysone as effector of specific changes in the pattern of gene activities of **D.hydei**. Chromosoma 22:274-293.
———. 1968. Electron microscopical mapping of giant chromosomes. DIS 43:115.
———. 1968. The effect of ecdysone analogues on the puffing pattern of **D.hydei**. DIS 43:145.
———. 1968. Differential patterns of gene activitty in relation to insect development (Abstr.). Proc. Int. Union Physiol. Sci. 6:154-155.
———. 1968. Factors involved in the expression of gene activity in polytene chromosomes. Chromosoma 24:418-437.
———. 1969. Induction and control of puffing. Ann. Embryol. Morphogen. Suppl. 1:153-164.
———. 1972. The control of puffing in **D.hydei**. In: Developmental studies on giant chromosomes, W. Beermann (ed.), Springer Verlag, Berlin.
———. 1973. Synthetic activity of polytene chromosomes. Int. Rev. Cytol. 35:61-116.
———, C. ALONSO, P.J. HELMSING, H.J. LEENDERS & J. DERKSEN. 1974. Structure and function in the genome of **D.hydei**. Cold Spring Harbor Sym. 38:645-654.
——— & W. BEERMANN. 1969. Biochemical activity of interphase chromosomes (polytene chromosomes). Handbook of Mol. Cytol. 15:500-519.
——— & J.B. BOYD. 1969. Structural and functional properties of polytene nuclei isolated from salivary glands of **D.hydei**. J. Cell Biol. 41:591-599.
——— & W.C. DE BRUIN. 1963. Submicroscopic structure of **D.hydei** salivary gland cells. Z. Zellforsch. 59:142-152.
——— & Th.K.H. HOLT. 1964. The induction of chromosomal activities by temperature shocks. Genen en Phaenen 9:1-7.
——— & ———. 1965. Differentiation of transplanted larval salivary glands of **D.hydei** in adults of the same species. J. Exp. Zool. 160:299-317.
——— & H.G. KEYL. 1967. Distribution of DNA in heterochromatin of polytene nuclei of **D.hydei**. Genetics 57:1-13.
——— & G.F. MEIJER. 1968. A specific chromosome element, the telomere of Drosophila polytene chromosomes. Chromosoma 25:184-197.
———, F.M.A. VAN BREUGEL & Th.K.H. HOLT. 1965. Experimental puffs in **D.hydei** salivary gland chromosomes. Chromosoma 16:35-47.
——— & E. WILLART. 1971. Ecdysone-related changes at the nuclear and cytoplasmic level of Malpighian tubule cells in Drosophila. J. Insect Physiol. 17:2337.
BISSELING, T., H.D. BERENDES & N.H. LUBSEN. 1976. RNA synthesis in puff 2-48 Bd after experimental induction in **D.hydei**. Cell 8:299-304.
BONACCORSI, S., S. PIMPINELLI & M. GATTI. 1981. Cytological dissection of sex chromosome heterochromatin of **D.hydei**. Chromosoma 84:391-403.
BOYD, J.B. 1968. Mass preparation of salivary glands from prepupae of **D.hydei**. DIS 43:181.
———. 1970. Characterization of an acid active deoxyribonuclease from the larval salivary gland of **D.hydei**. Bioch. Biophys. Acta 209:349-356.
———. 1970. Characterization of an alkaline active deoxyribonuclease from the prepupal salivary gland of **D.hydei**. Bioch. Biophys. Acta 209:339-348.
——— & H.D. BERENDES. 1968. Nuclei isolated from salivary glands of **D.hydei** - structural and functional aspects. (Abstr.). Genetics 60:164.
———, ——— & H. BOYD. 1968. Mass preparations of nuclei from the larval salivary glands of **D.hydei**. J. Cell Biol. 38:369-376.
——— & H. BOYD. 1970. Deoxyribonucleases of organs of **D.hydei** at the onset of metamorphosis. Biochem. Genet. 4:447-459.
——— & W.R. LOGAN. 1972. Developmental variations of a deoxyribonuclease in the salivary glands of **D.hydei** and **D.melanogaster**. Cell Differ. 1:107-118.
BRADY, T. & K. BELEW. 1978. Coenzyme (pyridoxine HCl) mediated activation of the tyrosine amino-transferase gene in **D.hydei** salivary polytene chromosomes. (Abstr.). J. Cell Biol. 79:343A.

_____ & _____. 1981. Pyridoxine induced puffing (II-48C) and synthesis of a 40KD protein in **D.hydei** salivary glands. Chromosoma 82:89-98.

BURKE, J.F., H.J. SINCLAIR, J.H. SANG & D. ISH-HOROWITZ. 1984. An assay for transient gene expressions in transfected Drosophila cells using [^{3}H] guanine incorporation. E.M.B.O.J. 3:2549-2554.

BUTTERWORTH, F.M. & H.D. BERENDES. 1974. Ecdysone-binding proteins in haemolymph and tissues of **D.hydei** larvae. J. Insect Physiol. 20:2195-2204.

CHATTERJEE, S.N. & A.S. MUKHERJEE. 1970. Puffing activity pattern in the X chromosome of **D.hydei** and their relation to male X hyperactivity and dosage compensation (Abstr.). Proc. Indian Sci. Congr. Assoc. 54:351-352.

_____ & _____. 1971. DNA replication pattern of the puffing sites in the X-chromosome of **D.hydei**. DIS 46:55.

_____ & _____. 1972. Chromosomal basis of dosage compensation in Drosophila. V. Puffwise analysis of gene activity in the X-chromosome of male and female **D.hydei**. Chromosoma 36:46-59.

CHILD, C.M. 1942. Differential dye reduction in ovaries of **Drosophila hydei**. Physiol. Zool. 15:13-29.

_____. 1943. Indophenol reaction and reduction in living ovaries of **D.hydei**. Physiol. Zool. 16:141-161.

CLAUSEN, R.E. 1923. Inheritance in **D.hydei**. I. White and vermilion eye-colors. Am. Nat. 57:52-58.

CLAGG, C.F. 1954. **D.hydei Sturtevant**. Proc. Hawaiian Ent. Soc. 15:271.

COUNCE, S.J. & N.R. RUDDLE. 1969. Strain differences in egg structure in **D.hydei**. Genetica 40:324-338.

DANGLI, A., C. GROND, P. KLOETZEL & E.K.F. BAUTZ. 1983. Heat shock puff 93D of **D.melanogaster**: accumulation of an RNP-specific antigen associated with giant particles of possible storage function. E.M.B.O.J.

DANIELI, G.A. 1969. Effetti della somministrazione dell'iprite azotata HN$_2$ sulla sintesi del DNA nelle ghiandole salivari di **D.hydei**. Atti dell'Instituto Veneto di Scienze, Letteri ed Arti CXXVII:337-350.

_____ & E. RODINO. 1967. Larval moulting cycle and DNA synthesis in **D.hydei** salivary glands. Nature 213:424-429.

_____ & _____. 1968. Variations of nucleic acid content in the salivary glands of **D.hydei** during late larval development. Experientia 24:89-90.

DERKSEN, J.W.M. 1975. The submicroscopic structure of synthetically active units in a puff of **D.hydei** giant chromosomes. Chromosoma 50:42-52.

_____. 1976. Ribonuclear protein formation at locus 2-48BC in **D.hydei**. Nature 263:438.

_____ & H.D. BERENDES. 1970. Polytene chromosome structure at the submicroscopic level. II. Length distribution of DNA molecules from polytene chromosomes of **D.melanogaster** and **D.hydei**. Chromosoma 31:468-477.

_____, _____ & E. WILLART. 1973. Production and release of a locus specific ribonucleoprotein product in polytene nuclei of **D.hydei**. J. Cell Biol. 59:661-668.

_____, _____ & _____. 1974. Modification of the nuclear envelope in polytene cells of **D.hydei** under the influence of vitamin B6. Cell Differ. 3:157-164.

_____ & E. WILLART. 1976. Cytochemical studies on RNP complexes produced by puff 2-48BC in **D.hydei**. Chromosoma 55:57-68.

DICKSON, E., J.B. BOYD & C.D. LAIRD. 1971. Sequence diversity of polytene chromosome DNA from **D.hydei**. J. Mol. Biol. 61:615-627.

DOANE, W.W. 1968. The amylase locus in **D.hydei**. DIS 43:143.

_____. 1969. Drosophila amylases and problems in cellular differentiation. In: Problems in Biology: DNA in development, pp. 73-109 (E.W. Hanly, ed.). Univ. Utah Press, Salt Lake City.

_____. 1971. Isoamylases in **D.hydei**: a system for the analysis of gene-specific puffing activity. DIS 47:100.

_____ & J. ABRAHAM. 1974. Starch degrading enzyme in **D.hydei** restricted to mature male accessory gland. Isoz. Bull. 7:38.

_____ & M.M. KOLAR. 1974. Molecular weight of the polypeptide chain of X-amylase from an Amy7 strain of **D.hydei**. Isoz. Bull. 7:32-33.

ECKES, B., B. GRUNERT, D. MISCHKE & M. SCHWOCHAU. 1980. Organization of the mitochondrial DNA in **D.melanogaster** and **D.hydei**. (Abstr.). Eur. J. Cell Biol. 22:125.

ELGIN, S.C.R. & J.B. BOYD. 1975. The proteins of polytene chromosomes of **D.hydei**. Chromosoma 51:135-145.

EMMERICH, H. 1969. Anreicherung von tritiummarkiertem Ecdyson in den Zellkernen der Speicheldrüsen von **D.hydei**. Exp. Cell Res. 58:261-270.

_____. 1970. Ecdysonbindende Proteinfraktionen in den Speicheldrüsen von **D.hydei**. Z. Vergl. Physiol. 68:385-402.

FIALA, Y. & J. NEUBERT. 1952. Der Erzeugung von Chromosomendislokationen durch Röntgenstrahlen in der Keimbahn und in den Geschlechtszellen verschiedenen Alters bei **D.hydei Sturt.** Chromosoma 4:577-584.

FOWLER, G.L. 1973. In vitro cell differentiation in the testes of **D.hydei**. Cell Differ. 2:33-42.

_____ & R. JOHANNISSON. 1976. Single-cyst in vitro spermatogenesis in **D.hydei**. In: Invertebrate tissue culture. E. Kurstak & K. Maramoroick (eds.), pp161-172.

_____ & J. UHLMANN. 1974. Single cyst in vitro spermatogenesis in **D.hydei**. DIS 51:81-83.

FRANZ, G., K.H. GLÄTZER & W. KUNZ. 1980. Evidence for the inactivity of the rRNA genes with an intervening sequence in **D.hydei**. (Abstr.). Eur. J. Cell Biol. 22:38.

_____ & W. KUNZ. 1981. Intervening sequences in ribosomal RNA genes and bobbed phenotype in **D.hydei**. Nature 292:638-640.

_____, _____ & C. GRIMM. 1983. Determination of the regions of rDNA involved in polytenization in salivary glands of **D.hydei**. Mol. Gen. Genet. 191:74-80.

FREI, H. 1972. Röntgen induced sex linked recessive lethal factors and mutation rates in **D.hydei**. (Abstr.). Arch. Genet. 45:99-100.

_____. 1973. New white alleles by recombination in **D.hydei**. DIS 50:165-166.

_____. 1973. Strahlenempfindlichkeit der meiotischen Prophase-Stadien in der Oogenese von **D.hydei**. Arch. Genet. 46:63-64.

_____. 1974. Unterschiedliche Strahlenempfindlichkeit in der Spermatogenese von **D.hydei**. Letalraten und die Bedeutung der arteigenen genetischen Organisation. Arch. Genet. 47:136-171.

_____. 1974. Radiation-induced dominant lethality in oocytes of **D.hydei**, with emphasis on the differential sensitivity of the immature stages in class B. Mut. Res. 24:125-134.

_____. 1975. Intragenic recombination at the white locus of **D.hydei**. Mol. Gen. Genet. 142:105-117.

_____. 1976/1977. Mutation, recombination and genetic organization in **D.hydei**, compared to other Drosophila species. Arch. Genet. 49/50:42-69.

_____. 1978. Chromosomenpaarung in der achiasmatischen Meiose von **D.hydei**-Männchen. Arch. Genet. 51:246.

_____. 1979. Gynandromorphs and XO males from unstable chromosome transmission in **D.hydei**. VIth Eur. Dros. Res. Conf., Kupari, Yugoslavia.

_____. 1979. The "collochore" function of the nucleolar organizing regions in meiosis of male **D.hydei**. VIth Eur. Dros. Res. Conf., Kupari, Yugoslavia.

_____ & H. BECK. 1976. Meiosis in **D.hydei** males with a detached X-chromosome. (Abstr.). Vth Eur. Dros. Res. Conf., Louvain-La-Neuve, Belgium.

_____ & _____. 1978. Eine unbestendiges X.Y^S-Chromosom von **D.hydei** führt zu Gynandern und XO-Männchen.   Arch. Genet. 51:42-43.

_____ & F.M.A. VAN BREUGEL. 1982. Drosophila species: new mutants. DIS 58:191-195.

_____ & A. SOURE-LEUTHOLD. 1976. Development of clones within the tergum anlage of **D.hydei**. Vth Eur. Dros. Res. Conf., Louvain-La-Neuve, Belgium.

GEER, B.W. & B.C. DOWNING. 1972. Changing in lipid and protein synthesis during spermatozoan development and thoracic tissue maturation in **D.hydei**. Wilh. Roux's Arch. 170:83-89.

_____, D.V. MARTENSEN, B.C. DOWNING & G.S. MUZYKA. 1972. Metabolic changes during spermatogenesis and thoracic tissue maturation in **D.hydei**. Dev. Biol. 28:390-406.

GLÄTZER, K.H. 1973. The status of Drosophila "pseudoneohydei". DIS 50:47.

_____. 1975. Visualization of gene transcription in spermatocytes of **D.hydei**. Chromosoma 53:371-375.

_____. 1979. Lengths of transcribed rDNA repeating units in spermatocytes of **D.hydei**: only genes without an intervening sequence are expressed. Chromosoma 75:161-175.

_____. 1979. The transcribed rDNA repeating units of all three nucleolus organizers of **D.hydei** are homogenous in size despite a 5.5 hB intervening sequence in one of the nucleolus organizers. Hoppe Seyler's Z. Physiol. Chem. 360:267.

_____. 1984. Preservation of nuclear RNP antigens in male germ cell development of **D.hydei**. Mol. Gen. Genet. 196:236-243.

_____ & P.M. KLOETZEL. 1985. Antigens of cytoplasmic RNP particles of **D.melanogaster** can be localized on distinct Y chromosomal structures in spermatocytes of **D.hydei**. DIS 61:84.

_____ & G.F. MEIJER. 1981. Morphological aspects of the genetic activity in primary spermatocyte nuclei of **D.hydei**. Biol. Cell 41:165-172.

_____, R. RENKAWITZ-POHL & W. KUNZ. 1980. Heterogeneity in rRNA lengths between different nucleolus organizers in **D.hydei**. (Abstr.). Eur. J. Cell Biol. 22:7.

GLOOR, H. 1951. Kältepolyploidie in Ganglienzellen von **D.hydei**. Rev. Suisse Zool. 54:520-521.

_____ & H.R. KOBEL. 1966. Antennepedia (ss^{Anp}), eine homoeotische Mutante bei **D.hydei Sturt**. Rev. Suisse Zool. 73:229-252.

_____ & H. STAIGER. 1954. Lethal-polyploid - a polyploid gene in **D.hydei**. J. Heredity 45:289-293.

_____, F.M.A. VAN BREUGEL & W.S. VOLKERS. 1967. Some observations on variegation in Drosophila. Genetica 38:95-114.

GREGG, T.G. 1957. The production of attached-X chromosomes in **D.hydei**. Studies in the genetics of Drosophila, Univ. Texas Publ. 5721:238-245.

_____. 1963. Nondisjunction in **D.hydei**. (Abstr.). Proc. 11th Int. Congr. Genet., The Hague 1:128.

_____. 1978. Crossing over and nondisjunction in interspecific hybrids between **D.hydei** and **neohydei**. Genetics 88(suppl.):33-34.

_____ & A.L. ALLENSPACH. 1971. Fine structural analysis of spermatocyte nuclei in **D.hydei**. (Abstr.). Genetics 68(suppl.):25.

_____ & J.W. DAY. 1965. Nondisjunction of the X-chromosome in females of **D.hydei**. Genetica 36:172-182.

_____ & L.A. SMUCKER. 1965. Pteridines and gene homologies in the eye color mutants of **D.melanogaster** and **D.hydei**. Genetics 52:1023-1034.

GRIMM, C. & W. KUNZ. 1980. Disproportionate rDNA replication does occur in diploid tissue in **D.hydei**. Mol. Gen. Genet. 180:23-26.

_____, _____ & G. FRANZ. 1984. The organ-specific rRNA gene number in **D.hydei** is controlled by sex heterochromatin. Chromosoma 89:48-54.

GROND, C.J. & J. DERKSEN. 1983. The banding pattern of the salivary gland chromosomes of **D.hydei**. Eur. J. Cell Biol. 30:144-148.

———, ——— & G.J. BRAKENHOFF. 1982. The banding pattern of the segment 46A-48C in **D.hydei** polytene chromosomes as studied by confocal scanning light microscopy. Exp. Cell Res. 138:458-461.

———, H.N. LUBSEN & H. BECK. 1982. Recombination frequency and DNA content of the distal part of the second chromosome of **D.hydei Sturtevant**. Experientia 38:328-329.

———, F.P.A.M.N. PETERS, J.W.M. DERKSEN & M. VAN DER PLOEG. 1983. Identification of the heat shock band 2-48B of **D.hydei** and determination of its haploid DNA content. Eur. J. Cell Biol.

———, R.G.J. RUTTEN & W. HENNING. 1984. Ultrastructure of the Y chromosomal lampbrush loops in primary spermatocytes of **D.hydei**. Chromosoma 89:85-95.

———, I. SIEGMUND & W. HENNING. 1983. Visualization of a lampbrush loop-forming fertility gene in **D.hydei**. Chromosoma 88:50-56.

GUNDELFINGER, E.D. 1983. Interaction of nucleic acid with the DNA-dependent RNA polymerases of Drosophila. F.E.B.S. Lett. 157:133-138.

——— & H. STEIN. 1982. RNA polymerase I from **D.hydei** prepupae: purification and partial characterization. F.E.B.S. Lett. 142:7-11.

HACKSTEIN, J.H.P., O. LEONCINI, H. BECK, G. PEELEN & W. HENNIG. 1982. Genetic fine structure of the Y chromosome of **D.hydei**. Genetics 101:257-277.

HANNA, P.J. 1981. Inhibition of Y-chromosome loop formation in primary spermatocyte nuclei of **D.hydei** by trimethylphosphate. Experientia 37:951-952.

HARISANOVA, N.T. & K.H. RALCHEV. 1985. The effect of 10°C temperature on the embryonic development of **D.hydei**. DIS 61:88.

HAWLEY, R.S. & K.D. TARTOF. 1983. The ribosomal DNA of **D.melanogaster** is organized differently from that of **D.hydei**. J. Mol. Biol. 163:499-503.

HELMSING, P.M. & H.D. BERENDES. 1971. Induced accumulation of nonhistone proteins in polytene nuclei of **D.hydei**. J. Cell Biol. 50:893-896.

——— & O. VAN EUPEN. 1972. Gene activity and histone patterns in salivary gland nuclei and chromatin of **D.hydei**. DIS 48:121-122.

HENNIG, I. 1978. Vergleichend-zytologische und -genetische Untersuchungen am Genom der Fruchtfliegen-Arten **D.hydei, neohydei** and **eohydei** (Diptera: Drosophilidae). Entomol. Germ. 4:211-223.

HENNIG, W. 1967. Untersuchungen zur struktur und Funktion des Lampenbürsten-Y-Chromosoms in der Spermatogenese von Drosophila. Chromosoma 22:294-357.

———. 1968. Ribonucleic acid synthesis of the Y-chromosome of **D.hydei**. J. Mol. Biol. 38:227-239.

———. 1972. Highly repetitive DNA sequences in the genome of **D.hydei**. I. Preferential localization in the X chromosomal heterochromatin. J. Mol. Biol. 71:407-417.

———. 1973. A remarkable status of **Drosophila pseudoneohydei**. DIS 50:121.

———. 1977. Gene interactions in germ cell differentiation. Adv. Enzyme Regul. Biosynth. 15:363-371.

———. 1978. The lampbrush Y chromosome of the fruit fly species **D.hydei** (Diptera: Drosophilidae). Entomol. Germ. 4:200-210.

———. 1985. Y chromosome function and spermatogenesis in **D.hydei**. Adv. Genet. 23:179-234.

———, I. HENNIG & O. LEONCINI. 1974. Some observations on spermatogenesis of **D.hydei**. DIS 51:127.

———, ——— & H. STEIN. 1970. Repeated sequences in DNA of Drosophila and their localization in giant chromosomes. Chromosoma 32:31-63.

———, P.HUIJSER, P. VOGT, H. JAECKLI & J.E. EDSTROEM. 1983. Molecular cloning of microdissected lampbrush loop DNA sequences of **D.hydei**. E.M.B.O. J. 2:1741-1746.

———, B. LINK & O. LEONCINI. 1975. The location of the nucleolus organizer regions in **D.hydei**. Chromosoma 51:57-63.

——— & B. MEER. 1971. Reduced polyteny of ribosomal RNA cistrons in giant chromosomes of **D.hydei**. Nature New Biol. 233:70-72.

———, G.F. MEIJER, I. HENNIG & O. LEONCINI. 1973. Structure and functions of the Y chromosome of **D.hydei**. Cold Spring Harbor Symp. Quant. Bio.. 38:673-683.

HERMANS, L. 1979. Purification of mitochondrial NADH dehydrogenase from **D.hydei** and comparison with the "heat-shock" polypeptides. Bioch. Biophys. Acta 567:125-134.

HERRNSTADT, C. & W. KUNZ. 1980. Comparison of rDNA restriction patterns in compensating and non-compensating organs in **D.hydei**. (Abstr.). Eur. J. Cell Biol. 22:7.

——— & M. SCHWOCHAU. 1980. Identification of functional nucleotide sequences of the Y-chromosome of **D.hydei**. (Abstr.). Eur. J. Cell Biol. 22:49.

——— & ———. 1981. Demonstration of Y-specific RNA in the primary spermatocytes of **D.hydei** by "in situ" DNA/RNA-transcript hybridization. (Abstr.). Eur. J. Cell Biol. 24:331.

HESS, O. 1964. Struktur-Differenzierungen in Y-Chromosom von **D.hydei** und ihre Beziehungen zu Genaktivitäten   I. Mutanten der Funktionsstrukturen. Verh. Dtsch. Ges. 1964:156-163.

———. 1965. Struktur-Differenzierungen im Y-Chromosom von **D.hydei** und ihre Beziehungen zu Gen-Aktivitäten. III. Sequenz und Lokalisation der Schleifen-Bildungsorte. Chromosoma 16:222-248.

———. 1965. The effect of X-rays on the functional structures of the Y chromosome in spermatocytes of **D.hydei**. J. Cell Biol. 25:169-173.

———. 1966. Funktionelle und strukturelle Organisation der Lampenbursten-Chromosomen. In: P. Sitte (Herausgeb.). Probleme der biologischen Reduplikation. Springer Verlag, pp. 29-54.

———. 1967. Morphologische Variabilität der chromosomalen Funktionsstrukturen in den Spermatocytenkernen von Drosophila Arten. Chromosoma 21:429-445.

———. 1967. Complementation of genetic activity in translocated fragments of the Y chromosome in **D.hydei**. Genetics 56:283-295.

———. 1968. Genetische Aktivität in translozierten Fragmenten des Y Chromosoms von **D.hydei**. Verhandl. Deutsch. Zool. Ges., Heidelberg, Akad. Verlagsges. Geest & Portig K-6, Leipzig.

———. 1968. The function of the lampbrush loops formed by the Y chromosome of **D.hydei** in spermatocyte nuclei. Mol. Gen. Genet. 103:58-71.

———. 1969. Veränderungen der Chromosomenstruktur und ihre Beziehungen zur genetischen Funktion. Rev. Suisse de Zool. 76:603-641.

———. 1969. Changes of the chromosomal structure and their relations to gene activity. Exc. Med. Int. Congr. Series 191. Third Int. Conf. Congenital Malformations, September.

———. 1969. Genetic activities of the Y chromosome in Drosophila. Ann. Embryol. Morphogen., Suppl. 1:165-176.

———. 1970. Genetic function correlated with unfolding of lampbrush loops by the Y chromosome in spermatocytes of **D.hydei**. Mol. Gen. Genet. 106:328-346.

———. 1970. Independence between modification of genetic position effects and formation of lampbrush loops by the Y chromosome of **D.hydei**. Mol. Gen. Genet. 107:224-242.

———. 1971. Lampenbürstenchromosomen. In: Handb. allg. Pathol. II/2, pp. 215-281. Springer Verlag, Berlin.

———. 1973. Genetic regulation of cell differentiation as revealed by Y chromosome lampbrush loops in **D.hydei**. In: J. Wahrmann & K.R. Lewis (eds.), Chromosomes today 4:117-125. John Wiley & Sons, New York.

———. 1974. Local structural variations of the Y chromosome of **D.hydei** and their correlation to genetic activity. Cold Spring Harbor Symp. Quant. Biol. 38:663-671.

———. 1975. De genetische regulatie van de celdifferentiatie, toegelicht aan de hand van de spermiogenese van **D.hydei**. In: Ontwikkelingsbiologie, Biologische Raad Reeks, pp. 169-179. Wageningen, Pudoc.

———. 1975. Funktion der Fertilitätsfaktoren auf dem Y-Chromosom von Drosophila. In: Rensing, L., H.H. Trepte & G. Birukow (Herausgeb.). Regulationsmechanismen der Genaktivität und Replikation bei Riesenchromosomen. Nachricht. Akad. Wiss. Göttingen II, Math.-Phys. Klasse 11:202-206.

———. 1975. Y-linked factors affecting male fertility in **D.melanogaster** and **D.hydei**. In: R. King (ed.). Handbook of Genetics 3:747-756, Plenum Publ. Co., New York.

———. 1976. Genetics of **D.hydei Sturtevant**. In: M. Ashburner & E. Novitski (eds.). The genetics and biology of Drosophila, Vol. 1c:1343-1363. Acad. Press, London.

———. 1981. Lampbrush chromosomes. In: The genetics and biology of Drosophila, Vol. 2d:1-38, M. Ashburner & T.R.F. Wright (eds.). Academic Press, London.

——— & M.M. GREEN. 1965. Report of new mutants of **D.hydei**. DIS 40:37-39.

——— & G.F. MEIJER. 1963. Chromosomal differentiation of the lampbrush type formed by the Y chromosome in **D.hydei** and **D.neohydei**. J. Cell Biol. 16:527-540.

——— & ———. 1963. Artspezifische funktionelle Differenzierungen der Y-Heterochromatins bei Drosophila-Arten der **D.hydei**-Subgruppe. Port. Act. Biol. A VII:29-46.

——— & ———. 1968. Genetic activities of the Y chromosome in Drosophila during spermatogenesis. Adv. Genetics 14:171-223.

HOLT, T.K.H. 1970. Local protein accumulation during gene activation. I. Quantitative measurements on dye binding capacity at subsequent stages of puff formation in **D.hydei**. Chromosoma 32:64-78.

———. 1971. Local protein accumulation during gene activation. II. Interferometric measurements of the amount of solid material in temperature induced puffs of **D.hydei**. Chromosoma 32:428-435.

——— & H.D. BERENDES. 1965. Experimental puffs on **D.hydei** polytene chromosomes, induced by temperature shocks. DIS 40:59.

——— & A.M.C. KUIJPERS. 1972. Induction of chromosome puffs in **D.hydei** salivary glands after inhibition of RNA synthesis by alpha-amanitin. Chromosoma 37:423-432.

——— & ———. 1972. Effects of alpha-amanitin on nucleolar structure and metabolism in **D.hydei**. Experientia 28:899-900.

HOWLAND, R.B., E.A. CLANCY & B.P. SONNENBLICK. 1937. Transplantation of wildtype and vermilion eye disks among four species of Drosophila. Genetics 22:434-442.

HUBBY, J.L. & L.H. THROCKMORTON. 1960. Evolution and pteridine metabolism in the genus Drosophila. PNAS USA 46:65-78.

HULSEBOS, T.J.M., J.H.P. HACKSTEIN & W. HENNIG. 1983. Involvement of Y chromosomal loci in the synthesis of **D.hydei** sperm proteins. Dev. Biol. 100:238-243.

———, ——— & ———. 1984. Lampbrush loop-specific protein of **D.hydei**. PNAS USA 81:3404-3408.

IMBERSKI, R.B. 1971. Isozymes of aldehyde oxidase in **D.hydei**. (Abstr.). Genetics 68:Suppl. 30.

_____ & M. OLDS. 1973. Electrophoretic analysis of alcohol dehydrogenase and octanol dehydrogenase in **D.hydei** and related species. DIS 50:95.

_____ & C. STROMMEN. 1972. Developmental changes in alcohol dehydrogenase activity in **D.hydei**. DIS 48:74.

IZQUIERDO, M., C. ARRIBAS & C. ALONSO. 1981. Isolation of a structural gene mapping to subregions 63F of **D.melanogaster** and 90B of **D.hydei** polytene chromosomes. Chromosoma 83:353-366.

KALISCH, W.-E. 1982. Electron microscopic mapping using surface-spread polytene chromosomes. Genetica 60:21-24.

_____. 1982. Chromosome spreading of salivary glands and Malpighian tubules in **D.hydei**. DIS 58:85-87.

_____, G. SCHWITALLA & T. WHITMORE. 1986. Computerized electron microscopic maps of surface-spread polytene chromosomes. II. Chromosome 4 in **D.hydei**. Cytobios 45:185-194.

_____, _____ & _____. 1986. Electron microscopic band-interband pattern of the X chromosome in **D.hydei**. Chromosoma 93:387-392.

_____, T. WHITMORE & G. SCHWITALLA. 1985. Electron microscopic map of surface-spread polytene chromosomes in **D.hydei**. Chromosoma 92:265-272.

KAMBYSELLIS, M.P. 1968. Comparative studies of oogenesis and egg morphology among species of the genus Drosophila. Univ. Texas Publ. 6818:71-92.

KAYANO, H. 1976. Linkage of esterase isozyme genes and leucine amino-peptidase isozyme gene, and inversion in the chromosome of **D.hydei**. (Abstr.). Jap. J. Genet. 51:417.

_____. 1978. Linkage disequilibrium between esterase loci of **D.hydei**. (Abstr.). Jap. J. Genet. 53:209-214.

_____. 1980. Genetic variation in natural populations of **D.hydei**-esterase and LAP genes. (Abstr.). Jap. J. Genet. 55:466.

_____ & A. KAYANO. 1979. A sterility gene of **D.hydei**. (Abstr.). Jap. J. Genet. 54:443.

_____ & _____. 1981. The effect on recombination of genes due to heterozygous reciprocal translocation in **D.hydei**. (Abstr.). Jap. J. Genet. 56:610.

KLAGES, G. & H. EMMERICH. 1978. Binding and activation of C18 juvenile hormone in larvae of **D.hydei**. (Abstr.). Gen. Comp. Endocrinol. 34:115.

_____ & _____. 1979. Juvenile hormone binding proteins in the haemolymph of third instar larvae of **D.hydei**. Insect Biochem. 9:23-30.

_____ & _____. 1979. Juvenile hormone metabolism and juvenile hormone esterase titer in hemolymph and peripheral tissues of **D.hydei**. J. Comp. Physiol. 132B:319-325.

_____, _____ & M.G. PETER. 1980. High-affinity binding sites for juvenile hormone I in the larval integument of **D.hydei**. Nature 286:282-283.

KLOETZEL, P.M., E. KNUST & M. SCHWOCHAU. 1981. Analysis of nuclear proteins in primary spermatocytes of **D.hydei**: the correlation of nuclear proteins with the function of the Y chromosomal loops. Chromosoma 84:67-86.

KOBEL, H.R. 1979. Gene content of the dot chromosome of **D.hydei**. VIth Eur. Dros. Res. Conf., Kupari, Yugoslavia.

