DROSOPHILA INFORMATION SERVICE

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Lawrence, Kansas 66045 - USA

DROSOPHILA INFORMATION SERVICE

Number 63

Including the Special Report:

THE MOLECULAR GENOME OF **Drosophila melanogaster:**
Catalogs of cloned DNA, breakpoints
and transformed inserts by chromosome location.

by

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June 1986

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For information regarding submission of manuscripts or other contributions to Drosophila Information Service, contact P.W. Hedrick, Editor, Division of Biological Sciences, University of Kansas, Lawrence, Kansas 66045 - USA.

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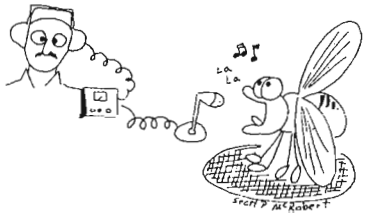

graphics by

Scott P. McRobert

ANNOUNCEMENTS

NOTICE OF PRICE CHANGE: Effective with Volume 65 (June 1987), the cost of a subsciption will rise to $7.00 per copy if paid in advance ($8.00 if invoiced). This is the first price raise that DIS has had in quite a few years and is due to rising costs of printing and postage, as usual. Back issues (vol. 64 and prior) will remain at $5/copy.

Notice of Publication: The next installment of The Genome of **Drosophila melanogaster** by Dan Lindsley and Georgianna Zimm will appear as DIS 64 in the fall of 1986. It is anticipated that it will include mutations beginning with the letter L (including the lethals), Duplications, Inversions, Rings, Translocations, and Transpositions. In addition, there will be an ordered list of rearrangement breakpoints and polytene gene localizations prepared by Andrew Cockburn from a computer search of the current data base for the revised edition, plus the as yet unrevised material from the Red Book.

27th ANNUAL DROSOPHILA RESEARCH CONFERENCE

Held April 10-13, 1986, at the Asilomar Conference Center in Pacific Grove, California.

Plenary Session - Chair: Larry Sandler — Friday, April 11
Speakers: M. Gatti, R.S. Hawley, A.J. Hilliker, A. Chovnick, L. Sandler

Concurrent Sessions: — Friday afternoon, April 11
Population genetics and evolution (Chair - Margaret Kidwell)
Pattern formation (Chair - Judith Lengyel)
Gene expression (Chair - Steven Henikoff)

Poster Session (odd-numbered posters) — Friday evening, April 11
Consecutive Workshops:
Oncogene homologues (Chair - John McDonald)
Heat shock (Chair - Nancy Petersen)

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Gene interactions (Chair - Paul Bingham)
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28th Annual Drosophila Research Conference will be held May 20-24, 1987, at the Bismarck Hotel in Chicago, Illinois. Local arrangements are being handled by Janice Spofford (University of Chicago); please write to get on the mailing list to: Office of Continuing Medical Education, SBRI J 131 (Box 139), University of Chicago, Chicago, Illinois 60637, or call 312-962-1056. The conference will start Wednesday evening (May 20) and run thru Sunday noon, May 24 at the Bismarck Hotel, which is close to the rapid transit from O'Hare airport. Room rates: $45 single and $55 double. All meetings will be at the hotel. William Engels is coordinating the program arrangements (Lab of Genetics, 509 Genetics Bldg, 445 Henry Mall, University of Wisconsin, Madison, WI 53706). The regular mailing will be sometime in the Fall 1986.

New Publication: **Diptera: Drosophilidae,** by Gerhard Bächli and Hans Burla, Vol. 7 (1985) of Insecta Helvetica, edited by the Swiss Entomological Society, 116 pages, 3 color plates; 20 Swiss Francs (about $9). This volume, written in German, is thought to include all Central European species. It contains information on collecting, biology and morphology, as well as illustrated keys to genera and species.

New laboratory: Einar Arnason has organised a new Drosophila laboratory at the Institute of Biology, University of Iceland, Grensásvegur 12, 108 Reykjavik, Iceland (tel: 354-1-685433). We request being put on mailing lists where such exist. Fields of interest are evolutionary biology: population genetics.

Stock not available: We have been receiving requests for the **Drosophila melanogaster** stock Barlike-eyes (3-94) Ble, which was erroneously listed in Stock List #3 (DIS 57). We regret to say that we do not, and never did, have the stock in question.

Recommends needed service: Prof. Robert Arking, Wayne State University, recommends Watkins Tweezer Refinishing (P.O. Box 1402, 1209 Hilltop Drive, Mount Dora, Florida 32757 Tel 904-386-8037) for repairing laboratory forceps at ca. $2.50/tweezers. He has been very satisfied with their work. In the past Prof. Arking had to rely on replacing damaged forceps and was pleased to find this service through a watchmaker friend.

ANNOUNCING A 1987 UCLA SYMPOSIUM:

Molecular Biology of Invertebrate Development
March 15-21, 1987, Park City, Utah. Organizer: J. Dennis O'Connor. Advisory Committee: Hans Bode, Eric Davidson, David Epel, James Fristrom, Anthony Mahowald & Gerald Wyatt.
Abstract Submission Deadline: November 21, 1986

The development of a single cell into a multicellular organism comprised of several differentiated cell types and organ systems requires the integration of gene expression, regulation of membrane transport, and the recognition of both intracellular and extracellular signals. These and other related phenomena are being investigated in diverse plant and animal species. Invertebrates continue to offer excellent experimental systems to study the molecular mechanisms involved in the regulation of development, due in no small measure to the facility with which they can be cultured, their relatively small genome, and the number and array of the critical signals (e.g., hormones, pheromones, neuropeptides). By focusing on invertebrate development, this meeting will permit a relatively detailed examination of the principal events of development in the most extensively investigated organisms, while at the same time permitting comparative analysis.

Plenary Session topics will include: Vitellogenesis/Oogenesis, Molecular Sequelae of Fertilization, Early Molecular Events in Cell Lineage, Segmentation and Early Pattern Events, Cell Lineages and Commitment, Molecular Neurobiology, Genetic and Cellular Mechanisms in Imaginal Disc Development, Juvenile Hormone/Gene Expression/Metamorphosis, Cis and Trans Regulatory Elements, Regulation at the Post-transcriptional Level.

Related 1987 UCLA Symposia include: (Abstract deadlines: * Oct 3, 1986; ** Oct 24, 1986; § Nov 21, 1986) **Steroid Hormone Action,** Jan 17-23, 1987*, Park City, Utah. **Molecular Paradigms for Eradicating Helminthic Parasites,** Jan 24-Feb 1, 1987*, Steamboat Springs, Colorado. **Molecular Biology of Intracellular Protein Sorting and Organelle Assembly,** Jan 30-Feb 5*, Taos, New Mexico. **Signal Transduction in Cytoplasmic Organization and Cell Motility,** Feb 15-21**, Lake Tahoe, California. **Mechanisms of Control of Gene Expression** (a Roche-UCLA Symposium), March 28-April 3§, Steamboat Springs, Colorado.

For further information, please call or write to: UCLA Symposia, Molecular Biology Institute, University of California, Los Angeles, CA 90024. Tel 213-206-6292. Telex: UCLA Symposia 9103427597. Applications will be accepted at any time for meetings which are not over-subscribed.

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Part 4: TEACHING NOTES (alphabetical by author)

Achary, P.M. and C.C. Das. Berhampur University, Berhampur, India. Evaluation of Hydroxyurea as an in vivo synchronizing agent of DNA replication on the polytene chromosomes of Drosophila.

Replicating polytene nuclei of Drosophila salivary glands exist in an asynchronous state with respect to their S-phase, thus representing a heterogenous population of polytene nuclei (Plaut et al. 1964; Rodman 1968; Lakhotia & Mukherjee 1970; Kalisch & Haegele 1973; Mukherjee et al. 1980; Achary et al. 1981).

Hydroxyurea, a known DNA synthesis inhibitor, is used in the present investigation for evaluating its in vivo synchronization effect on the replicating polytene nuclei.

The inhibitory action of Hydroxyurea on DNA synthesis is probably by blocking the activation of reductase activity by specific deoxyribonucleoside triphosphates (Swindlehurst et al. 1971).

Early third instar giant mutant female larvae of **Drosophila melanogaster** (gt w^a/Df(1)62 g^{18}) were fed on sucrose (1M) solution containing 1mg/ml Hydroxyurea for 48 hr using an apparatus designed for chemical mutagenesis (Achary & Dutta 1984). ^{3}H-TdR autoradiograms were prepared from larval salivary glands and the labelling patterns were scored following the classification of Chatterjee & Mukherjee (1975).

Table and histogram show predominance of mid-part of S-phase nuclei (3C and 3D patterns). While the frequencies of early part (DD, 1C and 2C patterns) was 13.75 percent and late part (2D and 1D patterns) was 11.89 percent, the mid-part (3C and 3D) showed a percentage as high as 73.29.

The synchronizing effect of Hydroxyurea at the mid-part of S-phase simulates those of FdUrd (Achary et al. 1981) and Aphidocolin and Ricin (Duttagupta & Banerjee 1984), where more than sixty-five percent of mid-part nuclei were recorded. Cold thymidine chase experiments, for releasing the block and to obtain further synchronization at other parts of S-phase, are in progress.

Acknowledgement: We gratefully acknowledge Dr. A.K. Duttagupta, Calcutta University, for providing laboratory facilities.

References: Achary, P.M. et al. 1981, Chromosoma 82:505-514; Achary, P.M. & P.K. Dutta 1984, DIS 60:209; Chatterjee, S.N. & A.S. Mukherjee 1975, Ind. J. Expt. Biol. 13:452-459; Duttagupta, A.K. & S. Banerjee 1984, DIS 60:89-90; Kalisch, W.E. & K. Haegele 1973, Chromosoma 44:265-283; Lakhotia, S.C. & A.S. Mukherjee 1970, J. Cell Biol. 47:18-33; Mukherjee et al. 1980, in: Development and Neurobiology of Drosophila (Hollaender, ed.) Plenum Pr NY, p57-84; Plaut, W. et al. 1966, J. Molec. Biol. 16:85-93; Rodman, T.C. 1968, Chromosoma 23:271-287.

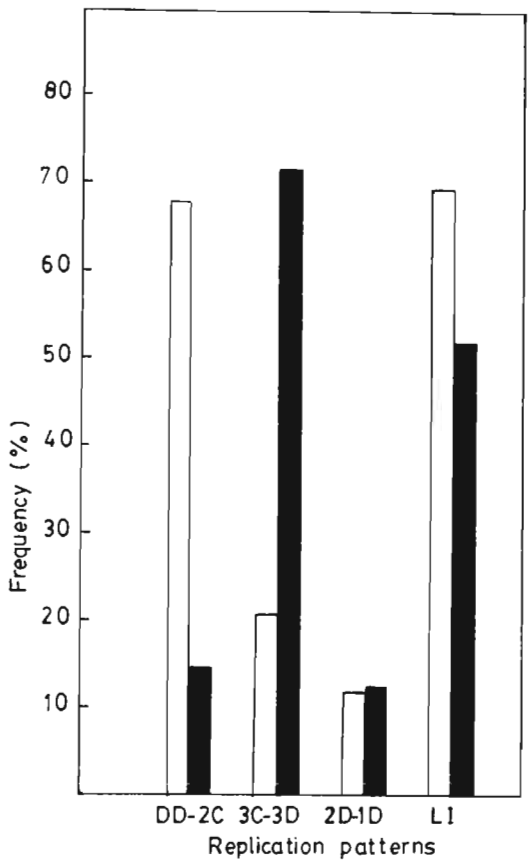


Table 1. Frequencies (in %) of different types of ^{3}H-TdR replicating patterns of salivary gland polytene nuclei of giant **Drosophila melanogaster** larvae fed with Hydroxyurea for 48 hr. Numbers in () show number of nuclei observed.

	Replication patterns								
	DD	1C	2C	3C	3D	2D	1D	U1	LI
CONTROL:	**tn** = in total nuclei					**ln** = in labelled nuclei			
tn	31.62	12.50	2.94	5.88	8.09	3.68	4.41	30.89	69.12
	(43)	(17)	(4)	(8)	(11)	(5)	(6)	(42)	(94)
ln	45.74	18.09	4.26	8.51	11.70	5.32	6.38	--	--
		68.09		20.21			11.70		
HYDROXYUREA:	**tn** = in total nuclei					**ln** = in labelled nuclei			
tn	0.00	2.96	4.15	16.60	20.36	5.34	2.37	48.22	51.78
	(0)	(15)	(21)	(84)	(103)	(27)	(12)	(244)	(262)
ln	0.00	5.73	8.02	32.06	39.31	10.31	4.58	--	--
		13.75		72.37			11.89		

Figure 1. Histogram showing frequencies of early (DD-2C), mid (3C-3D) and late (2D-1D) patterns within the labelled nuclei. The open bars represent control and solid bars represent experimental (Hydroxyurea). LI indicates labelling index.

Adell, J.C. & L.M. Botella. Universidad de Valencia, Spain. Effect of crowding in cultures of lethal (2) giant larvae.

A larval arrest in development of **Drosophila melanogaster** was first detected in crowded conditions by Mensua & Moya (1983) in different wild strains. Afterwards it has been also shown in other Drosophila species, other Drosophilids (Botella & Mensua 1985a), as well as in other holometabolous insects (**Tribolium castaneum**) data to be published elsewhere. Recently it has been shown (Botella & Mensua 1985b) that stopped larvae seem to have decreased levels of molting hormone. This fact has been used as a starting point to investigate the larval stop in mutants known to be affected in the levels of molting hormone, such as lethal giant larvae (Welch 1957).

For this purpose crowded cultures (70 larvae in 0.5 ml of Lewis medium) of the strain (ℓ(2)gl a px or/SM5 al^2 Cy lt^v cn sp^2) were reared at 25°C. At different days from the seeding day, different sets of crowded cultures were subjected to overfeeding (Moya & Mensua 1983) allowing larvae to migrate spontaneously to larger vials (10 x 25 cm with 10 ml of Lewis medium in an inclined position). In this way the initial population was divided into two subpopulations, inner (non-migrated individuals) and outer (individuals recovered by overfeeding following their spontaneous migration).

Two controls, uncrowded and crowded (never subjected to overfeeding), were also taken. A total of five replications were made of every set of vials.

Table 1 shows survival in inner, outer and total population, and mean developmental time in inner and outer populations throughout overfeedings and controls.

As can be seen from this table, the number of larvae recovered by overfeeding decreases as the cultures developed and becomes zero in the two last overfeedings.

As reflected by the mean developmental time (MDT), larvae recovered in the three first outer populations may be considered stopped.

However, from a certain age on (14 day old) no larvae were recovered by overfeeding, and hence third instar larvae of this age die, possibly as a consequence of the mutation ℓ(2)gl.

In Table 2 the larval mortality before and within the third instar are recorded expressed in %.

In contrast with other strains in which dead larvae cannot directly be seen, careful inspection of these cultures reveals the presence of dead larvae in third instar.

The main point to be noted is the increase in larval mortality both before and within third instar as the overfeedings are later. The situation of the crowded control becomes similar to the cultures overfed at 16th day.

The effect of competition seems to enhance the larval mortality, and we suspect that some heterozygous larvae for the mutant ℓ(2)gl die when the competition becomes stronger. These results are only a preliminary report to be further investigated but which appears to indicate a phenocopy effect of ℓ(2)gl due to an interaction between environment (competition) and heterozygous constitution for this mutant.

References: Botella, L.M. & J.L. Mensua 1985a, IXth E.D.R.C. Hungary; _____ 1985b, DIS 61:39-40; Mensua, J.L. & A. Moya 1983, Heredity 51:347-352; Welch, R.M. 1957, Genetics 42:5434-5559.

Table 1. Survival in inner, outer and total population, and mean developmental time (MDT) in inner and outer populations throughout overfeedings and control.

	Survival (inner)	Survival (outer)	Survival (total)	MDT (inner)	MDT* (outer)
Uncrowded control:					
	---	50.2±1.6	50.2±1.6	---	12.8±0.1
Crowded control:					
	13.5±2.6	---	13.5±2.6	13.9±0.3	---
Overfeeding (days):					
8	6.0±0.6	42.8±1.6	48.8±1.1	13.2±0.5	16.2±0.1
10	14.0±2.3	22.4±5.6	36.4±3.9	13.4±0.1	18.4±0.4
12	13.7±3.4	8.5±6.4	22.2±6.0	14.3±0.3	21.5±0.2
14	11.0±2.0	---	11.0±2.0	13.3±0.2	---
16	10.0±1.9	---	10.0±1.9	12.8±0.4	---

*Regression values: a(intercept at origin) = 5.41; b(slope) = 1.33; R^2 = 0.99.

Table 2. Larval mortality before and within 3rd instar.

	% dead larvae before 3rd instar	% dead larvae* in 3rd instar
Control (crowded)	44.4%	54.4%
Overfeeding (days):		
8	24.9%	2.7%
10	37.4%	4.6%
12	31.6%	20.9%
14	35.7%	58.2%
16	44.6%	54.1%

* this percentage of dead larvae was calculated referring to individuals which attain 3rd instar.

Adell, J.C. & L.M. Botella. Universidad de Valencia, Spain. Effect of long-term competition on development of **Drosophila melanogaster.**

It has been reported (Mensua & Moya 1983) that competition induces larvae of **D.melanogaster** to arrest their development in third larval instar. This is true both, at different temperatures (Botella & Mensua 1985a) and at different crowding densities (Mensua et al. 1983). Nevertheless, the number of arrested larvae depends on the degree of crowding (Mensua et al. 1983). On the other hand, long-term competition in other Drosophilids (such as **Chymomyza costata**) induces third instar larvae to enter diapause. This being so, and to further investigate on the effects of continuous crowding in **D.melanogaster**, two sets of experiments have been carried out in this species, reared at two temperatures (19 and 25°C). For this purpose two different sets of 5 x 0.8 cm vials with 0.5 ml of Lewis medium were seeded with 70 newly hatched larvae from an isogenic Oregon-R strain. A total of 30 vials were started at 19°C and 26 vials at 25°C. At different intervals, every 9 days at 25°C and every 18 days at 19°C, the still remaining larvae were extracted from the original cultures, and transferred by groups of 70 larvae to vials identical to the previous ones, keeping in this way the same crowding. The number of emerged adults was recorded daily until the exhaustion of the cultures.

Table 1 is a life table showing the following parameters: the total number of remaining larvae at the end of each transfer, the number of dead larvae, the total number of pupated larvae, the number of dead pupae and the number of emerged adults, each accompanied by their corresponding percentage referred to the total number of larvae in each period for 19°C and 25°C. Bearing in mind that previous experiments carried out in our laboratory have shown that mean developmental time in the same strain in uncrowded cultures is about 19-23 days at 19°C and about 9-13 days at 25°C, a large delay in mean developmental time can be observed in both temperatures as a consequence of long-term competition. In our case development has been prolongated until 32 days at 25°C and 53 days at 19°C. It is worth pointing out that pupal mortality increases as competition lasts longer. This effect may be due to the increased intoxication of larvae subjected to the competition process which are being forced to ingest their metabolic wastages (uric acid among them) (Botella et al. 1985). It is also interesting to note the higher rates of mortality obtained at 19°C owing to the fact that 25°C is a better temperature for Drosophila development than 19°C (Ashburner & Thompson 1978).

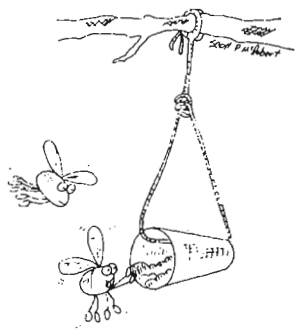
On the other hand, in contrast with the result obtained in Drosophilids with larval diapause, such as **Chymomyza costata** (Botella & Mensua 1985b), long-term competition cannot stop larval development for months in third instar. In fact, as can be seen from the table, all larvae finally pupated or died. In this sense larval arrest in Drosophila may be viewed rather as a kind of quiescent state susceptible to end as soon as environmental conditions become slightly more favourable throughout the successive transfers to fresh medium. This mechanism would be parallel to the adaptive meaning of larval diapause in non-diapausing species such as Drosophila, but in a more flexible way than diapause do.

Table 1. Life Table from the experiments of long-term competition in **Drosophila melanogaster** at 19°C and 25°C.

Temperature	Days[a]	Dead larvae(%)	Pupated larvae(%)	Dead Pupae(%)*	Emerged Adults(%)*	Remaining larvae(%)
19°C	1-18	255(12.1)	372(17.3)	99(26.6)	273(73.4)	1473(70.1)
	19-36	495(33.7)	908(61.8)	312(34.4)	596(65.6)	67(4.6)
	37-54	43(64.2)	24(35.8)	17(70.8)	7(29.2)	0
25°C	1-9	489(26.9)	350(19.3)	45(12.8)	306(87.2)	980(53.8)
	10-18[b]	216(23.7)	545(59.9)	69(12.7)	476(87.3)	149(16.4)
	19-27	50(35.7)	82(58.6)	33(40.2)	49(59.8)	8(5.7)
	28-36	5(72.5)	3(37.5)	3(100)	0	0

* % of emerged adults and dead pupae were referred to total no. of pupated larvae. a = days of culture. b = % of 910 larvae which were seeded.

References: Ashburner, M. & J.N. Thompson 1978, The Genetics and Biology of Drosophila, v.2a Academic Pr.; Botella, L.M. & J.L. Mensua 1985a, IXth E.D.R.C. Hungary; _____ 1985b, DIS 61:39-40; Botella, L.M., A. Moya, C. Gonzalez & J.L. Mensua 1985, J. Insect Physiol. 31:179-184; Mensua, J.L., L.M. Botella & A. Moya 1983, VIIIth E.D.R.C. Cambridge; Mensua, J.L. & A. Moya 1983, Heredity 51:347-352.



Alahiotis, S.N. and G.N. Goulielmos.
University of Patras, Patras, Greece.

Fertile F_1 males and females from crosses between **Drosophila mauritiana** females and **D.melanogaster** or **D.simulans** males.

It is known that in crosses between the sibling species **Drosophila simulans** and **D.mauritiana** (a species originated from **D.simulans**; Tsacas et al. 1981) fertile females and sterile males are yielded in the F_1 generation, independently of what species is used as female. Moreover, it is also known that in crosses between the sibling species **D.melanogaster** and **D.mauritiana**, sterile females or males (in respect with the melanogaster sex used) are yielded in the F_1 generation. In spite of this general consensus, we present now data showing that fertile males and females can be obtained from some crosses between the sibling species pair mentioned above. Thus, in one out of 55 pair matings, where **mauritiana** was used as female and **melanogaster** as male, fertile F_1 males and females appeared. The fertility of these individuals was tested by mating them and noticing if F_2 (or F_n) progeny are yielded. These interspecific fertile hybrids have been named mame and are kept with success in our laboratory as a stock for 10 generations so far. In the opposite cross (♀ **melanogaster** x **mauritiana** ♂), seven out of 43 pair matings were found to yield hybrid females (named mema) which are sterile when crossed with **melanogaster** or **mauritiana** males. It must be noted that as **melanogaster** we used the CyL^4/Pm stock, while in a previous effort using another **melanogaster** stock (homozygous for the malate dehydrogenase fast form), we failed to get one successful mating (yielded offspring) out of 92 performed.

In another interspecific cross-type where **mauritiana** was used as female and **simulans** as male, two out of 82 pair matings yielded fertile F_1 males and females. These interspecific fertile hybrids have been named masi and are kept in our lab as a stock, like mame. In the case where **mauritiana** is used as male and **simulans** as female, 63.41% (26/41) successful matings obtained. Each such successful pair mating yielded females and males which are sterile when crossed with each other but fertile when crossed with **mauritiana** (males) and **simulans** (males or females). When **mauritiana** females are crossed with the above described interspecific hybrids (named sima), no progeny are produced. The same is also true in the cross ♀ **mauritiana** x mame ♂. In crosses ♀ mame x masi ♂ (and the reciprocal), some pair matings are successful, yielding females and males which possibly get genes from three different species (**melanogaster, simulans, mauritiana**). In Table 1 we show all the above information described and in Table 2 we give a brief summary of the reproductive isolation status among the three sibling species examined as it was found in our lab (with the strains of the species we used).

It must be noted that the combination **melanogaster-simulans** was tested and our findings verify the previous consensus (see Table 2), that is, sterile F_1 females or males are produced, dependent on the **melanogaster** sex used. However, in a mass cross (6 ♀ **melanogaster** x **simulans** ♂ 6) a part of the sterile females obtained as expected, 3 hybrid males were also

Table 1. Successful and unsuccessful pair-matings between pairs of **D.melanogaster (mel)**, **D.simulans (sim)** and **D.mauritiana (maur)**, as well as between fertile interspecific hybrids* and their parents.

species pair	No. of matings successful	unsuccessful	total
♀ **sim** x **maur** ♂	26	15	41
♀ **maur** x **mel** ♂	1	54	55
♀ **maur** x **sim** ♂	2	80	82
♀ **mel** x **maur** ♂	7	36	43
♀ masi x **sim** ♂	15	27	42
♀ masi x **maur** ♂	16	1	17
♀ **maur** x masi ♂	0	49	49
♀ **sim** x masi ♂	1	3	4
♀ mame x **mel** ♂	13	0	13
♀ mame x **maur** ♂	1	7	8
♀ **maur** x mame ♂	0	38	38
♀ **mel** x mame ♂	8	0	8
♀ mema x **mel** ♂	0	14	14
♀ mema x **maur** ♂	0	28	28
♀ sima x **sim** ♂	7	3	10
♀ sima x **maur** ♂	3	3	6
♀ **maur** x sima ♂	0	16	16
♀ **sim** x sima ♂	9	0	9
♀ masi x mame ♂	5	18	23
♀ mame x masi ♂	7	25	32

*The name of the interspecific hybrids is composed of two syllables. The first is from the ♀ and the seond from the ♂ species-parent name.

Table 2. Hybridization possibilities between the 3 sibling species, **melanogaster, simulans, mauritiana**, as it has been found in our lab.

parents	♀	♂
♀ **mel** x **sim** ♂	sterile	---
♀ **sim** x **mel** ♂	---	sterile
♀ **mel** x **maur** ♂	sterile	---
♀ **maur** x **mel** ♂	fertile	fertile
♀ **sim** x **maur** ♂	fertile*	fertile*
♀ **maur** x **sim** ♂	fertile	fertile

*but sterile when crossed with each other (see text)

Table 3. Mating propensities in multiple choice experiments involving the interspecific fertile hybrids masi and its parental species, **mauritiana** and **simulans.**

Cross A x B	♀AxA♂	♀AxB♂	♀BxA♂	♀BxB♂	No. of chambers*	Sexual Isolation Index ± S.E.
sim x masi	25	22	13	27	6	0.195 ± 0.105
maur x masi	5	14	15	37	8	0.183 ± 0.116

* in each chamber existed 12 virgin females and 12 males from each stock.

noticed. They electrophorized for the alcohol dehydrogenase and exhibited an intermediate (between **melanogaster** and **simulans**) electrophoretic pattern.