_____ & H. GLOOR. 1971. Drosophila species: new mutants, **D.hydei**. Report of H. Gloor. DIS 47:47-52.

_____ & J.J. VAN DEN BOSCH. 1970. Abnormal-abdomen bei **D.hydei**. Genetica 41:119-140.

KNUST, E., P.M. KLOETZEL & M. SCHWOCHAU. 1979. Isolation and characterization of ribonucleoprotein particles from nuclei of the primary spermatocytes of **D.hydei**. (Abstr.). Hoppe Seyler's Z. Physiol. Chem. 360:305.

KONINKX, J.F.J.G. 1976. Protein synthesis in salivary glands of **D.hydei** after experimental gene induction. Biochem. J. 158:623.

KUNZ, W. 1980. Chromosome structure and functions in gametogenesis, with special consideration of insect oogenesis and spermatogenesis. Symp. Roy. Entomol. Soc. London 10:51-64.

_____. 1981. The "ribosomal insertion" in **D.hydei**: A mobile genetic element? (Abstr.). Eur. J. Cell Biol. 24:335.

_____ & K.H. GLÄTZER. 1979. Similarities and differences in the gene structure of the three nucleoli of **D.hydei**. (Abstr.). Hoppe Seyler's Z. Physiol. Chem. 360:313.

_____, C. GRIMM & G. FRANZ. 1981. Autonome Steuerung der rRNA-Genzahl bei der Organdifferenzierung von **D.hydei**: eine Abweichung von der Regel der Genomkonstanz. Verl. Dtsch. Zool. Ges. 1981:127-133.

_____, G. PETERSEN, R. RENKAWITZ-POHL, K.H. GLÄTZER & M. SCHÄFER. 1981. Distribution of spacer length classes and the intervening sequence among different nucleolus organizers in **D.hydei**. Chromosoma 83:145-158.

_____ & U. SCHÄFER. 1975. Unabhängige Kontrolle der rDNA-Replikation in Y-Chromosom von **D.hydei**. In: Rensing, L., H.H. Trepte & G. BIRUKOW (Herausgeb.). Regulationsmechanismen der Genaktivatät und Replikation bei Riesenchromosomen. Nachricht. Akad. Wiss. Göttingen, II, Math.-Phys. Klasse II:67-69.

_____ & _____. 1975. Variations in the ribosomal DNA content of the Y chromosome in **D.hydei**. In: Abstracts 10th Int. Congress F.E.B.S. 303, Paris.

_____ & _____. 1975. Schwankungen in Y chromosomalen r-DNA gehalt bei **D.hydei**. Verh. Dtsch. Zool. Ges.:115. G. Fischer, Stuttgart.

_____ & _____. 1976. Variations in the number of the Y chromosomal rRNA genes in **D.hydei**. Genetics 82:25-34.

LASSAK, H.F., W. NILLEN & M. SCHWOCHAU. 1980. Postmeiotic tubulin synthesis is controlled by the activity of the Y-chromosome in the meiotic prephase of **D.hydei**. (Abstr.). Eur. J. Cell Biol. 22:45.

_____, P.M. KLOETZEL, M. SCHWOCHAU & K.H. GLÄTZER. 1979. RNA synthesis, RNA storage and translation products in the spermatogenesis of **D.hydei**. (Abstr.). Hoppe Seyler's Z. Physiol. Chem. 360:314.

LEENDERS, H.J. 1971. Temperature induced puffs in Drosophila: their possible physiological origin. DIS 46:64.

———— & & P.J.A. BECKERS. 1973. The effect of changes in the respiratory metabolism upon genome activity. A correlation between induced gene activity and an increase in activity of a respiratory enzyme. Chromosoma 3:433-444.

———— & H.D. BERENDES. 1972. The effect of changes in the respiratory metabolism upon genome activty in Drosophila. Chromosoma 37:433-444.

————, ————, P.J. HELMSING, J. DERKSEN & J.F.J.G. KONINKZ. 1974. Nuclear-mitochondrial interactions in the control of mitochondrial respiratory metabolism. Sub-Cell Biochem. 3:119-147.

————, J. DERKSEN, P.M.J.M. MAAS & H.D. BERENDES. 1973. Selective induction of a giant puff in **D.hydei** by vitamin B6 and derivatives. Chromosoma 41:447-460.

————, A. KEMP., J.F.J.G. KONINKX & J. ROSING. 1974. Changes in cellular ATP, ADP and AMP levels following treatments affecting cellular respiration and the activity of certain nuclear genes in Drosophila salivary glands. Exp. Cell Res. 86:25-30.

LEENDERS, J.F.J.G. & L.M. BIRT. 1975. A correlation between newly induced gene activity and an enhancement of mitochondrial enzyme activity in the salivary glands of Drosophila. Exp. Cell Res. 92:275-282.

LEONCINI, O. 1977. Temperatursensitieve Mutanten im Y-Chromosom von **D.hydei**. Chromosoma 63:329-357.

LEWIS, M., P.J. HELMSING & M. ASHBURNER. 1975. Parallel changes in puffing activity and patterns of protein synthesis in salivary glands of Drosophila. PNAS USA 72:3604-3608.

LIEBRICH, W. 1981. In vitro differentiation of isolated single spermatocyte cysts of **D.hydei**. (Abstr.). Eur. J. Cell Biol. 24:335.

————. 1981. In vitro spermatogenesis in Drosophila. I. Development of isolated spermatocyte cysts from wild-type **D.hydei**. Cell Tissue Res. 220:251-262.

LIFSCHYTZ, E. 1974. Genes controlling chromosome activity. An X-linked mutation affecting Y-lampbrush loop activity in **D.hydei**. Chromosoma 47:415-427.

————. 1979. A procedure for the cloning and identification of Y-specific middle repetitive sequences in **D.hydei**. J. Mol. Biol. 133:267-277.

————, D. HAREVEN, A. AZRICH & H. BRODSLEY. 1983. DNA clones and RNA transcripts of four lampbrush loops from the Y chromosome of **D.hydei**. Cell 32:191-199.

LUBSEN, N.H. & P.J.A. SONDERMEIJER. 1978. The products of the "heat-shock" loci of **D.hydei**. Correlation between locus 2-36A and the 70.000 MW "heat-shock" polypeptide. Chromosoma 66:115-125.

————, ————, M. PAGES & C. ALONSO. 1978. In situ hybridization of nuclear and cytoplasmic RNA to locus 2-48BC in **D.hydei**. Chromosoma 65:199-212.

MARKOV, T.A. 1985. A comparative investigation of the mating system of **Drosophila hydei**. Anim. Behav. 33:775-781.

MEIJER, G.F. 1963. Die Funktionsstrukturen des Y-Chromosoms in den Spermatocytenkernen von **D.hydei**, **D.neohydei**, **D.repleta** und einige anderen Drosophila-Arten. ————————————?

————. 1968. Spermiogenese in normale und Y-defizienten Männchen von **D.melanogaster** und **D.hydei**. Z. Zellforsch. Micr. Anat. 84:141-175.

————. 1969. Experimental studies on spermiogenesis in Drosophila. Genetics (Suppl.) 61:79-92.

———— & W. HENNIG. 1974. The nucleolus in primary spermatocytes of **D.hydei**. Chromosoma 46:121-144.

———— & ————. 1974. Molecular aspects of the fertility factors in Drosophila. In: The functional anatomy of the spermatozoon. Ed. Afzelius, B.A. Oxford, New York.

———— & O. HESS. 1965. Strukturdifferenzierungen im Y-Chromosom von **D.hydei** und ihre Beziehungen zu Genaktivitaten. II. Effekt der RNS-Synthese-Hemmung durch Actinomycin. Chromosoma 16:249-270.

————, ———— & W. BEERMAN. 1961. Phasenspezifische Funktionsstrukturen in Spermatocytenkerne von **D.melanogaster** und ihre Abhängigkeit vom Y-Chromosomen. Chromosoma 12:676-716.

MULDER, M.P., P. VAN DUIJN & H. GLOOR. 1968. The replicative organization of DNA in polytene chromosomes of **D.hydei**. Genetica 39:385-428.

NATHANSON, M.A. & A.L. ALLENSPACH. 1971. Electron microscopic analysis of nuclear structures found in primary spermatocytes of **D.hydei**. (Abstr.). Amer. Zool. 11:685.

NELLEN, W. 1979. Male fertility in **D.hydei** correlated to storage of a specific mRNA. (Abstr.). Hoppe Seyler's Z. Physiol. Chem. 360:333.

NÖTHIGER, R. 1976. Spermatogenesis in larval testes of **D.hydei** cultured in vivo. Wilh. Roux's Arch. 180:R1.

PATTERSON, J.T. & W.S. STONE. 1952. Evolution in the genus Drosophila. Macmillan, New York.

PETERS, F.P.A.M.N., C.J. GROND, P.J.A. SONDERMEIJER & N.H. LUBSEN. 1982. Chromosomal arrangement of heat shock locus 2-48B in **D.hydei**. Chromosoma 85:237-249.

————, N.H. LUBSEN & P.J.A. SONDERMEIJER. 1980. Rapid sequence divergence in a heat shock locus of Drosophila. Chromosoma 81:271-280.

POELS, C.L.M. 1970. Time sequence in the expression of various developmental characters induced by ecdysterone in **D.hydei**. Dev. Biol. 23:210-225.

————. 1971. Changes in the function of salivary gland cells of Drosophila induced by the moulting hormone ecdyson. (Abstr.). IInd Eur. Dros. Res. Conf., Zurich, Switzerland.

_____. 1972. Functional and structural changes in Drosophila salivary gland cells triggered by ecdysone. (Abstr.). Neth. J. Zool. 22:217.

_____, A. DE LOOF & H.D. BERENDES. 1971. Functional and structural changes in Drosophila salivary gland cells triggered by ecdysterone. J. Insect Physiol. 17:1717-1729.

RALCHEV, K.H., N.T. HARISANOVA & G.H. KONSTANTINOV. 1978. Phosphorylation of chromatin proteins during **D.hydei** development. (Abstr.). 14th Int. Congr. Genet. Moscow, Part I. Contributed Paper Sessions: 603.

REILING, H., W.-E KALISCH, T. WHITMORE & K. TEGTMEIER. 1984. Computer map of salivary gland chromosomes in **D.hydei**. Eur. J. Cell Biol. 34:336-338.

REINHARDT, C.A. & H. GLOOR. 1981. Imaginal disc cells of mutant lethal polyploid larvae (1(3)pl) of **D.hydei** rarely contain centrioles. VIth Eur. Dros. Res. Conf., Kupari, Yugoslavia.

RENKAWITZ, R. 1978. Characterization of two moderately repetitive DNA components localized within the beta-heterochromatin of **D.hydei**. Chromosoma 66:225-236.

_____. 1978. Two highly repetitive DNA satellites of **D.hydei** localized within the alpha-heterochromatin of specific chromosomes. Chromosoma 66:237-248.

_____. 1979. Isolation of twelve satellite DNA's from **D.hydei**. Int. J. Biol. Macromolec. 1:133-136.

_____ & W. KUNZ. 1975. Independent replication of the ribosomal RNA genes in the polytrophic-meroistic ovaries of **Calliphora erythrocephala, D.hydei** and **Sarcophaga barbata.** Chromosoma 53:131-140.

RENKAWITZ-POHL, R. 1978. Number of the repetitive euchromatic 5S RNA genes in polyploid tissues of **D.hydei**. Chromosoma 66:249-258.

_____ & K.H. GLÄTZER. 1980. Clustering of rRNA genes with intervening sequences in the nucleolus organizer of **D.hydei**. (Abstr.). Eur. J. Cell Biol. 22:7.

_____, _____ & W. KUNZ. 1980. Characterization of cloned ribosomal DNA from **D.hydei**. Nucleic Acids Res. 8:4593-4611.

_____, _____ & _____. 1981. Ribosomal RNA genes with an intervening sequence are clustered within the X chromosomal ribosomal DNA of **D.hydei**. J. Mol. Biol. 148:95-101.

RUNGGER-BRÄNDLE, E. 1976. Abnormal microtubules in the mutant 'lethal-polyploid' (lpl) of **D.hydei**. Experientia 32:810.

_____. 1976. Spermatogenesis in larval testes of **D.hydei**. Wilh. Roux's Arch. 180:79-91.

_____. 1977. Abnormal microtubules in testes of the mutant 1(3)pl (lethal-polylpoid) of **D.hydei** cultured in vivo. Exp. Cell Res. 107:313-324.

_____. 1977. Morphogenetic deficiencies in transplanted gonadal anlagen of the mutant 1(3)pl (lethal-polyploid) in **D.hydei**. Exp. Cell Res. 107:301-312.

_____, W.W. FRANKA, M. OSBORN & K. WEBER. 1978. Indirect immunofluorescence microscopy of microtubular structures in male germ cells of wildtype and 1(3)pl (lethal-polyploid) **D.hydei**. Differentiation 10:187-191.

_____, M. JAMRICH & E.K.F. BAUTZ. 1981. Localization of RNA polymerase B and histones in the nucleus of primary spermatocytes of **D.hydei** studied by immunofluorescence microscopy. Chromosoma 82:399-407.

_____ & J.P. VAN DIJK. 1973. Differentiation capacities of imaginal disks of the mutant lethal polyploid (lpl) in **D.hydei**. Wilh. Roux's Arch. 173:333-345.

SCHÄFER, U. 1972. Some observations on bobbed in **D.hydei**. DIS 49:51.

_____. 1976. Hybrid breakdown in **D.hydei** x **D.neohydei** hybrids. (Abstr.). Genetics 83 (Suppl.):S67-S68.

_____. 1978. Sterility in **D.hydei** x **D.neohydei** hybrids. Genetica 49:205-214.

_____. 1979. Viability in **D.hydei** x **D.neohydei** hybrids and its regulation by genes located in the sex heterochromatin. Biol. Zbl. 98:153-161.

_____ & W. KUNZ. 1974. Evidence for a second nucleolus organizer on the Y chromosome of **D.hydei**. Genetics 77 (Suppl. 1):58.

_____ & _____. 1975. Two separated nucleolus organizers on the **D.hydei** Y chromosome. Mol. Gen. Genet. 137:365-368.

_____ & M. SCHÄFER. 1979. Localization of the nucleolus organizer regions in the **D.hydei**-subgroup. (Abstr.). Genetics 91:112-113.

SIN, Y.T. & H.J. LEENDERS. 1975. Studies on the mitochondrial NAD dependent isocitrate dehydrogenase of D. larvae after induction of gene activity by anaerobiosis. Insect Biochem. 5:447-457.

SINCLAIR, J.H., S.E. SAUNDERS, J.F. BURKE & J.H. SANG. 1985. Regulated expression of a **D.melanogaster** heat shock locus after stable integration in a **D.hydei** cell line. Mol. Cell Biol. 5:3208-3213.

SONDERMEIJER, P.J.A., J.W.M. DERKSEN & N.H. LUBSEN. 1980. Established cell lines of **D.hydei**. In vitro 16:913-914.

_____ & N.H. LUBSEN. 1978. Heat shock peptides in **D.hydei** and their synthesis in vitro. Eur. J. Biochem. 88:331-339.

_____ & _____. 1979. The activity of two heat shock loci of **D.hydei** in tissue culture cells and salivary gland cells as analyzed by in situ hybridization of complementary DNA. Chromosoma 72:281-291.

SPENCER, W.P. 1927. The X-chromosome of **D.hydei**. J. Exp. Zool. 47:441-466.

_____. 1928. Five autosomal mutants in **D.hydei**. Genetics 13:44-49.

_____. 1930. Primary non-disjunction in **D.hydei**. Ohio J. Sci. 30:221-228.

_____. 1930. Primary non-disjunction of the X-chromosome in females of **D.hydei**. Genetics 36:172-182.

_____. 1930. The phenotypic expression of the mutant squatty, in **D.hydei**. (Abstr.). Anat. Rec. 47:388-389.

_____. 1930. Mosaic orange - an asymmetrical eye color in **D.hydei**. J. Exp. Zool. 56:267-294.

_____. 1932. The vermilion mutant of **D.hydei** breeding in nature. Am. Nat. 66:474-479.

———. 1934. Duplication 33e 27 in **D.hydei**. Am. Nat. 68:77.

———. 1935. A teaching model of crossing-over in the X-chromosome of **D.hydei**. Am. Nat. 70:68-69.

———. 1937. Multiple alleles at the bobbed locus in populations of **D.hydei**. Genetics 23:170.

———. 1939. On the genetic structure of **D.hydei** populations. (Abstr.). Genetics 24:86-87.

———. 1940. Subspecies, hybrids and selection in **D.hydei** and **D.virilis**. Am. Nat.74:157-179.

———. 1942. Engrailed, a pupal lethal at high temperature in **D.hydei**. Am. Nat. 76:325-329.

———. 1944. Isoalleles at the bobbed locus in **D.hydei** populations. Genetics 29:520-536.

———. 1945. Interaction of alleles at the bobbed locus in **D.hydei**. Am. Nat. 79:85-88.

———. 1947. Mutations in wild populations in Drosophila. Adv. Genet. 1:359-402.

———. 1949. Gene homologies and the mutants of **D.hydei**. In: Genetics, Paleontology, and Evolution (Jepsen, Mayr & Simpson, eds.). Princeton Univ. Press, Princeton, N.J.:23-44.

SPINDLER, K.D. 1976. Intitial characterization of chitinase and chitobiase from the integument of **D.hydei**. Insect Biochem. 6:663-667.

SOBELS, F.H. et al. 1954. The distribution of the genus Drosophila in the Netherlands. Arch. Neerl. Zool. 10:357-374.

SRDIC, Z., H. BECK & H. GLOOR. 1976. The mutant l(3)gl (lethal giant larvae) of **D.hydei**. (Abstr.). Vth Eur. Dros. Res. Conf., Louvain-la-Neuve, Belgium.

———, ——— & ———. 1978. Yolk protein differences between species of Drosophila. Experientia 34:1572-1574.

SRDIC, Z. & P. BORNER. 1980. Hemolymph proteins in an ascitic condition, induced by lethal l(3)gl tumorous tissue in **D.hydei**. Experientia 36:921-923.

SRDIC, Z. & H. FREI. 1979. Karyotype anomalies in cultured l(3)gl brain cells of **D.hydei**. VIth Eur. Dros. Res. Conf., Kupari, Yugoslavia.

——— & ———. 1980. Developmental capacities of mixed normal and neoplastic l(3)gl imaginal discs and neuroblast tissue in **D.hydei**. Differentiation 17:187-192.

——— & H. GLOOR. 1976. Developmental capacities of adult primorida of the lethal mutant l(3)gl of **D.hydei**. (Abstr.). Vth Eur. Dros. Res. Conf., Louvain-la-Neuve, Belgium.

——— & ———. 1978. Le role hematopoietique des glandes lymphatiques de **D.hydei**. Rev. Suisse Zool. 85:11-19.

——— & ———. 1979. Spherule cells in Drosophila species. Experientia 35:1246-1249.

——— & ———. 1980. The fate of **D.hydei** Anlagen in heteroplastic transplantations. Wilh. Roux's Arch. 188:101-105.

——— & M. JACOBS-LORENA. 1978. Drosophila egg chambers develop to mature eggs when cultured in vivo. Science 202:641-643.

——— & C. REINHARDT. 1980. Histolysis initiated by "lymph gland" cells of Drosophila. Science 207:1375-1377.

———, ———, H. BECK & H. GLOOR. 1979. Autonomous yolk protein synthesis in ovaries of Drosophila cultured in vivo. Wilh. Roux's Arch. 187:255-266.

———, ———, ——— & ———. 1979. Autonomous yolk protein synthesis in ovaries of Drosophila cultured in vivo. (Abstr.). VIth Eur. Dros. Res. Conf., Kupari, Yugoslavia.

STAIGER, H. & H. GLOOR. 1952. Mitose hemming und polyploidie durch einen Letalfaktor (lpl=letal polyploid) bei **D.hydei**. Chromosoma 5:221-245.

STOCKERT, J.C. 1971. Alteration in the spermatocyte nuclei of a testis of **D.hydei**. DIS 47:63-64.

STONE, W.S. 1942. Heterosis in **D.hydei**. Univ. Texas Publ. 4228:16-22.

STUBBE, A.E. & H. LÜERS. 1941. Vergleichende Untersuchungen der Wildtypen verschiedener Arten an Hand von Transplantationen der Augenanlagen. Vergleich der Wildtypen von **D.melanogaster** und **D.hydei**. Z. Vererbungslehre 79:498-502.

STURTEVANT, A.H. 1921. The North American species of Drosophila. Carnegie Inst. Wash. Publ. 301:1-150.

———. 1946. On the dot chromosomes of **D.repleta** and **D.hydei**. Genetics 31:259-268.

——— & E. NOVITSKI. 1941. The homologies of the chromosome elements in the genus Drosophila. Genetics 26:517-541.

TEMPLETON, A.R. 1979. The parthenogenetic capacities and genetic structures of sympatric populations of **D.mercatorum** and **D.hydei**. Genetics 92:1283-1293.

TIWARI, R.K. & S.C. LAKHOTIA. 1984. Replication in Drosophila chromosomes XIII. Comparison of late replicating sites in two polytene cell types in **D.hydei**. Genetica 15:227-234.

VAN BREUGEL, F.M.A. 1965. Experimental puffs in **D.hydei** salivary gland chromosomes observed after treatment with CO, CO_2 and N_2. DIS 40:62.

———. 1966. Puff induction in larval salivary gland chromosomes of **D.hydei Sturtevant**. Genetica 37:17-28.

———. 1970. An analysis of white-mottled mutants in **D.hydei**, with observations on X-Y exchanges in the male. Genetica 41:589-625.

———. 1971. Drosophila species: new mutants, **D.hydei**. DIS 47:46.

———. 1971. The action of the Notch locus in **D.hydei**. Genetica 42:25-42.

———. 1971. X-Y exchanges in the males of **D.hydei** carrying the w^mCo duplication. Genetica 42:1-12.

———. 1973. Cell clustering and pleiotropy in white-variegated eyes and Malpighian tubes of **D.hydei**. Genetics 75:323-334.

——— & H.J. BOS. 1969. Some notes on differential chromosome activity in Drosophila. Genetica 40:359-378.

——— & C. GROND. 1980. Bristle patterns and clones along a compartment border in the anterior wing margin of **D.hydei**. Wilh. Roux's Arch. 188:195-200.

_____ & M. HUIZING. 1985. Partial hyperploidy of the X-chromosome in **D.hydei** leading to duplication of male gonadal and genital structure. Wilh. Roux's Arch. Dev. Biol. 194:491-494.

_____, A. RAY & H. GLOOR. 1968. A comparison of banding patterns in salivary gland chromosome of two species of Drosophila. Genetica 39:165-192.

_____, H.C. VAN DEN BROEK, C. GROND & F. HARTOG. 1981. Bristle patterns, clones and cell competitions along the anterior margin of Notch wings of **D.hydei**. Wilh. Roux's Arch. 190:40-48.

_____ & Q.J.M. VAN DER AART. 1979. A clonal analysis of the relationship of duplicated bristles in Drosophila. Wilh. Roux's Arch. 186:267-271.

_____ & B. VAN ZIJLL-LANGHOUT. 1983. The Notch locus of **D.hydei**: alleles, phenotypes and functional organization. Genetics 103:197-217.

_____, E. VERMET-ROZEBOOM & H.J. GLOOR. 1975. Phenocopies in **D.hydei** induced by actinomycin D and fluorouracil with special reference to Notch mutants. Wilh. Roux's Arch. 178:309-320.

VAN ZIJLL-LANGHOUT, B.W. & F.M.A. VAN BREUGEL. 1986. Wing reduction and cell death in **D.hydei**: Analysis of the mutants Notch and Costal-nick. Dev. Genet. 6:257-268.

VOGT, P. & W. HENNIG. 1983. Y chromosomal DNA of **D.hydei**. J. Mol. Biol. 167:37-56.

_____, _____ & L. SIEGMUND. 1982. Identification of cloned Y chromosomal DNA sequences from a lampbrush loop of **D.hydei**. PNAS USA 79:5132-5136.

VOSSEN, J.G.H.M., H.J. LEENDERS, J. DERKSEN & G. JEUCKEN. 1977. Chromosomal puff induction in salivary glands from **D.hydei** by arsenite. Exp. Cell Res. 109:277-283.

_____, _____ & W.G. KNOPPIEN. 1983. A change in the affinity of larval alpha-glycerophosphate dehydrogenase in relation to the activity of the heat shock genes of **D.hydei**. Insect Biochem.

WARTERS, M. 1944. Chromosomal abnormalities in wild populations of Drosophila. Univ. Texas Publ. 4445:129-174.

WASSERMAN, M. 1954. Cytological studies of the repleta group. Univ. Texas Publ. 5422:130-152.

_____. 1960. Cytological and phylogenetic relationships in the repleta group of the genus Drosophila. PNAS USA 46:842-859.

_____. 1962. Cytological studies of the repleta group of the genus Drosophila. IV. The hydei subgroup. Univ. Texas Publ. 6205:73-84.

WEBER-VON GROTTHUSS, E., F. HEVERT, U. ALSBACHER & A. WESSING. 1974. Influence of ouabain on Na$^+$ and K$^+$ concentration in haemolymph of **D.hydei** and appearance of Malpighian tubules. J. Insect Physiol. 20:1411-1420.

WHARTON, L.T. 1943. Analysis of the metaphase and salivary chromosomes morphology within the genus Drosophila. Univ. Texas Publ. 4313:282-319.

_____. 1944. Interspecific hybridization in the repleta group. Univ. Texas Publ. 4445:175-193.

WHITMORE, T. & W.-E. KALISCH. 1984. Hoechst 33258 staining of surface spread polytene chromosomes in **D.hydei**. DIS 60:206.

_____, _____ & H. REILING. 1984. An electron microscopic map of chromosome 6 of **D.hydei**. DIS 60:208.

WOLBURG, H. 1973. Concrements of larval primary urine of **D.hydei**. I. Structure. Cytobiologie 8:25-38.

WONG, P.T. 1968. Effect of X ray on DNA synthesis in salivary gland cells of **D.hydei**. (Abstr.). Genetics 60:239.

_____. 1970. Effects of X-irradiation on DNA synthesis in salivary gland cells of **D.hydei**. Diss. Abstr. 31B:531B.

YAMASAKI, N. 1977. Selective staining of Y chromosomal loops in **D.hydei, D.neohydei** and **D.eohydei**. Chromosoma 60:27-37.

_____. 1981. Differential staining of Y chromosomal lampbrush loops of **D.hydei**. Chromosoma 83:679-684.

ZACHARIAS, H., W. HENNIG & O. LEONCINI. 1982. Microspectrophotometric comparison of the genome sizes of **D.hydei** and some related species. Genetica 58:153-157.

ZAMBURLINI, P. & G.A. DANIELI. 1970. Acrylamide-gel electrophoresis of **D.hydei** proteins at different stages of larval development. DIS 45:85-86.

ZIEGLER, R. & H. EMMERICH. 1973. Phosphorylation of chromosomal proteins in **D.hydei**. DIS 50:180-182.

ZIELKE, U., D. MISCHKE & M. SCHWOCHAU. 1979. Molecular characterization of the mitochondrial RNA and DNA of **D.hydei**. Hoppe Seyler's Z. Physiol. Chem. 360:409-410.

_____, P.M. KLOETZEL & M. SCHWOCHAU. 1980. Mass isolation of a RNP-protein which binds selectively to the nuclear HN RNA of **D.hydei**. Eur. J. Cell Biol. 22:66.
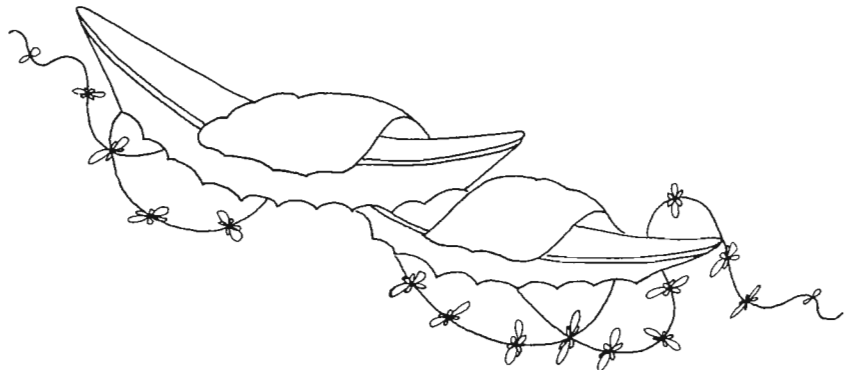
**Tübingen mutants and stocklist. Rick Tearle and Christiane Nüsslein-Volhard.
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The following is a summary of the mutants and rearrangements held in our laboratory at
Tübingen. This information is provided in two forms: a "Redbook" of genes and a DIS 57-style
stocklist.

Our version of the genes entries is given for those genes which were identified and/or substantially
investigated in this lab. The mutants all affect early development and were isolated in several screens
over the past decade. We have not included much molecular biology, as with a few exceptions that has
been carried out elsewhere. We have ommitted the category "Source" from the normal Lindsley and
Grell entry as this is more applicable to the alleles, rather than the genes.

The category "Discoverer" has also been omitted because the method of screening usually
precluded such an identification. However most of the original mutants were isolated in the original
screens (1st, 2nd and 3rd zygotics, and 3rd maternals) and the participants in those screens can be
ascertained from Table 1. There are two major exceptions to this. Rice and Garen isolated a number of
3rd chromosome maternal mutations and called them mat(3)1, mat(3)2 etc. We have renamed these
alleles rm1, rm2 etc. Most of the original 2nd chromosome maternal mutations were isolated by Trudi
Schüpbach, Eric Wieschaus and co-workers (given double letter names e.g. QP, HL). There is
considerable overlap with their material and we wish to acknowledge that here. We apologise to other
Drosophilists if we have inadvertently claimed their work as our own.

The DIS 57 style stocklist covers all of the stocks maintained in this lab. The stocklist is broken up
into genes and chromosomes to facilitate perusal. Although we are happy to fulfil stock requests of a
specific nature, many of the stocks are kept in the stock centres at Umea and Bowling Green and that
should be the source for initial requests. We are not willing to provide the complete set of relatively
uncharacterised 3rd chromosome maternals (fs(3)005-14 etc) because of the amount of work involved,
but will provide subsets if they are requested on biologically sensible grounds.

There are two major discrepancies between the genes list and stocklist. We have shortened many
of the allele names in the genes list but not yet in the stocklist (e.g. bsk^{IIP71} vs bsk^{IIP}). The shortened
version should be used. We are also in the process of converting our nomenclature for transpositions
and translocations to that suggested by Lindsley and Zimm, and there are differences in the
nomenclature between the genes list and stocklist.

The genes list and stocklist were compiled using a series of programs written by Rick Tearle (see
note in this issue of DIS).

Table 1. Summary of mutant screens.

Target	Marked Chromosome	Nomenclature	Participants	References
2nd zygotic embr. visibles	cn bw sp	IB, IIA etc	CN EW & HK (1979)	1
1st zygotic embr. visibles	y, w	YA, XM etc	EW GJ & CN (1980)	2
3rd zygotic embr. visibles	rucuca st e	odd nrs, 5C etc even nrs, 14K etc	GJ EW CN & HK (1980)	3
3rd maternal and fs	rusteca rutipa	even nrs, 260-3 etc odd nrs, 155-10 etc	CN GJ KA & RL (1982)	4,5,6,7
p,ri,kni,hb	rusteca	XT + nr	RL (1982)	8
Kr	IIb	IV, 5 etc	AP & HJ (1982)	9
Df(2L)dl-H	IIa	nrs for dl alleles HT-1 etc for lethals	RS & CN (1982)	10
pum,tsl,tub,bcd	thrips	690 etc	HF RL KA & CN (1983)	5,11
bcd,pum	thrips	variable	RL HF & CN (1983)	5,11
ea	rusteca	numerical	FM (1983)	12
Tl,pll,spz	rusteca	808, 111 etc	KA & CN (1984)	12
cact,spl,wbl, vas,stau	b pr	C8, D3 etc	UM RL & CN (1984)	
cact	IIa	P1	DM (1986)	
pum,osk,nos	rusteca	RA10, RC12 etc	RL UM AB & RT (1987)	

All EMS screens except that for p, ri, kni and hb (RL 1982) which was an X-ray screen.

Participants:
- KA = Kathryn Anderson
- AB = Anke Beermann
- HF = Hans Georg Frohnhöfer
- GJ = Gerd Jürgens
- HJ = Herbert Jäckle
- HK = Hildegard Kluding
- RL = Ruth Lehmann
- DM = David Marcey

- FM = Felix Müller-Holtkamp
- UM = Ulrike Mayer
- CN = Christiane Nüsslein-Volhard
- AP = Annette Preiss
- RS = Ruth Steward
- RT = Rick Tearle
- EW = Eric Wieschaus

Marker Chromosomes

IIa = b pr cn wx^{wxt} bw

IIb = b pr cn wx^{wxt} If

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

rusteca =   ru st e ca

thrips =   th st cp in ri p^p

References:
1. Roux Archives 193:267 (84)
2. Roux Archives 193:296 (84)
3. Roux Archives 193:283 (84)
4. Nature 311:223 (84)
5. Nature 324:120 (86)
6. Cell 47:141 (86)
7. Cell 46:457 (86)
8. Dev. Biol. 119:402 (87)
9. Nature 313:27 (85)
10. Genetics 113:665 (86)
11. Nature (in press)
12. Symp.Soc.Dev.Biol (86)

Tübingen Redbook

alb alberich
Location: 2- (unmapped)
References: R. Lehmann unpublished.
Phenotype: Maternal effect. Occasional embryo lacks
abdominal segments (c/f *osk*).
Alleles: EMS induced: 404.

Alp   Abnormal leg pattern
Location: 2-10.
References: G. Jürgens and S. Cohen unpublished.
Phenotype: Defined by 2 dominant gain-of-function
alleles. Heterozygotes viable with fusion of
metatarsal and 2nd tarsal segments. Transheterozygote
is pupal lethal with more extreme tarsal fusions.
Cytology: 23F6-24A1 (common breakpoint of
translocations).
Alleles: X-ray induced: T(2;3)XT1, T(2;4)X2.

aop   anterior open
Location: 2-12 (between S and Sp).
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Homozygous
embryos have anterior dorsal hole in epidermis. Brain
and sometimes gut extrude through the hole. Head
involution normal. Visible during dorsal closure.
Alleles: EMS induced: IP, IIS (plus 4 discarded
alleles).

arm   armadillo
Location: 1-1.2.
References: Roux 193:296 (84), Roux 195:49 (86),
Cell 49:177 (87).
Phenotype: Embryonic lethal. Embryonic
segmentation defective by germ band shortening.
Causes mirror image duplication of anterior third of
each segment. Cell autonomous in mosaic embryos.
Homozygous germ line clones in females arrested in
oogenesis.
Cytology: 1B14-1E4 (covered by Dp(1;Y)67g but
not by aneuploid segregant of T(1;2)Bld; not
uncovered by Df(1)A94).
Alleles: EMS induced: XK.

arr   arrow
Location: 2-66.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Additional denticle
bands anterior and posterior to normal denticle bands,
especially in ventral midline. Stronger at 18°.
Alleles: EMS induced: IB, IIW   (plus 6 discarded
alleles).

Asx   Additional sex combs
Location: 2-72.
References: Roux 193:267 (84), Nat 316:153 (85),
Jürgens unpublished.
Phenotype: Recessive embryonic lethal. Embryos
show partial transformation of head and thorax into
abdominal structures and A8 into A7; heterozygous

males have sex combs on second and third legs with
low penetrance.
Cytology: 51A1-B4 (uncovered by Df(2R)L+48).
Alleles: EMS induced: IIF, ET21. X-ray induced:
XT129. Hybrid dysgenic: P2 (P. Santamaria).

aur   aurora
Synonym: early-A, frühe-2
Location: 3-53.
References: D. Glover and C. Nüsslein-Volhard
unpublished.
Phenotype: Maternal. Embryos abnormal from
nuclear cycle 9 onwards. Multiple spindles assemble
in long arrays with shared centrosomes. polyploid
nuclei develop with multiple centrosomes. Larval
mitosis not investigated.
Cytology: 87A4-87B2 (uncovered by Df(3R)E229
and by Df(3R)kar-D1).
Alleles: 074, 175, 287.

baz   bazooka
Location: 1-56.7.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. Large ventral and
dorsal holes.
Cytology: 15A5-16A2 (covered by Dp(1;4)exd$^+$82b
and not uncovered by Df(1)r-D17).
Alleles: EMS induced: XI.

bcd   bicoid
Location: 3-48.
References: Nat 324:120 (86), Cell 47:735 (86), H-
G. Frohnhöfer, T. Berleth, W. Driever, M. Noll and
C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Gnathal and thoracic structures
deleted. Telson duplicated anteriorly. Anterior
abdominal segments often fused. In heterozygotes
cephalic fold is shifted anteriorly.
Mol. Biol: Cloned and sequenced. Transcript
contains a prd repeat, M repeat and homeobox. RNAs
transcribed from at least stage 8 of oogenesis but
translated in early embryo. RNAs and protein both
localised at anterior pole of embryo (RNA more than
the protein).
Cytology: 84A (uncovered by Df(3R)LIN &
Df(3R)9A99but not by Df(3R)Ns-R$^+$17).
Alleles: EMS induced: 085, 111, 2-13 (wk), 23-16
(stg), 33-5(stg), E1(stg), E2(stg), E3(ts), E4, E5(wk),
GB(stg).

bch   branch
Location: 3-46.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Incomplete fusion of
denticle bands.
Alleles: EMS induced: 10E.

bhe   broad head
Location: 2-0 (approx)
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos have incompletely involuted heads. In double mutants with *Pc* like mutants abdominal transformations occur.
Alleles: EMS induced: IJ, IM (plus 6 discarded alleles).

bib   big brain
Location: 2-34.7.
References: Roux 192:62 (83), Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos lack ventral epidermis and show hypertrophy of the CNS.
Cytology: 30A9-F (Campos-Ortega).
Alleles: EMS induced: ID5, ID6, IJ, IIP, IIV, IIID (all strong).

bic   bicaudal
Location: 2-67.0.
References: J Exp Zool 161:221 (66), Roux 183:249 (77), Symp. Soc. Dev. Biol.:185 (79).
Phenotype: Maternal. *bic*/Df(2R)vg-B females produce embryos with anterior duplications (symmetrical and asymmetrical) of posterior segments.
Cytology: 49D3-E6 (uncovered by Df(2R)vg-B and Df(2R)vg-D).
Alleles: 1.

BicC   Bicaudal-C
Location: 2-51.
References: Genetics 112:803 (86).
Phenotype: Recessive female sterile. Oogenesis is blocked at stage 8-9. Follicle cells continue to differentiate, resulting in very small, cup-like eggs. Dominant haplo-insufficient temperature sensitive (best at 25°) produces bicaudal embryos at low frequency.
Cytology: 35D4-35E6 (uncovered by Df(2L)osp29 but not by Df(2L)75c).
Alleles: EMS induced: C96, WC.

BicD   Bicaudal-D
Location: 2-52.9.
References: Genetics 112:803 (86).
Phenotype: Maternal. Recessive alleles have 16 nurse cells and no oocyte. Dominant gain of function alleles give bicaudal embryos, homozygotes more penetrant.
Cytology: 36C2-9 (uncovered by Df(2L)TW119 and not Df(2L)MHS5).
Alleles: EMS induced: 7134, IIIE. X-ray induced: 7134r26. Polygenic: YC.

bicF   bicaudal-F
Location: 3- (unmapped).
References: C. Nüsslein-Volhard unpublished.

Phenotype: Dominant maternal effect, giving bicaudals in some genetic backgrounds (e.g. with CyO, DTS513). High temperature and short egg shape may enhance this effect. Homozygous phenotype is probably collapsed eggs.
Alleles: 1.

bip   bipolar oocyte
Location: 3-10.
References: C. Nüsslein-Volhard unpublished.
Phenotype: Female sterile. No eggs laid. Oocytes often have nurse cell clusters at both ends (15 nurse cells altogether). Mature oocytes can have micropyles at both ends, no dorsal appendages and no polarity.
Alleles: EMS induced: 653.

brh   brown head
Location: 2-61.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Defect in head involution and denticle bands abnormal.
Alleles: EMS induced: IB, IID (plus 6 discarded alleles).

bsk   basket
Location: 2-33.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos have a large anterior dorsal hole in cuticle.
Cytology: 31B-E (uncovered by Df(2L)J27).
Alleles: IIJ, IIP, IL*.

btd   buttonhead
Location: 1-31.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. Head involution incomplete.
Cytology: 8A5-9A1 (segmental aneuploidy).
Alleles: IIIA.

cact   cactus
Location: 2-51.2
References: T. Schupbach, D. Godt, S. Roth, D. Marcey and C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Recessive phenotype of embryos ventralised i.e. denticle bands expanded dorsally and dorsal cuticle reduced or absent (c/f $Tl^{Dominant}$ alleles). Haplo-insufficiency causes a weak ventralisation. Some alleles are zygotic lethal.
Cytology: 35E6-36A8 (uncovered by $Df(2L)sna2.4^L$ GYL^R but not by Df(2L)osp3 nor Df(2L)H20).
Alleles: EMS induced: 99, D12, D13, G8, S1, U7 (all zygotic lethal), A2, F11, IIIG (all very stg), H4, O9, O11, PD, SG (all stg), H8, HE, Q6, Y11 (all wk and ts). Hybrid dysgenic: UK, UL, UW, VQ, P2.

ci-D   cubitus interruptus-Dominant
Location: 4-0.

References: Nat 287:795 (80), Roux 193:296(84), DIS 62:63 (85).
Phenotype: Embryonic lethal. Recessive phenotype of duplication of denticle bands at the expense of naked cuticle (segment polarity). Dominant wing phenotype probably unrelated to recessive lethality.
Cytology: 102A3.
Alleles: 1, l(4)13.

cli   clift
Location: 2-17.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Defect in head involution. No segmental movements. In combination with *Pc* like mutants shows abdominal transformations.
Alleles: EMS induced: IID ,IIE.

cno   canoe
Location: 3-49.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Embryo open dorsally.
Alleles: EMS induced: 8A (ts), 9K (stgst), 10B1 (plus 11 discarded alleles).

cra   crack
Location: 2-77.
References: Roux 193:267 (84)
Phenotype: Embryonic lethal. Defect in head involution. In combination with *Pc* like mutants shows abdominal transformations.
Alleles: EMS induced: IJ, IIG.

crb   crumbs
Location: 3-82.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Many small holes in cuticle. Hypertrophy of VNS differs from that of neurogenic loci (Campos-Ortega).
Cytology: 95E-96A (uncovered by aneuploid segregant of T(Y;3)H173 but not T(3;Y)G73).
Alleles: EMS induced: 8F, 11A (plus 4 discarded alleles).

dib   disembodied
Location: 3-12.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Cuticle and head structures undifferentiated.
Cytology: 62D-64C (uncovered by aneuploid segregant of T(2;3)Dll but not Tp(3;Y)H141).
Alleles: EMS induced: 10F, 10K, 10L.

Dl   Delta
Location: 3-66.2.
References: Roux 192:62 (83), Roux 193:283 (84), DIS 62:86 (85).
Phenotype: Embryonic lethal. Lacks ventral cuticle. Hypertrophy of ventral nervous system. Dominant haplo-insufficient phenotype of thickened wing veins.

Cytology: 92A1,2 (uncoverd by Df(3R)bxd110 and Df(3R)Dl-X43).
Alleles: EMS induced: 41, 5F, 6B, 6L, 9D, 9K (wkst), 9M, 9P (stgst), 9Q (ts), 10G ,IL79N.

dl   dorsal
Location: 2-52.9
References: Symp. Soc. Dev. Biol.37:185 (79), Nat 283:474 (80), EMBO 2:1695 (83), Gen 113:665 (86).
Phenotype: Maternal. Embryos dorsalised i.e. dorsal pattern expanded to cover whole of embryonic circumference. Dominant haplo-insufficient ts phenotype is partially dorsalised (mainly lacks mesoderm).
Cytology: 36C2-36C9 (uncovered by Df(2L)TW137 & Df2L)TW119 but not Df(2L)M-HS5).
Alleles: EMS induced: 1, 2 (ts), 3, 4, 5 (wk), 6, 8, 103 (dom), 160 dom), 7607 (dom), B10, O11, PZ (ts), Q17, QF, QD, SC, SG, U5. X-ray induced: H, T (both inversions), I5.

Dll   Distalless
Synonym: Brista (Ba).
Location: 2-107.8.
References: Roux 196:124 (87), S. Cohen and G.Jürgens unpublished.
Phenotype: Recessive embryonic lethal. Peripheral sensory structures associated with appendage-forming imaginal discs absent. Haplo-insufficient dominant adult phenotype with heterozygotes showing antenna-to-leg transformation.
Mol. Biol: Region cloned and gene tentatively identified (S. Cohen, G. Jürgens and H. Jäckle unpublished).
Cytology: 60E5-6 (breakpoint of T(2;3)Dll; not uncovered by Df(2R)ES1).

E(spl)   Enhancer of split
Location: 3-89.
References: Roux 192:62 (83), Roux 193:283 (84).
Phenotype: Recessive embryonic lethal. Amorphic alleles lack ventral cuticle and show hypertrophy of CNS. All alleles appear to be deficiencies.
Cytology: 96F (uncovered by Df(3R)Espl-8D).
Alleles: EMS induced: 8D (associated with a small deficiency in 96F).

ea   easter
Location: 3-57.
References: Symp. Soc. Dev. Biol. 192:177 (86), Nat 311:223 (84).
Phenotype: Maternal. Embryos dorsalised. Two dominant alleles (5.13 and D288) show a lateralised phenotype which can be reverted back to dorsalised. Dorsalised phenotype rescuable by polyA$^+$ RNA injection.
Cytology: 88E-88F.
Alleles: EMS induced: 1 (stg), 2, 3, 4, 5, 5.13 (dom), 6, 7, 8, 9, 10, 11, 111 (wk), 125, 818 (ts), 831, 84b (dom), D288 (dom). X-ray induced: D288-RX1 (revertant of dom).

ely-C   early-C
Location: 3- (unmapped).
References: C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Embryos show abnormal development during early cleavage and cease development soon after.
Alleles: EMS induced: 043, 230.

ems   empty spiracle
Location: 3-53.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. No filzkörper or antennal sense organ, head open.
Cytology: 88A1-10 (uncovered by Df(3R)red-31 but not by Df(3R)red-P93).
Alleles: EMS induced: 7D, 10A (plus 3 discarded alleles).

en   engrailed
Location: 2-62
References: PNAS 78:1095 (81), Roux 193:267 (84).
Phenotype: Embryonic lethal. Anterior margin of each segment defective. Pair rule defects in naked cuticle of T1,T3,A2,A4,A6 & A8.
Cytology: 48A2.
Alleles: EMS induced: IK (stg), IM (ts), IO, IIB (stg), IIT, IIIB.

eve   even-skipped
Location: 2-58.
References: Roux 193:267 (84), CSH 50:145 (85), Cell 47:721 (86).
Phenotype: Embryonic lethal. Cuticle of amorphic embryos show unsegmented lawn. Weaker alleles show pair-rule segmentation defects with the denticle band and adjacent cuticle of T1,T3,A2,A4,A6 and A8 deleted, giving larvae with half the normal number of segments.
Cytology: 46C3-11 (uncovered by Df(2R)eve1.27).
Alleles: EMS induced: ID (ts), IIR (wk), R13 (stg). X-ray induced: 3.77 (med).

exd   extradenticle
Location: 1-54.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. T2 and T3 resemble T1. A1 resembles posterior abdominals. In combination with *Pc* like mutants shows abdominal transformations.
Cytology: 13F-14B1 (uncovered by Df(1)sd72b).
Alleles: EMS induced: XP, YO.

exu   exuperantia
Location: 2-93.
References: Roux 195:302 (86), Genes Dev (in press).

Phenotype: Maternal. Anterior structures replaced by posterior midgut & proctodeum. Enhanced by *bcd*. With *vas* gives dicephalic embryos. *bcd*[+] activity homogenously distributed.
Cytology: 56F-57B (tentative localisation by segmental aneuploidy).
Alleles: EMS induced: PJ, SB, SC, QR (Schüpbach). Hybrid dysgenic: VL (Schupbach). All strong.

fai   faint
Location: 2-61.
References: Roux 193:267 (84).
Phenotype: Larval lethal. Cuticle and mouthparts of larvae unpigmented.
Alleles: EMS induced: IIA, IIV (plus 4 discarded alleles).

fas   faint sausage
Location: 2-68.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos have poorly developed cuticle with necrotic patches and an abnormal head. First visible in extended germ band stage (6h).
Alleles: EMS induced: IC, IIA (plus 2 discarded alleles).

fch   fragile chorion
Location: 3-55.
References: C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Chorion very thin or non-existent.
Alleles: EMS induced: 055, 267.

fkh   forkhead
Location: 3-95.
References: Roux 193:283 (84), D. Weigel and G. Jürgens unpublished.
Phenotype: Embryonic lethal. Head skeleton forked, lack anal plates and Malpighian tubules. Homeotic transformation of anterior head and posterior tail regions.
Mol. Biol: Cloned (D. Weigel, G. Jürgens and H. Jäckle unpublished).
Cytology: 98D2,3 (common breakpoint of several translocations).
Alleles: EMS induced: E200(=1), 2, 3, 12D*. X-ray induced: XT4, XT5 (wk), XT6, XT7, XT8 (wk).

flb   faint little ball
Location: 2-101.
References: Roux 193:267 (84), H-G. Frohnhöfer and C. Nüsslein-Volhard unpublished.
Phenotype: Embryonic lethal. Embryos form a ball of dorsal hypoderm with the internal organs extruded anteriorly. Ventral cuticle absent or strongly reduced. First visible in extended germ band stage (6h). No maternal effect as shown by pole cell transplantation.

Alleles: EMS induced: IF(ts), IK(stg), IP, IIC, IIE (ts), IIG, IIL, IIW (ts), IIX, IIIB41, IIIB92, IIIC81, IIIC87, IIID, IIIF.

flz   filzig
Location: 2-59.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Denticles and hairs of larvae have an abnormal, fuzzy texture. Mouth hooks are poorly developed.
Cytology: 44F-46D (uncovered by aneuploid segregants of Tp(2;3)eve1.18 and Tp(2;3)eve2.28).
Alleles: EMS induced: IP, IIG (plus 3 discarded alleles).

fog   folded gastrulation
Location: 1-65.
References: Roux 193:296 (84), Dev Biol 111:359 (85).
Phenotype: Embryonic lethal. Cellular blastoderm normal and gastrulation starts normally. No posterior midgut formed and germband does not elongate but forms a series of transverse ventral folds. No maternal effect. Homozygous germ line clones give viable embryos.
Cytology: 20A3-B (covered by Y^{67g} but not by B^SY).
Alleles: EMS induced: 4a6.

Fs(2)D10   Female sterile(2)D10
Synonym: D10-9.
Location: 2- (unmapped).
References: U. Mayer, R. Lehmann and C. Nüsslein-Volhard unpublished.
Phenotype: Dominant female sterile. Development ceases before cellularisation of blastoderm. Cleavage appears normal.
Alleles: One EMS induced allele.

Fs(2)X10   Female sterile(2)X10
Synonym: X10-11.
Location: 2- (unmapped)
References: U. Mayer, R. Lehmann and C. Nüsslein-Volhard unpublished.
Phenotype: Dominant female sterile (100% of eggs). Gives cup-like eggs.
Alleles: One EMS induced allele.

Fs(2)Y12   Female sterile(2)Y12
Synonym: Y12-13.
Location: 2- (unmapped).
References: U. Mayer, R. Lehmann and C. Nüsslein-Volhard unpublished.
Phenotype: Semidominant female sterile giving cup like eggs.
Alleles: One EMS induced allele.

ftd   faintoid
Synonym: quicksilver
Location: 1-36.

References: Roux 193:296 (84).
Phenotype: Embryonic lethal. Denticles and mouthparts unpigmented.
Cytology: 10F1-10 (uncovered by Df(1)RA47).
Alleles: EMS induced: IV8.

ftz   fushi tarazu
Location: 3-47.5
References: Dev Biol 102:147 (84), Roux 193:283 (84) Cell 37:825, 833, 843 (84).
Phenotype: Embryonic lethal. Pair rule mutant phenotype. Deletion of parasegments 2,4,6,8,10,12 and 14.
Cytology: 84B1,2.
Alleles: EMS induced: 7B, 9H , 9O, E66, E193.

fzy   fizzy
Location: 2-51.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Ventral hypdoderm and ventral nervous system degenerate.
Alleles: EMS induced: IB, IH (plus 6 discarded alleles).

gd   gastrulation defective
Synonym: fs(1)18.
Location: 1-37
References: Gen 81:683 (75), Gen (77), Symp. Soc.Dev. Biol.:177 (86).
Phenotype: Maternal. Embryos dorsalised.
Cytology: 11A1-7 (uncovered by Df(1)KA10).
Alleles: EMS induced: 1(ts), 2, 3, 4, 5, 6, 7 (stg). 6 complements 3 and partially complements 2. Synonyms: 190=190-5, 1=573, 2=11-1524, 3=12-4955, 4=13-935, 5=13-1697, 6=13-1853, 7=14-743 (alleles 2-7 from Mohler).

gho   ghost
Location: 2-68.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Cuticle undifferentiated with traces of mouthparts visible.
Alleles: EMS induced: IB, IP, IIU*.

gnu   giant nuclei
Location: 3-44.
References: Cell 46:457 (86).
Phenotype: Maternal. Uncouples nuclear division from other cytoplasmic events of mitosis during early cleavage. DNA replicates, centrosomes divide and migrate to the cortex but no nuclear division occurs. Develop 2-4 giant nuclei. Larval mitoses appear normal.
Cytology: 70D2-70E8 (uncovered by Df(3L)fz-m21 but not Df(3L)fz-d21.
Alleles: EMS induced: 305.

gra   grain
Location: 3-47.

References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Filzkörper not
elongated and head skeleton defective.
Alleles: EMS induced: 7J, 7L.

grh　grainy head
Synonym: l(2)IM45.
Location: 2-86.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Head skeleton
defective and denticles weak.
Cytology: 55A-F (uncovered by Df(2R)PC4).
Alleles: EMS induced: IM.

gsb　gooseberry
Location: 2-107.
References: Nat 287:795 (80), Roux 193:267 (84),
Cell 47:1033 (86).
Phenotype: Embryonic lethal. Segment polarity
mutant. On the ventral side of the embryo the naked
cuticle is absent in each segment, being replaced by
mirror image duplication of denticle bands.
Mol. Biol: gsb^+ function appears to be provided by
closely linked duplicated genes, which probably
explains the low mutagenesis frequency with EMS
and the recovery of deficiencies.
Cytology: 60E9-F1 (uncovered by Df(2R)IIX62 and
partially by Df(2R)SB1).
Alleles: All alleles are deficiencies e.g. IIX62.

gt　giant
Location: 1-0.9 (left of tko).
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. Segments A5-7 and
head defective.
Cytology: 3A2.
Alleles: YA.

h　hairy
Synonym: barrel.
Location: 3-26.5
References: Nat 287:795 (80); Roux 193:283 (84),
Genetics 111:463 (85), CSH 50:135 (85), Nat
318:439 (85).
Phenotype: Embryonic lethal. Pair rule phenotype.
Deletion of denticle bands of T1,T3,A2,A4,A6 & A8
and naked cuticle of T2,A1,A3,A5,A7. h^1 is a
hypomorphic viable allele affecting bristle pattern.
Cytology: 66D8-12 (Ish-Horowicz).
Alleles: EMS induced: 8K, 12C, 14H, IK, ILK.
EMS induced in h^1 background: 5H (stg), 7H,
9K(wk), 11D.

hau　haunted
Location: 3-48.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Cuticle
undifferentiated. Only head skeleton visible.
Alleles: EMS induced: 11, 9G, IM (plus 2 discarded
alleles). All strong.

hb　hunchback
Location: 3-48.
References: Roux 193:283 (84), Dev Biol 119:402
(87), Dev Biol 119:418 (87), Nat 327:383 (87).
Phenotype: Embryonic lethal. Gap mutant. Gnathal
and thoracic segments missing. A8 also defective.
Maternal expression in germline mosaics, dispensible
for survival of hb/+ progeny.
Cytology: 85A (uncovered by Df(3R)p-XM66 and
Df(3R)p-XT118).
Alleles: EMS induced: 349 (stg), 11C, 14C
(homeotic), 14F (stgst), 6N (stgst), 7L, 7M (stgst),
7O, 9K49 (homeotic), 9K57 (homeotic), 9K67, 9Q
(stgst), 9R (wkst), C8, IIU (wk). X-ray induced: FB
(stgst), FF (stgst), XT. X-ray induced revertants of
Rg-pbx: G1, G2 (E. Lewis).

hh　hedgehog
Location: 3-81.
References: Nat 287:795 (80), Roux 193:283 (84).
Phenotype: Embryonic lethal. Segment polarity
mutant. Posterior naked section of ventral part of
each segment deleted and replaced by mirror image of
anterior denticle belts. Appear to lack segment
boundaries.
Cytology: 94E (uncovered by aneuploid segregants
of T(Y;3)B27 and not by T(Y;3)R13).
Alleles: EMS induced: 6L (wk), 6N (ts), 9K (ts),
10B (wk), 11C, 13C*, 13E, IJ (stg), IIK, IIO, IIX.

hnt　hindsight
Location: 1-7.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. Germ band does not
retract. Embryo U-shaped with head facing
posteriorly.
Cytology: 4B1-C15 (uncovered by Df(1)RC40 but
not Df(1)JC70).
Alleles: EMS induced: XE.

hth　homothorax
Location: 3-48.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Thoracic segments
similar to each other. Denticles appear to be
intermediate between T1 and T2.
Cytology: 85E-86B (uncovered by
Df(3R)G42^{P}R36^D, synthetic deficiency from 2
·T(Y;A)'s).
Alleles: EMS induced: 5E.

kay　kayak
Location: 3-99.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Embryos remain open
dorsally.
Cytology: 99A-100A (uncovered by aneuploid
segregant of T(Y;3)B172 but not T(Y;3)A113).
Alleles: EMS induced: 7P, 9B.

kkv   krotzkopf verkehrt
Location: 3-49.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Head skeleton crumbled and denticle bands narrower. Embryos sometimes inverted in egg case.
Alleles: EMS induced: 14C, 7J, IB (plus 21 discarded alleles)

kni   knirps
Location: 3-47.0.
References: Roux 193:283 (84), R. Lehmann unpublished.
Phenotype: Embryonic lethal. Gap mutant. A1 to A7 fused into a single remaining denticle belt.
Cytology: 77E1-4 (uncovered by Df(3L)XT1 and Df(3L)XT106).
Alleles: EMS induced: 17, 19 (stg), 301 (stg), 357 (stg), 5F, 7G (stg), 14F (wkst), IL (stg), IID (stg), IIE (stg), IIV, T. X-ray induced: FC (stg).

knk   knickkopf
Location: 3-49.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Head skeleton defective and denticle bands narrowed. Embryos rarely inverted in egg case.
Cytology: 85E-F (uncovered by Df(3R)by10).
Alleles: 5C, 7A, 13B* (wk), 14D, T.

Kr   Krüppel
Location: 2-107.6.
References: Nat 287:795 (80), Roux 193:267 (84), Dev Biol 104:172 (84), Nat 317:40 (85), Nat 319:336 (86).
Phenotype: Embryonic lethal. Gap mutant. Homozygous Kr embryos lack segments T1 to A5 with A6 mirror image duplicated. Visible during early gastrulation. No maternal effect as shown by mitotic recombinant clones in the germ line. $Kr/+$ larvae and adults sometimes have small defects in T2.
Cytology: 60F2 (uncovered by Df(2R)B80 but not Df(2R)IIX62).
Alleles: EMS induced: 2 (stg), 3 (stg), 7 (stg), 8 (stg), 9 (stg), 6A69 (stg), IV (i/m), V (wk), VI (i/m), VII (wk), VIII (wk), IX (wk), X (wk), XI (wk), XII (wk), IO (wk), IIE (wk), IIIA (i/m), J1 (stg).

kug   kugelei
Location: 3-47.
References: Nüsslein-Volhard unpublished.
Phenotype: Female sterile. Mature eggs not laid and almost round. Chorion morphology appears normal.
Alleles: EMS induced: 003 (wk), 040, 054, 113, 258, 381, 602, 622, 649, 674.

l(2)IA109   lethal(2)IA109
Location: 2-60.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Dorsal anterior hole.
Alleles: One EMS induced allele.

l(3)88Ab   lethal(3)88Ab
Synonym: l(3)K43, l(1)SG44.
Location: 3-51.1.
References: PNAS 68:2594 (71), Dev Biol 93:240 (82), DIS 65.
Phenotype: Pupal lethal. Discs much reduced. Mitotic lesion. Viable allele l(3)88Ab^2 is female sterile with chorion thin or non-existent. Amplification of chorion genes affected (Spradling).
Alleles: EMS induced: 88Ab^2=fs(3)293-19.

lds   lodestar
Synonym: early-3, early-B, early-5.
Location: 3-47.
References: D. Glover and C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. In nuclear cycles 9-10 multiple complex anaphase spindles share several centrosomes. Multiple groups of chromosomes are often directed to the same pole. Elongated polyploid nuclei develop. Mitotic abnormalities in larval brain cells (Gatti).
Alleles: 042, 074, 098, 298.

lea   leak
Location: 2-3.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Head involution incomplete. In combination with Pc like mutants abdominal transformations occur.
Cytology: 21F2-22B1 (uncovered by Df(2L)S-2 but not Df(2L)S-3).
Alleles: 25, IIS.

lin   lines
Location: 2-59.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Small anterior portion of each segment deleted. A8, spiracles and anal plates absent. Head abnormal.
Cytology: 44F-46D (uncovered by aneuploid segregant of Tp(2;3)eve1.18 and Tp(2;3)eve2.28).
Alleles: EMS induced: 5E, IIF, IIU. All similar.

mam   mastermind
Location: 2-71.
References: Roux 192:62 (83), Roux 193:267 (84).
Phenotype: Embryonic lethal. Hypertrophy of CNS. Tsp during stages 6-12. Maternal effect in germline clones.
Cytology: 50E (breakpoint of In(2R)N-2G).
Alleles: EMS induced: IB (stg), IF (stg), IJ, IL4, IL1, IIH, IIJ, IIL, IIV.

mid   midline
Location: 2-16.
References: Roux 193:267 (84).

Phenotype: Embryonic lethal. Deletion of anterior three rows of denticles in every adominal segment. *mid^{IIID}* has additional rows of denticles posterior to abdominal denticle bands.
Cytology: 25E-25F1,2 (included in Df(2L)cl-1, Df(2L)cl-7 & Df(2L)GdhA).
Alleles: IK, IIS, IIID.

mmy   mummy
Location: 2-16.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Mouthparts and denticles poorly differentiated.
Alleles: EMS induced: IK, IL (ts), (plus 3 discarded alleles).

ncn-I   nurse cell number-I
Location: 3-68.
References: Nüsslein-Volhard unpublished.
Phenotype: Female sterile. No eggs are laid. Follicles have increased numbers of nurse cells and are often tumorous.
Alleles: EMS induced: 018, 077.

ndl   nudel
Synonym: mat(3)2.
Location: 3-17.
References: Nat 311:223 (84), Symp Soc Dev Biol :177 (86).
Phenotype: Maternal. Eggs slightly collapsed and embryos dorsalised. Cytoplasmic transplantation not feasible.
Alleles: EMS induced: 046, 093, 111, 133, 169, 260, rm2. All strong.

neu   neuralised
Location: 3-50.
References: Roux 192:62 (83), Roux 193:283 (84).
Phenotype: Embryonic lethal. Hypertrophy of CNS (neurogenic).
Cytology: 85D (uncovered by Df(3R)p-XT9 but not by Df(3R)p-XT103).
Alleles: EMS induced: 9L (stg), 11B, 12H (cts),IF (cts), IN, IIIA.

nkd   naked
Location: 3-47.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Incomplete deletion of denticle bands.
Cytology: 75D-76B (uncovered by Df(3L)L14^P L131^D, synthesised from 2 T(Y;A)'s).
Alleles: EMS induced: 6J (wk), 7E (stg), 7H (stg), 9G, 9H.

nos   nanos
Location: 3-66.2.
References: R. Lehmann unpublished.