The situation where **mauritiana** females do not give offsprings with masi or mame males, was further investigated in an attempt to see if this situation is due to pre- or post-mating isolation mechanism. Thus, performing multiple choice experiments (see for methodology Kilias & Alahiotis 1982), the mating propensities of our interspecific hybrids with their parental species were determined. As it is shown in Table 3, **mauritiana** females copulate with masi males, a fact which demonstrates that the absolute reproductive isolation observed between them is not due to premating mechanisms. Furthermore the same table shows that no significant sexual isolation toward homogametic mating has been developed. Taking into consideration the **melanogaster**-mame combination, we observed in preliminary experiments, that all the four possible mating types can be obtained, while this is not true in the case of **mauritiana**-mame combination where **mauritiana** females do not seem to copulate with mame males.

The situation described here regarding the reproductive isolation status between three sibling species in the **D.melanogaster** subgroup differs with that which it was so far known. These differences may be based on the genetic composition of our strains used (e.g. CyL^4/Pm for **D.melanogaster**; **D.simulans** was captured recently from a Greek natural population) or on some evolutionary changes of **D.mauritiana** under the laboratory conditions where lately is maintained.

The implication of these hybrids to the study of the speciation mechanisms is obvious and can be proved important in understanding the evolution of interspecific reproductive isolation. The elucidation of the detailed genetic organization of these hybrids (the status of which was also verified by electrophoretic and cytogenetic criteria) will contribute greatly to the approach of the above purpose.

References: Kilias, G. & S.N. Alahiotis 1982, Evolution 36:121-131; Tsacas et al. 1981, in: The Genetics and Biology of Drosophila, Ashburner, Carson & Thompson (eds.), Acd. London, Vol 3a:197-259.

Albers, K.B.M. and R. Bijlsma. University of Groningen, Haren, Netherlands. Selection for increase in tolerance with respect to xenobiotics in **Drosophila melanogaster.**

It is well known that **D.melanogaster** can readily become tolerant for a wide range of toxic chemicals when present in its environment. To obtain insight in the process of developing tolerance, two different populations of **D.melanogaster** were subjected to selection on five xenobiotics. Selection was performed by rearing the flies in cages on food supplemented with the xenobiotics. During the experiment the concentrations were increased regularly in 7-8 steps and the initial concentrations and the concentrations after 17 months (the moment the tolerance levels were determined) of the different chemicals are given: (1) phenobarbital (sodium salt): 250 → 1300 ppm; (2) rotenon (a commercial anti-flea powder containing 0.9% rotenon was used): 6 → 24 ppm; (3) malathion (a commercial preparation containing 50% malathion was used): 0.15 → 0.65 ppm; (4) carbaryl (also a commercial preparation containing 50% carbaryl was used): 6 → 28 ppm; (5) DDT: 15 → 65 ppm. The first chemical is used as a drug; the other four are or have been used as insecticides.

For the experiments two different sets of each six population cages were established. The Bogota populations were initiated with 36 independent lines isolated from the Bogota base population as described by Bijlsma (1980). The 50 x 50 populations were initiated with 40 lines from the second reisolation from the original 50 x 50 base population as described by Bijlsma & Van Delden (1977). All cages of each set were provided with standard food (for description of the food see Bijlsma 1980) for the first weeks to get them well established. Thereafter one cage of each set was kept on this food (control) while the others were supplied with standard food supplemented with one of the five toxic compounds. To standardize the selection pressures somewhat, the initial concentrations were chosen in such a way that the larval viability was approximately 40-60%; as a results the density in the cages was kept well over a thousand individuals. When the concentrations were increased during the experimental period, it was also ensured that the population density stayed above this level.

Table 1. Mean egg-to-adult survival (mean ± S.E. in angles) for the selected populations on food supplemented with the different xenobiotics together with the survival of the control populations at the same concentration.

		Bogota		50 x 50	
		selected	control	selected	control
phenobarbital	1750 ppm	48.46±1.04	13.47±1.34	53.77±1.19	8.34±1.62
rotenon	30 ppm	43.44±3.97	28.05±0.83	50.71±5.36	14.15±2.26
malathion	0.90 ppm	59.14±0.94	5.18±1.11	55.02±2.60	0
carbaryl	36 ppm	50.41±1.29	0	51.82±1.98	0.96±0.96
DDT	90 ppm	53.46±1.02	17.34±1.50	51.83±1.86	3.67±1.68

Table 2. Estimated concentrations of the xenobiotics (in ppm) that give an egg-to-adult survival of 50% both for the selected populations and the control populations together with the ratio of these two.

	Bogota			50 x 50		
	selected population	control population	ratio select/contr.	selected population	control population	ratio select/contr.
phenobarbital	3500	950	3.7	4425	750	5.9
rotenon	25.5	12.5	2.0	56	16	3.5
malathion	2.90	0.18	16.1	2.0	0.15	13.3
carbaryl	41	10	4.1	42	10	4.2
DDT	210	17	12.4	210	9	23.2

The level of tolerance was determined by measuring the egg-to-adult survival of both selected and control flies on food supplemented with a series of different concentrations of the xenobiotic studied (selected flies were tested only on the chemical they were reared on). For the tests flies were collected from the cages and reared for one generation on normal food. Thereafter females were allowed to lay eggs on 2% agar gels for 6 h, after which the eggs were collected and cultured in vials, 100 eggs per vial. Table 1 shows the result when selected and control flies were reared at the same concentration. It is clear that the selected flies of both populations show a much higher survival than the control flies for all xenobiotics. Student-t testing of the difference in survival between selected and control flies showed that the difference was highly significant for all comparisons ($P<0.001$). This indicates a significant increase in tolerance in all selected populations. As the egg-to-adult survival was measured on different concentrations of the xenobiotics, it was possible to estimate the concentrations at which 50% survival would be expected. These concentrations are shown in Table 2 for both selected and control flies. Furthermore the relative increase in tolerance in the selected populations (represented by the ration of these two) is shown in this Table. It is clear that the two populations have reacted in more or less the same way; the relative increase for each xenobiotic shows the same order of magnitude for both. On the other hand there are marked differences between the compounds.

Phenobarbital, rotenon and carbaryl show an increase in tolerance of 2-5 times, whereas the increase in tolerance for malathion and DDT is much greater, 15-20x. Both malathion and DDT treated flies seem to have become resistant to concentrations much higher than they have experienced during the experiment (highest concentrations used, respectively, 0.65 and 65 ppm). Especially the difference in tolerance increase between carbaryl ($\sim$4 x) and malathion ($\sim$15 x) is remarkable, as it is well known that both insecticides bring about their toxic effect in the same way by inhibiting acetyl-choline esterase activity in insects. The reason why some xenobiotics cause a much higher increase in tolerance is not clear at the moment. One possibility is that malathion and DDT have caused higher selection pressures in the cages and therefore might have effected a different tolerance mechanism. Investigation of the genetic bases of the increase in tolerance and the possible existence of cross-resistance is needed to elucidate this question.

References: Bijlsma, R. 1980, Biochem. Genet. 18:699-715; Bijlsma, R. & W. van Delden 1977, Genet. Res. 30:221-236.

Alcorta-Azcue, E., E. Garcia-Vazquez and F. Sanchez-Refusta. University of Oviedo, Spain. Influence of the altitude in Asturian communities of Drosophilidae.

The Drosophila genus is a material usually used for the genetic research, because flies are easily captured and reproduced in laboratory; their fecundity is high (Fowler 1973) and their vital cycle is short (Ashburner & Thompson 1978). Drosophila has 15 subgenus (Wheeler 1983); in our work we study two of them: Dorsilopha (one species, **D.buskii**), and Drosophila (49 species reported in Europe).

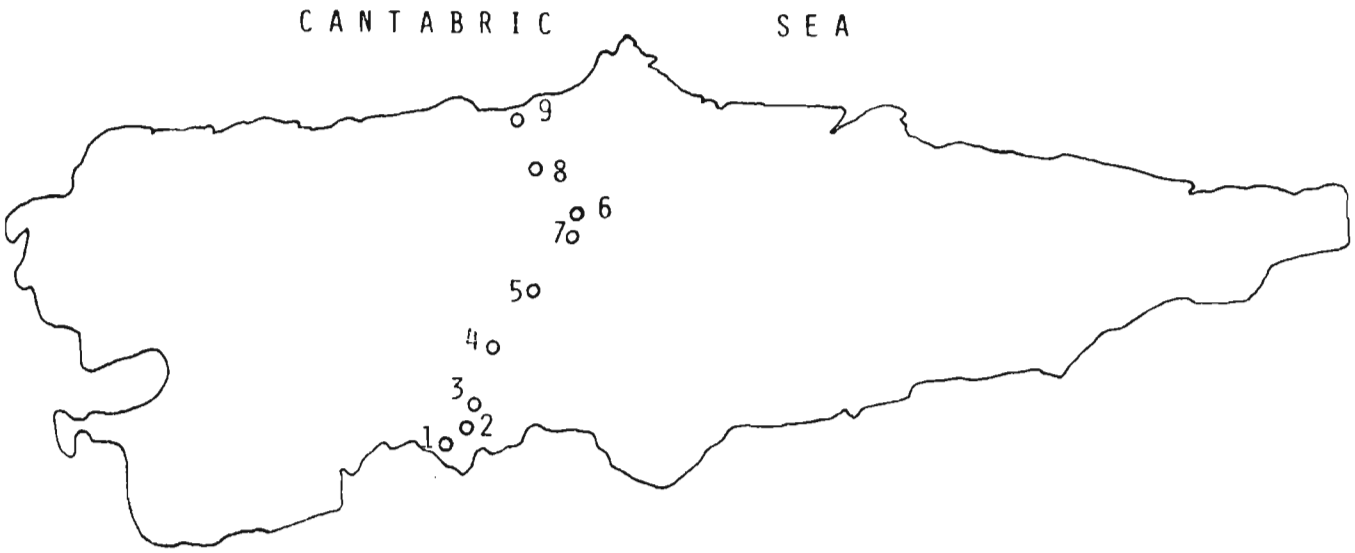
We sample Drosophilidae along the river valley Paramo-Nalon, in Asturias (North of Spain). This valley goes along 60 km. from 1300 m. of altitude to sea level, in S-N direction. His climate has a gradient from cold maritime in the high area to temperate maritime in the coast belt (Felicisimo & Alvarez 1980). This is why this valley is adequate for studying altitude influence over the Drosophilidae communities.,

So, we can bring some new data about Drosophila species ecology and distribution.

Capture sites are in the map of Figure 1, as well as the ecosystem type of each one. We show in Table 1 the ecologic and climatic parameters that we have measured for each capture site. Thermopluviometric data are obtained in the nearest meteorologic stations. These data are missing for those sites where pluviosity and temperature records couldn't be taken.

Flies were captured during the last week of August. Eight traps with banana and yeast have been placed in nature for three days. Begg & Hogben (1946) and Parsons & Stanley (1981) have described this method as effective for capturing the species of Drosphila in the wild, though banana is not their natural nutritive source. Data are shown reuniting both sexes.

We have found 10 species of the Drosophila genus (Table 2). **D.simulans** and **D.melanogaster** are constant in all collection stations. **D.obscura, D.buskii, D.immigrans** and **D.funebris** are less constant, while the other four species are rare.



Sites	Ecosystem	Sites	Ecosystem
1. Puerto Ventana	Beech Forest	6. Trubia nc.	Orchard
2. Cabanas	Meadow	7. Trubia c.	Poplar Forest
3. Fresnedo	Chestnut Forest	8. Sandiche	Orchard
4. Entrago	Meadow	9. Soto del Barco	Shrubbery
5. Proaza	Apple Forest		

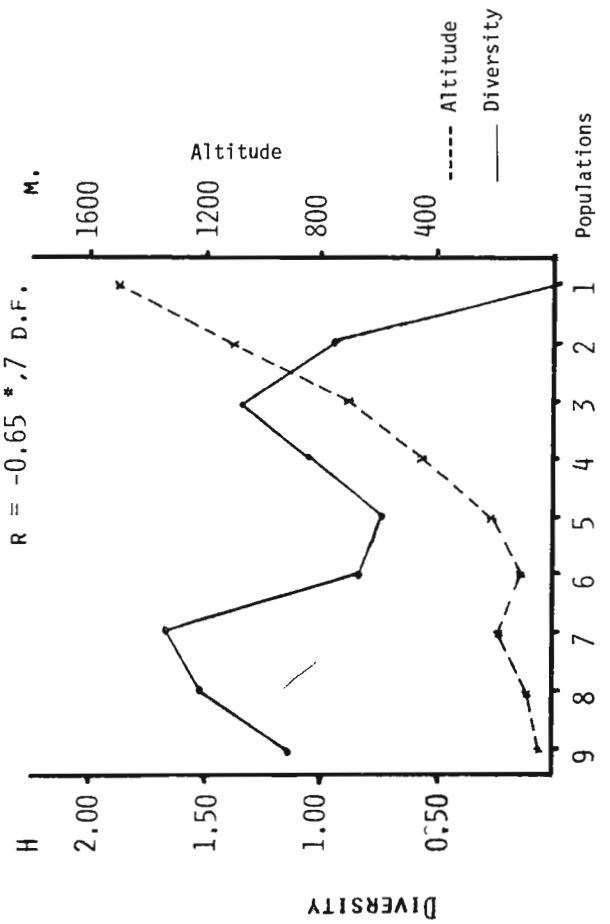
Figure 1. Capture sites in Asturias, along the Paramo-Nalon valley.