Phenotype: Maternal. Lacks abdominal segments but has normal pole cells and pole plasm. Pole plasm has no posterior activity.
Cytology: 91F-92A.
Alleles: EMS induced: 18, 53, L7.

odd   odd skipped
Location: 2-8.
References: Nat 287:795 (80), Roux 193:267 (84), CSH 50:145 (85).
Phenotype: Embryonic lethal. Pair rule phenotype. Partial deletion of denticle bands of T2,A1,A3,A5 and A7.
Cytology: 24A1-3 (uncovered by Df(2L)odd-1.10 and Df(2L)odd-5.1).
Alleles: EMS induced: 7L (stg), 9P (wk), IIIC (wk), IIID (stg). X-ray induced: 1.36 (stg).

opa   odd paired
Location: 3-48.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Pair rule phenotype. All alleles are variable.
Cytology: 82A-E.
Alleles: EMS induced: 5H, 7N, 9C (wk), 9O, 13D (wkst), E8, IIC, IIP (stg), T.

osk   oskar
Location: 3-48.4
References: Cell 47:141 (86).
Phenotype: Maternal. Lacks pole cells, pole plasm and abdominal segments. Removes posterior activity required for establishment of pole cells and abdominal segments.
Cytology: 85B (uncovered by Df(3R)p-XT26 but not Df(3R)p-XT118).
Alleles: EMS induced: 54, 084, 88, 123, 150, 166 (stg), 255 (wk), 301 (ts), 346.

otd   orthodenticle
Location: 1-26.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. All denticles in abdominal segments point posteriorly. Head also affected.
Cytology: 7F1-8A5 (uncovered by Df(1)RA2 and Df(1)KA14).
Alleles: EMS induced: YH.

pbl   pebble
Location: 3-26.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. Embryo forms a faint ball of cuticle. Mitosis defective resulting in fewer and much larger cells in late embryo.
Cytology: 66A-C (uncovered by aneuplod segregant of Tp(3;Y)G130 and Tp(3;Y)B162 but not Tp(3;Y)A76).
Alleles: EMS induced: 5B, 5D, 7O, 8J, 11D. All cold ts.

Pc   Polycomb
Location: 3-48.
References: Dev Biol 100:399 (83), Jürgens
unpublished.
Phenotype: Embryonic lethal. Shows strong
homeotic transformation of head and abdominal
segments toward A8. *Pc*/+ males have sex combs on
2nd or 3rd legs.
Cytology: 78D1-79C1 (uncovered by Df(3L)Pc =
Df(3L)ASC).
Alleles: EMS induced: 9M, E213, ET12, ET23. X-
ray induced: XH1, XM1, XM75, XM80, XL5,
XT109.

Pcl   Polycomb like
Location: 2-86.
References: Genet 102:49 (82), Genet 105:357 (83),
Nat 316:153 (85), G. Jürgens unpublished.
Phenotype: Embryonic lethal. Shows weak posterior
transformation of abdominal segments which is
strongly enhanced in combination with other *Pc* like
mutants. *Pcl*/+ males sometimes have sex combs on
2nd or 3rd legs.
Cytology: 55A-C (uncovered by Df(2R)Pcl-7B and
Df(2R)Pcl-11B).
Alleles: EMS induced: E3, E90 (both G. Struhl),
E501. X-ray induced: X21, XF21, XM3, XT9*,
XT56 (wk).

phm   phantom
Location: 1-64.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. Poorly differentiated
cuticle. Embryo contracted posteriorly.
Cytology: 17A-18A (uncovered by Df(1)N19).
Alleles: EMS induced: XE.

pim   pimples
Location: 2-30.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Poorly differentiated
cuticle with necrotic patches. Head abnormal.
Cytology: 31B-E (uncovered by Df(2L)J27).
Alleles: EMS induced: IL.

pip   pipe
Location: 3-47.
References: Nat 311:223 (84), Symp Soc Dev Biol
:177 (86).
Phenotype: Maternal. Embryos dorsalised. Not
rescuable by cytoplasmic transplantation.
Alleles: EMS induced: 386, 664 (both stg).

ple   pale
Location: 3-18.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Cuticle and head
skeleton unpigmented.

Cytology: 65A-E (uncovered by aneuplod segregant
of Tp(3;Y)G130 and Tp(3;Y)A76 but not
Tp(3;Y)B162).

Alleles: EMS induced: 7F*, 7O, 11F* (ts), 14C.

pll   pelle
Location: 3-92.
References: Nat 311:223 (84), Symp Soc Dev Biol
:177 (86), Dev Biol 110:238 (85).
Phenotype: Maternal. Embryos dorsalised.
Rescuable by injection of polyA$^+$ RNA.
Cytology: 97F (uncovered by Df(3R)9Q-RXP but
not Df(3R)9Q-RXI).
Alleles: EMS induced: 019 (med), 74 (stg), 078
(stgst), 122 (cts), 312 (stg), 316 (wk), 385 (stg), 628
(wk), 864 (cts), rm8 (stg).

pnr   pannier
Location: 3-58.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Open dorsally.
Cytology: 89B4-10 (uncovered by Df(3R)sbd45).
Alleles: EMS induced: 7G, 9L.

pnt   pointed
Location: 3-79.
References: Roux 193:283 (84), U. Mayer
unpublished.
Phenotype: Embryonic lethal. Denticle bands
narrow. CNS broad and less dense than wildtype.
Head skeleton has fused ventral arms. Sensory organs
(maxillary, antennal and keilins organs) spread.
Germline viable with no maternal component.
Cytology: 94E (uncovered by aneuploid segregant of
T(Y;3)B27 but not T(Y;3)R13).
Alleles: EMS induced: 8B, 9J.

prd   paired
Location: 2-45
References: Nat 287:795 (80), Roux 193:267 (84),
CSH 50:145 (85), Cell 47:735.
Phenotype: Embryonic lethal. Pair rule mutant.
Deletion of denticle bands of T1,T3,A2,A4 & A6 and
deletion of naked cuticle of T2,A1,A3,A5 & A7. No
head fold visible in gastrulation. Tsp during
blastoderm.
Cytology: 33C1,2 (uncovered by Df(2L)prd-1.25; in
situ data).
Alleles: EMS induced: 6L (stg), IIB (wk), IIN (ts),
IIW (wk), FR1 (stg). X-ray induced: 2.45, 32.12, X3
(all strong).

Psc   Posterior sex combs
Location: 2-67.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos show partial
transformation of head and thorax into abdominal
structures. Enhanced in combination with other *Pc*
like mutants. *Psc*/+ males sometimes have sex
combs on 2nd and 3rd legs.

Cytology: 49D3-E6 (uncovered by Df(2R)vg-D) and Df(2R)vg-B but not Df(2R)vg-C).
Alleles: EMS induced: IIN.

ptc   patched
Synonym: patch (pat), already occupied.
Location: 2-55.
References: Nat 287:795 (80), Roux 193:267 (84).
Phenotype: Embryonic lethal. Segment polarity mutant. Mirror image duplication of segment boundaries and adjacent cuticle of all segments, with deletion of the remainder of the segment. Visible during extended germ band stage (6h).
Cytology: 44B-F (included in Df(2R)eve-1.18 but not in Df(2R)eve-2.28).
Alleles: EMS induced: 6C*, 6P, 7M, 8H, 9B, IF (ts), IN (stg), IIB, IIC2, IIC8, IIE, IIR (wk), IIU (wk), IIW, IIX. Strong unless otherwise noted.

Pu   Punch
Synonym: unpigmented (upi).
Location: 2-97.
References: Redbook, Roux 193:267 (84).
Phenotype: Recessive embryonic lethal. Cuticle and mouthparts unpigmented. Dominant eye color phenotype.
Cytology: 57B-C (common breakpoint of *Pu* rearrangements).
Alleles: EMS induced: IN, IIK (plus 2 discarded alleles).

pum   pumilio
Location: 3-48.5.
References: R. Lehmann and C. Nüsslein-Volhard (in press).
Phenotype: Maternal. Mimics *osk* group in lacking abdominal segments but has pole cells which may be fertile. Posterior organising activity is present in embryos but unable to reach abdomen. Most alleles semi-lethal, escapers having abnormal bristles.
Cytology: 85C-85D (uncovered by Df(3R)p-XT9 but not by Df(3R)p-XT103).
Alleles: EMS induced: 21, 680 (stg), ET1, ET2, ET3 (cts), ET4, ET5, ET6, ET7 ET8, ET9, ET10, ET11.

put   punt
Location: 3-58 (approx).
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Embryos open antero-dorsally.
Cytology: 88C3-E2 (uncovered by aneuploid segregant of Tp(3;2)XM54 and Tp(3;1)kar5l).
Alleles: EMS induced: 135.

qua   quail
Location: 2-53 (to the right of *dl*).
References: Genetics 113:665 (86), Schüpbach unpublished.
Phenotype: Female sterile. Oogenesis is abnormal with nurse cell contents not being deposited in the oocyte, resulting in very small cup-like eggs.

Cytology: 36C2-9 (uncovered by Df(2L)TW119 but not by Df(2L)MHS5).
Alleles: EMS induced: WP.

raw   raw
Location: 2-19.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos appear to cease development at 14h. Dorsal closure is incomplete and there is no cuticular differentiation.
Alleles: EMS induced: IG, IIF (plus one discarded allele).

rho   rhomboid
Location: 3-3.
References: Roux 193:283 (84), U. Mayer unpublished.
Phenotype: Embryonic lethal. Denticle bands and VNS narrow. Fusion of medial parts of denticle bands. Ventral arms of head skeleton fused. Anal pads reduced. Keilins organs, maxillary and antennal sense organs reduced. Germline viable with no maternal component.
Cytology: 61F-62D (uncovered by aneuploid segregant of Tp(3;Y)H141 but not T(Y;3)A114).
Alleles: EMS induced: 7M.

rib   ribbon
Location: 2-88.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Lateral extent of denticle belts narrow. Adjacent denticle bands are fused in the ventral midline. Denticle differentiation is abnormal and dorsal closure defective.
Alleles: EMS induced: IK, IIB (wk), (plus 7 discarded alleles).

rtv   retroactive
Location: 1-38.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. Mouthparts darkly sclerotinised. Embryos sometimes inverted in egg case.
Cytology: 10A7-11 (uncovered by Df(1)RA37 but not be Df(1)GA112).
Alleles: EMS induced: YA.

run   runt
Location: 1-67.
References: Nat 287:795 (80), Roux 193:296 (84), Dev Biol 109:321 (85).
Phenotype: Embryonic lethal. Pair rule phenotype. Anti-*runt* phenotype in embryos with three doses of *run*[+]. Autonomous in gynanders.
Cytology: 19E1-F1 (tentatively; between Df(1)mal8 and Df(1)DCB1).
Alleles: EMS induced: YC, YP (ts).

S   Star
Location: 2-1.3

References: Redbook, Roux 193:267 (84), U. Mayer
unpublished.
Phenotype: Recessive embryonic lethal. Denticle
bands narrow and head skeleton ventral arms fused.
Anal pads reduced. Transverse commissures of VNS
reduced. Keilin organs, maxillary and antennal sense
organs strongly reduced. Lethal in germline.
Dominant eye phenotype.
Cytology: 21D2-22A1 (uncovered by Df(2L)S2 and
Df(2L)S3).
Alleles: EMS induced: 54, IIN.

sad   shadow
Synonym: karmoisin ghost.
Location: 3-51.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. No differentiation of
cuticle and head skeleton. Posterior of embryo is
condensed.
Cytology: 86F6-87A7 (uncovered by Df(3R)E229
but not Df(3R)kar-D1).
Alleles: EMS induced: 5F, 10D.

sal   spalt
Location: 2-44.
References: Roux 193:267 (84), Jürgens
(submitted).
Phenotype: Embryonic lethal. Shows partial
homeotic transformation of labium to prothorax and
of A9 to A8.
Cytology: 33A1-2 (uncovered by Df(2L)Prl but not
Df(2L)prd-1.7; and in situ data).
Alleles: EMS induced: 445, IIA, IIB.

scb   scab
Location: 2-73.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos have small
mid-dorsal hole with a necrotic rim.
Alleles: EMS induced: IID, IIG (plus 2 discarded
alleles).

Scm   Sex combs on midleg
Location: 3-48.5.
References: Nat 316:153 (85).
Phenotype: Recessive embryonic lethal and
dominant haplo-abnormal, both phenotypes very
similar to *Pcl*.
Cytology: 85EF (uncoverd by Df(3R)by10 and
Df(3R)by62).
Alleles: EMS induced: E502, ET16, ET19*. X-ray
induced: XF24.

Scr   Sex combs reduced
Location: 3-47.
References: Nat 316:153 (85), G. Jürgens
unpublished.
Phenotype: Embryonic lethal. Homeotic
transformation of T1 to T2 and labium to maxilla.
Scr/+ males have fewer sex combs than wildtype.
Cytology: 84B1-2.

Alleles: EMS induced: 7F, 8B, 9G, 13A, E4B, EH1,
ET6, ET16, ET21. X-ray induced: X24, XF5, XF9,
XH21, XH44, XH45, XT12, XT63, XT66, XT127,
XT130, XT145, XTA1.

scw   screw
Synonym: l(2)IG76.
Location: 2-53.
References: Roux 193:267 (84), K. Arora and C.
Nüsslein-Volhard unpublished.
Phenotype: Embryonic lethal. Partially ventralised.
Cephalic furrow shifted dorsally with defects in
germband extension. Ventral denticles extended
laterally. Spiracles and 2-3 terminal segments
retracted into embryo.
Cytology: 37F-38A7 (uncovered by Df(2L)TW50
but not Df(2L)E55).
Alleles: EMS induced: C13, IG (wk), N5, O5, S12.

sdt   stardust
Location: 1-23.
References: Roux 193:296 (84).
Phenotype: Embryonic lethal. Hypoderm almost
totally absent. Only small remains of cuticle found.
Cytology: 7D10-F2 (uncovered by Df(2L)RA2 but
not Df(2L)KA14).
Alleles: EMS induced: XM.

seg   short egg
Location: 1-
References: Symp Soc Dev Biol 36: (78),
Wieschaus and Kluding unpublished.
Phenotype: Maternal effect. Eggs shorter and broader
than normal but density of blastoderm nuclei and egg
volume not affected. Segmentation usually normal.
Alleles: EMS induced: 1.

sha   shavenoid
Location: 2-62.
References: Roux 193:267 (84), Nüsslein-Volhard
unpublished.
Phenotype: Trichomes missing or very short. Flies
cannot fly or walk on glass. In larvae the number of
denticles is reduced with remaining denticles thin and
bent. Hairs are largely absent but sensory hairs not
affected. Autonomous in nuclear transplants.
Cytology: 47E3-48B2 (uncovered by Df(2R)en-A
and Df(2R)en-B).
Alleles: EMS induced: IN, IIP (plus 2 discarded
alleles).

shd   shade
Location: 3-41.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. No differentiation of
cuticle or head skeleton.
Cytology: 70D-71C (uncovered by aneuploid
segregant of Tp(3;Y)B162 but not Tp(3;Y)G145.
Alleles: EMS induced: 6J, 7C, 9K (plus 2 discarded
alleles).

shg   shotgun
Location: 2-92.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryonic cuticle has many small holes with necrotic rims. Head grossly distorted. Weak alleles show head defects and irregular flaws in segmentation pattern.
Alleles: EMS induced: IG, IH (plus 16 discarded alleles).

shm   schmal
Synonym: l(3)S8.
Location: 3-52.
References: DIS 56:65 (81), U. Mayer unpublished.
Phenotype: Embryonic lethal. Denticle bands of all segments narrow. Both head skeleton ventral arms and anal plates fused. In the VNS transverse commissures lacking entirely. Mesectodermal cells missing. Germline viable with no maternal component.
Cytology: 87E1 (close to ry).
Alleles: EMS induced: E320, RD.

shn   schnurri
Location: 2-62.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos lack dorsal hypoderm. Internal organs appear normal and extruded through the open dorsal side of the embryo. Ventral hypoderm contracted.
Cytology: 47E3-48B2 (uncovered by Df(2R)en-B and Df(2R)en-A).
Alleles: EMS induced: IB, IM (ts), (plus 15 discarded alleles). Hybrid dysgenic: TD5 (P. Gergen).

sic   sichel
Location: 3-48.8.
References: U. Mayer unpublished.
Phenotype: Maternal. Most embryos show narrow denticle bands and head skeleton with fused ventral arms. Embryos may also be normal and hatch, or die early with irregular cell cleavage patterns. Germline dependent but may also have a somatic component.
Cytology: 85D7-12 (uncovered by Df(3R)ß$_2$t and Df(3R)by10).
Alleles: EMS induced: 215 (wk), 256, 371 ,612. All cold ts.

slf   schlaff
Location: 2-15.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Cuticular differentiation normal but arrangement of cuticle around embryo abnormal. Cuticle is detached from the body. Head skeleton tilted backwards. Posterior segments contracted, anterior segments stretched around egg tip.
Alleles: EMS induced: IG, IJ (plus 4 discarded alleles).

sli   slit
Location: 2-77.

References: Roux 193:267 (84), G. Jürgens unpublished.
Phenotype: Embryonic lethal. Head involution abnormal. In combination with *Pc* like mutants abdominal transformations occur.
Alleles: EMS induced: IG2, IG7.

slp   sloppy paired
Location: 2-8.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos lack parts of naked cuticle of T2,A1,A3,A5 and A7 in an irregular fashion.
Cytology: 24C-D (uncovered by Df(2L)ed-SZ1).
Alleles: EMS induced: IIM (wk), L12 (stg), 7L* (stg).

smo   smooth
Location: 2-4.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. All denticles in abdominal segments point posteriorly. At 18° naked cuticle deleted and denticle belts of adjacent segments fused and locally arranged as mirror image duplications.
Alleles: EMS induced: IIG, IIX, Q14. All cold ts.

sna   snail
Synonym: l(2)br28.
Location: 2-51.
References: Genet 105:615 (83), Roux 193:267 (84), H-G. Frohnhöfer and C. Nüsslein-Volhard unpublished.
Phenotype: Embryonic lethal. Partially dorsalised. Ventral furrow is missing and embryo twisted or coiled in egg case, often with the posterior side up. No maternal effect in germ line chimeras. *sna*/+ embryos have delayed ventral furrow formation.
Cytology: 35C3-D (uncovered by Df(2L)75c but not Df(2L)A72).
Alleles: EMS induced: HG, IIG. X-ray induced: 4.26, 18.19. All strong.

snk   snake
Location: 3-52.
References: Nat 311:223 (84), Symp Soc Dev Biol (86), Nat 323:688 (86).
Phenotype: Maternal. Embryos show extreme dorsalised phenotype - only dorsally derived cuticle remains. Responds most strongly of all maternal dorsalized mutants to rescue by injection of cytoplasm and polyA$^+$ RNA.
Mol. Biol: Appears to encode a serine protease.
Cytology: 87D10-12 (uncovered by Df(3R)ry36 and not Df(3R)kar-lG27).
Alleles: EMS induced: 073, 229, 233, rm4. All strong.

sog   short gastrulation
Location: 1-53.
References: Roux 193:296 (84).

Phenotype: Embryonic lethal. Germ band extension incomplete. Probably a weak ventralised phenotype.
Alleles: EMS induced: XM.

spg sponge
Synonym: early-D, mat3(6).
Location: 3-95
References: C. Nüsslein-Volhard and E. Wieschaus unpublished.
Phenotype: Maternal. Embryos show abnormal cleavage.
Alleles: EMS induced: 145, 242, 805, 842, rm6.

spi spitz
Location: 2-54.
References: Roux 193:267 (84), U. Mayer unpublished.
Phenotype: Embryonic lethal. Denticle bands narrow and head skeleton ventral arms fused. Anal pads reduced. Transverse commissures of ventral nervous system reduced. Keilin organs, maxillary and antennal sense organs all reduced in size. Semilethal in germline.
Cytology: 37E2-38A1 (uncovered by Df(2L)E55 and Df(2L)TW9).
Alleles: EMS induced: IIA, IIT, IIIA*.

spn-A spindle-A
Location: 3-96.
References: C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Egg shape affected. In extreme cases dorsal appendages are lacking and eggs have little or no dorsal-ventral polarity. Some eggs have one fused dorsal appendage. Low fecundity. Eggs often slightly collapsed.
Alleles: EMS induced: 003, 050, 057, 215.

spn-B spindle-B
Location: 3-52.
References: C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Similar to spn-A but normal eggs also recovered. Eggs always unfertilised and abnormal eggs often long.
Cytology: 88A10-C3 (uncovered by Df(3R)red-31 & Df(3R)red-P93 but not by Df(3R)red-P52).
Alleles: EMS induced: 056, 153, 225.

spn-C spindle-C
Location: 3-17.
References: C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Similar to spn-A.
Alleles: EMS induced: 094, 422, 660.

spn-D spindle-D
Location: 3-91.
References: C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Similar to spn-A. Embryos sometimes hatch.
Alleles: EMS induced: 150 (wk), 349 (stg).

spn-E spindle-E
Location: 3-62.
References: C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Similar to spn-A.
Alleles: EMS induced: 616, 653.

spn-F spindle-F
Location: 3-100.
References: C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Similar to spn-A.
Alleles: EMS induced: 234.

spo spook
Location: 3-19.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. No differentiation of cuticle and mouthparts.
Alleles: EMS induced: 4G, 7J.

spz spätzle
Location: 3-92.
References: Nat 311:223 (84), Symp Soc Dev Biol :177 (86).
Phenotype: Maternal. Embryos dorsalised. Germline dependent.
Cytology: 97D13-E4 (uncovered by Df(3R)9Q-RX1 but not Df(3R)ro80b nor Df(3R)D605)
Alleles: EMS induced: 145 (wk), 197 (stg), 670 (ts), rm7 (stg).

sro shroud
Location: 3-100.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. No differentiation of cuticle and mouthparts.
Cytology: 99A-C (uncovered by aneuploid segregant of T(3;Y)B172 and Tp(3;Y) but not T(3;Y)A113).
Alleles: EMS induced: 8A, 11C (plus 2 discarded alleles).

srp serpent
Synonym: spt (pre-occupied).
Location: 3-58.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Germ band shortening incomplete.
Cytology: 89A1-B4 (uncovered by Df(3R)sbd105 but not Df(3R)sbd45).
Alleles: EMS induced: 288, 6G, 9L (plus 3 discarded alleles).

srw shrew
Location: 3-15 (approx.).
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Embryos partially ventralised.
Alleles: EMS induced: 10K.

stau   staufen
Location: 2-83.5
References: Roux 123:12-23 (87).
Phenotype: Maternal. Lacks pole cells, pole plasm and abdominal segments (c/f *osk*). Anterior part of head also defective.
Cytology: 55A-C (uncovered by Df(2R)Pcl-7B and Df(2R)Pcl-11B).
Alleles: EMS induced: D3 (stg),G2 (stg), HL (wk), C8 (ts).

stg   string
Location: 3-99.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Denticle bands reduced. Failure of post blastoderm mitoses with fewer and larger cells in all tissues. No neuropile.
Cytology: 99A (D. Weigel and G. Jürgens).
Alleles: EMS induced: 4B (ts), 7B, 7L, 7M (stgst), 8A, 9A, 9K (wkst), 13D, IIB, IO.

str   slater
Location: 2-17.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos lack dorsal hypoderm. Internal organs appear normal and are extruded through the open dorsal side of the embryo. Ventral hypoderm contracted.
Alleles: EMS induced: IO, IIB.

swa   swallow
Synonym: fs(1)1502, fs(1)M44.
Location: 1-14.
References: Dev Biol 47:419 (75), Genetics 113:695 (86), Genes Dev (in press), Dev Biol (in press).
Phenotype: Maternal. Embryo shows head defects (similar to weak *bcd* alleles) and abdominal defects. Many eggs already defective in early cleavage. Cold sensitive at 18°. Affects *bcd*+ localisation.
Mol. Biol: Cloned (E. Stephenson).
Cytology: 5E6-7 (uncovered by Df(1)JF5; Stephenson and Mahowald).
Alleles: EMS induced: 14 (stg),15 (wk), 82 (stg, most eggs develop), 99 (stg) T573 (wk). 14 and 15 from M. Gans, 82 and 99 from D. Mohler, T573 from E. Stephenson.

thi   thickhead
Location: 2-72.
References: Roux 193:287 (84), G. Jürgens unpublished.
Phenotype: Embryonic lethal. Head broad. In combination with *Pc* like mutants abdominal transformations occur.
Alleles: EMS induced: IIM, IIN.

thr   three rows
Location: 2-86.
References: Roux 193:267 (84).

Phenotype: Embryonic lethal. Denticle bands have fewer rows, all pointing posteriorly. Denticles larger than normal. Apparently no mitoses after the 1st post blastoderm mitosis, resulting in embryos with fewer and larger cells than normal. Tsp 4-9h.
Cytology: 55A-F (uncovered by Df(2R)PC4).
Alleles: EMS induced: IB (stg), IL, IIV (ts), (plus 4 discarded alleles). Hybrid dysgenic: BH (P.Gergen).

Tl   Toll
Location: 3-91.
Synonym: mat(3)9
References: Nat 311:223 (84), Cell 42:779 (85), Cell 42:791 (85).
Phenotype: Maternal. Dominant alleles are partially ventralised, recessive hypomorphic alleles are lateralised and null alleles (revertants of dominants) are dorsalised. In Tl⁻ embryos a normal dorsoventral pattern can be restored by injection of wild-type cytolplasm, the ventral position being determined by the site of injection.
Cytology: 97D1-2 (based on breakpoints of several revertants).
Alleles: EMS induced dominants: 1, 5B, 9Q, 84c. EMS induced (hypomorphic) recessives: r26, r444, r632, rm9. X-ray induced (amorphic) revertants of dominants: 1-RXA, 1-RXD, 1-RXH, 5B-RXV, 9Q-RX etc. EMS induced (amorphic) revertants of dominants: 5B-REQ, 5B-REK, 9Q-RE.

tld   tolloid
Location: 3-86.
References: Roux 193:283 (84), H-G. Frohnhöfer and C. Nüsslein-Volhard unpublished.
Phenotype: Embryonic lethal. Partially ventralised. Cephalic furrow shifted dorsally and defects in germband extension. Denticle belts extended laterally. No maternal effect in pole cell transplants. Complex intra-allelic complementation pattern.
Cytology: 96B-D (uncovered by Df(3R)XTA1).
Alleles: EMS induced: 5H (wk), 6B, 6P4, 6P7, 7H, 7M, 7O, 8L, 9B, 9D (ts), 9K, 9Q1, 9Q7, 10E (stg), 10F, FF, T.

tll   tailless
Location: 3-102.
References: Roux 193:283 (84), Dev Biol 113:64 (86).
Phenotype: Embryonic lethal. Segmentation gene with anterior and posterior defects. Anterior defects in head skeleton but labrum is present. Posteriorly the Malpighian tubules, anal pads and A8 are missing. The posterior midgut, proctodeum and pole cells are normal.
Cytology: 100A5-B2 (breakpoint of In(3R)C; Strecker and Merriam).
Alleles: EMS induced: L10.

tny   tiny
Location: 3-91.
References: K. Anderson unpublished.
Phenotype: Female sterile. Eggs are very small.

Cytology: 97D9-15 (uncovered by Df(3R)ro80b but not by Df(3R)ro-XB3).
Alleles: EMS induced: 4.

tor   torso

Location: 2-57 (left of *cn*).
References: Roux 195:302 (86), M. Klingler, H-G. Frohnofer, J. Szabad, M. Erdelyi and C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Anterior and posterior most structures are deleted (labrum, dorsal bridge, telson, A8, part A7). Shows pole hole phenotype. Gain of function antimorphic alleles have opposite phenotype: segment defects in middle of embryo, terminal structures present.
Cytology: 43E9,10 (uncovered by Df(2L)H23 & Df(2L)tor-rx5 but not by Df(2L)CA53). Lefevre's maps are incorrectly labelled in this region and published localisation of *cn* is incorrect (C. Nüsslein-Volhard).
Alleles: EMS induced recessives: HH, HM, QA, QK2, QK4, QL, PM, RI, WK (all stg). EMS induced antimorphs: RL3 (rec), Y9 (semidom), 4021 (dom). EMS induced revertants of antimorphs: re1, re2, AL1...AL18, AY1...AY41. X-ray induced revertants of antimorphs: rx2, rx8, rx9.

tov   tiny ovaries

Location: 3- (unmapped).
References: C. Nüsslein-Volhard unpublished.
Phenotype: female sterile; no eggs laid; rudimentary ovaries; may be agametic.
Alleles: EMS induced: 012, 155, 267, 345, 404.

trh   trachealess

Location: 3- -1.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Tracheae are absent and filzkorper not elongated.
Cytology: 61E-F (uncovered by aneuploid segregant of T(Y;3)A114 and of Tp(3;Y)G130).
Alleles: EMS induced: 5D, 7J.

tsg   twisted gastrulation

Location: 1-42.
References: Roux 193:296 (84), Dev Biol 111:359 (85).
Phenotype: Embryonic lethal. Gastrulation abnormal, similar to weak ventralising mutants.
Cytology: 11A1-11A7 (uncovered by Df(1)KA10).

tsl   torso like

Location: 3-71.
References: H-G. Frohnofer and C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Anterior and posterior defects similar to tor. Soma dependent in pole cell transplants.
Cytology: 93F (uncovered by Df(3R)e-F4 but not by Df(3R)e-D7 nor Df(3R)e-R1).

Alleles: EMS induced: 035, 135 (ts), 146 , 691, 174, MK (wk).

tub   tube

Location: 3-47.
References: Nat 311:223 (84), Symp Soc Dev Biol :177 (86).
Phenotype: Maternal. Dorsalised. Rescuable with wildtype cytoplasm and RNA.
Alleles: 118, 238 (both stg).

tud   tudor

Location: 2-97.
References: Cell 43:97 (85), Roux 195:302 (86).
Phenotype: Maternal. Embryos lack pole plasm and pole cells. Abdominal segmentation defective (similar to osk but less extreme). Escapers may hatch and develop into sterile adults.
Cytology: 57B13-57C1 (uncovered by Df(2R)Pu-rP133).

tup   tailup

Location: 2-54.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Head broad. Germ band shortening apparently ceases early resulting in the posterior most three segments remaining on dorsal side of embryo.
Alleles: IIIB, IIIE.

twi   twist

Location: 2-100.
References: Genet 105:615 (83), Roux 193:267 (84) Frohnhofer and Nüsslein-Volhard unpublished.
Phenotype: Embryonic lethal. Partially dorsalised. Ventral furrow is missing and embryo twisted or coiled in egg case, often with the posterior side up. Similar to *sna*. No maternal effect in germ line chimeras. *twi*/+ embryos have delayed ventral furrow formation.
Cytology: 59B6-D5 (uncovered by Df(2R)31 but not by Df(2R)23).
Alleles: EMS induced: ID, IIE, IIH, D5, O5, X10 (all stg), IG (ts).

ush   u-shaped

Synonym: l(2)19.
Location: 2-0.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Germ band does not shorten. Lateral fusion of anterior and posterior hypoderm.
Cytology: 21C8-E1 (uncovered by Df(2L)al).
Alleles: 19, IIA (both stg).

vas   vasa

Location: 2-51.
References: Roux 195:302 (86).

Phenotype: Maternal. Lacks pole cells, pole plasm and abdominal segments (c/f *osk*). Differs from other *osk* group mutants in that eggs also have abnormal shape.
Cytology: 35B.
Alleles: EMS induced: B5, D1, O11, O14, Q6, Q7, PD.

vls   valois
Location: 2-53.
References: Roux 195:302 (86).
Phenotype: Maternal. Lacks pole cells, pole plasm and abdominal segments (c/f *osk*). Differs from other *osk* group mutants in that the majority of embryos die during early syncytial divisions.
Cytology: 38A-E (uncovered by Df(2L)TW2 and Df(2L)TW84).
Alleles: EMS induced: PE, PG, RB.

vss-1   variable size and shape
Location: 3-
References: C. Nüsslein-Volhard unpublished.
Phenotype: Maternal. Produces eggs of variable size and shape.
Alleles: EMS induced: 258, 675.

wg   wingless
Location: 2-30.
References: Nat 287:795 (80), Roux 193:267 (84).
Phenotype: Embryonic lethal. Segment polarity phenotype. Denticle bands are duplicated at the expense of naked cuticle.
Cytology: 28A (N. Baker).
Alleles: EMS induced: 6K, 7L, IG, IJ, IN, IIS, IIID (all stg), IL (ts).

yrt   yurt
Location: 3-52.
References: Roux 193:283 (84).
Phenotype: Embryonic lethal. Embryos have a dorsal posterior hole.
Cytology: 87E12-F12 (uncovered by Df(3R)126 but not by Df(3R)kar-SZ8).
Alleles: EMS induced: 5G*, 9G,10H (wk).

zip   zipper
Location: 2-107.
References: Roux 193:267 (84).
Phenotype: Embryonic lethal. Embryos open anteriorly.
Cytology: 60E9-F1 (uncovered by Df(2R)IIX62 but not by Df(2R)SB1).
Alleles: EMS induced: ID (stg), IIF (med), (plus 16 discarded alleles).

Tübingen Stocklist - Genes

$+^{\pi}$ 1533
a 152
abdA^{M1} 651 1052
AbdB^{M1} 1051 1052
AbdB^{M5} 652
ac^3 1
Adh^{47} 148
Adh^{n2} 155 156 396 404 411 416 469
    472 544 560 1470 1473
    1476-1478 1511 1512 1517 1518
Adh^{n5} 462
Adh^{nB} 378
Adh^{nf3} 409
al 149-152 400 420 1457
alb^{404} 653
amx 2
Antp^{4B} 1293
Antp^{73b} 654
Antp$^{73b+R40}$ 655
Antp^{B} 656
aop^{IIS} 153
aop^{IP} 154
ap^{Xa} 1508
arm^{XK} 123
arr^{IB69} 278
arr^{IIW84} 279
ast 494
Asx^{IIF51} 280
Asx^{XF23} 637
aur^{074} 1187
aur^{175} 1257
aur^{287} 1188
B 30 66
B^{S} 3 140 1465 1497
B^{S-} 4
b 149-152 155-235 392 400 404 405
    411 416-420 427 435 448 460 461
    465 469 472 489-492 502-504 509
    512 513 517 525-527 544 548 560
    567 603 1455 1457 1463 1470 1473
    1476-1478 1511 1512 1517 1518
b^{XL1} 379 380 1509
Ba^5 236
baz^{XI} 124
bb 9
Bc 237
bcd^{085} 657
bcd^{111} 1365
bcd$^{2-13}$ 658 1366 1367
bcd$^{23-16}$ 1368 1369
bcd$^{33-5}$ 1370 1402
bcd^{E1} 1255 1294 1351 1403 1404 1407
bcd^{E2} 1189 1295
bcd^{E3} 1190 1364
bcd^{E4} 1191 1296
bcd^{E5} 1072 1256
bcd^{GB} 659

bch^{10E113} 1297
bga1^{ftzP} 1528
bhe^{IJ119} 238
bhe^{IM112} 239
bib^{ID05} 240
bib^{IIID118} 241
bib^{IIP39} 242
bib^{IIV46} 243
bib^{IJ66} 244
bic 245-247
BicC^{C96} 248
BicC^{WC} 249-251
BicD^{21} 252
BicD$^{23-10}$ 253
BicD^{52} 254
BicD^{7134} 157-161 255 489 490
BicD$^{7134-R26}$ 491
BicD^{IIIE} 162 163 256-258
BicD^{YC71} 259
bicF 660
Binsn 5
bip^{653} 1074
Bl 150 204 205 435 451 461 465 528
brh^{IB} 281
brh^{IID61} 282
bsk^{IIJ04} 260
bsk^{IIP71} 261
btd^{IIIA} 125
bw 152-154 158 159 161 163 172 178
    189-202 206 209 220 226-229
    238-244 246 247 249-251 256 257
    260-265 267-301 303-355 357-375
    387 388 392 396 417 418 422-425
    447 449 454 460 470 473 476 479
    480 482-488 490 492 493 495 499
    505 506 508 509 514 523 529-534
    538-542 547 548 551 556 557 559
    564-566 571-584 586-588 590-592
    594-596 598 601 602 604 605 607
    608 612 613 615 616 618 619
bw^{+} 1464
bw^{11} 381
bw^{V1} 517 1468
bx 698 755 1442
bx^3 1282
bx^{34e} 661
bxd^{110} 1442
bxd^{13D49} 1081
c 150-152 204 435 458 461 465 525
C(1)DX 6-8
C(1)M3 9
C(1)RM 10 11
ca 500 651 657 670-672 675 676
    679-686 708 725 727-731 733
    779-781 784-932 934-939 943-985
    992 993 999 1000 1013-1018 1023
    1029 1030 1038 1040 1051 1052
    1058 1067 1068 1074-1077
    1079-1082 1085-1138 1140-1153
    1155-1164 1167 1170-1176
    1178-1188 1190-1204 1207-1234

Tübingen Stocklist - Chromosomes

CHROMOSOME 1 CHROMOSOMES

1	ac^3	D268
2	amx lz^9 v	M120
3	B^S Y	A301 A302 F302-F350
		G301-G317 G319-G347
		G349-G359 G361-G383
		G385-G397 G399 G801-G829
4	B^{S-} v$^+$ Y	D104
5	Binsn	D129 D130
6	C(1)DX,w cv	A102
7	C(1)DX,y f	D103 D107
		D118-D122 D720
8	C(1)DX,y w f	D101 D108 D116
		D125 D126 D128
		D132 D135 D144
		D145 D148 D152
9	C(1)M3,y^2 bb	D806
10	C(1)RM,y	D260 D261
11	C(1)RM,y^2	A326 D826
12	C1B	A103 M102 M103
13	ct^{YI} w	B113
14	Df(1)16-3-22	D147
15	Df(1)259-4	D143
16	Df(1)62g18	D119
17	Df(1)64c18,g sd	D116
18	Df(1)64c4	D120
19	Df(1)A113	D132
20	Df(1)A94	D112
21	Df(1)bb^{1158},y	D102
22	Df(1)C149	D140
23	Df(1)C246	D123
24	Df(1)C52	D151
25	Df(1)ct$^{268-42}$,y	D146
26	Df(1)ct^{J4}	D144
27	Df(1)D15,v f	D148
28	Df(1)DCB1-35b	D142
29	Df(1)dm^{75e19},f^{36a}	D133
30	Df(1)g^1,f B	D124
31	Df(1)GA112	D104
32	Df(1)HA32	D106
33	Df(1)HF366	D134
34	Df(1)JA26	D130
35	Df(1)JA27	D136
36	Df(1)JC19	D117
37	Df(1)JC70	D115
38	Df(1)JF5,f	D105
39	Df(1)KA10,y Hw	D131
40	Df(1)KA14	D150
41	Df(1)KA7	D145
42	Df(1)mal^3 Dp(1;1)sc^{V1},y^2 ct^6 f y$^+$	D121
43	Df(1)N$^{264-39}$,y w	D122
44	Df(1)N^8,y f^{36a}	D129
45	Df(1)N105	D139
46	Df(1)N12,ras v	D110
47	Df(1)N19	D137
48	Df(1)N73	D141
49	Df(1)RA2	D149
50	Df(1)RC40	D111
51	Df(1)run^{III2},y f^{36a}	D114
52	Df(1)S39	D113
53	Df(1)sc^8 In(1)sc^8,w^a	D101
54	Df(1)sc^{J4},sc^{J4}	D107
55	Df(1)sc^{S2},y	D212
56	Df(1)sd^{72b}	D109

57	Df(1)svr,svr spl ras^2 fw	D103
58	Df(1)v^{L15},y	D152
59	Df(1)v^{L3}	D153
60	Df(1)w$^{258-45-64}$,y^2 sn^3	D125
61	Df(1)w^{67k30}	D128
62	Df(1)X12,y sc	D118
63	Dp(1;f)z^9,z	D107
64	FM0	M125-M128
65	FM3	M106 M108 M109 M111 M112
66	FM4,B	D143 D146
67	FM6	A101 D110 D112 D113 D123
		D127 D137-D142 D147 D151
68	FM7	A104 B101-B122 D104 D105
		D109 D114 D115 D117 D136
		D149 M105 M110 M122-M124
69	FM7,1(1)TW9	A105 M101 M104
70	FM7a	D111
71	FM7c	D106 D131 D133 D134 D150
72	fs(1)1182,v^{24}	M111
73	fs(1)572,v^{24}	M109
74	fs(1)K10 w f mal	M103
75	fs(1)K10^{254}	M101
76	fs(1)M22^{1136}	M128
77	fs(1)M23^{1444}	M125
78	fs(1)M23^{5087}	M127
79	fs(1)M57^{341}	M126
80	fs(1)M57^{358}	M129
81	fs(1)N^{211} g f	M123
82	fs(1)ph^{1901} v g f	M122
83	fs(1)pmd^{758}	M130
84	fu^{ECN}	M102
85	gd^{190}	M104
86	gt^{YA} w	B110
87	In(1)AM	D124
88	In(1)B^{M1} Df(1)mal^8,y v	D108
89	M(1)o^{Sp}	A101
90	N^{XK} y	B118
91	ot	D138
92	ovo^{D1} v^{24}	M113
93	ras v Ub1	B103
94	ras v Ub1^h	B105
95	sc cv ct v g^2 f	A104
96	sc ec cv swa^{15}	M112
97	sc^8 w$^+$ Y	A105
98	sdt^{XN} y	B120
99	seg cv v	M107
100	spl^{7L48}	Z761
101	svb^{YD} w	B109
102	swa^{14} ct v	A512 M105
103	swa^{14} v fs(1)N^{211}	M132
104	swa^{T573} ct v	M106
105	T(1;Y)2,y$^+$	D127
106	v cv run^{YP}	B106
107	v gd^2	M116
108	v gd^3	M115
109	v gd^4	M114
110	v gd^5	M117
111	v gd^6	M118
112	v gd^7	M119

113　v^{24} gd^1............M108
114　w............A102 A210 D267 D717
115　w otd^{YH}............B102
116　w rtv^{YA}............B115
117　w run^{YC}............B112
118　w sog^{XM42}............B122
119　w tsg^{YB}............B119
120　w^+ Y............D116 D118-D120
121　w^a N^{55e11}............D126
122　y............A216 A324 A326 D269 D272
123　y arm^{XK}............B114
124　y baz^{XI}............B121
125　y btd^{IIIA}............B111
126　y cv $fs(1)pmd^{151}$ f car............M124
127　y cv swa^{82} v f............M110
128　y cv swa^{99} v f............M121
129　y ec cv ct v exd^{XP}............B108
130　y fog^{4a6}............B116
131　y ftd^{IV8}............B101
132　y hnt^{XE}............B104
133　y $l(1)E12^{ts}$ f^{36a}............A103
134　Y mal^+ y^+............D114
135　y phm^{XE}............B107
136　y tsg^{XB}............B117
137　y w N^{264-47}............A105
138　y w^a f^{36a}............A106
139　y^+ mal^+ Y............D142
140　y^+ w^+ B^S Y............D122
141　y^+ Y............D101 D102
142　y^+ Y mal............D108
143　y^+ Y mal^+............D121
144　y^+ Y mal^{106}............D138
145　y^2 Y............D103
146　$Y^S X.Y^L$ In(1)EN,y............D260 D261
147　z w mh^{1182}............M131

CHROMOSOME 2 CHROMOSOMES

148　Adh^{47} sna^{HG} cn vg............B222
149　al dp b dl pr cn sca sp............M201
150　al dp b pr Bl c px sp............A202
151　al dp b pr c px sp............A201
152　al dp b pr cn vg c a px bw mr sp...A207
153　aop^{IIS} cn bw sp............Z718
154　aop^{IP} cn bw sp............2258
155　b Adh^{n2} pr cn $eve^{3.77}$ sca............B219
156　b Adh^{n2} pr cn sca............A208
157　b $BicD^{7134}$ pr cn $stau^{D3}$............M749
158　b $BicD^{7134}$ $stau^{HL}$ bw............M748
159　b $BicD^{7134}$ tor^{RL3} cn bw............M733
160　b $BicD^{7134}$ tor^{WK} cn............M202
161　b $BicD^{7134}$ tud^{WC} bw............M282
162　b $BicD^{IIIE}$ cn............M244
163　b $BicD^{IIIE}$ cn $stau^{HL}$ bw............M747
164　b $cact^{A2}$ pr............M717
165　b $cact^{D12}$ pr............M718
166　b $cact^{D13}$ pr............M713
167　b $cact^{F11}$ pr............M711

168　b $cact^{F6}$ pr............M708
169　b $cact^{G8}$ pr............M719
170　b $cact^{H4}$ pr............M710
171　b $cact^{H8}$ pr............M720
172　b $cact^{HE}$ cn bw............M297
173　b $cact^{L14}$ pr............M721
174　b $cact^{O11}$ pr............M714
175　b $cact^{O9}$ pr............M715
176　b $cact^{Q6}$ pr............M707
177　b $cact^{S1}$ pr............M716
178　b $cact^{SG}$ cn bw............M296
179　b $cact^{U7}$ pr............M712
180　b $cact^{Y11}$ pr............M709
181　b dl pr cn vg^D............M208
182　b dl^{103} cn sca............K203
183　b dl^2............M210
184　b dl^3............M216
185　b dl^{B10} pr............M299
186　b dl^{I5}............M252
187　b dl^{O11} pr............M701
188　b dl^{U5} pr............M298
189　b dp cn bw............D264
190　b $l(2)Bld^{M6}$ pr cn wx^{wxt} bw............A222
191　b $l(2)HT1^{F1}$ pr cn wx^{wxt} bw............A227
192　b $l(2)HT1^{M9}$ pr cn wx^{wxt} bw............A223
193　b $l(2)HT10^{M10}$ pr cn wx^{wxt} bw......A224
194　b $l(2)HT11^{M11}$ pr cn wx^{wxt} bw......A225
195　b $l(2)HT2^{F2}$ pr cn wx^{wxt} bw............A228
196　b $l(2)HT4^{F6}$ pr cn wx^{wxt} bw............A230
197　b $l(2)HT5^{F7}$ pr cn wx^{wxt} bw............A231
198　b $l(2)HT6^{F4}$ pr cn wx^{wxt} bw............A229
199　b $l(2)HT7^{F13}$ pr cn wx^{wxt} bw......A232
200　b $l(2)HT8^{F20}$ pr cn wx^{wxt} bw......A233
201　b $l(2)HT9^{M5}$ pr cn wx^{wxt} bw............A221
202　b plw^{M36} pr cn wx^{wxt} bw............A226
203　b pr............A212
204　b pr Bl c If............A203
205　b pr Bl L^2 sp............A205
206　b pr cn sca exu^{PJ} bw............M285
207　b pr cn sca tud^{B36}............M235
208　b pr cn sha^{IN98}............Z249
209　b pr cn wx^{wxt} bw............A204
210　b pr cn wx^{wxt} If............A220
211　b pr eve^{R13}............Z771
212　b pr Pcl^{E90}............B231
213　b pr $stau^{C8}$............M729
214　b pr $stau^{G2}$............M284
215　b pr tk............D266
216　b pr twi^{D5}............Z769
217　b pr twi^{O5}............Z770
218　b pr twi^{X10}............Z774
219　b pr wbl^{E4}............M706
220　b pr wbl^{RP} bw............M270
221　b pr wbl^{T6}............M705
222　b scw^{C13} pr............B218
223　b scw^{N5} pr............B225
224　b scw^{O5} pr............B213

225	b scw^{S12} pr........................B216	
226	b sna$^{18.19}$ pr cn wx^{wxt} bw.........B224	
227	b sna$^{4.26}$ pr cn wx^{wxt} bw.........B226	
228	b tor^{PM} cn bw....................M741	
229	b tor^{WK} cn stau^{HL} bw..............M736	
230	b vas^{011} pr cn...................M723	
231	b vas^{014} cn.....................M728	
232	b vas^{D1} pr cn...................M724	
233	b vas^{D5} pr cn...................M727	
234	b vas^{Q6} pr cn...................M726	
235	b vas^{Q7} pr......................M725	
236	Ba^5.............................B230	
237	Bc Gla..................A206 K204 K210	
238	bhe^{IJ119} cn bw sp................Z231	
239	bhe^{IM112} cn bw sp................Z246	
240	bib^{ID05} cn bw sp.................Z210	
241	bib^{IIID118} cn bw sp..............Z749	
242	bib^{IIP39} cn bw sp................Z711	
243	bib^{IIV46} cn bw sp................Z728	
244	bib^{IJ66} cn bw sp.................Z227	
245	bic L^2...........................M245	
246	bic L^2 exu^{PJ} bw..................M735	
247	bic L^2 stau^{HL} bw.................M734	
248	BicC^{C96} pr cn sca................M249	
249	BicC^{WC} cn bw sp.................M221	
250	BicC^{WC} cn exu^{PJ} bw..............M740	
251	BicC^{WC} cn stau^{HL} bw.............M738	
252	BicD^{21}..........................M281	
253	BicD$^{23-10}$........................K202	
254	BicD^{52}..........................M283	
255	BicD^{7134} pr cn sca...............M241	
256	BicD^{IIIE} cn tud^{WC} bw.............M702	
257	BicD^{IIIE} pr cn exu^{PJ} bw..........M745	
258	BicD^{IIIE} pr cn sca...............M246	
259	BicD^{YC71}.........................M238	
260	bsk^{IIJ04} cn bw sp................Z296	
261	bsk^{IIP71} cn bw sp................Z712	
262	bw...........................D268	
263	cact^{99} cn bw sp.................M231	
264	cact^{IIIG} cn bw sp................M232	
265	cact^{PD} cn bw............M243 M261	
266	cact^{PD} dl....................M203	
267	cact^{UK} pr cn bw................M294	
268	cact^{UL} pr cn bw.......M233 M237 M247	
269	cact^{UW} pr cn bw.........M248 M250	
270	cact^{VQ} pr cn bw.........M251 M256	
271	cap^{HK} cn bw sp.................M732	
272	cap^{RK} cn bw sp.................M731	
273	ck^{IIB33},cn bw sp................Z265	
274	ck^{IIQ106},cn bw sp...............Z714	
275	ck^{IIU116},cn bw sp...............Z725	
276	cli^{IID18} cn bw sp................Z276	
277	cli^{IIE107} cn bw sp...............Z283	
278	cn arr^{IB69} bw sp................Z205	
279	cn arr^{IIW84} bw sp...............Z732	
280	cn Asx^{IIF51} bw sp...............Z285	
281	cn brh^{IB} bw sp.................Z204	

282	cn brh^{IID61} bw sp...............Z277	
283	cn bw egl^{WU} sp.................M222	
284	cn bw Kr.....................A504 B206	
285	cn bw sp............A217 B318 D270 D271	
286	cn bw sp Kr^2....................Z757	
287	cn bw sp Kr^{IIE57}................Z282	
288	cn bw sp Kr^{IIIA102}..............Z738	
289	cn bw sp Kr^{IO66}................Z252	
290	cn bw sp zip^{ID16}...............Z211	
291	cn bw sp zip^{IIF107}..............Z287	
292	cn cra^{IIG44} bw sp...............Z291	
293	cn cra^{IJ23} bw sp................Z226	
294	cn en^{IIB86} bw sp................Z269	
295	cn en^{IIIB24} bw sp...............Z739	
296	cn en^{IIT84} bw sp................Z722	
297	cn en^{IK57} bw sp.................Z233	
298	cn en^{IM99} bw sp.................Z245	
299	cn en^{IO34} bw sp.................Z251	
300	cn eve^{ID} bw sp................Z212	
301	cn eve^{IIR59} bw sp...............Z715	
302	cn exu^{PJ} If Kr^{XII}.............B201	
303	cn exu^{QR} bw.....................M260	
304	cn exu^{SB} bw.....................M273	
305	cn exu^{SC} bw.....................M272	
306	cn fai^{IIA25} bw sp...............Z260	
307	cn fai^{IIV56} bw sp...............Z729	
308	cn fas^{IC67} bw sp................Z209	
309	cn fas^{IIA92} bw sp...............Z262	
310	cn flb^{IF26} bw sp................Z214	
311	cn flb^{IIC82} bw sp...............Z273	
312	cn flb^{IIE07} bw sp...............Z279	
313	cn flb^{IIG31} bw sp...............Z290	
314	cn flb^{IIIB41} bw sp..............Z741	
315	cn flb^{IIIB92} bw sp..............Z742	
316	cn flb^{IIIC81} bw sp..............Z743	
317	cn flb^{IIIC87} bw sp..............Z744	
318	cn flb^{IIIF18} bw sp..............Z752	
319	cn flb^{IIL65} bw sp...............Z702	
320	cn flb^{IIW74} bw sp...............Z731	
321	cn flb^{IIX51} bw sp...............Z735	
322	cn flb^{IK35} bw sp................Z232	
323	cn flb^{IP02} bw sp................Z254	
324	cn flz^{IIG68} bw sp...............Z292	
325	cn flz^{IP04} bw sp................Z255	
326	cn gho^{IB104} bw sp...............Z207	
327	cn gho^{IP107} bw sp...............Z257	
328	cn hsk^{IM45} bw sp................Z243	
329	cn l(2)IA109 bw sp................Z201	
330	cn lin^{IIF103} bw sp..............Z286	
331	cn lin^{IIU35} bw sp...............Z723	
332	cn mam^{IB99} bw sp................Z206	
333	cn mam^{IF33} bw sp................Z215	
334	cn mam^{IIH57} bw sp...............Z295	
335	cn mam^{IIJ14} bw sp...............Z297	
336	cn mam^{IIL61} bw sp...............Z701	
337	cn mam^{IIV06} bw sp...............Z726	
338	cn mam^{IJ113} bw sp...............Z230	

339	cn mam^{IL115} bw sp....................Z242	
340	cn mam^{IL42} bw sp....................Z238	
341	cn pim^{IL97} bw sp....................Z240	
342	cn Psc^{IIN48} bw sp....................Z708	
343	cn Pu^{IIK105} bw sp....................Z299	
344	cn Pu^{IN18} bw sp....................Z247	
345	cn rib^{IIB44} bw sp....................Z267	
346	cn rib^{IK81} bw sp....................Z235	
347	cn scb^{IID83} bw sp....................Z278	
348	cn scb^{IIG113} bw sp....................Z293	
349	cn sha^{IIP118} bw sp....................Z713	
350	cn shg^{IG29} bw sp....................Z221	
351	cn shg^{IH81} bw sp....................Z224	
352	cn shn^{IB} bw sp....................Z202	
353	cn shn^{IM56} bw sp....................Z244	
354	cn sli^{IG107} bw sp....................Z223	
355	cn sli^{IG23} bw sp....................Z218	
356	cn stau^{D3}....................M254	
357	cn stau^{HL} bw....................M258	
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1469	T(1;2)Bld	K201 K209 K211
1470	T(1;2)odd$^{1.10}$,b Adh^{n2} pr cn sca	D229
1471	T(1;2)OR64	K201 K207-K209 K211
1472	T(1;2)rb^{+71g},ct^6 v	D135
1473	T(Y;2)eve$^{2.37}$,b Adh^{n2} pr cn sca	D249
1474	T(Y;2)G	D266
1475	T(Y;2)G44,y$^+$	D260
1476	T(Y;2)odd$^{2.31}$,b Adh^{n2} pr cn sca	D248
1477	T(Y;2)odd$^{4.13}$,b Adh^{n2} pr cn sca	D254
1478	T(Y;2)odd$^{4.25}$,b Adh^{n2} pr cn sca	D276
1479	T(Y;2)R31,y$^+$	D261

CHROMOSOME 1-3 CHROMOSOMES

1480	Dp(1;3)sc^{J4},M(3)i^{55}	A324
1481	Dp(1;3)sn^{13a1}	D144
1482	Dp(1;3)w^{+67k27}	D125
1483	Dp(1;3)w^{m49a}	D128
1484	Dp(3;1)P115,y	D342
1485	Dp(3;1)P115,y^2	D386
1486	Dp(3;Y)p^{92};st hb^{14F} e	D825
1487	Dp(3;Y)P92,p$^+$ Rg-pbx	D803
1488	T(1;3)FA11,ru st e ca	D364
1489	T(1;3)FA62,ru st e ca	D363
1490	T(1;3)FC8,ru st e ca	D333
1491	T(1;3)FM7^TM3	A512
1492	T(1;3)kar^{51}	D394
1493	T(1;3)O5,D	D339
1494	T(1;3)OR60	A302 D347
		K302-K307 K309
1495	T(1;3)OR60,y	A301
1496	T(1;3)ry^{35},D^3 cu kar	D338

1497 T(Y;3)B172 In(3R)B172 y^+ B^S........D806
1498 ·T(Y;3)MA9^{P}A109^D,y^+.................D826
1499 T(Y;3)MA9,ru st e ca..............D367
1500 T(Y;3)R87^{P}A121^D,y^+.................D398
1501 T(Y;3)SCB^{XT21}.......................D395

CHROMOSOME 1-4 CHROMOSOMES

1502 Dp(1;4)r^+,f^+......................D148

CHROMOSOME 2-3 CHROMOSOMES

1503 Dp(2;3)osp^3........................D263
1504 Dp(2;3)P32.........................D719
1505 Dp(2;3)sna$^{+2.40}$.....................D710
1506 Dp(2;3)Yan.........................D264
1507 Dp(3;2)ry^{+70hR}...............A501 D328
1508 T(2;3)ap^{Xa},ap^{Xa}...................B338
1509 T(2;3)CyO,b^{XL1} sca^{XL1}.............A209
1510 T(2;3)D11,Pc^3.....................D703
1511 T(2;3)eve$^{1.18}$,b Adh^{n2} pr cn sca....D232
1512 T(2;3)eve$^{5.5}$,b Adh^{n2} pr cn sca.....D251
1513 T(2;3)FC10,ru st e ca..............D368
1514 T(2;3)FC34,ru st e ca..............D320
1515 T(2;3)FC9,ru st e ca..............D393
1516 T(2;3)Me.........................D319
1517 T(2;3)odd$^{3.29}$,b Adh^{n2} pr cn sca....D250
1518 T(2;3)odd$^{5.1}$,b Adh^{n2} pr cn sca.....D275
1519 T(2;3)p^{XM54},Msc....................D314
1520 T(2;3)p^{XT126},ru st e ca...........D385
1521 T(2;3)P,dp b cn P e................D281
1522 T(2;3)P10.........................D343
1523 T(2;3)prd$^{2.27.3}$,b Adh^{n2} pr cn sca..D246
1524 T(2;3)RXE,ru st....................M353
1525 T(2;3)SM1^TM2.....................A501
1526 T(2;3)sna$^{2.40}$,b Adh^{n2} pr cn sca....D278
1527 Tf(3;2)262,ry$^+$ lac-z^{Hsp26} cn.......D280
1528 Tf(3;2)4304,ry$^+$ bgal^{ftzP}..........A507
1529 Tp(3;2)FA12,ru st e ca.............D384
1530 Tp(3;2)FC82,st e..................D379
1531 Tp(3;2)XT89,ru st e ca.............D383

CHROMOSOME 1-2-3 CHROMOSOMES

1532 T(Y;2)prd$^{5.12}$ T(2;3)prd$^{5.12}$,b Adh^{n2} pr
      cn sca........................D258

OTHER CHROMOSOMES

1533 +$^\pi$.......................A508 A509
1534 Canton-S..........................A506
1534 Oregon-R....................A502 A505

Tübingen Stocklist - Stocks

A: marker and balancer stocks
B: zygotic lethals (others and ours)
D: rearrangements
F: 3rd chromosome female steriles (from unpublished 1982 screen)
G: 3rd chromosome maternals (from unpublished 1982 screen)
H: 3rd chromosome egg shape (from unpublished 1982 screen)
K: stocks requiring careful maintenance
M: maternals (others and ours)
Z: zygotic lethals (from 1979 and 1980 screens published in Roux Archives 1984)

A101 $M(1)o^{SP}/FM6$
A102 $C(1)DX,w\ cv/w$
A103 $y\ 1(1)E12^{ts}\ f^{36a}/ClB$
A104 $sc\ cv\ ct\ v\ g^2\ f/FM7$
A105 $FM7,1(1)TW9/y\ w\ N^{264-47}/sc^8\ w^+\ Y$
A106 $y\ w^a\ f^{36a}$

A201 $al\ dp\ b\ pr\ c\ px\ sp/CyO$
A202 $al\ dp\ b\ pr\ Bl\ c\ px\ sp/SM1$
A203 $b\ pr\ Bl\ c\ If/CyO,1(2)100^{DTS}$
A204 $b\ pr\ cn\ wx^{wxt}\ bw$
A205 $b\ pr\ Bl\ L^2\ sp/CyO$
A206 $In(2L)t\ In(2R)Cy,Roi\ cn^2\ bw\ sp\ or/Bc$
      Gla
A207 $al\ dp\ b\ pr\ cn\ vg\ c\ a\ px\ bw\ mr\ sp/SM1$
A208 $b\ Adh^{n2}\ pr\ cn\ sca$
A209 $In(2LR)Pm,dp\ b\ bw^{V1};\ T(2;3)CyO,b^{XL1}$
      sca^{XL1}
A210 $w/Dp(2;1)TE59,w^+\ b/CyO$
A211 $pr\ pk\ pwn/CyO,pk$
A212 $b\ pr$
A213 $1(2)91^{DTS}\ b\ pr\ cn\ sca/CyO$
A214 $In(2R)mam^{N-2G},S\ Sp\ Tft\ mam^{N-2G}\ Pu^2/CyO$
A216 $y;\ CyO/In(2LR)Pm\ Dp(1;2)sc^{19},y^+\ bw^{V1}$
A217 $cn\ bw\ sp$
A218 $rdo\ hk\ Ddc^{n7}\ pr/CyO$
A219 $lt\ stw^3$
A220 $b\ pr\ cn\ wx^{wxt}\ If$
A221 $b\ 1(2)HT9^{M5}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A222 $b\ 1(2)Bld^{M6}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A223 $b\ 1(2)HT1^{M9}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A224 $b\ 1(2)HT10^{M10}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A225 $b\ 1(2)HT11^{M11}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A226 $b\ plw^{M36}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A227 $b\ 1(2)HT1^{F1}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A228 $b\ 1(2)HT2^{F2}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A229 $b\ 1(2)HT6^{F4}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A230 $b\ 1(2)HT4^{F6}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A231 $b\ 1(2)HT5^{F7}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A232 $b\ 1(2)HT7^{F13}\ pr\ cn\ wx^{wxt}\ bw/CyO$
A233 $b\ 1(2)HT8^{F20}\ pr\ cn\ wx^{wxt}\ bw/CyO$

A301 $B^S\ Y;\ T(1;3)OR60,y;\ dsx^D\ Sb\ e/TM6$
A302 $B^S\ Y;\ T(1;3)OR60;\ dsx^D/TM8$
A303 $ru\ st\ ri^{XT2}\ e\ ca/TM3,Sb$
A304 $Pr\ Dr/TM3,Sb\ tld^T$
A305 $ru\ h\ th\ st\ cu\ sr\ e^s\ ca$
A306 $ru\ h\ th\ st\ cu\ sr\ e^s\ Pr\ ca/TM6$