Table 1. Parameters determined in each site of capture.

Sites	Altitude	Temperature averages	Pluviosity	Orientation	Dominant species	Total no. of individuals	Ratio: **D.sim./D.mel.**
Puerto Ventana	1500 m.	13.3°	9.0 mm	E (90°)	**D.obscura**	7	--
Cabanas	1090 m.	13.3°	8.2 mm	NW (330°)	**D.simulans**	9	7.04
Fresnedo	690 m.	18.0°	--	SE (120°)	**D.simulans**	95	16.13
Entrago	450 m.	--	35.0 mm	N (0°)	**D.simulans**	68	4.25
Proaza	210 m.	20.2°	27.2 mm	SE (110°)	**D.simulans**	877	8.06
Trubia nc.	180 m.	19.2°	32.4 mm	E (50°)	**D.simulans**	478	4.08
Trubia c.	90 m.	19.2°	32.4 mm	N (0°)	**D.simulans**	942	3.42
Sandiche	60 m.	19.6°	29.8 mm	Mid-valley	**D.melanogaster**	640	0.67
Soto del Barco	10 m.	18.0°	32.0 mm	NW (320°)	**D.melanogaster**	149	0.33

Table 2. Number of individuals of each captured species in each site.

Sites	**D.melano-gaster**	**D.simu-lans**	**D.ob-scura**	**D.subob-scura**	**D.fune-bris**	**D.bus-kii**	**D.pha-lerata**	**D.immi-grans**	**D.tes-tacea**	**D.hydei**
Puerto Ventana	--	--	7	--	--	--	--	--	--	--
Cabanas	1	7	1	--	--	--	--	--	--	--
Fresnedo	4	64	19	--	8	--	--	--	--	--
Entrago	12	51	4	1	--	--	--	--	--	--
Proaza	94	752	10	--	9	11	--	--	--	--
Trubia nc.	72	293	28	--	--	11	3	71	--	--
Trubia c.	212	724	1	--	2	1	1	1	--	--
Sandiche	342	230	17	2	--	42	--	5	1	1
Soto del Barco	102	34	13	--	--	--	--	--	--	--



Drosophila abundance, measured as the total number of captured individuals, is the first index of the habitat resources for this genus (Table 1). Altitude is a limitant factor, though at 700 m. we captured a number of individuals not different from that of the captures at sea level (Garcia-Vazquez et al. 1985).

Diversity (Figure 2), as index of the community's variety, is measured by the Shannon formula. We find a negative correlation between diversity and altitude ($r = -0.65$, 7 df., $p<0.05$), mainly due to influence of the higher areas (above 700 m.). Climatic conditions in this area are not suitable for the Drosophila development. So, in Puerto Ventana only the resistant **D.obscura** develops. **D.melanogaster** and **D.simulans** appear in Cabanas (1090 m.), but they are very less abundant than in the lowest areas. Favourable environment in low areas enable more diverse communities development.

Altitude has a different importance for each captured species. **D.funebris** is associated to forest ecosystems, although it seems to be independent from the dominant tree species. **D.funebris** does not compete with the other species because there is not a dimunition of them when **D.funebris** is more abundant. Altitude is not a decisive factor for this species.

D.buskii and **D.immigrans** appear respectively from Proaza and Trubia nc. to low areas; altitude

Figure 2. Diversity and altitude variation along the valley. Correlation between these parameters.

seems to be a limitant factor (stronger in **D.immigrans** than for **D.buskii**), as we cannot associate their appearance with other ecological factor.

D.simulans is the most abundant species in this sampling. It is dominant in most of the capture sites, though **D.melanogaster** coexists with it. **D.melanogaster** and **D.simulans** are sibling species, which compart the same habitat (Parsons 1975); they have an effective sexual isolation (Mourad & Mallah 1960). Nevertheless we find a different development for both species according to altitude. **D.melanogaster** has a negative correlation between altitude and abundance (measured as the number of captured individuals, see Table 2): $r = -0.67$, 6 df., $p<0.05$); **D.simulans** seems to be the most resistant to adverse ambiental conditions, and it accepts a greater ambiental variation. it is established in more extensive areas than **D.melanogaster**, and easily becomes the dominant species.

In high areas, temperature has much day-night variation; furthermore, the minimum temperature is less than in low areas. The fact that **D.simulans** have been favoured in that environment is contrary to findings of other authors. Parsons (1975) found that **D.melanogaster** is favoured against **D.simulans** when temperature variation is great; Cuesta & Comendador (1982) proved that **D.simulans** is less resistant to low temperatures than **D.melanogaster.**

Also, a seasonal substitution of **D.melanogaster** by **D.simulans** is cited: **D.melanogaster** is dominant in spring, whereas **D.simulans** is dominant in autumn (McKenzie & Parsons 1974). We think that this replacement is more rapid in high areas, under unfavourable ambiental conditions.

References: Ashburner, M. & J.N. Thompson Jr 1978, in: Genetics and Biology of Drosophila, Ashburner & Wright (eds.), v.2a:1-109, Academic Pr. London; Begg, M. & L. Hogben 1946, Proc. Roy. Soc. Lond. Ser. B 133:1-19; Cuesta, L. & M.A. Comendador 1982, Medio Ambiente 6(1):33-42; Felicisimo, A.M. & M.A. Alvarez 1980, in: Avances sobre la Investigacion en Bioclimatologia, VII Simposio de Bioclimatologia, Sevilla (Spain); Fowler, G.L. 1973, Adv. Genet. 17:293-360; Garcia-Vazquez, E., F. Sanchez-Refusta & E. Alcorta-Azcue 1985, Rev. Biol. Univ. Oviedo 3:31-4; McKenzie, J.A. & P.A. Parsons 1974, Aust. J. Zool. 22:175-187; Mourad, A.M. & G.S. Mallah 1960, Evolution 14:166-170; Parson, P.A. 1975, Quart. Rev. Biol. 50:151-169; _____ 1982, Evol. Biol. 14:297-350; Parson, P.A. & S.M. Stanley 1981, in: Genetics and Biology of Drosophila, Ashburner, Carson & Thompson (eds.), v.3a:349-393, Academic Pr. London; Wheeler, M.R. 1983, in: Genetics and Biology of Drosophila, Ashburner, Carson & Thompson (eds.), v. 3a:1-97, Academic Pr. London.

Alexandrov, I.D. Research Institute of Medical Radiology, Obninsk, USSR. Modification of radiation-induced rates of intra- and inter-genic changes at the black locus of **Drosophila melanogaster** by the genotype, caffeine, actinomycin-D, sodium fluoride and radiation quality.

In the same experiments described earlier (Alexandrov 1985), two hundred and forty-seven different black mutations (region 34D of 2L chromosomes) were recovered simultaneously with the yellow ones among the F_1 progeny resulting from the mating of the irradiated males to $In(1)sc^{S1}L-sc^{8R}$ + dl-49, y sc^{S1} sc^8 w^a; b cn vg females. Each of the black mutants was at first mated 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, when all was said and done, the black homozygotes. According to data of the genetical analysis, all the black mutations, as with the yellow ones, have been classified into 3 main and regularly occurring mutant types: (1) sterile F_1 visibles (SV) (80 out of 247 black mutations scored), (2) transmissible visibles with recessive lethality (LV) (76 out of 167 fertile F_1 mutants), and (3) transmissible and viable in homozygote visibles (VV) (all the other fertile mutants).

A further analysis of LV showed that one third of them (25 out of 76) had a dominant sterility and a low viability (LV^S) (i.e., the F_2 heterozygotes b^x/b Pm or b^x/In(2LR)Cy, when inter-se crossed, yielded no progeny at all), but the rest of the LV were fertile ones (LV^F) (51 out of 76). Some of the latter, when b^xBl heterozygotes constructed were inter-se crossed, had a recessive lethality inseparable from the black phenotype (true LV^F being deficiencies, inversions, etc., according to data of the genetical [Alexandrov 1984] and cytogenetical [Alexandrov & Alexandrova, this issue: New Mutants] analysis), whereas the others had an independent recessive lethal separated from the black mutation by crossing over in the b^x/Bl female heterozygotes (so-called "twin" black mutants). Polytene chromosome analysis of LV^F and the 68 VV preserved was also carried out, and the number of VV associated (VV^{ch}) as well as unassociated (VV^g) with chromosome rearrangements was detected (Alexandrov & Alexandrova, ibid.).

A knowledge of a genetical nature of the black mutations scored has made it possible now to estimate the relative proportion of intra-locus changes (VV^g) versus all inter-genic (i.e., chromosome SV, LV^S, LV^F and VV^{ch}) alterations affecting the chromosome region of interest after action of the variable studied (Table 1) (LV^F are given as so far non-tested for the presence of independent recessive lethals). Corrected a.m.f. for VV^g (i.e., taking account of the "twin" black LV^F, among which gamma-ray-induced "twin" mutants are found to arise nearly twice as frequently as the neutron-induced ones) and for the chromosome rearrangements as a whole are listed in Table 2.

TABLE 1.

Conditions of experiment§	Number of mutations: v^g	SV	LV^S	LV^F	v^{ch}: Micro deletions*	v^{ch}: Visible rearrangements	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								
	9	12	7	11	2			41
/a.m.f./=	/1.2/			/4.1/				/5.3/
2. gamma-rays, 40 Gy, D-18, No. = 156127								
	19	14	3	5				41
/a.m.f./=	/3.0/			/3.5/				/6.5/
3. caffeine (0.2%) + gamma-rays, 40 Gy, D-32, No. = 91729								
	18	4	1	2	1			26
/a.m.f./=	/4.9/			/2.2/				/7.1/
4. actinomycin-D (100 µg/ml) + gamma-rays, 40 Gy, D-32, No. = 69046								
	8	9	6	5	1			29
/a.m.f/=	/2.9/			/7.6/				/10.5/
5. sodium fluoride (0.2%) + gamma-rays, 40 Gy, D-32, No. = 30861								
	8	6		3				17
/a.m.f./=	/6.5/			/7.3/				/13.8/
6. 0.1, 0.35 and 0.85 MeV fission neutrons, 8-10 Gy, D-32, No. = 79839								
	2	10	5	13	1			31
/a.m.f./=	/2.5/			/36.3/				/38.8/
7. ^{252}Cf, 14 Gy, D-32, No. = 24072								
	2	2	1	1	1	In		8
/a.m.f./=	/5.9/			/17.8/				/23.7/
8. 0.85 MeV fission neutrons, 10 Gy + gamma-rays, 10 Gy, D-32, No. = 13377								
	1	4		1	3			9
/a.m.f./=	/3.7/			/29.9/				/33.6/
9. X- or gamma-rays, 40 Gy, c(3)G, No. = 143305								
	10	16	2	8				38
/a.m.f./=	/1.7/			/4.5/				/6.6/
10. 0.35 and 0.85 MeV fission neutrons, c(3)G, 5 Gy, No. = 20605								
	1	2						3
/a.m.f./=	/9.7			/19.4/				/29.1/
....., ..., 10 Gy, No. = 10357								
	0	1		2		T(2;3)		4
/a.m.f./=	/0.0/			/38.6/				/38.6/

* Sterile in homozygote black mutants without visible rearrangements;
** a.m.f. = average mutation frequency, locus/r x 10^{-8}.

As seen, v^g (corrected a.m.f.) arise nearly as frequently as chromosome rearrangements in the wild-type (D-32, D-18) and c(3)G male germ cells (the post-meiotic stages as a whole) after gamma-irradiation. On the other hand, fission neutrons and ^{252}Cf are more efficient than gamma-rays in producing both the chromosome and the gene black mutations in different genotypes although this effect is more profound for the first than for the latter. Therefore, the relative proportion of intra- versus intergenic changes induced by high-LET radiations at the locus in question turn out to be 1:4 and 1:2 for the wild-type and c(3)G male germ cells, respectively. It is of interest that the consecutive irradiation by neutrons and gamma-rays increases the yield of the chromosome black mutations as well as v^g. This finding for the black v^g is analogous to that for v^g at the yellow locus (Alexandrov 1985). However, unlike the latter, pre-treatment of the D-32 males with caffeine or actinomycin-D (other things being equal) enhances the occurring of v^g whereas the actinomycin-D, but not the caffeine, increases the yield of the black chromosome mutations (cf. the yellow ones, Alexandrov 1985).

The modifying effect of sodium fluoride (the males were fed on 0.2% NaF in 10% sucrose for 24 hr prior to irradiation) is marked for both gene and chromosome black mutations (Table 2), and this picture is found to be exactly opposite to that for the sex-linked loci. In particular, among 15522 X chromosomes studied in the same experiments, not one yellow mutation and merely 9 white mutants (among which only one was v^g) were scored. Therefore, sodium fluoride significantly increases the yield of gamma-ray-induced autosomal, but not sex-linked, visibles mutations, at least under our experimental conditions. To answer the question, whether the action of sodium fluoride is a chromosome-

Table 2. Corrected (i.e., with accounting of the "twin" black mutants*) a.m.f.** for the gene (VV^g) and chromosome black mutations scored in different experiments listed in Table 1.