A307 $ftz\ ry^{506}/TM3,ry^{MC}$
A308 $Sb\ H^{32g}/TM2$
A309 $In(3L)P,gm/R\ Ly$
A310 $ru\ h\ th\ st\ cu\ sr\ e^s\ Tl^{5BRXV}\ ca/TM1,p^P$
      opa^T
A311 $ru\ th\ st\ ri\ roe\ p^P\ e^s\ ca$
A312 $ru\ st\ e\ ca$
A313 $Pr\ Dr/TM3,knk^T\ Sb$
A314 $Sb\ Tb/TM6C$
A315 $1(3)4^{DTS}/TM3,Sb$
A316 $Ki\ Sb\ M(3)w^{124}/TM2$
A317 $1(3)7^{DTS}\ st\ tra\ p^P/TM3,Sb$
A318 $ru\ h\ th\ st\ cu\ sr\ e^s\ Pr\ ca/TM6B$
A319 $Df(3R)4SCB,th\ st\ p^P/TM3,st^{X2}\ Sb$
A320 $st\ e$
A321 $ru\ kls\ st\ e\ tld^{6P41}/TM3,ru\ kls\ Sb$
A322 $TM1,kni^T\ p^P/TM2$
A323 $sbd^2\ bx^3\ pbx\ e^{11}/TM1$
A324 $y;\ Dp(1;3)sc^{J4},M(3)i^{55}/TM1,mwh$
A325 $Dfd/TM3$
A326 $C(1)RM,y^2/y;\ TM3,y^+/Sb$
A327 $TM3/TM1,p^P$
A328 $th\ st\ cp\ in\ ri\ p^P$
A329 $mwh\ th\ st\ ri\ roe\ p^P\ cu\ sr\ e^s$
A330 $cu\ kar\ red\ sbd$
A331 $st\ W\ cu\ ry\ ss/TM3$
A332 $hb^{14F}\ ry^{506}/TM3,ry^{MC}$

A401 $1(4)13/ey^D$
A402 $ci-D/Df(4)G$

A501 $Dp(3;2)ry^{+70hR};\ T(2;3)SM1^{\wedge}TM2;\ MRS$
A502 Oregon-R
A503 $sal^{IIA}\ pr/CyO;\ ry^{506}$
A504 $cn\ bw\ Kr/SM1;\ ry^{506}$
A505 Oregon-R
A506 Canton-S
A507 $Tf(3;2)4304,ry^+\ bgal^{ftzP}$
A508 $+^{\pi}$
A509 $+^{\pi};\ ru\ h\ th\ st\ cu\ sr\ e^s\ Pr\ ca/TM6$
A510 $CyO/1(2)amd^2\ Bl;\ TM3,Sb/Gl$
A511 $CyO/Ddc^{n27}\ pr;\ TM3,Sb/Gl$
A512 $T(1;3)FM7^{\wedge}TM3;\ swa^{14}\ ct\ v;\ st\ bcd^{E4}\ e$

B101 $y\ ftd^{IV8}/FM7$
B102 $w\ otd^{YH}/FM7$
B103 $ras\ v\ Ubl/FM7$
B104 $y\ hnt^{XE}/FM7$
B105 $ras\ v\ Ubl^h/FM7$

B106   v cv run^{YP}/FM7
B107   y phm^{XE}/FM7
B108   y ec cv ct v exd^{XP}/FM7
B109   svb^{YD} w/FM7
B110   gt^{YA} w/FM7
B111   y btd^{IIIA}/FM7
B112   w run^{YC}/FM7
B113   ct^{YI} w/FM7
B114   y arm^{XK}/FM7
B115   w rtv^{YA}/FM7
B116   y fog^{4a6}/FM7
B117   y tsg^{XB}/FM7
B118   N^{XK} y/FM7
B119   w tsg^{YB}/FM7
B120   sdt^{XN} y/FM7
B121   y baz^{XI}/FM7
B122   w sog^{XM42}/FM7

B201   cn exu^{PJ} If Kr^{XII}/CyO
B202   pr cn thr^{BH9} bw/CyO
B203   prd^{FR1}/CyO
B204   shn^{TD5}/CyO
B205   sna^{IIG} cn twi^{IIH07} bw sp/CyO
B206   cn bw Kr/SM1
B207   pr cn thr^{BH9} bw/CyO
B208   sna^{IIG} sha^{IN}/CyO
B209   vg^{D} Asx^{XF23} Pcl^{XM3}/CyO
B210   prd$^{32.12}$/CyO
B211   net dpp^{27} ed Su(dx)^{2}/CyO
B212   Pcl^{XM3}/CyO
B213   b scw^{O5} pr/CyO
B214   dp b esc^{6} cn bw/CyO
B215   dpp^{4}/CyO
B216   b scw^{S12} pr/CyO
B217   odd^{IIID} Sp cn eve^{ID}/CyO
B218   b scw^{C13} pr/CyO
B219   b Adh^{n2} pr cn eve$^{3.77}$ sca/CyO
B220   odd^{IIID} dp prd^{IIB} cn eve^{ID}/CyO
B221   prd$^{2.45}$ b Adh^{n2} pr cn sca/CyO
B222   Adh^{47} sna^{HG} cn vg/CyO
B223   shn^{TD5}/CyO
B224   b sna$^{18.19}$ pr cn wx^{wxt} bw/CyO
B225   b scw^{N5} pr/CyO
B226   b sna$^{4.26}$ pr cn wx^{wxt} bw/CyO
B227   Pcl^{X21}/CyO
B228   pr cn thr^{BH9} bw/CyO
B229   shn^{TD5}/CyO
B230   Ba^{5}/SM5
B231   b pr Pcl^{E90}/CyO
B232   odd$^{1.36}$ b Adh^{n2} pr cn sca/CyO

B301   kni^{IID} hb^{7M} cu sr e^{s} ca/TM3,Sb
B302   th st cp in ri kni^{301} p^{P} Ubx/TM6
B303   kni^{IID} hb^{9K67} cu sr e^{s} ca/TM3,Sb
B304   hb^{G1}/TM1
B305   th st cp in ri bcd$^{2-13}$ hb^{9R12}/TM3
B306   hb^{FF8}/TM3,Sb

B307   th st cp in ri p^{P} hb^{349}/TM3,Sb
B308   Df(3R)PXT15,st p^{P} e t11^{L10}/TM3,Sb
B309   st kni^{17} e/TM3
B310   th st cp kni^{301} in ri p^{P} e t11^{L10}/TM3,Sb
B311   st kni$^{1-19}$ e/TM3
B312   st tsl^{146} t11^{L10}/TM3,Sb
B313   ri Scr^{XF9} p^{P} Ubx/TM1
B314   h^{ILK} th st ftz^{7B} cu sr e^{s} ca/TM3,Sb
B315   Pcl^{XL5}/TM3,Sb
B316   AbdB^{M5}/TM1
B317   mwh jv st red sbd AbdB^{M1} e^{11} ro ca/TM1
B318   cn bw sp; MRS,Sb/kar^{2} shm^{S8}
B319   In(3R)Msc p^{P}/TM3,Sb
B320   mwh jv st red sbd Ubx^{XM1} abdA^{M1} AbdB^{M1} e^{11} ro ca/TM1
B321   h^{ILK} th st opa^{5H} cu sr e^{s} ca/TM3,Sb
B322   th st cp in ri p^{P} shm^{E320}/TM3,Sb
B323   abdA^{M1} mwh jv st red sbd e ro ca/TM1
B324   Antp^{B}/TM1
B325   Antp$^{73b+R40}$/TM3,Sb
B326   ru st hb^{XT79} e ca/TM3,Sb
B327   Scr^{XT130}/TM3,tld^{T}
B328   Antp^{73b}/TM3,Sb
B329   zen^{W36} red e/TM3,Sb
B330   th st cp kni^{357} in ri p^{P}/TM3,Sb
B331   th st cp kni^{301} in ri p^{P}/TM3,Sb
B332   st kni^{FC} e/TM3
B333   st hb^{FB} e/TM3,Sb
B334   E(spl)^{R1}/TM3
B335   ru kls th st cp in ri ftz^{E193} p^{P}/TM3,Sb ru kls
B336   Ubx^{C1}/TM1
B337   th st pb p^{P}/TM3,Sb
B338   In(3R)Rg(pbx); T(2;3)ap^{Xa},ap^{Xa}
B339   ru h th st cu Ubx^{79f} sr e^{s} ca/TM1
B340   neu^{IIIA}/TM3,Sb
B341   th st cp in ri ftz^{E66} p^{P}/TM3,Sb
B342   th st cp in ri ftz^{E193} p^{P}/TM3,Sb
B343   th st cu sr e^{s} fkh^{E200}/TM3,Sb
B344   th st in ri kni^{301} p^{P} nos^{L7} e/TM3
B345   kni^{IL} pum^{680}/TM3
B346   kni^{IL} roe p^{P} osk^{301}/TM3
B347   Df(3R)9A99,th st cp in ri kni^{301} cu e^{s} ca/TM3
B348   hb^{C8}/TM3,Sb
B349   kni^{IL} tsl^{691} e/TM3
B350   th st ri p^{P} pum^{TE3} e t11^{L10}/TM3

D101   C(1)DX,y w f/Df(1)sc^{8} In(1)sc^{8},w^{a}/y^{+} Y
D102   Df(1)bb^{1158},y/y^{+} Y; K-pn ca
D103   C(1)DX,y f/Df(1)svr,svr spl ras^{2} fw/y^{2} Y
D104   Df(1)GA112/FM7/B^{S-} v^{+} Y
D105   Df(1)JF5,f/FM7
D106   Df(1)HA32/FM7c
D107   C(1)DX,y f/Df(1)sc^{J4},sc^{J4}; Dp(1;f)z^{9},z

D108 $C(1)DX,y$ w $f/In(1)B^{M1}$ $Df(1)mal^8,y$ v/y^+ Y mal
D109 $Df(1)sd^{72b}/FM7$
D110 $Df(1)N12,ras$ v/FM6
D111   Df(1)RC40/FM7a
D112   Df(1)A94/FM6
D113   Df(1)S39/FM6
D114 $Df(1)run^{III2},y$ $f^{36a}/FM7/Y$ mal^+ y^+
D115   Df(1)JC70/FM7
D116 $C(1)DX,y$ w $f/Df(1)64c18,g$ sd/w^+ Y
D117   Df(1)JC19/FM7
D118 $C(1)DX,y$ $f/Df(1)X12,y$ sc/w^+ Y
D119 $C(1)DX,y$ $f/Df(1)62g18/w^+$ Y
D120 $C(1)DX,y$ $f/Df(1)64c4/w^+$ Y
D121 $C(1)DX,y$ $f/Df(1)mal^3$ $Dp(1;1)sc^{V1},y^2$ ct^6 f y^+/y^+ Y mal^+
D122 $C(1)DX,y$ $f/Df(1)N^{264-39},y$ w/y^+ w^+ B^S Y
D123   Df(1)C246/FM6
D124 $Df(1)g^1,f$ B/In(1)AM
D125 $C(1)DX,y$ w $f/Df(1)w^{258-45-64},y^2$ sn^3; $Dp(1;3)w^{+67k27}/+$
D126 $C(1)DX,y$ w f/w^a N^{55e11}; Dp(1;2)51b/+
D127 $T(1;Y)2,y^+/FM6$
D128 $C(1)DX,y$ w $f/Df(1)w^{67k30}$; $Dp(1;3)w^{m49a}/+$
D129 $Df(1)N^8,y$ $f^{36a}/Binsn$
D130   Df(1)JA26/Binsn
D131   Df(1)KA10,y Hw/FM7c
D132 $C(1)DX,y$ w $f/Df(1)A113$; $Dp(1;2)w^{+64b}/+$
D133 $Df(1)dm^{75e19},f^{36a}/FM7c$
D134   Df(1)HF366/FM7c
D135 $C(1)DX,y$ w f; $T(1;2)rb^{+71g},ct^6$ v
D136   FM7/Df(1)JA27
D137   Df(1)N19/FM6
D138   ot/FM6/y^+ Y mal^{106}
D139   Df(1)N105/FM6
D140   Df(1)C149/FM6
D141   Df(1)N73/FM6
D142   Df(1)DCB1-35b/FM6/y^+ mal^+ Y
D143   Df(1)259-4/FM4,B
D144 $C(1)DX,y$ w $f/Df(1)ct^{J4}$; $Dp(1;3)sn^{13a1}/Ki$
D145 $C(1)DX,y$ w $f/Df(1)KA7$; $Dp(1;2)v^{+65b}/+$
D146 $Df(1)ct^{268-42},y/FM4,B$
D147   Df(1)16-3-22/FM6
D148 $C(1)DX,y$ w $f/Df(1)D15,v$ f; $Dp(1;4)r^+,f^+/+$
D149   Df(1)RA2/FM7
D150   Df(1)KA14/FM7c
D151   Df(1)C52/FM6
D152 $C(1)DX,y$ w $f/Df(1)v^{L15},y$; $Dp(1;2)v^{+75d}/+$
D153 $C(1)DX,y$ w $f/Df(1)v^{L3}$; $Dp(1;2)v^{+63i}$

D201 $Df(2R)vg^D,b$ Adh^{n2} pr cn /CyO
D202 $Df(2R)vg^D/CyO,l(2)513^{DTS}$
D203 $Df(2L)osp^{29},Adh^{nf3}$ pr cn/CyO
D204 $Df(2R)vg^D,L^2$ sp/CyO
D205 $Df(2R)vg^B/CyO$
D206 $Df(2R)vg^C,L^2$ sp/CyO
D207 $Df(2L)VA18,pr/CyO$ $Dp(2;2)M(2)m^+$
D208 $Df(2R)ST1,noc^3$ Adh^{n5} pr/CyO

D209   Df(2L)TW9,Tft cn/CyO
D210 $Df(2L)TW137,cn$ bw/CyO $Dp(2;2)M(2)m^+$
D211   M(2)H-S5/CyO
D212 $Df(1)sc^{S2},y/+$; $CyO/Df(2)sc^{S2}$ $Dp(1;2)sc^{S2},cn$ M(2)S7
D213   Df(2L)TW84/CyO
D214 $Df(2L)TW50,cn/CyO$ $Dp(2;2)M(2)m^+$
D215   Df(2L)TW2/CyO
D216   Df(2R)PC4/CyO
D217 $Df(2R)L^{+48},Bl/CyO$
D218 $Df(2R)pk^{78k},sp/CyO$
D219 $Df(2L)TW119,cn$ bw/CyO,bw^{11}
D220 $Df(2L)TW201,b/CyO$ $Dp(2;2)M(2)m^+$
D221   Df(2L)PR31/SM1
D222 $Df(2R)pk^{78s}$ $In(2R)pk^{78s}/CyO$
D223   Df(2L)C/SM1
D224   Df(2L)75c In(2L)75c/CyO,b^{XL1}
D225 $Df(2L)esc^{P2}/CyO$
D226 $Df(2R)cn^{84.4}/CyRoi$
D227 $Df(2R)vg^D,l(2)91^{DTS}$ b Adh^{n2} pr cn/CyO
D228 $Df(2R)M-c^{33a}/In(2LR)bw^{V32g}$
D229 $T(1;2)odd^{1.10},b$ Adh^{n2} pr cn sca; CyO
D230 $Df(2R)cn^{12}/CyRoi$
D231 $Df(2R)cn^9/CyRoi$
D232 $T(2;3)eve^{1.18},b$ Adh^{n2} pr cn sca; CyO
D233 $Df(2L)S^3/SM1$
D234 $Df(2L)S^2/In(2L)Cy$ In(2R)Cy,E(S) Cy cn^2
D235   Df(2R)42,cn/SM1
D236 $Df(2L)M^{zB}/SM1$
D237 $Df(2R)cn^{84h80}/SM5$
D238   Df(2L)al/CyO
D239 $Df(2R)bw^5/CyO,bw^{11}$
D240   Df(2L)TW158,cn bw/CyO
D241   Df(2L)E55,rdo hk pr/CyO
D242 $Df(2R)cn^{79b9}/SM5$
D243   Df(2L)J-der-2/SM1
D244   Df(2L)Prl/CyO
D245 $Df(2R)UR1,b$ pr Bl c $If^R/SM1$
D246 $T(2;3)prd^{2.27.3},b$ Adh^{n2} pr cn sca; CyO
D247 $Df(2R)en^A/CyO$
D248 odd^{IIID} dp prd^{IIB} cn eve^{ID}/CyO; $T(Y;2)odd^{2.31},b$ Adh^{n2} pr cn sca
D249 odd^{IIID} dp prd^{IIB} cn eve^{ID}/CyO; $T(Y;2)eve^{2.37},b$ Adh^{n2} pr cn sca
D250 $T(2;3)odd^{3.29},b$ Adh^{n2} pr cn sca; CyO
D251 $T(2;3)eve^{5.5},b$ Adh^{n2} pr cn sca; CyO
D252 $Df(2L)cl^7/CyO$
D253 $Df(2R)AP1,b$ pr Bl c $If^R/SM1$
D254 odd^{IIID} dp prd^{IIB} cn eve^{ID}/CyO; $T(Y;2)odd^{4.13},b$ Adh^{n2} pr cn sca
D255   Df(2L)GdhA,dp L/CyO
D256   Df(2L)J-der-27/SM1
D257 $Df(2L)64j,L^2/CyO,Adh^{nB}$
D258 odd^{IIID} dp prd^{IIB} cn eve^{ID}/CyO; $T(Y;2)prd^{5.12}$ $T(2;3)prd^{5.12},b$ Adh^{n2} pr cn sca
D259 $Df(2R)cn^{79b13}/SM5$
D260 $C(1)RM,y/Y^S X.Y^L$ In(1)EN,y; In(2L)Cy In(2R)Cy,Cy cn^2; $T(Y;2)G44,y^+$

D261　$C(1)RM,y/Y^{Sx}.Y^{L}$ In(1)EN,y; In(2L)Cy
　　　In(2R)Cy,Cy cn^{2}; T(Y;2)R31,y^{+}
D262　Df(2L)A72,b cn bw/CyO
D263　Dp(2;3)osp^{3}/TM3
D264　b dp cn bw; Dp(2;3)Yan/TM2
D265　Df(2L)TE116-prx4,dp b cn sp/CyO
D266　T(Y;2)G; b pr tk
D267　w; Df(2R)en^{B}/CyO
D268　Dp(2;Y)bw^{+},sc^{8} bw^{+}/ac^{3}; bw
D269　y; Df(2R)B80,y^{+}/SM1
D270　Dp(2;Y)L12,B^{S}; cn bw sp
D271　Dp(2;Y)L9,sp^{+}; cn bw sp
D272　y; Df(2R)Rsp^{Ins31}/Gla
D273　Df(2L)TE116-prx3,dp b cn bw/CyO
D274　Df(2L)$b^{75.2}$/CyO
D275　T(2;3)$odd^{5.1}$,b Adh^{n2} pr cn sca; CyO
D276　odd^{IIID} dp prd^{IIB} cn eve^{ID}/CyO;
　　　T(Y;2)$odd^{4.25}$,b Adh^{n2} pr cn sca
D277　Df(2L)TE116-prx18,dp b cn bw/CyO
D278　T(2;3)$sna^{2.40}$,b Adh^{n2} pr cn sca; CyO
D279　Df(2L)esc^{P3}/CyO
D280　Tf(3;2)262,ry^{+} lac-z^{Hsp26} cn; ry
D281　T(2;3)P,dp b cn P e; CyO; TM1
D282　Df(2L)TW1,Tft l(2)74i/CyO
D283　Df(2R)SB1,b pr B1 c If^{R}/SM1
D284　Df(2L)H68,dp b M/CyO Dp(2;2)M(2)m^{+}
D285　Df(2L)VA21,pr/CyO Dp(2;2)M(2)m^{+}
D286　Df(2L)TW161,cn bw/CyO
D287　Df(2L)CA53/CyO
D288　Df(2L)tkv^{SZ2},al b/SM1
D289　Df(2R)42490/CyO
D290　In(2LR)Sco^{R+1}/CyO
D291　Df(2L)T317,b Adh^{n2} pr cn sca/CyO
D292　Df(2L)H20,b Adh^{n2} pr/CyO
D293　Df(2L)$prd^{1.7}$,b Adh^{n2} pr cn sca/CyO
D294　odd^{IIID} dp prd^{IIB} cn eve^{ID}/CyO;
　　　Df(2L)$prd^{1.25}$ T(Y;2)$prd^{1.25}$,b pr cn sca
D295　Df(2R)$eve^{1.27}$,cn sca bw sp/CyO
D296　Dp(2;2)GYL,dp cn bw/CyO,esc^{2}
D297　Df(2R)gsb^{ES1},b pr cn wx^{wxt} If/CyO
D298　Df(2L)ed^{SZ1},al b/SM5
D299　Df(2R)H23/CyRoi

D701　In(2LR)TE146^{GR15}/CyO
D702　Df(2L)cl^{1},Adh^{n2} cn bw/CyO
D703　T(2;3)Dll,Pc^{3}; SM1
D704　Df(2R)Pu^{+D17},nw^{D} Elp^{RD17} Pin^{4+}/SM1
D705　Df(2R)Pu^{rP133},c px sp/SM1
D706　dpp^{48} Sp cn/CyO; Dp(2;1)G146
D707　Df(2L)DTD2/Dp(2;2)MVD2,ast ed dp cl
D708　In(2LR)TE146^{SZ4}/CyO
D709　Df(2R)S60,b pr cn bw/CyO
D710　Df(2L)$sna^{2.40L}$ Dp(2;2)GYL^{R}/CyO;
　　　Dp(2;3)$sna^{+2.40}$/TM3,Sb
D711　Df(2R)tor^{40R5}/CyRoi
D712　Df(2R)tor^{40R6}/CyRoi
D713　Dp(2;1)G146/+; dpp^{37} Sp cn bw/CyO
D714　In(2LR)Gla Dp(2;2)DTD48,cn/CyO
D715　Dp(2;1)G146/+; Df(2L)DTD2/CyO
D716　In(2LR)TE146^{SR14}/CyO

D717　w; Dp(1;2)TE116,al dp b w^{+} cn sp/SM5
D718　Df(2R)XM82/CyO
D719　Dp(2;3)P32/TM1
D720　C(1)DX,y f/Dp(2;Y)R31; exu^{QR}/CyO

D301　Df(3L)vin^{3},ru h gl e ca/TM3,Sb
D302　Df(3L)vin^{7},ru h gl e ca/TM3,Sb
D303　Df(3L)Ly,mwh/TM1,jv
D304　Df(3L)th^{SS117},h ri e^{S}/TM1
D305　Df(3L)st^{SS106},h ri e^{S}/TM1
D306　Df(3L)ri^{79c},th st/TM3,Sb
D307　Df(3R)SCB^{XL2}/TM3,Sb
D308　Df(3L)ASC,cp in ri p^{P}/TM3,Sb
D309　Df(3R)Ns^{RC7},e^{11}/TM3,Sb
D310　Df(3R)Scr,p^{P} e/TM2,p^{P}
D311　Df(3R)R10/TM1
D312　Df(3R)9A99,ru h th st cu sr e^{S} ca/TM6
D313　Df(3R)roe^{X54},st e/TM3,Sb
D314　T(2;3)p^{XM54},Msc; TM1,p^{P}
D315　Df(3R)4SCB/TM3,Sb
D316　Df(3R)roe^{XM4},ru h th st Msc/TM3,Sb
D317　Df(3R)dsx^{D+R5} bx sr e^{S}/TM3,Sb
D318　Df(3R)cu^{X1},$M-S31^{-}$/TM1
D319　Df(3R)M-S31; T(2;3)Me
D320　T(2;3)FC34,ru st e ca; TM3
D321　Df(3R)kar^{D3} In(3R)AFA,e/cu kar H e^{4}
D322　Df(3R)E229/TM3,Sb
D323　Df(3R)T-63A,ri cu sr e^{S}/TM3,Sb
D324　Df(3R)kar^{D1} In(3R)AFA e/cu kar Sb
D325　Df(3R)kar^{D2} In(3R)AFA,e/cu kar Sb
D326　Df(3R)p^{XM66},Msc/TM3,Sb
D327　Df(3R)kar^{3J},ru h th st sr e^{S} ca/TM3,Sb
D328　Dp(3;2)ry^{+70hR}; Df(3R)ry^{85}/MKRS,Sb
D329　Df(3R)kar^{31}/MRS,Sb
D330　Df(3R)ry^{61h}/MKRS,Sb
D331　Df(3R)by^{62},red e/TM1,p^{P}
D332　Df(3R)ry^{36}/MKRS,Sb
D333　T(1;3)FC8,ru st e ca; TM3
D334　Df(3R)red^{31}/MRS,Sb
D335　Df(3R)red^{P52}/TM1
D336　Df(3R)red^{P93},l(3)tr Sb/In(3L)P
　　　In(3R)P18,Me Ubx e^{4}
D337　Df(3R)red/TM1
D338　T(1;3)ry^{35},D^{3} cu kar; TM3,Sb
D339　T(1;3)O5,D; TM3,Sb
D340　LVM/Df(3R)sbd^{105},p sbd^{105} bx sr e^{S}
D341　Df(3R)bxd^{100},red/TM1
D342　Dp(3;1)P115,y; Df(3R)P115,mwh jv red
　　　e^{11}/TM1
D343　T(2;3)P10; CyO; TM3
D344　Tp(3)S462/TM1
D345　Df(3R)Ubx^{109},gl e^{S}/Dp(3;3)P5,Sb
D346　Df(3R)P9/Dp(3;3)P5
D347　Df(3R)XTA1,M(3)w^{-} th st ri roe
　　　p^{P}/TM3,Sb; T(1;3)OR60
D348　Df(3R)by^{10},red e/TM2,Ubx e
D349　Tp(3)bxd^{110},bx bxd^{110} sr e^{S}/bx^{34e} Mc
D350　Df(3R)e^{F1} In(3R)C,cd/cu H e^{4}
D351　Df(3R)$5B^{RXP}$,kni^{IID} ca/TM3,Sb
D352　Df(3R)$Antp^{Ns+R17}$/TM3,Sb

D353 Df(3R)dsx$^{Mas+R48}$/TM2

D354 Df(3R)ftz^{RARU} Df(3L)ASC,ri/TM3,Sb

D355 Df(3R)4SCB/Dp(3;3)Antp^{BL}Msc^R,p^p

D356 Df(3R)p^{13},e/TM3,Sb

D357 Msc^{T2} mwh tuh-3/TM3,Sb

D358 Mc/Df(3R)C4

D359 Df(3R)e^{N19}/TM2

D360 Df(3R)ry^{1402}/MKRS,Sb

D361 Df(3R)sbd^{45},mwh e/TM2

D362 Df(3R)Msc^{T1},mwh tuh-3/TM3,Sb

D363 T(1;3)FA62,ru st e ca; TM3

D364 T(1;3)FA11,ru st e ca; TM3

D365 Df(3R)e^{D7}/TM1,cu

D366 Df(3R)e^{N12}/TM1,cu

D367 T(Y;3)MA9,ru st e ca; Ms(3)R24/TM3,Sb

D368 T(2;3)FC10,ru st e ca; TM3

D369 Df(3R)D1^{102}/DcxF

D370 Df(3R)D1^{A143}/DcxF

D371 Df(3L)ri^{XT104},ru st e ca/TM1

D372 Dp(3;3)bxd^{110} Df(3R)D1^{X43}/In(3R)?,Sb Tb

D373 Df(3R)Scx^{w+RX2},red e/TM3,Sb

D374 Dp(3;3)D1/TM3

D375 Dp(3;3)Su^{33}-M(3)w/TM1

D376 Dp(3;3)Su^1-M(3)w,e^S E(spl)/TM3,Sb

D377 Df(3R)p^{XT9},st e ca/TM3,Sb

D378 Df(3R)e^{R1}/TM3,Sb

D379 Tp(3;2)FC82,st e; TM3

D380 Df(3R)D605/TM3

D381 Df(3R)p^{XT9},st/In(3R)Antp^B,p^p

D382 Df(3R)e^{F4} In(3R)C,cd/H Pr e^4 ca

D383 Tp(3;2)XT89,ru st e ca; TM3,Sb

D384 Tp(3;2)FA12,ru st e ca; TM3

D385 T(2;3)p^{XT126},ru st e ca; TM3

D386 Dp(3;1)P115,y^2/FM6; Df(3R)P9,red/TM1

D387 Df(3R)D1^{X43},roe p^p/TM1,p^p

D388 Df(3R)126c,kar^2/MKRS

D389 Df(3R)126d,p^{126d} kar^2 red/MKRS

D390 Df(3R)1C4a/MRS

D391 In(3LR)p^{XT117},ru st e ca/TM3

D392 Df(3R)kar^{SZ8}/TM3

D393 T(2;3)FC9,ru st e ca; TM3

D394 T(1;3)kar^{51}; MKRS

D395 T(Y;3)SCB^{XT21}; ru h th st cp in ri
      Scr^{XF9} p^p cu sr e^s ca/TM3,Sb

D396 Df(3R)LIN,th st cp in ri e/TM3,Sb

D397 Df(3R)Ser$^{+R82f24}$,Tb/TM6

D398 T(Y;3)R87^{P}A121^D,y$^+$; TM6

D399 Df(3R)ro^{82b},Pr/TM3,Sb

D801 Df(3R)ro^{80b}/TM6

D802 Df(3R)ro^{XB3},Tb/TM1

D803 Dp(3;Y)P92,p$^+$ Rg-pbx; Df(3R)p^{XT118},ru
      st e ca/TM3

D804 Df(3R)roe^{ST1}/TM3,Sb

D805 Df(3R)p^{XT104},ru st e ca/TM3

D806 C(1)M3,y^2 bb/Y^{S}X.Y^L In(1)EN,y;
      T(Y;3)B172 In(3R)B172 y$^+$ B^S; TM6

D807 Df(3R)p^{XT103},ru st e ca/TM3

D808 Df(3R)p^{XT27},ru st e ca/TM3

D809 Df(3R)p^{XT26},ru st e ca/TM3

D810 Df(3R)p^{XT15},ru st e ca/TM3

D811 In(3L)ri^{XT101},ru st e ca/TM3

D812 Df(3L)ri^{XT1},ru st e ca/TM3

D813 Df(3L)ri^{XT106},ru st e ca/TM3

D814 In(3L)ri^{XT102},ru st e ca/TM3

D815 Df(3R)9Q^{RX1},mwh th st ri roe p^p sr e
      ca/TM3

D816 Df(3R)5B^{REK1},ca/TM3

D817 Df(3R)5B^{RXQ},ru h th st cu sr e^s ca/TM3

D818 Df(3R)5B^{RXI},e ca/TM3

D819 Df(3R)5B^{RXP},e ca/TM3

D820 Df(3R)N74/TM3,Sb

D821 Df(3R)AP37/TM3,Sb

D822 Df(3R)ry^{1608}/MKRS,Sb

D823 Df(3R)LIN,st p^p e/TM3,Sb

D824 Df(3R)RPB,ru st e/TM3,Sb

D825 Dp(3;Y)p^{92};st hb^{14F} e/TM3

D826 C(1)RM,y^2/Y^{S}X.Y^L In(1)EN,y;
      T(Y;3)MA9^{P}A109^D,y$^+$; TM1

F301 ru st kug^{003} e ca/TM3,Sb

F302 B^S Y; fs(3)005-14,ru st e ca/TM3,Sb

F303 B^S Y; fs(3)006-17,ru st e ca/TM3,Sb

F304 B^S Y; tov^{012},ru st e ca/TM3,Sb

F305 B^S Y; ru st ncn-I^{018} e ca/TM3,Sb

F306 B^S Y; fs(3)020-3,ru st e ca/TM3,Sb

F307 B^S Y; ru st kug^{040} e ca/TM3,Sb

F308 B^S Y; fs(3)050-17,ru st e ca/TM3,Sb

F309 B^S Y; ru st kug^{054} e ca/TM3,Sb

F310 B^S Y; fs(3)074-3,ru st e ca/TM3,Sb

F311 B^S Y; ru st ncn-I^{077} e ca/TM3,Sb

F312 B^S Y; fs(3)092-11,ru st e ca/TM3,Sb

F313 B^S Y; ru st e spn-A^{094} ca/TM3,Sb

F314 B^S Y; fs(3)102-19,ru th st ri roe p^p e^s
      ca/TM3,Sb

F315 B^S Y; ru th st kug^{113} ri roe p^p e^s
      ca/TM3,Sb

F316 B^S Y; fs(3)118-6,ru th st ri roe p^p e^s
      ca/TM3,Sb

F317 B^S Y; fs(3)127-4,ru th st ri roe p^p e^s
      ca/TM3,Sb

F318 B^S Y; fs(3)131-19,ru th st ri roe p^p e^s
      ca/TM3,Sb

F319 B^S Y; fs(3)135-17,ru th st ri roe p^p e^s
      ca/TM3,Sb

F320 B^S Y; tov^{155},ru th st ri roe p^p e^s
      ca/TM3,Sb

F321 B^S Y; fs(3)160-9,ru th st ri roe p^p e^s
      ca/TM3,Sb

F322 B^S Y; fs(3)173-11,ru th st ri roe p^p e^s
      ca/TM3,Sb

F323 B^S Y; fs(3)176-9,ru th st ri roe p^p e^s
      ca/TM3,Sb

F324 B^S Y; fs(3)179-15,ru th st ri roe p^p e^s
      ca/TM3,Sb

F325 B^S Y; fs(3)192-9,ru th st ri roe p^p e^s
      ca/TM3,Sb

F326 B^S Y; fs(3)193-16,ru th st ri roe p^p e^s
      ca/TM3,Sb

F327 B^S Y; fs(3)217-3,ru st e ca/TM3,Sb

F328 B^S Y; fs(3)227-17,ru st e ca/TM3,Sb
F329 B^S Y; fs(3)245-19,ru st e ca/TM3,Sb
F330 B^S Y; tov^{267},ru st e ca/TM3,Sb
F331 B^S Y; ru st kug^{285} e ca/TM3,Sb
F332 B^S Y; fs(3)298-5,ru st e ca/TM3,Sb
F333 B^S Y; fs(3)335-18,ru th st ri roe p^p e^s ca/TM3,Sb
F334 B^S Y; fs(3)337-12,ru th st ri roe p^p e^s ca/TM3,Sb
F335 B^S Y; tov^{345},ru th st ri roe p^p e^s ca/TM3,Sb
F336 B^S Y; fs(3)371-7,ru th st ri roe p^p e^s ca/TM3,Sb
F337 B^S Y; ru th st kug^{381} ri roe p^p e^s ca/TM3,Sb
F338 B^S Y; fs(3)401-18,ru st e ca/TM3,Sb
F339 B^S Y; tov^{404},ru st e ca/TM3,Sb
F340 B^S Y; ru st kug^{602} e ca/TM3,Sb
F341 B^S Y; ru st kug^{622} e ca/TM3,Sb
F342 B^S Y; fs(3)633-7,ru st e ca/TM3,Sb
F343 B^S Y; fs(3)643-7,ru st e ca/TM3,Sb
F344 B^S Y; ru st kug^{649} e ca/TM3,Sb
F345 B^S Y; ru bip^{653} st e ca/TM3,Sb
F346 B^S Y; fs(3)668-18,ru st e ca/TM3,Sb
F347 B^S Y; ru st kug^{674} e ca/TM3,Sb
F348 B^S Y; fs(3)690-7,ru st e ca/TM3,Sb
F349 B^S Y; fs(3)691-9,ru st e ca/TM3,Sb
F350 B^S Y; fs(3)813-8,ru st e ca/TM3,Sb

G301 B^S Y; fs(3)011-18,ru st e ca/TM3,Sb
G302 B^S Y; ru st e pll^{019} ca/TM3,Sb
G303 B^S Y; fs(3)020-17,ru st e ca/TM3,Sb
G304 B^S Y; fs(3)034-4,ru st e ca/TM3,Sb
G305 B^S Y; ru st e tsl^{035} ca/TM3,Sb
G306 B^S Y; fs(3)039-12,ru st e ca/TM3,Sb
G307 B^S Y; fs(3)041-14,ru st e ca/TM3,Sb
G308 B^S Y; ru st lds^{042} e ca/TM3,Sb
G309 B^S Y; $ely-c^{043}$,ru st e ca/TM3,Sb
G310 B^S Y; ru ndl^{046} st e ca/TM3,Sb
G311 B^S Y; fs(3)046-6,ru st e ca/TM3,Sb
G312 B^S Y; fs(3)062-15,ru st e ca/TM3,Sb
G313 B^S Y; ru st snk^{073} e ca/TM3,Sb
G314 B^S Y; ru st lds^{074} e ca/TM3,Sb
G315 B^S Y; ru st aur^{074} e ca/TM3,Sb
G316 B^S Y; fs(3)076-11,ru st e ca/TM3,Sb
G317 B^S Y; ru st e pll^{078} ca/TM3,Sb
G318   ru st osk^{084} e ca/TM3,Sb
G319 B^S Y; bcd^{085},ru st e ca/TM3,Sb
G320 B^S Y; fs(3)090-11,ru st e ca/TM3,Sb
G321 B^S Y; ru ndl^{093} st e ca/TM3,Sb
G322 B^S Y; fs(3)098-1,ru st e ca/TM3,Sb
G323 B^S Y; ru ndl^{111} th st ri roe p^p e^s ca/TM3,Sb
G324 B^S Y; ru th st ri roe p^p ea^{111} e^s ca/TM3,Sb
G325 B^S Y; fs(3)115-6,ru th st ri roe p^p e^s ca/TM3,Sb
G326 B^S Y; fs(3)115-8,ru th st ri roe p^p e^s ca/TM3,Sb

G327 B^S Y; fs(3)115-18,ru th st ri roe p^p e^s ca/TM3,Sb
G328 B^S Y; fs(3)117-19,ru th st ri roe p^p e^s ca/TM3,Sb
G329 B^S Y; ru th st tub^{118} ri roe p^p e^s ca/TM3,Sb
G330 B^S Y; ru th st ri roe p^p e^s $p11^{122}$ ca/TM3,Sb
G331 B^S Y; ru ndl^{133} th st ri roe p^p e^s ca/TM3,Sb
G332 B^S Y; ru th st ri bcd^{E1} roe p^p e^s ca/TM3,Sb
G333 B^S Y; fs(3)143-15,ru th st ri roe p^p e^s ca/TM3,Sb
G334 B^S Y; ru th st ri roe p^p e^s spg^{145} ca/TM3,Sb
G335 B^S Y; fs(3)145-18,ru th st ri roe p^p e^s ca/TM3,Sb
G336 B^S Y; skf^{153},ru th st ri roe p^p e^s ca/TM3,Sb
G337 B^S Y; fs(3)154-19,ru th st ri roe p^p e^s ca/TM3,Sb
G338 B^S Y; fs(3)158-5,ru th st ri roe p^p e^s ca/TM3,Sb
G339 B^S Y; fs(3)159-12,ru th st ri roe p^p e^s ca/TM3,Sb
G340 B^S Y; ru th st ri roe p^p osk^{166} e^s ca/TM3,Sb
G341 B^S Y; ru ndl^{169} th st ri roe p^p e^s ca/TM3,Sb
G342 B^S Y; ru th st ri roe p^p e^s tsl^{174} ca/TM3,Sb
G343 B^S Y; ru th st ri roe p^p aur^{175} e^s ca/TM3,Sb
G344 B^S Y; fs(3)176-4,ru th st ri roe p^p e^s ca/TM3,Sb
G345 B^S Y; fs(3)194-15,ru th st ri roe p^p e^s ca/TM3,Sb
G346 B^S Y; fs(3)197-8,ru th st ri roe p^p e^s ca/TM3,Sb
G347 B^S Y; ru kls st e ca/TM3,Sb
G348   ru st sic^{215} e ca/TM3,Sb
G349 B^S Y; fs(3)217-6,ru st e ca/TM3,Sb
G350 B^S Y; ru st snk^{229} e ca/TM3,Sb
G351 B^S Y; fs(3)230-5,ru st e ca/TM3,Sb
G352 B^S Y; $ely-c^{230}$,ru st e ca/TM3,Sb
G353 B^S Y; fs(3)233-13,ru st e ca/TM3,Sb
G354 B^S Y; ru st snk^{233} e ca/TM3,Sb
G355 B^S Y; ru st tub^{238} e ca/TM3,Sb
G356 B^S Y; ru st e spg^{242} ca/TM3,Sb
G357 B^S Y; ru st bcd^{E2} e/TM3,Sb
G358 B^S Y; fs(3)255-3,ru st e ca/TM3,Sb
G359 B^S Y; ru st osk^{255} e ca/TM3,Sb
G360   ru st sic^{256} e ca/TM3,Sb
G361 B^S Y; fs(3)257-18,ru st e ca/TM3,Sb
G362 B^S Y; ru ndl^{260} st e ca/TM3,Sb
G363 B^S Y; fs(3)268-4,ru st e ca/TM3,Sb
G364 B^S Y; fs(3)269-3,ru st e ca/TM3,Sb
G365 B^S Y; fs(3)271-5,ru st e ca/TM3,Sb
G366 B^S Y; ru st aur^{287} e ca/TM3,Sb
G367 B^S Y; fs(3)288-6,ru st e ca/TM3,Sb

G368　B^S Y; fs(3)297-12,ru st e ca/TM3,Sb
G369　B^S Y; ru st lds^{298} e ca/TM3,Sb
G370　B^S Y; fs(3)301-8,ru th st ri roe p^p e^s ca/TM3,Sb
G371　B^S Y; ru th st ri roe p^p osk^{301} e^s ca/TM3,Sb
G372　B^S Y; gnu^{305},ru th st ri roe p^p e^s ca/TM3,Sb
G373　B^S Y; ru th st ri roe p^p e^s pll^{312} ca/TM3,Sb
G374　B^S Y; ru th st ri roe p^p e^s pll^{316} ca/TM3,Sb
G375　B^S Y; fs(3)330-7,ru th st ri roe p^p e^s ca/TM3,Sb
G376　B^S Y; spg^{335},ru th st ri roe p^p e^s ca/TM3,Sb
G377　B^S Y; fs(3)337-8,ru th st ri roe p^p e^s ca/TM3,Sb
G378　B^S Y; fs(3)338-16,ru th st ri roe p^p e^s ca/TM3,Sb
G379　B^S Y; fs(3)340-4,ru th st ri roe p^p e^s ca/TM3,Sb
G380　B^S Y; ru th st ri bcd^{E5} roe p^p e^s ca/TM3,Sb
G381　B^S Y; fs(3)352-3,ru th st ri roe p^p e^s ca/TM3,Sb
G382　B^S Y; fs(3)352-16,ru th st ri roe p^p e^s ca/TM3,Sb
G383　B^S Y; fs(3)358-7,ru th st ri roe p^p e^s ca/TM3,Sb
G384　ru th st ri roe p^p sic^{371} e^s ca/TM3,Sb
G385　B^S Y; fs(3)374-13,ru th st ri roe p^p e^s ca/TM3,Sb
G386　B^S Y; fs(3)377-16,ru th st ri roe p^p e^s ca/TM3,Sb
G387　B^S Y; fs(3)382-8,ru th st ri roe p^p e^s ca/TM3,Sb
G388　B^S Y; spg^{383},ru th st ri roe p^p e^s ca/TM3,Sb
G389　B^S Y; fs(3)384-9,ru th st ri roe p^p e^s ca/TM3,Sb
G390　B^S Y; ru th st ri roe p^p e^s pll^{385} ca/TM3,Sb
G391　B^S Y; ru th st pip^{386} ri roe p^p e^s ca/TM3,Sb
G392　B^S Y; fs(3)405-2,ru st e ca/TM3,Sb
G393　B^S Y; fs(3)416-7,ru st e ca/TM3,Sb
G394　B^S Y; fs(3)421-4,ru st e ca/TM3,Sb
G395　B^S Y; fs(3)422-15,ru st e ca/TM3,Sb
G396　B^S Y; fs(3)425-12,ru st e ca/TM3,Sb
G397　B^S Y; fs(3)610-15,ru st e ca/TM3,Sb
G398　ru st sic^{612} e ca/TM3,Sb
G399　B^S Y; fs(3)624-16,ru st e ca/TM3,Sb

G801　B^S Y; ru st e Tlr^{632} ca/TM3,Sb
G802　B^S Y; fs(3)640-2,ru st e ca/TM3,Sb
G803　B^S Y; fs(3)651-14,ru st e ca/TM3,Sb
G804　B^S Y; fs(3)651-18,ru st e ca/TM3,Sb
G805　B^S Y; fs(3)652-13,ru st e ca/TM3,Sb
G806　B^S Y; ru st pip^{664} e ca/TM3,Sb
G807　B^S Y; ru st bcd^{E3} e ca/TM3,Sb

G808　B^S Y; fs(3)665-19,ru st e ca/TM3,Sb
G809　B^S Y; ru st e spz^{67} ca/TM3,Sb
G810　B^S Y; fs(3)672-16,ru st e ca/TM3,Sb
G811　B^S Y; fs(3)677-6,ru st e ca/TM3,Sb
G812　B^S Y; fs(3)679-7,ru st e ca/TM3,Sb
G813　B^S Y; ru st pum^{680} e ca/TM3,Sb
G814　B^S Y; fs(3)681-1,ru st e ca/TM3,Sb
G815　B^S Y; fs(3)689-14,ru st e ca/TM3,Sb
G816　B^S Y; ru st e tsl^{691} ca/TM3,Sb
G817　B^S Y; fs(3)805-2,ru st e ca/TM3,Sb
G818　B^S Y; ru st e spg^{805} ca/TM3,Sb
G819　B^S Y; fs(3)806-14,ru st e ca/TM3,Sb
G820　B^S Y; fs(3)807-19,ru st e ca/TM3,Sb
G821　B^S Y; fs(3)808-1,ru st e ca/TM3,Sb
G822　B^S Y; fs(3)808-12,ru st e ca/TM3,Sb
G823　B^S Y; ru st ea^{818} e ca/TM3,Sb
G824　B^S Y; fs(3)819-13,ru st e ca/TM3,Sb
G825　B^S Y; fs(3)820-12,ru st e ca/TM3,Sb
G826　B^S Y; fs(3)836-8,ru st e ca/TM3,Sb
G827　B^S Y; ru st bcd^{E4} e ca/TM3,Sb
G828　B^S Y; ru st e spg^{842} ca/TM3,Sb
G829　B^S Y; fs(3)842-16,ru st e ca/TM3,Sb

H301　B^S Y; ru st e spn-A^{003} ca/TM3,Sb
H302　B^S Y; fs(3)010-6,ru st e ca/TM3,Sb
H303　B^S Y; fs(3)013-1,ru st e ca/TM3,Sb
H304　B^S Y; fs(3)016-16,ru st e ca/TM3,Sb
H305　B^S Y; fs(3)021-1,ru st e ca/TM3,Sb
H306　B^S Y; fs(3)022-2,ru st e ca/TM3,Sb
H307　B^S Y; fs(3)045-3,ru st e ca/TM3,Sb
H308　B^S Y; ru st e spn-A^{050} ca/TM3,Sb
H309　B^S Y; ru st fch^{055} e ca/TM3,Sb
H310　B^S Y; ru st spn-B^{056} e ca/TM3,Sb
H311　B^S Y; ru st e spn-A^{057} ca/TM3,Sb
H312　B^S Y; fs(3)074-19,ru th st ri roe p^p e^s ca/TM3,Sb
H313　B^S Y; fs(3)076-8,ru st e ca/TM3,Sb
H314　B^S Y; fs(3)089-17,ru st e ca/TM3,Sb
H315　B^S Y; fs(3)092-15,ru st e ca/TM3,Sb
H316　B^S Y; fs(3)093-18,ru st e ca/TM3,Sb
H317　B^S Y; fs(3)096-11,ru st e ca/TM3,Sb
H318　B^S Y; fs(3)097-12,ru st e ca/TM3,Sb
H319　B^S Y; fs(3)108-17,ru th st ri roe p^p e^s ca/TM3,Sb
H320　B^S Y; fs(3)112-13,ru th st ri roe p^p e^s ca/TM3,Sb
H321　B^S Y; fs(3)116-11,ru th st ri roe p^p e^s ca/TM3,Sb
H322　B^S Y; fs(3)120-19,ru th st ri roe p^p e^s ca/TM3,Sb
H323　B^S Y; fs(3)121-14,ru th st ri roe p^p e^s ca/TM3,Sb
H324　B^S Y; fs(3)122-7,ru th st ri roe p^p e^s ca/TM3,Sb
H325　B^S Y; fs(3)131-11,ru th st ri roe p^p e^s ca/TM3,Sb
H326　B^S Y; fs(3)131-13,ru th st ri roe p^p e^s ca/TM3,Sb