No. of exp.	Total LV^F scored	No. of "twin" LV^F	Corrected a.m.f. for VV^g ***	Chromosome rearr.***
1.	11	8	2.6 (17)	2.7 (24)
2.	5	non-tested	3.0 (19)	3.5 (22)
3.	2	0	4.9 (18)	2.2 (8)
4.	5	3	4.0 (11)	6.5 (18)
5.	3	1	7.3 (9)	6.5 (8)
6.	13	4	7.5 (6)	31.3 (25)
7.	1	0	5.9 (2)	17.8 (6)
8.	3	2	11.2 (3)	22.4 (6)
9.	8	4	2.4 (14)	19.4 (20
10.	2	0	9.7 (1)	19.4 (2)

* See text; ** See Table 1; *** Figures in () show number of mutations recovered.

or locus-specific, the effect of the NaF upon the radiomutability of the other autosomal loci needs to be studied. Such research with both cinnabar and vestigial mutations scored simultaneously with the black ones is in progress now.

When comparing data secured for both the black (this Note) and the yellow (Alexandrov 1985) loci, one must point out that, in the picture of the radiomutability of the two loci in question after action of the variables studied, there are very marked features in common (e.g., the same proportion of intra- versus inter-genic alterations after gamma-irradiation predominate the inter- over intra-geneic changes among neutron-induced mutations, etc.) as well as obvious distinctions (e.g., the different effects of the modifiers studied upon the yield of the yellow and black VV^g) which appear to be conditioned by unique features of the fine structure of the loci themselves and/or of the neighbouring chromatin.

References: Alexandrov, I.D. 1984, DIS 60:45-47; _____ 1985, DIS 61:25-26.

Alexandrova, M.V. Research Institute of Medical Radiology, Obninsk, USSR.

Genetic and cytogenetic mapping of the chromosome rearrangement breakpoints induced by high- and low-LET radiations around the black locus of **D.melanogaster.**

The first results of the cytological analysis of lethal black mutations induced by gamma-rays in c(3)G male sperms or spermatids had revealed that the location of chromosome rearrangement breakpoints around the black locus, which have been mapped by Woodruff & Ashburner (1979) in 34D6-8 region of 2L chromosome, appear to be clearly non-random. To study the "hot" points supposed in region of interest, more precisely the breakpoints of 31 radiation-induced chromosome black mutations preserved, were determined by a cytological mapping on the polytene chromosomes as well as by the genetic testing against nub, j, rk and pu.

According to the data obtained (Table 1) in 34D1.2-D4 division, lying just to the distal of b, the breakpoints of rearrangements induced by both low- and high-LET radiations were recovered at a disproportionately high frequency relative to ones mapping in the other division of the 34A-34D1.2 region studied, namely 27 independent hits in the first versus 2, 1, 1 and 2 hits in the latter, respectively (Figure 1). In contrast, the distribution of the breakpoints in the proximal of b region 34D8-35E seems to be more or less at random, although in 34D8-E1.2 division direct adjoining to b the frequency of the breakpoints is higher than in the others as well.

As a whole, 35 (55%) out of 64 radiation-induced breakpoints recovered in 34A-35E region of 2L chromosome were mapped in 34d3-8 subregion. They apparently surround the locus black situated there. Since a high frequency of chromosome breakpoints is one of the main features of intercalary heterochromatin in **D.melanogaster** (Hannah 1951), our data make it possible to assume that the 34D3-8 region appears to be also intercalary heterochromatin, one in which the black locus is apparently embedded. It seems to be the particular position of the locus in question that brings about both the predominance of deficiencies among the radiation-induced rearrangements in the region of interest (Table 1) and the high frequency of point mutations at the b locus after action of low- or high-LET radiation (Alexandrov, this issue).

The data obtained allow a decision to be also made concerning the more exact location of the j, rk and pu on the chromosome map than it was as yet known. Indeed, the twin j rk phenotype found to have all the j rk/Df(2L) heterozygotes lacking the bands 34E4.5, but not 34E1.2 (e.g., cf. Df(2L)b79b4 and Df(2L)b801). Therefore, the both loci in question are closely linked and located in the bands (or interbands) 34E4-5. Results of the marker analysis demonstrate too that the pu locus is placed more far to the right of rk (probably, in the bands 35B2-4, Figure 1) than it was previously mapped with respect to rk and lethal

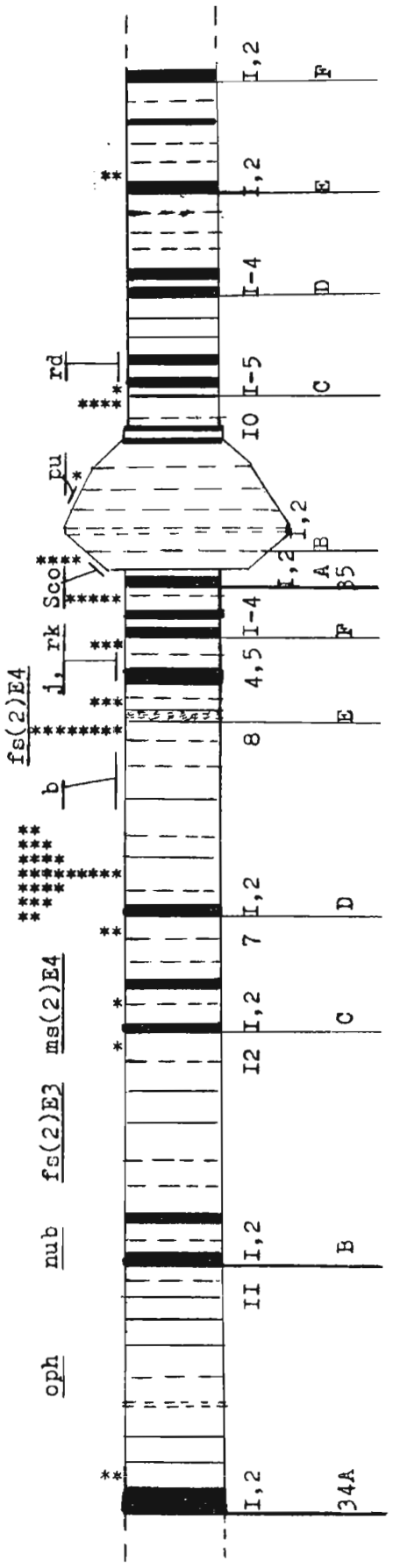


Figure 1. Results of cytogenetic and genetic mapping of the radiation-induced chromosome breakpoints (*) in the 34A - 35E region of 2L chromosome of **D.melanogaster.** One asterisk corresponds to one breakpoint. Genetic and chromosome maps as described by Lindsley & Grell (1968).

Table 1. Cytology and genetics of the radiation-induced chromosome rearrangements recovered as the lethal black mutations.

Rearrangements	Cytology	Genetics
Tp(2)b71k1	34D2-4; 34D8-E1.2; 43C2-4	nub^+- b^- - j^+ - rk^+- pu^+
Df(2L)b74c6	34D2-4; 34D8-E1.2	nub^+- b^- - j^- - rk^+- pu^+
Df(2L)b77c	34D2-4; 34F4-35A1.2	nub^+- b^- - j^- - rk^-- pu^+
Df(2L)b78j	34D2-4; 35A3-4	nub^+- b^- - j^- - rk^-- pu^+
Df(2L)b79a3	34D2-4; 34D8-E1.2	nub^+- b^- - j^+ - rk^+- pu^+
Df(2L)b79b3	34C7-D1.2; 35A4	b^- - j^- - rk^-- pu^+
Df(2L)b79b4	34D2-4; 34E6-F1.2	nub^+- b^- - j^- - rk^-- pu^+
Df(2L)b79b8	34D2-4; 34F4-35A1.2	nub^+- b^- - j^- - rk^-- pu^+
In(2L)b79d5	$34D4^{\pm}$; $35B10^{\pm}$	nub^+- b^- - j^+ - rk^+- pu^+
T(2,3)b79d6	34A2-3; 34D8-E2; 79B; 80C	b^- - j^+ - rk^+- pu^+
Tp(2)b79h1	34D2-4; 34D8-E2; 41	nub^+- b^- - j^+ - rk^+- pu^+
Df(2L)b80k	34D2-4; 35B10-C1	nub^+- b^- - j^- - rk^-- pu^-
Df(2L)b801	34D2-4; 34E2-E4.5	nub^+- b^- - j^+ - rk^+- pu^+
Tp(2)b81a	34D2-4; 34D8-E2; 41D1-E1	nub^+- b^- - j^+ - rk^+- pu^+
Df(2L)b81f1	34D2-4; 35A3-4	nub^+- b^- - j^- - rk^-- pu^+
Ef(2L)b81f2	34D2-4; 35B4-6	nub^+- b^- - j^- - rk^-- pu^-
*In(2L)b81f3	34D2-4; $35B10^{\pm}$	nub^+- b^- - j^+ - rk^+- pu^+
In(2L)b8117	34D2-4; 40F	nub^+- b^- - j^+ - rk^+- pu^+
*In(2L)b82c44	34D2-4; 40F	nub^+- b^- - j^+ - rk^+- pu^+
In(2L)b83b22	34D2-4; $35B10^{\pm}$	nub^- - b^- - j^+ - rk^+- pu^+
Df(2L)b83d29a	34D2-4; 35E2-E6	nub^+- b^- - j^- - rk^-- pu^-
Df(2L)b8311	34D2-4; 34E2-E5	nub^+- b^- - j^+ - rk^+- pu^+
Df(2L) + T(2;3)b8312	Df(2L)34C2; 34E5.6 + T(2;3)34B12-C1; 35A2-3; 83A7-8; 83C	b^- - j^- - rk^-- pu^+
Df(2L)b84h14	34D2-4; 34F4-35A1.2	nub^+- b^- - j^- - rk^-- pu^+
Df(2L)b84h50	34D2-4; 35C1-C3	nub^+- b^- - j^- - rk^-- pu^-
Df(2L)b85b1	34D2-4; 34E2-E4.5	nub^+- b^- - j^+ - rk^+- pu^+
Df(2L)b85b2	34D2-4; 34D8-E1.2	nub^+- b^- - j^+ - rk^+- pu^+
Df(2L)b85c1	34D2-4; 34F4-35A1.2	nub^+- b^* - j^- - rk^-- pu^+
*T(2,3)b85c2	34C7-D1.2; 34E1.2; 95C4-D1	b^- - j^+ - rk^+- pu^+
Df(2L)b85f1	34D2-4; 34F3-4	nub^+- b^- - j^- - rk^-- pu^+
Df + In(2L)b85f2	Df(2L)34D2-4; 34E6-F1 + In(2L)33A1-2; 35E3-4	b^- - j^- - rk^-- pu^+

* Viable in homozygote.

mutations in the region of interest (Woodruff & Ashburner 1979). Indeed, the pu phenotype shown to have first the pu/Df(2L)b81f2 heterozygote (lacking the bands up to 35B4), but not the pu/Df(2L)b81f1 one (lacking the bands up to 35A3). All the other and larger deficiencies, when they are heterozygous for pu, have the pu phenotype too (Table 1).

Acknowledgement: I am grateful to Dr. R.C. Woodruff, Bowling Green, Ohio, for supplying the nub, j, rk and pu stocks.

References: Hannah, A. 1951, Adv. Genet. 4:87-125; Lindsley, D.L. & E.H. Grell 1968, Carn. Inst. Wash. Publ. 627; Woodruff, R.C. & M. Ashburner 1979, Genetics 92:133-149.

Al-Taweel, A.A. Iraqi A.E.C., Agriculture and Biology Faculty, Baghdad, Iraq. The frequency of spontaneous and induced recessive lethals in the sex chromosome of the **Drosophila melanogaster** wild type from Iraq.

The frequency of spontaneous sex-linked recessive lethals which occurred in nature and the induced gamma-radiation were determined by the Muller-5 technique in two Iraqian populations of **Drosophila melanogaster.** The first population was collected from Mosul, 396 km north of Baghdad (lat. 36; long. 43), and the second population was collected from Basrah, 545 km south of Baghdad (lat. 29.5; long. 47). Collections were made during June-August 1980. Immediately upon collection the flies were maintained in culture at 25°C in the laboratory in half-pint bottles with 50 ml food medium. The ingredients of the food medium in 1000 ml of water were agar (20 gm), date syrup (100 gm), Wheat (100 gm), yeast (50 gm), propionic acid (5 ml) and nepagin (15 ml) and were designated as M and B, respectively.

After several generations the frequency of spontaneous and induced SLRL-Mutations by 0.5 kGy of gamma radiation were determined by mating each male to virgin female of Muller-5 [(Sc B In-S W Sc) (Muller-5 originated from Genetics Dept., Aberdeen University)]. The F1 females were individually mated to Muller-5 male and the criterion of scoring the F1 as lethal was the complete absence of wild type males and the presence of at least 10-15 Muller-5 males.

Table 1 showed the frequency of spontaneous and induced SLRL-mutations by gamma-radiation in natural populations of **D.melanogaster** in comparison with that of the Oregon-K and from these results we can draw the following conclusions:

Table 1. Frequency of spontaneous and induced SLRL-mutations by gamma-rays in natural **D.melanogaster** populations collected from different localities in Iraq.

Population	CONTROL No. Chromosomes tested	%lethal	0.5 kGy No. Chromosomes tested	%lethal
M	1443 (10)**	0.69	405 (20)**	4.93
B	1035 (8)	0.77	468 (19)	4.06
Oregon-K*	2737 (13)	0.47	1547 (112)	7.23

* Oregon-K sent to us from Genetics Dept., Aberdeen U.
** numbers in () represent lethal chromosomes recorded.

1. No significant differences were observed between the spontaneous or induced SLRL-mutations by gamma-radiation in both Iraqian population (Chi-square test based on the observed frequencies were 0.0529 & 0.3935, respectively).

2. Also, no significant differences were noticed if the spontaneous SLRL-mutations were compared with that of the Oregon-K (M. vs. Oregon K X^2=0.8205; B vs. Oregon-K X^2=1.2069).

3. Just not significant and a significant difference were observed if the induced SLRL-mutations were compared with that of the Oregon-K (M. vs. Oregon-K X^2=2.6982; B vs. Oregon-K X^2=5.981).

Finally the Iraqian populations have shown a resistance to induce SLRL-mutations in comparison with Oregon-K because they have lived for a long time under natural environmental conditions and recently adapted to the laboratory conditions while the Oregon-K has been adapted to the laboratory conditions for several years.

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Influences on courtship and copulation of space and photoperiod during the first week of the male imago-life.

The sexual behavior of drosophila has been described by many authors, Ewing (1983) being one of the most recent. They emphasize the stereotyped components of male courtship giving rise to the choice of the female between males of different species. In spite of this cliche there is an individual variability which is not only a genetic effect.

Several authors have shown that the courtship behavior of drosophila is modified by the social experience of the male. For example, Mainardi (1967) has demonstrated that the male choice between two kinds of females (savage Oregon or yellow) depends on its individual experience. "Males reared in isolation perform their courtship at random" and court the Oregon female when reared in groups. This indicates that social experience during the beginning of the male imago life may modify its courtship.

More recently, Siegel & Hall (1979) found that a male having courted an unreceptive female presents an experience-dependent modification designated as "conditioned courtship" which lowers the possibility of copulation with a new female. This demonstrates that social experience immediately preceding courting may also modify the male's courtship.

The present paper shows that the physical-environmental factors (under which the male is bred during the first week of the imaginal life) do influence its sexual behavior.

Table 1. Analysis of variance of data.

(A) Precopulatory duration:

Source	D.F.	Mean square	F value
Space	1	19.85	6.4 *
Photoperiod	1	37.50	12.1 ***
Interaction	1	4.81	1.5
Residual	198		

(B) Copulatory duration

Space	1	.25	3.6
Photoperiod	1	.33	4.8 *
Interaction	1	.59	8.5 **
Residual	113		

* P<0.05; ** P<0.01; *** P<0.001

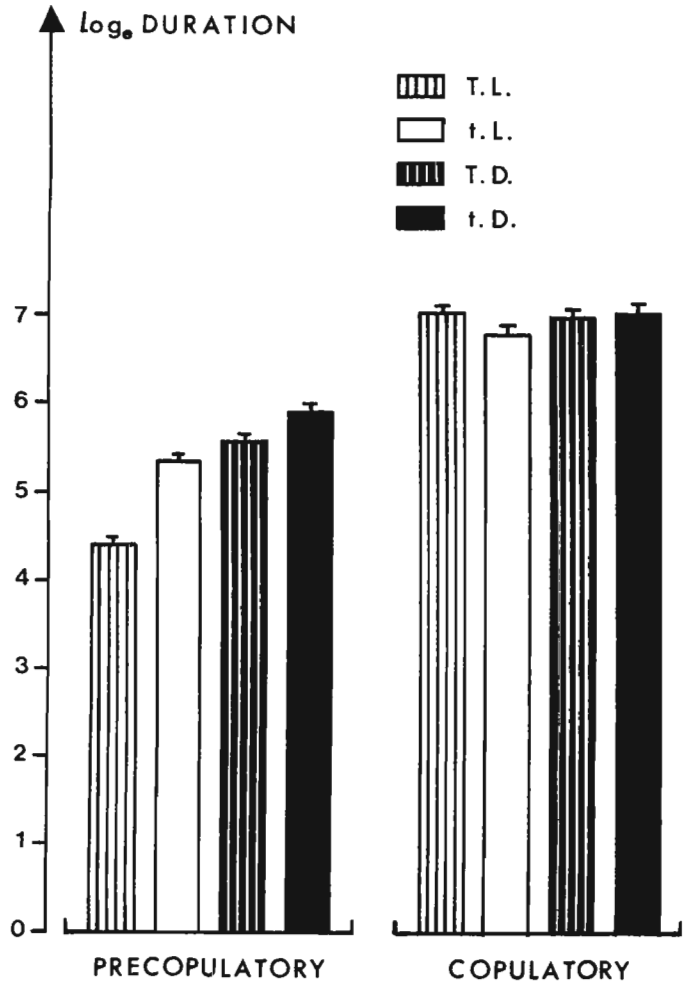

Figure 1. Precopulation and copulatory durations. Mean of Log_e duration.

Males from an outbred strain of **Drosophila melanogaster** (Brazzaville) are stored, just after hatching, individually in a test-tube and fed with a synthetic chemically defined medium (Marenco 1983). Four kinds of test-tubes are used: (1) glass test-tube about 75 cm^3 (T.L), (2) darkened glass test-tube about 75 cm^3 (T.D), (3) transparent plastic test-tube about 5 cm^3 (t.L), and (4) dark plastic test-tube about 5 cm^3 (t.D).

Females are stored in groups of ten in a 75 cm^3 glass test-tube with the same nutritive medium. For all the flies, this nutritive medium was changed every three days (under red light for T.D and t.D). Breeding and experiments are carried out under controlled conditions of humidity (80%), temperature (25°C), and for T.L and t.L photoperiod (L.D: 12/12, 100 lux from 8 a.m. to 8 p.m.). Courtship occurs in an observation-cell: a 20 mm high cylinder with an internal diameter of 25 mm lit by a 500 lux white light. Without anesthesia, male and female are individually placed in small transparent test-tubes. These two tubes are put into two opposite funnels driving the flies to the obsevation-cell. One to four minutes later, a sliding stick which separates the funnel from the cylinder, is withdrawn and the female is able to get into the observation-cell. Then the stick is pushed back and the same procedure is applied to the male. Observation begins when the male enters the cell and for 30 minutes we note the occurrence of all courtship elements. We only give below results concerning the precopulatory duration (from the time the male enters to copulation) and the copulatory duration.

A two-ways analysis of variance was made with a logarithmic transformation (Log_e) of these time periods (owing to an asymetry in the distribution of raw scores). Table 1(A) presents this analysis; both dimensions show a significant effect, without interaction. As it can be seen in Figure 1, the fastest couples are those whose males were bred in a large test-tube (T.L), under a photoperiod. The precopulatory time-periods are considerably longer for the couples whose male was of t.L type, longer still for the couples whose male was T.D and the longest for couples whose male was bred in a small dark test tube (t.D).

The same statistical treatment was performed for the duration of copulation. The two-way analysis of variance (on the logarithmic transformation of the duration, Table 1(B)) shows the significant effect of the absence of photoperiod during the first week of the imago life. This effect interacts with the breeding space where the male is confined (5 cm^3 or 75 cm^3).

Such results are not only reducible to the consequences of a strong lightening occurring after a week of darkness for T.D and t.D groups. Such males are not only slow in action for their courtship, but also

in copulation--even if they mate in less than 30 minutes--we think that the male-adaptation to the light is over before copulation, nevertheless the behavior of the couple is influenced by this lightening deficiency. The influence of space, even though less important than that of light, modifies courtship: couples whose males are bred in small tubes have a longer precopulatory period.

These preliminary results show that imaginal breeding conditions (individual experience) must influence the sexual behavior of **Drosophila melanogaster.**

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Band, H.T. Michigan State University, East Lansing, Michigan. Emergence of **D.simulans** and other Drosophila from a variety of fruit/nut substrates.

D.simulans has become a colonizing species. Neither McCoy (1962) nor Sabath (1974) included it among the drosophilids collected in their studies in Indiana. However, it has been consistently collected in Michigan along with other Drosophila in the 1980's (Band & Band 1983; Band et al. 1984). Its emergence has now been documented from an array of substrates in Fall 1985, along with other drosophila. This is shown in Table 1.

Table 1. Emergence of Drosophilids from a variety of substrates in Fall 1985. D.m.=**D.melanogaster**; D.s.=**D.simulans**; D.b.=**D.busckii**; D.i.=**D.immigrans**; C.a.=**Chymomyza amoena**; o.c.= ornamental crabapples.

			females	males				
site	date	substrate	D.m. + D.s.	D.m.	D.s.	D.b.	D.i.	C.a.
A	Oct.	apples	8	2	7		3	1
A	Oct.	walnuts				4	3	2
B	Oct.	o.c.	21	4	3			1
B	Sept.	pears	270	194	1		4	

Site A is the farm west of Lansing, site B a residential area of East Lansing. Both have been used in the past for studies on **C.amoena** overwintering in apples, walnut husks and ornamental crabapples (Band & Band 1982, 1984). On 27 Oct. 1985, apples and walnuts were collected at site A. Ten of each were inspected and those with drosophilid larvae and pupae held to verify their presence. Earlier 12 pears (on a tree) and 60 ornamental crabapples (on the ground) at site B had been collected. Four pears and 15 ornamental crabapples with drosophilid eggs and larvae were retained.

Emergence of **D.simulans** from ornamental crabapples at site B has been reported previously (Band & Band 1983). Carson (1965) found **D.busckii** breeding in walnut husks. However, **D.immigrans** has not been reported emerging from the walnut substrate.

The presence of **D.melanogaster, D.simulans, D.busckii** and **D.immigrans** at site A in 1985 represents an invasion since the site was last employed in **C.amoena** cold hardiness work. Apple and walnut trees are adjacent; fruits lie mixed on the ground. Selective substrate utilization at site A is evident. **D.melanogaster** and **D.simulans** occur in the rotting fruits; **D.busckii** in the walnut husks; **D.immigrans** and **C.amoena** in both substrates at site A.

The fact that **D.busckii** emerged from green fallen unripe apples in the mid-south (Band 1985) but from walnut husks from the site A farm is in agreement with the observations that this species does not ordinarily use fermenting fruits (Carson 1965; Atkinson & Shorrocks 1977). Emergence of **D.immigrans** from apples and walnuts agrees with previous findings that it is a generalist species (Carson, loc cit; Atkinson & Shorrocks, loc cit). Peak emergence at the site A farm had probably already occurred by the 27 Oct. collecting date. Numbers of **D.immigrans** and **D.busckii** emerging are lower than the numbers of pupae; many were used to determine cold hardiness characteristics. The incidence of **D.immigrans** is too low at site B to say whether or not this species is able to use ornamental crabapples for breeding; however, its emergence from this substrate has not been noted previously (Band & Band 1983).

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Band, H.T. Michigan State University, East Lansing, Michigan. Evidence that Drosophila oviposit on ripe and rotting fruits on trees.

McCoy (1962) presented data that **D.melanogaster** in tomato fields in Indiana oviposit only on damaged fruits less than 8 inches off the ground. He concluded from his study that Drosophila used only damaged fruits at or near ground level.

Cavener and Clegg (1981) reported that **D.melanogaster** in apple orchards in Georgia were ovipositing on fruits on trees. Band et al. (1984) used **D.melanogaster** emerging from overripe pears pulled from a tree in 1982 in their study of the existence of LSP-1β^S polymorphisms in mid-Michigan **D.melanogaster.**

Data were accumulated in Sept. 1985 to establish that at least 4 species oviposit on ripe-to-rotting fruits on trees. All pears come from the same tree as in 1982. The umbrella shape of the canopy and moderate height insure accessible fruits. Twenty-five **D.melanogaster** (and **simulans**) flew out of the first pear plucked; the single file emergence of the escapees from the hole at the calyx made them easy to count. Nine pears gathered on 9/22/84 and three on 9/29/85 were dissected. Four of the first group and one of the second, containing Drosophila eggs or larvae, were placed in individual jars and capped with a sturdy grade tissue. Potting soil was added as a substrate when 2 pears became watery and many larvae drowned. Table 1 shows the emergence data to 10/14 when all cultures were discarded because of mites.

Table 1. Emergence of Drosophila from frassy and rotting pears on a tree. Fruits taken from various heights but mostly within 5-8 feet from ground level. D.m.=**D.melanogaster**; D.s.=**D.simulans**; D.i.=**D.immigrans.**

collecting		females	males		
date	pear #	D.m. ± D.s.	D.m.	D.s.	D.i.
9/22/85	1	81	76	1	
9/22/85	2	74	55		
9/22/85	3	94	43		
9/29/85	1	21	20	_	4
Total		270	194	1	4

In each collection one pear contained only dead larvae. A pH of 3 was recorded. All pears were frassy or had begun to rot when gathered and one had 46 Drosophila eggs in frass along a tunnel from the center to the calyx. Two also had **Chymomyza amoena** eggs and lesser apple worm eggs (**Graptolitha prunivora**) but cultures were discarded before the longer developing species could emerge. These pears did not contain codling moth larvae (**Cydia pomonella**) although two others did (but had no evidence of drosophilid invasion).

Although urea/uric acid have been found to have a negative effect on **D.melanogaster** development (Botella et al. 1985), the Drosophila species ovipositing on and their emergence from parasitized fruits in Michigan parallels findings of Drosophila breeding in fallen frassy unripe apples in the mid-South (Band 1985). The total difference between the laboratory larvae in medium, yeast and temperature and natural substrates, diversity of microflora and fluctuating temperatures may minimize the effects of the presence of excreta, both that of the initial occupant and of co-occurring individuals.

In any event, whatever the source of the attraction to ripe and overripe fruits on trees, **D.melanogaster** females do not oviposit only on fruits at or close to ground level. Neither do other Drosophila in Michigan.