H327 B^S Y; fs(3)131-17,ru th st ri roe p^P e^s
ca/TM3,Sb

H328 B^S Y; fs(3)133-1,ru th st ri roe p^P e^s
ca/TM3,Sb

H329 B^S Y; fs(3)133-14,ru th st ri roe p^P e^s
ca/TM3,Sb

H330 B^S Y; fs(3)143-11,ru th st ri roe p^P e^s
ca/TM3,Sb

H331 B^S Y; fs(3)147-5,ru th st ri roe p^P e^s
ca/TM3,Sb

H332 B^S Y; ru th st ri roe p^P e^s spn-D^{150}
ca/TM3,Sb

H333 B^S Y; fs(3)152-18,ru th st ri roe p^P e^s
ca/TM3,Sb

H334 B^S Y; ru th st ri roe p^P spn-B^{153} e^s
ca/TM3,Sb

H335 B^S Y; fs(3)157-14,ru th st ri roe p^P e^s
ca/TM3,Sb

H336 B^S Y; fs(3)159-5,ru th st ri roe p^P e^s
ca/TM3,Sb

H337 B^S Y; fs(3)159-13,ru th st ri roe p^P e^s
ca/TM3,Sb

H338 B^S Y; fs(3)173-5,ru th st ri roe p^P e^s
ca/TM3,Sb

H339 B^S Y; fs(3)176-11,ru th st ri roe p^P e^s
ca/TM3,Sb

H340 B^S Y; fs(3)180-4,ru th st ri roe p^P e^s
ca/TM3,Sb

H341 B^S Y; fs(3)181-8,ru th st ri roe p^P e^s
ca/TM3,Sb

H342 B^S Y; fs(3)183-13,ru th st ri roe p^P e^s
ca/TM3,Sb

H343 B^S Y; fs(3)185-18,ru th st ri roe p^P e^s
ca/TM3,Sb

H344 B^S Y; fs(3)187-18,ru th st ri roe p^P e^s
ca/TM3,Sb

H345 B^S Y; fs(3)189-3,ru th st ri roe p^P e^s
ca/TM3,Sb

H346 B^S Y; fs(3)204-5,ru st e ca/TM3,Sb

H347 B^S Y; fs(3)205-4,ru st e ca/TM3,Sb

H348 B^S Y; fs(3)211-16,ru st e ca/TM3,Sb

H349 B^S Y; fs(3)215-9,ru st e ca/TM3,Sb

H350 B^S Y; fs(3)220-1,ru st e ca/TM3,Sb

H351 B^S Y; fs(3)224-8,ru st e ca/TM3,Sb

H352 B^S Y; fs(3)225-19,ru st e ca/TM3,Sb

H353 B^S Y; fs(3)226-6,ru st e ca/TM3,Sb

H354 B^S Y; fs(3)229-15,ru st e ca/TM3,Sb

H355 B^S Y; fs(3)230-2,ru st e ca/TM3,Sb

H356 B^S Y; ru st e spn-F^{234} ca/TM3,Sb

H357 B^S Y; fs(3)234-14,ru st e ca/TM3,Sb

H358 B^S Y; fs(3)247-19,ru st e ca/TM3,Sb

H359 B^S Y; fs(3)249-12,ru st e ca/TM3,Sb

H360 B^S Y; vss-I^{258},ru st e ca/TM2,Ubx

H361 B^S Y; fs(3)260-15,ru st e ca/TM3,Sb

H362 B^S Y; ru st fch^{267} e ca/TM3,Sb

H363 B^S Y; fs(3)272-9,ru st e ca/TM3,Sb

H364 B^S Y; fs(3)289-11,ru st e ca/TM3,Sb

H365 B^S Y; fs(3)293-19,ru st e ca/TM3,Sb

H366 B^S Y; fs(3)294-6,ru st e ca/TM3,Sb

H367 B^S Y; fs(3)298-12,ru st e ca/TM3,Sb

H368 B^S Y; fs(3)302-4,ru th st ri roe p^P e^s
ca/TM3,Sb

H369 B^S Y; fs(3)304-11,ru th st ri roe p^P e^s
ca/TM3,Sb

H370 B^S Y; fs(3)316-14,ru th st ri roe p^P e^s
ca/TM3,Sb

H371 B^S Y; fs(3)329-9,ru th st ri roe p^P e^s
ca/TM3,Sb

H372 B^S Y; fs(3)335-3,ru th st ri roe p^P e^s
ca/TM3,Sb

H373 B^S Y; ru th st ri roe p^P osk^{336} e^s
ca/TM3,Sb

H374 B^S Y; fs(3)343-11,ru th st ri roe p^P e^s
ca/TM3,Sb

H375 B^S Y; fs(3)344-1,ru th st ri roe p^P e^s
ca/TM3,Sb

H376 B^S Y; fs(3)346-14,ru th st ri roe p^P e^s
ca/TM3,Sb

H377 B^S Y; ru th st ri roe p^P e^s spn-D^{349}
ca/TM3,Sb

H378 B^S Y; fs(3)350-7,ru th st ri roe p^P e^s
ca/TM3,Sb

H379 B^S Y; fs(3)350-13,ru th st ri roe p^P e^s
ca/TM3,Sb

H380 B^S Y; fs(3)360-5,ru th st ri roe p^P e^s
ca/TM3,Sb

H381 B^S Y; fs(3)365-6,ru th st ri roe p^P e^s
ca/TM3,Sb

H382 B^S Y; fs(3)370-11,ru th st ri roe p^P e^s
ca/TM3,Sb

H383 B^S Y; fs(3)373-16,ru th st ri roe p^P e^s
ca/TM3,Sb

H384 B^S Y; fs(3)378-12,ru th st ri roe p^P e^s
ca/TM3,Sb

H385 B^S Y; fs(3)383-3,ru th st ri roe p^P e^s
ca/TM3,Sb

H386 B^S Y; fs(3)384-13,ru th st ri roe p^P e^s
ca/TM3,Sb

H387 B^S Y; fs(3)389-8,ru th st ri roe p^P e^s
ca/TM3,Sb

H388 B^S Y; fs(3)402-6,ru st e ca/TM3,Sb

H389 B^S Y; fs(3)404-3,ru st e ca/TM3,Sb

H390 B^S Y; fs(3)419-6,ru st e ca/TM3,Sb

H391 B^S Y; fs(3)421-2,ru st e ca/TM3,Sb

H392 B^S Y; ru spn-C^{422} st e ca/TM3,Sb

H393 B^S Y; fs(3)603-17,ru st e ca/TM3,Sb

H394 B^S Y; fs(3)615-13,ru st e ca/TM3,Sb

H395 B^S Y; ru st spn-E^{616} e ca/TM3,Sb

H396 B^S Y; fs(2)617-6,ru st e ca/TM3,Sb

H397 B^S Y; fs(3)640-9,ru st e ca/TM3,Sb

H398 B^S Y; fs(3)646-4,ru st e ca/TM3,Sb

H399 B^S Y; fs(3)649-13,ru st e ca/TM3,Sb

H801 B^S Y; ru st spn-E^{653} e ca/TM3,Sb

H802 B^S Y; fs(3)655-15,ru st e ca/TM3,Sb

H803 B^S Y; ru spn-C^{660} st e ca/TM3,Sb

H804 B^S Y; fs(3)665-11,ru st e ca/TM3,Sb

H805 B^S Y; fs(3)669-1,ru st e ca/TM3,Sb

H806 B^S Y; fs(3)671-1,ru st e ca/TM3,Sb

H807 B^S Y; vss-1^{675},ru st e ca/TM3,Sb

H808 B^S Y; fs(3)676-14,ru st e ca/TM3,Sb

H809 B^S Y; fs(3)678-2,ru st e ca/TM3,Sb

H810 B^S Y; fs(3)678-7,ru st e ca/TM3,Sb

H811 B^S Y; fs(3)680-17,ru st e ca/TM3,Sb

H812 B^S Y; fs(3)802-13,ru st e ca/TM3,Sb

H813 B^S Y; fs(3)806-8,ru st e ca/TM3,Sb

H814 B^S Y; fs(3)807-9,ru st e ca/TM3,Sb

K201 Fs(2)D; T(1;2)Bld; T(1;2)OR64

K202 $BicD^{23-10}$/CyO

K203 b dl^{103} cn sca/CyO,b^{XL1} sca^{XL1}

K204 dl^{7607}/CyRoi/Bc Gla

K206 tor^{Y9} cn sca/CyO

K207 Fs(2)X10,b pr/CyO; T(1;2)OR64

K208 Fs(2)D10,b pr/CyO; T(1;2)OR64

K209 dl^{160}; T(1;2)Bld; T(1;2)OR64

K210 tor^{4021}/CyRoi/Bc Gla

K211 dl^{103} pr cn bw; T(1;2)Bld; T(1;2)OR64

K301 ru th st Tl/Ms(3)R24/TM3,st^{X2} Sb

K302 ru h th st cu sr e^s Tl^{9Q} ca/TM3,Sb; T(1;3)OR60

K303 ru h th st cu sr e^s Tl^{5B} ca/TM3,Sb; T(1;3)OR60

K304 th st cp in ri p^P ea^{831} srp^{288-12}/TM3,Sb; T(1;3)OR60

K305 ru st e Tl^{84c} ca/TM3,Sb; T(1;3)OR60

K306 th st cp in ri p^P $ea^{5.13}$/TM3,Sb; T(1;3)OR60

K307 Fs(3)R12,ru h th st cu/TM3,Sb; T(1;3)OR60

K308 mwh snk^{rm4} red e Tl^{9Q} ca/Ms(3)R24/TM3,Sb

K309 ru ndl^{260} st sr e^s Tl^{5B} ca/TM3,Sb; T(1;3)OR60

K310 ru st ea^{125} e ca/TM3,Sb; T(1;3)OR60

K311 Fs(3)49/TM3/DcxF

K312 Fs(3)4102/TM3/DcxF

K313 Fs(3)5022/TM3/DcxF

M101 fs(1)$K10^{254}$/FM7,l(1)TW9

M102 fu^{ECN}/ClB

M103 fs(1)K10 w f mal/ClB

M104 FM7,l(1)TW9/gd^{190}

M105 swa^{14} ct v/FM7

M106 swa^{T573} ct v/FM3

M107 seg cv v/C(1)DX,y f

M108 v^{24} gd^1/FM3

M109 fs(1)572,v^{24}/FM3

M110 y cv swa^{82} v f/FM7

M111 fs(1)1182,v^{24}/FM3

M112 sc ec cv swa^{15}/FM3

M113 C(1)DX,y f/ovo^{D1} v^{24}

M114 v gd^4/FM3

M115 v gd^3/FM3

M116 v gd^2/FM3

M117 v gd^5/FM3

M118 v gd^6/FM3

M119 v gd^7/FM3

M120 C(1)DX,y f/amx lz^g v

M121 y cv swa^{99} v f/FM3

M122 fs(1)ph^{1901} v g f/FM7

M123 fs(1)N^{211} g f/FM7

M124 y cv fs(1)pmd^{151} f car/FM7

M125 fs(1)$M23^{1444}$/FM0

M126 fs(1)$M57^{341}$/FM0

M127 fs(1)$M23^{5087}$/FM0

M128 fs(1)$M22^{1136}$/FM0

M129 fs(1)$M57^{358}$/FM3

M130 fs(1)pmd^{758}/FM3

M131 z w mh^{1182}/FM3

M132 swa^{14} v fs(1)N^{211}/FM7

M201 al dp b dl pr cn sca sp/CyO

M202 b $BicD^{7134}$ tor^{WK} cn/CyO

M203 $cact^{PD}$ dl/CyO

M204 dl cn sca/CyO

M205 dl cn sca/CyO,$1(2)100^{DTS}$

M206 dl^8 pr cn wx^{wxt} bw/CyO

M207 $1(2)91^{DTS}$ b dl^2 c px sp/CyO

M208 b dl pr cn vg^D/CyO

M209 dl^4 pr vg^{np}/CyO,$1(2)100^{DTS}$

M210 b dl^2/CyO,$1(2)100^{DTS}$

M211 $1(2)91^{DTS}$ b tor^{WK} cn/CyO

M212 trk^{HD} cn bw/CyO

M213 tor^{WK} cn/CyO

M214 grk^{WG} b bw sp/CyO,$1(2)100^{DTS}$

M215 tud^{WC} bw sp/CyO,$1(2)100^{DTS}$

M216 b dl^3/CyO

M217 dl^6 pr cn wx^{wxt} bw/CyO

M218 sax^{WO18} cn bw sp/CyO,$1(2)100^{DTS}$

M219 okr^{WS1} cn bw sp/CyO,$1(2)100^{DTS}$

M220 cta^{WU31} cn bw sp/CyO,$1(2)100^{DTS}$

M221 $BicC^{WC}$ cn bw sp/CyO,$1(2)100^{DTS}$

M222 cn bw egl^{WU} sp/CyO

M223 vas^{PD} cn exu^{PJ} bw/CyO,$1(2)100^{DTS}$

M224 In(2L)dl^H,b dl^T pr/CyO

M225 dl^5 pr cn wx^{wxt} bw/CyO

M226 qua^{WP} cn bw sp/CyO

M227 kel^{WB} cn bw sp/CyO

M228 grk^{HK} cn bw sp/CyO,$1(2)100^{DTS}$

M229 cn top^{Q41} bw sp/CyO,$1(2)100^{DTS}$

M230 Fs(2)102,cn bw sp/CyO

M231 $cact^{99}$ cn bw sp/CyO

M232 $cact^{IIIG}$ cn bw sp/CyO

M233 $cact^{UL}$ pr cn bw/CyO,$1(2)100^{DTS}$

M234 dp b $BicD^{7134}$/CyO

M235 b pr cn sca tud^{B36}/CyO

M236 In(2L)dl^T,b dl^T pr cn sca/CyO

M237 $cact^{UL}$ pr cn bw/CyO,$1(2)100^{DTS}$

M238 $BicD^{YC71}$/CyO

M239 $spir^{339}$ cn sca/CyO

M240 dp b $BicD^{7134-R26}$/CyO

M241 $BicD^{7134}$ pr cn sca/CyO

M242 $spir^{224}$/CyO

M243 $cact^{PD}$ cn bw/CyO; ru kls

M244 b $BicD^{IIIE}$ cn/CyO

M245 bic L^2/CyO

M246 BicD^{IIIE} pr cn sca/CyO

M247 cact^{UL} pr cn bw/CyO,l(2)100^{DTS}

M248 cact^{UW} pr cn bw/CyO,l(2)100^{DTS}

M249 BicC^{C96} pr cn sca/CyO

M250 cact^{UW} pr cn bw/CyO,l(2)100^{DTS}

M251 cact^{VQ} pr cn bw/CyO,l(2)100^{DTS}

M252 b dl^{I5}/CyO

M253 alb^{404}/CyO

M254 cn stau^{D3}/CyO

M255 Df(2R)PC4, exu^{PJ} bw/CyO

M256 cact^{VQ} pr cn bw/CyO,l(2)100^{DTS}

M257 vas^{PD} cn bw/CyO

M258 cn stau^{HL} bw/CyO

M259 pr cact^{HE} cn sca/CyO

M260 cn exu^{QR} bw/CyO

M261 cact^{PD} cn bw/CyO

M262 stau^{D3} exu^{QR}/CyO

M263 dl^{PZ} cn bw/CyO

M264 slp^{PX},cn bw/CyO

M265 tor^{RL3} cn bw/CyO

M266 dl^{QF} cn bw/CyO,l(2)100^{DTS}

M267 dl^{SC} cn bw/CyO,l(2)100^{DTS}

M268 dl^{QD} cn bw/CyO,l(2)100^{DTS}

M269 dl^{Q17} cn bw/CyO,l(2)100^{DTS}

M270 b pr wbl^{RP} bw/CyO

M271 dl^{SG} cn bw/CyO,l(2)100^{DTS}

M272 cn exu^{SC} bw/CyO,l(2)100^{DTS}

M273 cn exu^{SB} bw/CyO,l(2)100^{DTS}

M274 tor^{RL3} cn stau^{HL} bw/CyO

M275 dl cn sca/CyO,l(2)100^{DTS}; ru kls

M276 trk^{PI} cn bw/CyO

M277 cn tor^{RL3} bw trk^{PZ}/CyO

M278 tor^{rx8}/CyRoi

M279 tor^{PM} cn bw/CyO

M280 tor^{HH} cn bw/CyO

M281 BicD^{21}/CyO

M282 b BicD^{7134} tud^{WC} bw/CyO

M283 BicD^{52}/CyO

M284 b pr stau^{G2}/CyO

M285 b pr cn sca exu^{PJ} bw/CyO

M286 tor^{rx9}/CyRoi

M287 cn stau^{HL} tud^{WC} bw/CyO

M288 vls^{PE} cn stau^{HL} bw/CyO

M289 vas^{PD} cn stau^{HL} bw/CyO

M290 vls^{RB} cn bw/CyO

M291 vas^{PD} cn tud^{WC} bw sp/CyO

M292 vls^{PG} cn bw/CyO

M293 vls^{PE} cn bw/CyO

M294 cact^{UK} pr cn bw/CyO,l(2)100^{DTS}

M295 tor^{rx7}/CyRoi

M296 b cact^{SG} cn bw/CyO

M297 b cact^{HE} cn bw/CyO

M298 b dl^{U5} pr/CyO

M299 b dl^{B10} pr/CyO

M701 b dl^{O11} pr/CyO

M702 BicD^{IIIE} cn tud^{WC} bw/CyO

M703 Fs(2)Y12-13,b pr/CyO

M704 pr tor^{TC} cn bw/CyO,l(2)100^{DTS}

M705 b pr wbl^{T6}/CyO

M706 b pr wbl^{E4}/CyO

M707 b cact^{Q6} pr/CyO

M708 b cact^{F6} pr/CyO

M709 b cact^{Y11} pr/CyO

M710 b cact^{H4} pr/CyO

M711 b cact^{F11} pr/CyO

M712 b cact^{U7} pr/CyO

M713 b cact^{D13} pr/CyO

M714 b cact^{O11} pr/CyO

M715 b cact^{O9} pr/CyO

M716 b cact^{S1} pr/CyO

M717 b cact^{A2} pr/CyO

M718 b cact^{D12} pr/CyO

M719 b cact^{G8} pr/CyO

M720 b cact^{H8} pr/CyO

M721 b cact^{L14} pr/CyO

M722 pr cn exu^{VL} bw/CyO,l(2)100^{DTS}

M723 b vas^{O11} pr cn/CyO

M724 b vas^{D1} pr cn/CyO

M725 b vas^{Q7} pr/CyO

M726 b vas^{Q6} pr cn/CyO

M727 b vas^{D5} pr cn/CyO

M728 b vas^{O14} cn/CyO

M729 b pr stau^{C8}/CyO

M730 pr tor^{TC} cn bw/CyO,l(2)513^{DTS}

M731 cap^{RK} cn bw sp/CyO,l(2)100^{DTS}

M732 cap^{HK} cn bw sp/CyO,l(2)100^{DTS}

M733 b BicD^{7134} tor^{RL3} cn bw/CyO

M734 bic L^2 stau^{HL} bw/CyO

M735 bic L^2 exu^{PJ} bw/CyO

M736 b tor^{WK} cn stau^{HL} bw/CyO,b^{XL1}

M737 tor^{rx2}/CyRoi

M738 BicC^{WC} cn stau^{HL} bw/CyO

M739 tor^{re2}/CyRoi

M740 BicC^{WC} cn exu^{PJ} bw/CyO

M741 b tor^{PM} cn bw/CyO

M742 Df(2R)vg^D,stau^{HL} bw/CyO

M743 Df(2R)vg^D,exu^{PJ} bw/CyO

M744 dp b BicD^{7134} cn exu^{PJ} bw/CyO

M745 BicD^{IIIE} pr cn exu^{PJ} bw/CyO

M746 vls^{RB} pr cn exu^{PJ} bw/CyO

M747 b BicD^{IIIE} cn stau^{HL} bw/CyO

M748 b BicD^{7134} stau^{HL} bw/CyO

M749 b BicD^{7134} pr cn stau^{D3}/CyO

M750 cn tor^{QA} bw/CyO,l(2)100^{DTS}

M751 cn tor^{QK25} bw/CyO,l(2)100^{DTS}

M752 cn tor^{RI} bw/CyO,l(2)100^{DTS}

M753 cn tor^{QK45} bw/CyO,l(2)100^{DTS}

M754 cn tor^{QL} bw/CyO,l(2)100^{DTS}

M755 cn tor^{HM} bw/CyO,l(2)100^{DTS}

M301 bcd^{GB} p^P/TM3,Sb

M302 ru e wo ro pll^{122} ca/TM3,Sb

M303 ru tsl^{MK} ca/TM3

M304 +/CyO,1(2)513^{DTS}; bicF/TM3,Sb
M305 th st ri p^P ts1^{174} e^s/TM3
M306 fs(3)6.16/TM3,Sb
M307 st bcd^{E2} p^P osk^{166}/TM3
M308 Fs(3)X25/TM3/TM1
M309 st nos^{18} e/TM3
M310 st bcd^{E1} ri pum^{680}/TM3
M311 st nos^{53} e/TM3,Sb
M312 ru th st ri roe p^P ea^{111} e^s ca/TM3,Sb
M313 ru st osk^{255} e ca/TM3,Sb
M314 ru h th st cu ea^1/TM8
M315 mwh ndl^{rm2} red e/TM1
M316 mwh snk^{rm4} red e/TM1
M317 mwh red e spz^{rm7}/TM1
M318 ru th st ri roe p^P Tl^{rm9}/TM3
M319 th st in ri roe p^P osk^{366}/TM3,Sb
M320 st ea^2 e/TM8
M321 st ea^3 e/TM8
M322 st ea^4 e/TM8
M323 st osk^{O84}/TM3
M324 th st in ri roe p^P osk^{301}/TM3
M325 st ea^7 e/TM8
M326 nos^{L7} st e/TM3,Sb
M327 th st in ri roe p^P osk^{166}/TM3,Sb
M328 st pum^{680}/TM3,Sb
M329 ru h th st cu e Tl^{r26} ca/TM3
M330 e Tl^{9QRE} ca/TM3
M331 mat(3)1,mwh red e/TM1
M332 mat(3)3,mwh red e/TM1
M333 mwh ndl^{rm5} red e/TM1
M334 st ri roe p^P osk^{166} e ts1^{691} ca/TM3,Sb
M335 mwh red e pll^{rm8}/TM1
M336 st pum^{680} e ts1^{691} ca/TM3,Sb
M337 st ri roe bcd^{E1} p^P nos^{L7} e/TM3,Sb
M338 ru kls
M339 th st ri bcd^{E1} roe p^P e ts1^{035}/TM3,Sb
M340 In(3LR)5B^{RXV},h th st cu ca/TM3
M341 In(3R)5B^{REN},e ca/TM3
M342 In(3R)5B^{REW},ca/TM3
M343 Tl^{5BREQ} ca/TM3
M344 st pum^{680} nos^{L7} e/TM3
M345 Df(3R)p^{XT103},st nos^{L7} e/TM3
M346 th st ri roe p^P osk^{166} nos^{L7} e/TM3
M347 st pum^{21} e/TM3,Sb
M348 st osk^{123} e/TM3,Sb
M349 st osk^{54} e/TM3,Sb
M350 st osk^{88} e/TM3,Sb
M351 ru h st e Tl^{1-RXA}/TM3
M352 ru st e Tl^{1-RXD}/TM3
M353 T(2;3)RXE,ru st/TM3
M354 ru st e Tl^{1-RXH}/TM3
M355 st osk^{150} e/TM3,Sb
M356 ru kls e Tl^{rm10}/TM3,Sb
M357 th st cp in ri bcd$^{2-13}$ p^P/TM3,Sb
M358 th st cp in ri bcd$^{23-16}$ p^P/TM3,Sb
M359 th st ri bcd$^{33-5}$ e/TM3,Sb
M360 th st cp in ri bcd^{111} p^P/TM3,Sb
M361 st ts1^{135}/TM3,Sb

M362 st ts1^{146}/TM3,Sb
M363 th st cp in ri bcd$^{33-5}$ p^P/TM3,Sb
M364 st nos^{L7} e/TM3,Sb
M365 spz^{145} ca/TM1
M366 pll^{74} ca/TM3
M367 e spz^{197} ca/TM1
M368 ru st e pll^{864} ca/TM3
M369 spz^{67} ca/TM1
M370 pll^{628} ca/TM3
M371 st e Tl^{r444}/TM3
M372 th st ri bcd^{E1} roe p^P/TM3
M373 spg^{rm6} ru th st cu sr e/TM1
M374 st bcd^{E4} e/TM3,Sb
M375 ri bcd^{E5} roe p^P/TM3,Sb
M376 Fs(3)T11-14/TM3; T(1;3)OR60
M377 Fs(3)D14/TM3; T(1;3)OR60
M378 st snk^{073} ea^1 sr e^s ca/TM3,Sb
M379 st ea^{818} Tl^{9QRE1} ca/TM3
M380 ru st ea^{818} spz^{67} ca/TM1
M381 ru th st pip^{386} ri roe p^P ea^1/TM3,Sb
M382 th st cu ea^1 Tl^{9QRX} ca/TM3
M383 ru h th st cu ea^1 e Tl^{rm9}/TM3
M384 st tub^{238} e Tl^{9QRE1} ca/TM3,Sb
M385 ru st tub^{118} e Tl^{rm9}/TM3
M386 st pip^{386} e Tl^{9QRE1} ca/TM3,Sb
M387 ru st snk^{229} e Tl^{rm9}/TM1
M388 ru st snk^{073} Tl^{9QRE1} ca/TM3,Sb
M389 ru ndl^{093} st Tl^{9QRE1} ca/TM3,Sb
M390 ru ndl^{260} st e Tl^{rm9}/TM3,Sb
M391 ru st pip^{664} e Tl^{rm9}/TM3,Sb
M392 bcd$^{2-13}$ p^P e/TM3,Sb
M393 th st bcd^{E3} e/TM3,Sb
M394 e ts1^{691}/TM1
M395 e ts1^{035} ca/TM1
M396 th st cp in ri bcd$^{23-16}$ p^P e/TM3,Sb
M397 ru st e tny$^{4-14}$ ca/TM3
M398 Tl^{r632} ca/TM3,Sb
M399 st snk^{073} e/TM8

M801 st ea^{84b} e/TM3,Sb
M802 th st cp pum^{ET1} in ri p^P/TM3,Sb
M803 th st cp pum^{ET2} in ri p^P/TM3,Sb
M804 th st cp pum^{ET3} in ri p^P/TM3,Sb
M805 th st cp pum^{ET4} in ri p^P/TM3,Sb
M806 th st cp pum^{ET5} in ri p^P/TM3,Sb
M807 th st cp pum^{ET6} in ri p^P/TM3,Sb
M808 th st cp pum^{ET7} in ri p^P/TM3,Sb
M809 th st cp pum^{ET8} in ri p^P/TM3,Sb
M810 th st cp pum^{ET9} in ri p^P/TM3,Sb
M811 th st cp pum^{ET10} in ri p^P/TM3,Sb
M812 th st cp pum^{ET11} in ri p^P/TM3,Sb
M813 th st ri roe bcd^{E1} osk^{166} p^P e ts1^{491}
      ca/TM3,Sb

Z201 cn 1(2)IA109 bw sp/CyO
Z202 cn shn^{IB} bw sp/CyO
Z203 cn thr^{IB} bw sp/CyO
Z204 cn brh^{IB} bw sp/CyO

Z205   cn arr^{IB69} bw sp/CyO

Z206   cn mam^{IB99} bw sp/CyO

Z207   cn gho^{IB104} bw sp/CyO

Z208   fzy^{IB115} cn bw sp/CyO

Z209   cn fas^{IC67} bw sp/CyO

Z210   bib^{ID05} cn bw sp/CyO

Z211   cn bw sp zip^{ID16}/CyO

Z212   cn eve^{ID} bw sp/CyO

Z213   cn twi^{ID96} bw sp/CyO

Z214   cn flb^{IF26} bw sp/CyO

Z215   cn mam^{IF33} bw sp/CyO

Z216   ptc^{IF85} cn bw sp/CyO

Z217   wg^{IG22} cn bw sp/CyO

Z218   cn sli^{IG23} bw sp/CyO

Z219   slf^{IG24} cn bw sp/CyO

Z220   raw^{IG26} cn bw sp/CyO

Z221   cn shg^{IG29} bw sp/CyO

Z222   scw^{IG76} cn bw sp/CyO

Z223   cn sli^{IG107} bw sp/CyO

Z224   cn shg^{IH81} bw sp/CyO

Z225   fzy^{IH108} cn bw sp/CyO

Z226   cn cra^{IJ23} bw sp/CyO

Z227   bib^{IJ66} cn bw sp/CyO

Z228   wg^{IJ69} cn bw sp/CyO

Z229   slf^{IJ83} cn bw sp/CyO

Z230   cn mam^{IJ113} bw sp/CyO

Z231   bhe^{IJ119} cn bw sp/CyO

Z232   cn flb^{IK35} bw sp/CyO

Z233   cn en^{IK57} bw sp/CyO

Z234   mmy^{IK63} cn bw sp/CyO

Z235   cn rib^{IK81} bw sp/CyO

Z236   mid^{IK97} cn bw sp/CyO

Z237   mmy^{IL07} cn bw sp/CyO

Z238   cn mam^{IL42} bw sp/CyO

Z239   cn thr^{IL62} bw sp/CyO

Z240   cn pim^{IL97} bw sp/CyO

Z241   wg^{IL114} cn bw sp/CyO

Z242   cn mam^{IL115} bw sp/CyO

Z243   cn hsk^{IM45} bw sp/CyO

Z244   cn shn^{IM56} bw sp/CyO

Z245   cn en^{IM99} bw sp/CyO

Z246   bhe^{IM112} cn bw sp/CyO

Z247   cn Pu^{IN18} bw sp/CyO

Z248   wg^{IN67} cn bw sp/CyO

Z249   b pr cn sha^{IN98}/CyO

Z250   ptc^{IN108} cn bw sp/CyO

Z251   cn en^{IO34} bw sp/CyO

Z252   cn bw sp Kr^{IO66}/SM1

Z253   str^{IO78} cn bw sp/CyO

Z254   cn flb^{IP02} bw sp/CyO

Z255   cn flz^{IP04} bw sp/CyO

Z256   l(2)IP85,cn bw sp/CyO

Z257   cn gho^{IP107} bw sp/CyO

Z258   aop^{IP} cn bw sp/CyO

Z259   spi^{IIA14} cn bw sp/CyO

Z260   cn fai^{IIA25} bw sp/CyO

Z261   sal^{IIA} cn bw sp/CyO

Z262   cn fas^{IIA92} bw sp/CyO

Z263   ush^{IIA102} cn bw sp/CyO

Z264   str^{IIB09} cn bw sp/CyO

Z265   ck^{IIB33},cn bw sp/CyO

Z266   prd^{IIB} cn bw sp/CyO

Z267   cn rib^{IIB44} bw sp/CyO

Z268   sal^{IIB} cn bw sp/CyO

Z269   cn en^{IIB86} bw sp/CyO

Z270   ptc^{IIB98} cn bw sp/CyO

Z271   ptc^{IIC22} cn bw sp/CyO

Z272   Ddc^{IIC67} cn bw sp/CyO

Z273   cn flb^{IIC82} bw sp/CyO

Z274   ptc^{IIC84} cn bw sp/CyO

Z275   ptc^{IIC99} cn bw sp/CyO

Z276   cli^{IID18} cn bw sp/CyO

Z277   cn brh^{IID61} bw sp/CyO

Z278   cn scb^{IID83} bw sp/CyO

Z279   cn flb^{IIE07} bw sp/CyO

Z280   cn twi^{IIE27} bw sp/CyO

Z281   ptc^{IIE51} cn bw sp/CyO

Z282   cn bw sp Kr^{IIE57}/CyO

Z283   cli^{IIE107} cn bw sp/CyO

Z284   raw^{IIF08} cn bw sp/CyO

Z285   cn Asx^{IIF51} bw sp/CyO

Z286   cn lin^{IIF103} bw sp/CyO

Z287   cn bw sp zip^{IIF107}/CyO

Z288   sna^{IIG} cn bw sp/CyO

Z289   smo^{IIG26} cn bw sp/CyO

Z290   cn flb^{IIG31} bw sp/CyO

Z291   cn cra^{IIG44} bw sp/CyO

Z292   cn flz^{IIG68} bw sp/CyO

Z293   cn scb^{IIG113} bw sp/CyO

Z294   cn twi^{IIH07} bw sp/CyO

Z295   cn mam^{IIH57} bw sp/CyO

Z296   bsk^{IIJ04} cn bw sp/CyO

Z297   cn mam^{IIJ14} bw sp/CyO

Z298   l(2)IIJ59,cn bw sp/CyO

Z299   cn Pu^{IIK105} bw sp/CyO

Z701   cn mam^{IIL61} bw sp/CyO

Z702   cn flb^{IIL65} bw sp/CyO

Z703   cn thi^{IIM44} bw sp/CyO

Z704   slp^{IIM105} cn bw sp/CyO

Z705   s^{IIN23},cn bw sp/CyO

Z706   prd^{IIN27} cn bw sp/CyO

Z707   cn thi^{IIN43} bw sp/CyO

Z708   cn Psc^{IIN48} bw sp/CyO

Z709   Ddc^{IIN49} cn bw sp/CyO

Z710   l(2)IIO32,cn bw sp/CyO

Z711   bib^{IIP39} cn bw sp/CyO

Z712   bsk^{IIP71} cn bw sp/CyO

Z713   cn sha^{IIP118} bw sp/CyO

Z714   ck^{IIQ106},cn bw sp/CyO

Z715   cn eve^{IIR59} bw sp/CyO

Z716   ptc^{IIR87} cn bw sp/CyO

Z717   mid^{IIS21} cn bw sp/CyO

Z718   aop^{IIS} cn bw sp/CyO

Z719	wg^{IIS34} cn bw sp/CyO
Z720	lea^{IIS51} cn bw sp/CyO
Z721	spi^{IIT25} cn bw sp/CyO
Z722	cn en^{IIT84} bw sp/CyO
Z723	cn lin^{IIU35} bw sp/CyO
Z724	ptc^{IIU112} cn bw sp/CyO
Z725	ck^{IIU116}, cn bw sp/CyO
Z726	cn mam^{IIV06} bw sp/CyO
Z727	cn thr^{IIV27} bw sp/CyO
Z728	bib^{IIV46} cn bw sp/CyO
Z729	cn fai^{IIV56} bw sp/CyO
Z730	prd^{IIW29} cn bw sp/CyO
Z731	cn flb^{IIW74} bw sp/CyO
Z732	cn arr^{IIW84} bw sp/CyO
Z733	ptc^{IIW109} cn bw sp/CyO
Z734	smo^{IIX43} cn bw sp/CyO
Z735	cn flb^{IIX51} bw sp/CyO
Z736	Df(2R)gsb^{IIX62}, cn bw sp/CyO
Z737	ptc^{IIX103} cn bw sp/CyO
Z738	cn bw sp $Kr^{IIIA102}$/CyO
Z739	cn en^{IIIB24} bw sp/CyO
Z740	tup^{IIIB29} cn bw sp/CyO
Z741	cn flb^{IIIB41} bw sp/CyO
Z742	cn flb^{IIIB92} bw sp/CyO
Z743	cn flb^{IIIC81} bw sp/CyO
Z744	cn flb^{IIIC87} bw sp/CyO
Z745	odd^{IIIC91} cn bw sp/CyO
Z746	wg^{IIID23} cn bw sp/CyO
Z747	odd^{IIID} dp b pr cn bw sp/CyO
Z748	mid^{IIID67} cn bw sp/CyO
Z749	$bib^{IIID118}$ cn bw sp/CyO
Z750	tup^{IIIE16} cn bw sp/CyO
Z751	l(2)$IIIF12$, cn bw sp/CyO
Z752	cn flb^{IIIF18} bw sp/CyO
Z753	ush^{19} cn bw sp/CyO
Z754	lea^{25} cn bw sp/CyO
Z755	S^{54}, cn bw sp/CyO
Z756	lin^{5E116}/CyO
Z757	cn bw sp Kr^{2}/SM1
Z758	wg^{6K64}/CyO
Z759	prd^{6L07}/CyO
Z760	ptc^{6P43}/CyO
Z761	slp^{7L48}/CyO
Z762	wg^{7L74}/CyO
Z763	odd^{7L82}/CyO
Z764	ptc^{7M59}/CyO
Z765	ptc^{9B28}/CyO
Z766	odd^{9P77}/CyO
Z767	slp^{L12}/CyO
Z768	Kr^{L14}/SM1
Z769	b pr twi^{D5}/CyO
Z770	b pr twi^{O5}/CyO
Z771	b pr eve^{R13}/CyO
Z772	smo^{Q14} b pr/CyO
Z773	prd^{X3} b pr/CyO
Z774	b pr twi^{X10}/CyO

Z301	st $Antp^{4B}$ e/TM3,Sb
Z302	st e stg^{4B51}/TM3,Sb
Z303	spo^{4G28} st e/TM3,Sb
Z304	ru pbl^{5B65} h th st cu sr e^{S} ca/TM3,Sb
Z305	ru h th st knk^{5C77} cu sr e^{S} ca/TM3,Sb
Z306	trh^{5D55} ru h th st cu sr e^{S} ca/TM3,Sb
Z307	ru pbl^{5D85} h th st cu sr e^{S} ca/TM3,Sb
Z308	th st hth^{5E04} cu/TM3,Sb
Z309	ru h th st cu sad^{5F45} sr e^{S} ca/TM3,Sb
Z310	ru h th st cu sr Dl^{5F} e^{S} ca/TM3,Sb
Z311	ru h th st kni^{5F107} cu sr e^{S} ca/TM3,Sb
Z312	l(3)$5G83$,ru h th st cu sr e^{S} ca/TM3,Sb
Z313	ru h^{5H07} th st cu sr e^{S} ca/TM3,Sb
Z314	ru h th st cu sr e^{S} tld^{5H56} ca/TM3,Sb
Z315	ru h th st opa^{5H} cu sr e^{S} ca/TM3,Sb
Z316	st Dl^{6B} e/TM3,Sb
Z317	st e tld^{6B69}/TM3,Sb
Z318	st srp^{6G54} e/TM3,Sb
Z319	st nkd^{6J48} e/TM3,Sb
Z320	shd^{6J102} st e/TM3,Sb
Z321	st e hh^{6L93}/TM3,Sb
Z322	st e hh^{6N16}/TM3,Sb
Z323	st hb^{6N47} e/TM3,Sb
Z324	st e tld^{6P41}/TM3,Sb
Z325	st e tld^{6P117}/TM3,Sb
Z326	ru h th st knk^{7A69} cu sr e^{S} ca/TM3,Sb
Z327	ru h th st ftz^{7B} cu sr e^{S} ca/TM3,Sb
Z328	ru h th st cu sr e^{S} stg^{7B69} ca/TM3,Sb
Z329	ru h shd^{7C94} th st cu sr e^{S} ca/TM3,Sb
Z330	ru h th st cu ems^{7D99} sr e^{S} ca/TM3,Sb
Z331	ru h th st nkd^{7E89} cu sr e^{S} ca/TM3,Sb
Z332	l(3)$7E103$,ru h th st cu sr e^{S} ca/TM3,Sb
Z333	ru h th st Scr^{7F28} cu sr e^{S} ca/TM3,Sb
Z334	ru h th st kni^{7G75} cu sr e^{S} ca/TM3,Sb
Z335	ru h th st cu pnr^{7G111} sr e^{S} ca/TM3,Sb
Z336	ru h th st nkd^{7H16} cu sr e^{S} ca/TM3,Sb
Z337	ru h th st cu sr e^{S} tld^{7H41} ca/TM3,Sb
Z338	ru h^{7H94} th st cu sr e^{S} ca/TM3,Sb
Z339	ru spo^{7J09} h th st cu sr e^{S} ca/TM3,Sb
Z340	ru h th st kkv^{7J22} cu sr e^{S} ca/TM3,Sb
Z341	trh^{7J83} ru h th st cu sr e^{S} ca/TM3,Sb
Z342	ru h th st gra^{7J86} cu sr e^{S} ca/TM3,Sb
Z343	ru h th st hb^{7L06} cu sr e^{S} ca/TM3,Sb
Z344	ru h th st gra^{7L12} cu sr e^{S} ca/TM3,Sb
Z345	ru h th st cu sr e^{S} stg^{7L105} ca/TM3,Sb
Z346	ru rho^{7M43} h th st cu sr e^{S} ca/TM3,Sb
Z347	ru h th st hb^{7M48} cu sr e^{S} ca/TM3,Sb
Z348	ru h th st cu sr e^{S} stg^{7M53} ca/TM3,Sb
Z349	ru h th st cu sr e^{S} tld^{7M89} ca/TM3,Sb
Z350	ru h th st opa^{7N57} cu sr e^{S} ca/TM3,Sb
Z351	ru h th st hb^{7O14} cu sr e^{S} ca/TM3,Sb
Z352	ru h th st cu sr e^{S} tld^{7O47} ca/TM3,Sb
Z353	ru pbl^{7O79} h th st cu sr e^{S} ca/TM3,Sb
Z354	ru h th st cu sr e^{S} kay^{7P54} ca/TM3,Sb
Z355	st e sro^{8A34} ca/TM3,Sb
Z356	st cno^{8A62} e/TM3,Sb

Z357 st e stg^{8A83}/TM3,Sb

Z358 st Scr^{8B48} e/TM3,Sb

Z359 st e pnt^{8B74}/TM3,Sb

Z360 st e E(spl)^{8D06}/TM3,Sb

Z361 st e crb^{8F105}/TM3,Sb

Z362 h^{8K115} st e/TM3,Sb

Z363 st e tld^{8L38}/TM3,Sb

Z364 ru h th st cu sr e^s stg^{9A19} ca/TM3,Sb

Z365 ru h th st ftz^{9A99} cu sr e^s ca/TM3,Sb

Z366 ru h th st cu sr e^s kay^{9B33} ca/TM3,Sb

Z367 ru h th st cu sr e^s tld^{9B66} ca/TM3,Sb

Z368 ru h th st opa^{9C96} cu sr e^s/TM3,Sb

Z369 ru h th st cu sr Dl^{9D} e^s ca/TM3,Sb

Z370 ru h th st cu sr e^s tld^{9D36} ca/TM3,Sb

Z371 ru h th st hau^{9G14} cu sr e^s ca/TM3,Sb

Z372 ru h th st nkd^{9G33} cu sr e^s ca/TM3,Sb

Z373 ru h th st cu yrt^{9G53} sr e^s ca/TM3,Sb

Z374 ru h th st Scr^{9G84} cu sr e^s ca/TM3,Sb

Z375 ru h th st ftz^{9H34} cu sr e^s ca/TM3,Sb

Z376 ru h th st nkd^{9H52} cu sr e^s ca/TM3,Sb

Z377 ru h th st cu sr e^s pnt^{9J31} ca/TM3,Sb

Z378 ru h th st cu sr e^s stg^{9K21} ca/TM3,Sb

Z379 ru h th st cu sr Dl^{9K} e^s ca/TM3,Sb

Z380 ru h th st hb^{9K49} cu sr e^s ca/TM3,Sb

Z381 ru h th st hb^{9K57} cu sr e^s ca/TM3,Sb

Z382 ru h^{9K78} th st cu sr e^s ca/TM3,Sb

Z383 ru h shd^{9K84} th st cu sr e^s ca/TM3,Sb

Z384 ru h th st cu sr e^s tld^{9K88} ca/TM3,Sb

Z385 ru h th st cu sr e^s hh^{9K94} ca/TM3,Sb

Z386 ru h th st cno^{9K104} cu sr e^s ca/TM3,Sb

Z387 ru h th st cu srp^{9L06} sr e^s ca/TM3,Sb

Z388 ru h th st neu^{9L119} cu sr e^s ca/TM3,Sb

Z389 ru h th st Pc^{9M21} cu sr e^s ca/TM3,Sb

Z390 ru h th st cu sr Dl^{9M} e^s ca/TM3,Sb

Z391 ru h^{9N53} th st cu sr e^s ca/TM3,Sb

Z392 ru h th st opa^{9O24} cu sr e^s ca/TM3,Sb

Z393 ru h th st ftz^{9O93} cu sr e^s ca/TM3,Sb

Z394 ru h th st cu sr Dl^{9P} e^s ca/TM3,Sb

Z395 ru h th st hb^{9Q17} cu sr e^s ca/TM3,Sb

Z396 ru h th st cu sr e^s tld^{9Q19} ca/TM3,Sb

Z397 ru h th st cu sr e^s tld^{9Q74} ca/TM3,Sb

Z398 ru h th st cu sr Dl^{9Q} e^s ca/TM3,Sb

Z399 th st hb^{9R12} cu/TM3,Sb

Z801 st ems^{10A37} e/TM3,Sb

Z802 st cno^{10B01} e/TM3,Sb

Z803 st e hh^{10B54}/TM3,Sb

Z804 st sad^{10D104} e/TM3,Sb

Z805 st e tld^{10E95}/TM3,Sb

Z806 st bch^{10E113} e/TM3,Sb

Z807 dib^{10F27} st e/TM3,Sb

Z808 st e tld^{10F102}/TM3,Sb

Z809 st Dl^{10G} e/TM3,Sb

Z810 st yrt^{10H59} e/TM3,Sb

Z811 srw^{10K28} st e/TM3,Sb

Z812 dib^{10K92} st e/TM3,Sb

Z813 dib^{10L34} st e/TM3,Sb

Z814 ru h th st cu sr e^s crb^{11A22} ca/TM3,Sb

Z815 ru h th st neu^{11B116} cu sr e^s ca/TM3,Sb

Z816 ru h th st cu sr e^s sro^{11C49} ca/TM3,Sb

Z817 ru h th st cu sr e^s hh^{11C89} ca/TM3,Sb

Z818 ru h th st hb^{11C102} cu sr e^s ca/TM3,Sb

Z819 ru pbl^{11D05} h th st cu sr e^s ca/TM3,Sb

Z820 ru h^{11D24} th st cu sr e^s ca/TM3,Sb

Z821 h^{12C89} st e/TM3,Sb

Z822 st neu^{12H56} e/TM3,Sb

Z823 ru h th st Scr^{13A59} cu sr e^s ca/TM3,Sb

Z824 ru st opa^{E8} e ca/TM3,Sb

Z825 ru h th st cu sr e^s stg^{13D22} ca/TM3,Sb

Z826 ru h th st cu bxd^{13D49} sr e^s ca/TM3,Sb

Z827 ru h th st opa^{13D92} cu sr e^s ca/TM3,Sb

Z828 ru h th st cu sr e^s hh^{13E02} ca/TM3,Sb

Z829 ple^{14A82}, st e/TM3,Sb

Z830 st kni^{14B107} e/TM3,Sb

Z831 st hb^{14C69} e/TM3,Sb

Z832 st kkv^{14C73} e/TM3,Sb

Z833 st knk^{14D79} e/TM3,Sb

Z834 st hb^{14F21} e/TM3,Sb

Z835 h^{14H103} st e/TM3,Sb

Z836 hau^{11}/TM3,Sb

Z837 Dl^{41}/TM3,Sb

Z838 put^{135}/TM3,Sb

Z839 tll^{L10}/TM3,Sb

Z840 kkv^{IB22}/TM3,Sb

Z841 neu^{IF65}/TM3,Sb

Z842 hh^{IJ35}/TM3,Sb

Z843 h^{IK93}/TM3,Sb

Z844 kni^{IL}/TM3,Sb

Z845 Dl^{IL79N}/TM3,Sb

Z846 h^{IL79K}/TM3,Sb

Z847 hau^{IM112G}/TM3,Sb

Z848 neu^{IN94}/TM3,Sb

Z849 opa^{IIC71}/TM3,Sb

Z850 kni^{IID}/TM3,Sb

Z851 kni^{IIE72}/TM3,Sb

Z852 hh^{IIK74}/TM3,Sb

Z853 hh^{IIO107}/TM3,Sb

Z854 opa^{IIP32}/TM3,Sb

Z855 ru h th st hb^{IIU}/TM3

Z856 kni^{IIV95}/TM3,Sb

Z857 hh^{IIX57}/TM3,Sb

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