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Band, H.T. Michigan State University, East Lansing, Michigan. Occurrence of three chymomyzid species at Mt. Lake Biological Station in Virginia.

Chymomyza amoena and at least two additional undescribed species of chymomyzids exist at Mt. Lake Biological Station. On the station grounds all three could be observed displaying on freshly cut oak (Quercus sp.) wood in the early morning (7-8:30 a.m.) and early evening (7-8:30 p.m.). The first unidentified species was observed in mid-July. Five flies were trapped 7/18/85; three still survived on apples 7/22/85 but only one male was successfully mailed to the University of Chicago. A female, which escaped, had a shovel-like ovipositor. By the time trapping and media availability for mailing a series of captured flies could be coordinated, the first species had been replaced by a second unknown species. Seven males were successfully mailed to the University of Chicago on 8/16/85. Trapping methods were as previously employed for MI adult **C.amoena** and consist of placing an empty receptacle over displaying flies, causing them to fly upward into the trap.

Table 1. Observations on 3 chymomyzid species, July - August 1985, at MLBS in Virginia. Two with unbanded wings plus **C.amoena**; m=males; f=females; p=progeny.

date	time	# unbanded	#**C.amoena**	trapped	remarks
7/18	p.m.	7	0	5 (4m,1f)	unident. sp #1
7/19	a.m.	7	2	1 C.a.m.	x MIf; p
7/22	a.m.	15	2		
7/29	p.m.	20	2		
8/6	p.m.	9	0		
8/12-14	p.m.	at least 7	0	7m	unident. sp #2

Chymomyza amoena co-occurred with at least one unknown chymomyzid species and was the only species with banded wings. Numbers of all three species were low although **C.amoena** was consistently less frequent than adults of either of the other two species. This is shown in Table 1. Wheeler (1952) lists chymomyzids as being found around peeled areas of tree trunks: aspen, alder, fir, pine. Attraction to Oak is not mentioned. However, **C.amoena** has a lesser tendency to be associated with wood than other species (Wheeler, ibid). Watabe (1985) has also reported the morning and evening display times of chymomzid males at timberyards in Japan. Oaks were among the cut logs.

All chymomyzids observed at MLBS were aggressive. All were wing-wavers, a typical chymomyzid characteristic that has been absent in some species in New Guinea (Okada 1981). Males attempted to mate indiscriminately between species. All could be kept alive on ripe apples although there was no record of emergence of either of the undescribed species from apples collected at 3 sites outside the station grounds in studies on **C.amoena** oviposition behavior; no unidentified chymomyzid larvae or unemerged pupae were later found in apples brought back to Michigan State University to suggest apples might be feeding/overwintering sites for larvae of the unknown species.

Only two adult **C.amoena** were observed at one of the three sites where apples were collected although a total of 95 **C.amoena** emerged from those found to contain **C.amoena** eggs. Eighty-four adults emerged from apples on the ground and 11 from apples pulled from trees. Pupation was mostly within apples; only 23 pupal cases were recovered from soil substrates supplied for individual records per apples with **C.amoena** eggs.

A single **C.amoena** male successfully trapped was mated with newly emerged Michigan **C.amoena** (from apples) on 7/19/85. Eggs were observed by 7/30/85, hatching by 8/3/85. Emergence to oviposition in 10 days and oviposition to hatching in 4 days is consistent with unpublished studies on interpopulation crosses in Michigan from apple substrates and interstate crosses between Michigan and Missouri **C.amoena.**

The total of 12 males, representing 3 species, and 1 female trapped on separate occasions agrees with Japanese findings that males predominate in collections. Differences in courtship displays were not noted but see Watabe (1985). Numbers of all species are lower than for chymomyzid collections at the Jyozankei timberyard (Watabe 1985). However, total population sizes at any one time were also small.

Acknowledgements: Thanks are gratefully extended to Lynn Thockmorton for establishing the existence of two undescribed chymomyzid species in the Virginia mountains, to Bruce Wallace at VPI&SU and the Drosophila group at the University of Virginia for supplying media and mailing cartons on different occasions, and to Jerry Wolff, Director of MLBS, for supplying space for research.

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Band, H.T., A.M. Gonzalez*, and R.N. Band.
Michigan State University, East Lansing; *Universidad de la Laguna, Tenerife, Canary Islands, Spain. Comparison of the two numbering systems for the larval hemolymph proteins of **Drosophila melanogaster.**

Two different numbering systems have been applied to the larval hemolymph proteins of **D.melanogaster.** Gonzalez et al. (1982) used the numbering system of Loukas et al. (1974) for D.subobscura (see also Cabrera et al. 1983). Singh et al. (1982) and Singh & Coulthart (1982) applied the numbering system of Doane & Treat-Clemons (1981); they surmised that proteins 16, 15, 11, 10, and 9 were SGS-3, LSP-2, LSP-1α, LSP-1β, and LSP- 1 of Akam et al. (1978) and Roberts & Roberts (1979). Band et al. (1984) used 2-dimensional electrophoresis to partition LSP-1 at pH 7 into the 3 component proteins of Roberts & Roberts (1979) in order to study the existence of LSP-$1\beta^S$ in northern U.S. populations; it verified that the numbering system of Doane & Treat-Clemons (1981, 1982) was the appropriate one for **D.melanogaster.** There was no way to relate the two numbering systems to one another.

To correlate the two numbering systems, stocks of **D.melanogaster** from Tenerife, Canary Islands, Spain and Cordoba, Spain, were mailed to East Lansing. Hemolymph proteins from larvae (and pupae) were electrophoresed along with OR-R controls as described by Band et al. (1984) and then compared to a

Table 1. Comparison of the numbering systems that have been applied to the larval hemolymph proteins of **Drosophila melanogaster.**

larval hemolymph protein	Singh et al. (1982)	Gonzalez et al. (1982)
SGS-3	pt. 16	pt. 15
LSP-2	pt. 15	pt. 10
LSP-1 α	pt. 11	
LSP-1 β	pt. 10	pt. 8
LSP-1 γ	pt. 9	pt. 7

SGS = salivary glue secretion (salivary gland); LSP = larval serum protein (fat body).

photograph of **D.melanogaster** larvae showing proteins numbered according to the system of Loukas et al. (1974) and applied by Gonzalez et al. (1982).

It is possible to construct the following identities between the two systems. This is given in Table 1. The results thus indicate that pt. 10 and pt. 7 and 8 represent fat body proteins in an array of species: **D.simulans**, **D.mauritiana** (Gonzalez et al. 1982); **D.pseudoobscura** (Lewontin 1974; Singh & Coulthart 1982; Cabrera et al. 1983); **D.guanche, D.madeirensis, D.subobscura, D.ambigua** (Cabrera et al. 1983); **D.persimilis** (Lewontin 1974).

Of special interest is the fact that LSP-2^F, which is rare in **D.melanogaster** (Singh et al. 1982) and carries a lethal or semilethal (Hoogwerf & Roberts 1982), is the predominant polymorphism in **D.simulans** where it also may tend to lethality or semilethality as a homozygote (Band, unpubl.); it is the form of LSP-2 in **D.mauritiana** (Gonzalez et al. 1982). Other species do not seem to have fixed this allele of LSP-2. LSP-2 as pt. 10 may also be involved in intra- and interpopulation cold resistance in **D.pseudoobscura** pupae (Marinkovic et al. 1969; Jefferson et al. 1974; Coyne et al. 1983). β is on chromosome 2; LSP-2 as pt. 10 is on chromosome 3 in **D.pseudoobscura** (Lewontin 1974). This is the protein called larval hemolymph protein (LHP) by Beverley & Wilson (1982, 1984) and has been used by them to construct phylogenetic trees among the drosophilids and other dipterans.

The failure to detect all 3 LSP-1 proteins by Gonzalez et al. (1982) may be a consequence of the technique, if limited migration prevents proper separation of the α and β proteins. LSP-$1\alpha^F$ was present in at least one larva in the photograph sent to E. Lansing, so the faster migrating alleles for β may represent, respectively, α^F homozygotes and α^F heterozygotes.

However, LSP-1 over an array of drosophilids does not display 3 independently migrating proteins (Brock & Roberts 1983; Band & Band, unpubl.). Also, separate α and β proteins are difficult to discern in **D.simulans** when β is β^F. Included as an allele of β, between species comparisons do enable determination of the existence of comparable rapid migrating LSP-1 proteins across an array of species. LSP-1 proteins in the **obscura** group tend to migrate at a faster rate than in the **melanogaster** group; LSP-2 tends to migrate slower among the **obscura** group than in the **melanogaster** group (Gonzalez et al. 1982; Cabrera et al. 1983).

Thus, speciations both within and between the 2 groups have involved the larval fat body proteins and their polymorphisms. Since to date there is no evidence that drosophilids are synthesizing glycerol, other polyols or relying on sugars for cold tolerance (Krunic et al. 1980; Band & Band 1980, 1982; Kimura 1982) while the fat body is reconstituted in drosophilids that show a discrete adult diapause (Carson & Stalker 1948; Begon 1976) and fat deposition increases in at least one species, **D.subobscura**, which doesn't have an adult diapause (Begon ibid.), fat body proteins may play a role in overwintering in addition to functioning as storage proteins for development.

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Bashkirov, V.N., M.Sh. Kubaneishvili, M.E. Yalakas and N.G. Schuppe. N.I. Vavilov Institute of General Genetics, USSR Academy of Sciences, Moscow. Unstable redundancy of 18S and 28S rRNA genes in **D.melanogaster.**

Ritossa & Spiegelman (1965) have shown that the rDNA quantity was directly proportional to the number of nucleolar organisers (NO) in genome. However, Ritossa et al. (1966) revealed that **D.melanogaster** females with four NO per genome lost about half of the rRNA genes over a period of several generations. Krider & Plaut (1972) also found that in homozygous females with four NO, X chromosomes despite having two NO frequently carried mutations for the bobbed locus. The partial loss of rDNA during several generations of multinucleolar flies was also observed by Kubaneishvili et al. (1983). These data are in contradiction with those obtained by Spear (1974) who did not observe instabilities in the rDNA content over 10 generations of multinucleolar genotypes, though in one of the generations he noted rRNA gene number variation but accounted it as artifact.

One shortcoming was shared by all these experiments: in all of them the $In(1)sc^{4R}sc^{8L}$ chromosome was used for synthesis of multinucleolar genotypes, though the regions important for maintenance of rRNA gene number stability are affected in this chromosome. Therefore, we performed the synthesis of female genotypes with three NO (termed trinucleolar females) using only structurally normal X chromosomes. An additional rRNA gene dosage was introduced into genome with an extra Y chromosome. The rRNA gene number was determined by using RNA/DNA hybridization technique. The results of typical experiments are presented in Table 1. The data obtained for other trinucleolar genotypes are represented in Fig. 1. It is obvious that in all these cases there is a considerable loss of rRNA gene copies in comparison with the theoretically expected values. This loss amounts to 70-300 genes. The rRNA gene reduction in a single NO of trinucleolar genotypes is termed by us retrocompensation. The matings between individuals from the stock reared with selection of trinucleolar females and wild (with respect to NO) individuals showed that trinucleolar females transmitted fewer ribosomal cistrons to the progeny than their normal brothers with two NO. The trinucleolar females in the

Table 1. rDNA content in the 5th-6th generations of the stock reared with selection of the females y ac sc w/y ac sc $w/B^{S}w^{+}y^{+}Y$.

Genotype	rDNA (%)	rRNA gene no.	X	Y	Δ
♀♀ y ac sc w/y ac sc w	0.470	537±12	269	--	
♂♂ y ac sc $w/B^{S}w^{+}y^{+}Y$	0.426	486±13	269	217	
♀♀ y ac sc w/yac sc $w/B^{S}w^{+}y^{+}Y$	0.333	419±15			335

X - number of X chromosomal rRNA genes. $X = \frac{\text{rRNA gene number in ♀♀ XX}}{2}$. Y - number of Y chromosomal rRNA genes. Y^2 = (rRNA gene number in ♂♂ XY)-X. Δ - difference between the observed and theoretically expected (T) rRNA gene number in XXY females. T = 2X + Y.

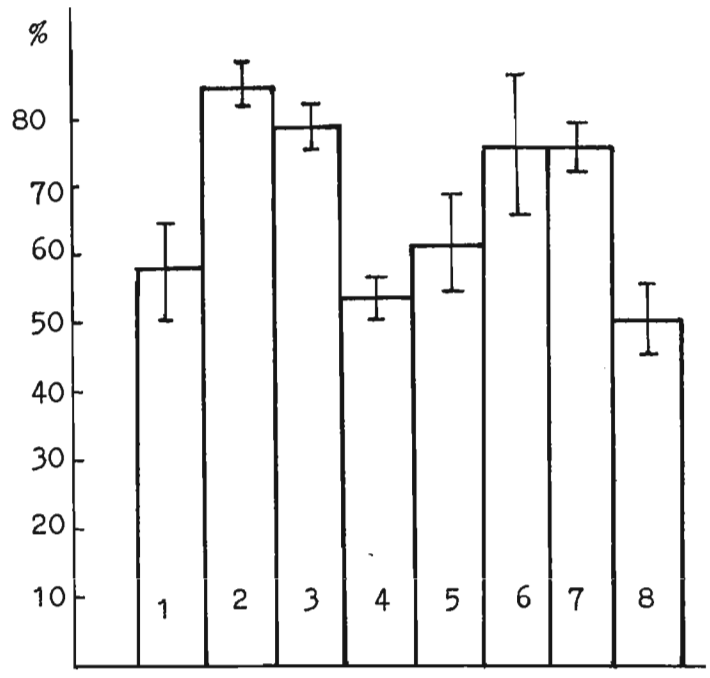


Figure 1. Relative rDNA contents in XXY females of different genetic backgrounds (the % ratio of the observed rDNA contents to theoretically expected ones). **1** - y ac sc w/y ac sc $w/B^{S}Y\ y^{+}$; **2** - y ac sc w/y ac sc $w/bw^{+}Y\ y^{+}$; **3** - Canton S/Canton $S/B^{S}Y\ y^{+}$; **4** - y ac sc w/y ac sc $w/B^{S}w^{+}y^{+}Y$; **5** - $y^{2}su(w^{a})\ w^{a}/y^{2}su(w^{a})\ w^{a}/B^{S}Y\ y^{+}$; **6** - Swedish b/Swedish $b/B^{S}Y\ y^{+}$; **7** - $In(1)sc^{8}/In(1)sc^{8}/B^{S}Y\ y^{+}$; **8** - $In(1)w^{m4}/In(1)w^{m4}/B^{S}Y\ y^{+}$.

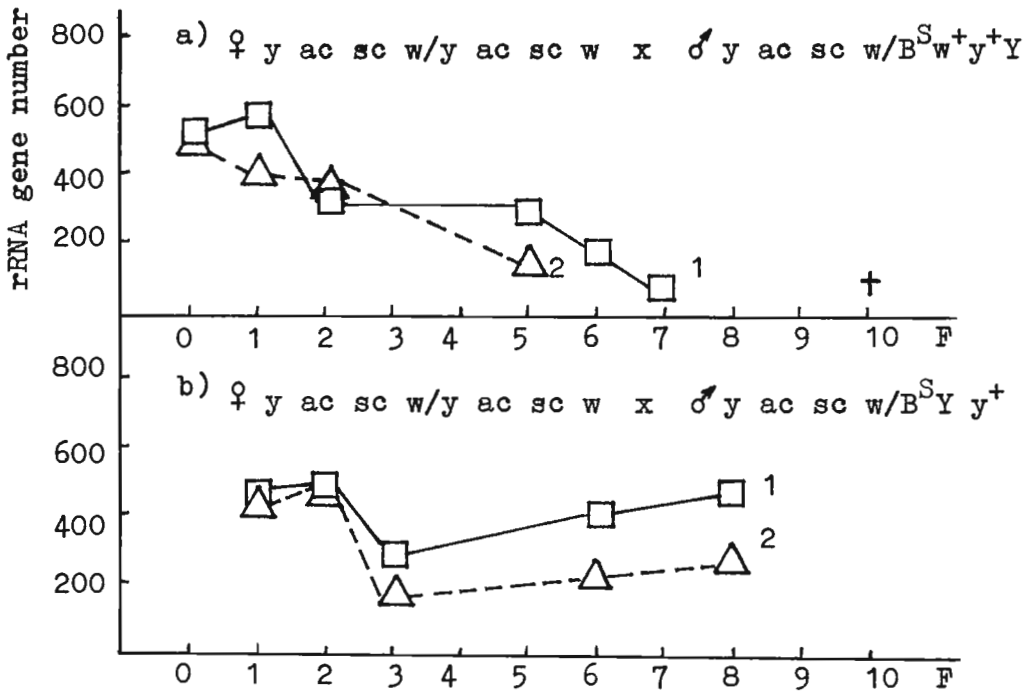


Figure 2. rRNA gene number during several generations of two-nucleolar offsprings of XXY gemales. **1** - ♀♀ ; **2** - ♂♂ ; **+** - death of stock; **F** - number of generation.

cross: ♀ y ac sc w/y ac sc w/B^{S}w^{+}y^{+}Y x ♂ y/Y transmitted with the X chromosome 313 ± 11 genes to the F_1 progeny. The F_1 progeny from the control cross ♀ y/y x ♂ y ac sc w/B^{S}w^{+}y^{+}Y inherited with the y ac sc w chromosome 429 ± 9 rRNA genes.

The results clearly show that the retrocompensation is at least partially reversible and the rDNA quantity in NO is increased during 1-2 generations after the introduction of a chromosome from trinucleolar genome into normal genotypic conditions. However, there is no stabilization of the rDNA content in the successive generations of the XXY female progeny with two NO. This is seen from the data presented in Fig. 2, where the results of rRNA gene number determinations performed during several generations in two stocks established from the two-nucleolar progeny of XXY females are shown.

The observed modulations of the rDNA content in these cases are probably provoked by the fact that during restoration of the rRNA gene number in the "retrocompensating" chromosomes the rDNA quantity rose above the normal level. This, in turn, reactivated the regulatory genes provoking retrocompensation. The system controlling the number of rRNA genes appears to be reorganized not always in time. Thus, the number of rRNA genes sometimes fails to be corrected in a necessary direction even when this can result in lethality. For example, see Fig. 2a where it is shown that the decrease in the rDNA content starting in the early generations did not stop leading to the death of stock in the 10th generation.

According to our results, retrocompensation does not occur in the first generation of trinucleolar females. The F_1 females with additional NO demonstrate additivity of contributions of all three NO into the total rDNA content. Hence, retrocompensation is a stepwise process and continues throughout several generations. Thus, in continuously maintained genotypes with an additional NO, retrocompensation occurs over a period of several generations which results in an essential DNA loss, probably in each of three NO. rDNA retrocompensation is in a certain sense opposed to magnification.

References: Krider, H.M. & W. Plaut 1972, J. Cell Sci. 11:689; Kubaneishvili, M.Sh., N.V. Vorobyova, V.T. Kakpakov & N.G. Schuppe 1983, Molec. Gen. Genetics 190:331; Ritossa, F.M., K.C. Atwood, D.L. Lindsley & S. Spiegelman 1966, Natl. Cancer Inst. Monogr. 23:449; Ritossa, F.M. & S. Spiegelman 1965, PNAS USA 53:737; Spear, B.B. 1974, Chromosoma 48:159.

Bélo, M., M.J.D. Cunha, M.E. Hunch and S.M. Cardoso. UNESP, Jaboticabal, Brazil.
Crosses with two **D.ananassae** strains.

Two **D.ananassae** strains (Ana-2 and Ana-5) were started with three females collected in the same place at Olimpia city (SP) Brazil, and were kept up isolated in a corn-wheat flour medium during 2435 days. The data in Table 1 show the values obtained for the descendant of the crosses with the two strains, F values of the analysis of variance and $w_{0.05}$ (Tukey's test) for comparison among the means. Those means followed by the same letter are statistically equal; if not, they are different.

Table 1. Values of the number of flies (NFL), biomass (BI), viability (VI) and developmental time (DT) for flies obtained from crosses with the two **D.ananassae** strains.

	Type of Crosses					
Female Male	Ana-2 x Ana-2	Ana-5 x Ana-5	Ana-2 x Ana-5	Ana-5 x Ana-2	Values of F	Values of $w_{0.05}$
NFL	245.17±24.70c	115.67±13.42b	40.75±12.29a	65.75±14.20ab	29.01**	63.72
BI	209.01±19.96c	116.12±13.18b	36.64±10.53a	58.85±12.61a	28.22**	55.01
VI	0.95± 0.01	0.93± 0.01	0.81± 0.06	0.87± 0.05	2.41^{ns}	--
DT	16.15± 0.23b	15.87± 0.19b	14.68± 0.28a	14.95± 0.22ab	4.21*	1.31

ns = not significant (P>0.05); * = significant (P<0.05); ** = significant (P<0.01).

The viability (VI) was measured by the formula: number of flies (NFL) divided by the sum of the number of flies, number of larvae and number of pupae. For development time (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 (1/4 of liter) and the first imago counting; t' is the interval between two countings (one day) and T is the maintenance time of the paternal flies in the bottles (three days). Each cross type was mounted with 20 couples and 12 repetitions.

The results concerned with the number of flies and biomass (mg) showed that crosses between the two different strains had lower productivity than the crosses of the same strains individuals, which seems to indicate any reproductive isolation degree between the two strains, and the Ana-2 strain was superior to Ana-5 in productivity. There was no variation concerned with viability, and the developmental time of the hybrid individuals were shorter than the homozygote flies. On the other hand, the crosses were not enough to show any maternal effect of the flies.

Reference: Carvalho, S. de 1981, PhD Thesis, Universidade de São Paulo, Fac. de Med. de Ribeirão Preto.

Botella, L.M. & J.L. Mensua. Universidad de Valencia, Spain.
Arrested development in second and third larval instars of **D.melanogaster** in highly crowded cultures.

Mensua & Moya (1983) put into evidence the existence of a larval stop in the third larval instar of **D.melanogaster** when reared in crowded conditions. Further studies (Mensua et al. 1983) have shown that the larval arrest occurs at third instar in a range of densities from 21 to 83 larvae in 0.5 ml of food at 25°C. Nevertheless, Perez Tome (1980) reported that in highly crowded cultures of **D.hydei** larval size showed two peaks corresponding, respectively, to the 2nd and 3rd instar. In these conditions this author reported that only a small fraction of the total competing population progresses towards the third instar.

In order to establish a comparison between this phenomenon occurring in **D.hydei** and the larval stop reported in **D.melanogaster**, the following experiments were designed. Different sets of 5 (5 x 0.8 cm) vials supplied with 0.5 ml of Lewis medium were seeded with either 150, or 300, or 500 recently hatched larvae of an isogenic Oregon-R strain. Cultures were incubated at 25°C. At different times from the seeding day (8th, 10th, 12th, 14th and 16th days), crowding was interrupted by the overfeeding technique (Moya & Mensua 1983). The results obtained concerning larva-adult viability and mean developmental time are shown in Figure 1. As regards viability, the decreases are sigmoidal-like, though far more marked when the initial density is of 300 or 500 larvae, the reduction in 50% of the initial population being reached two days earlier in these latter densities than at the density of 150 larvae.

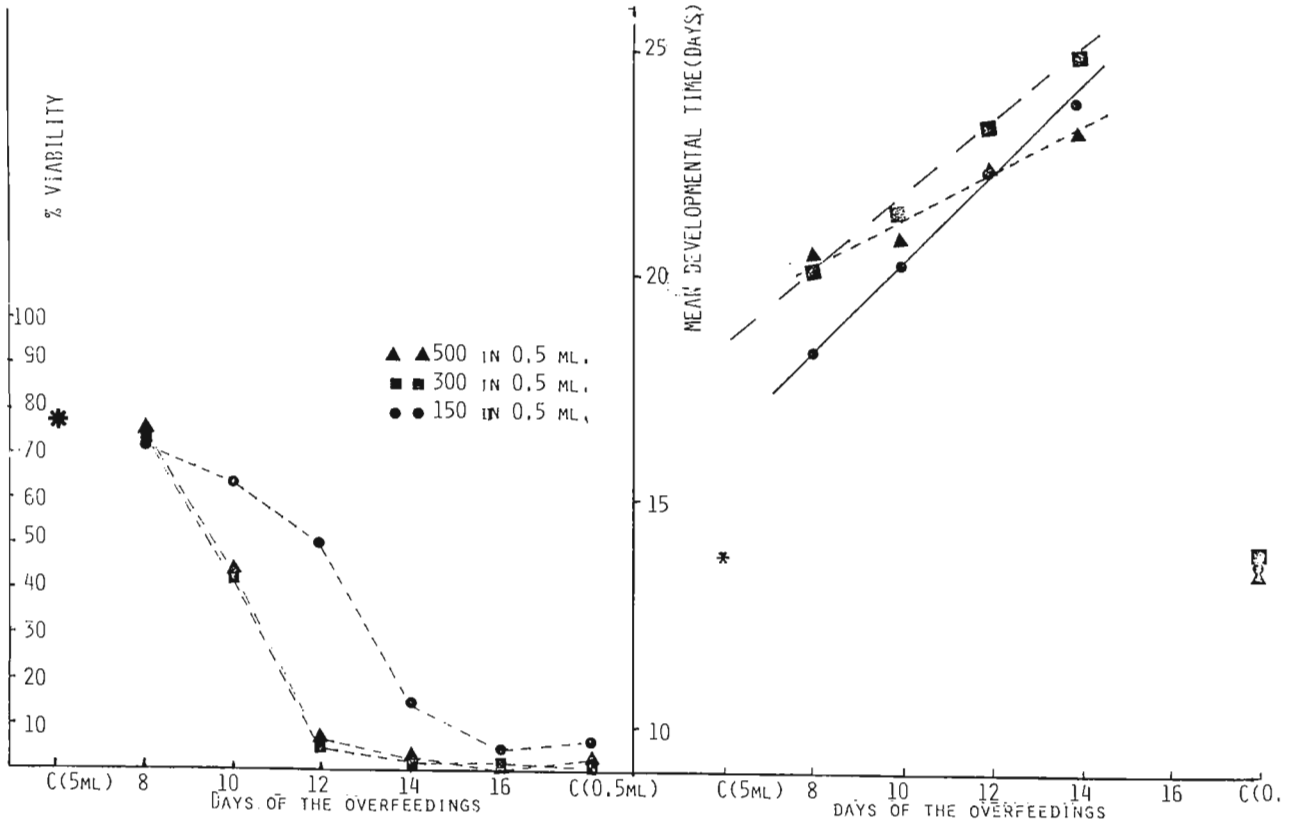


Figure 1. Larva-adult viability and mean developmental time in highly crowded cultures of **D.melanogaster.**

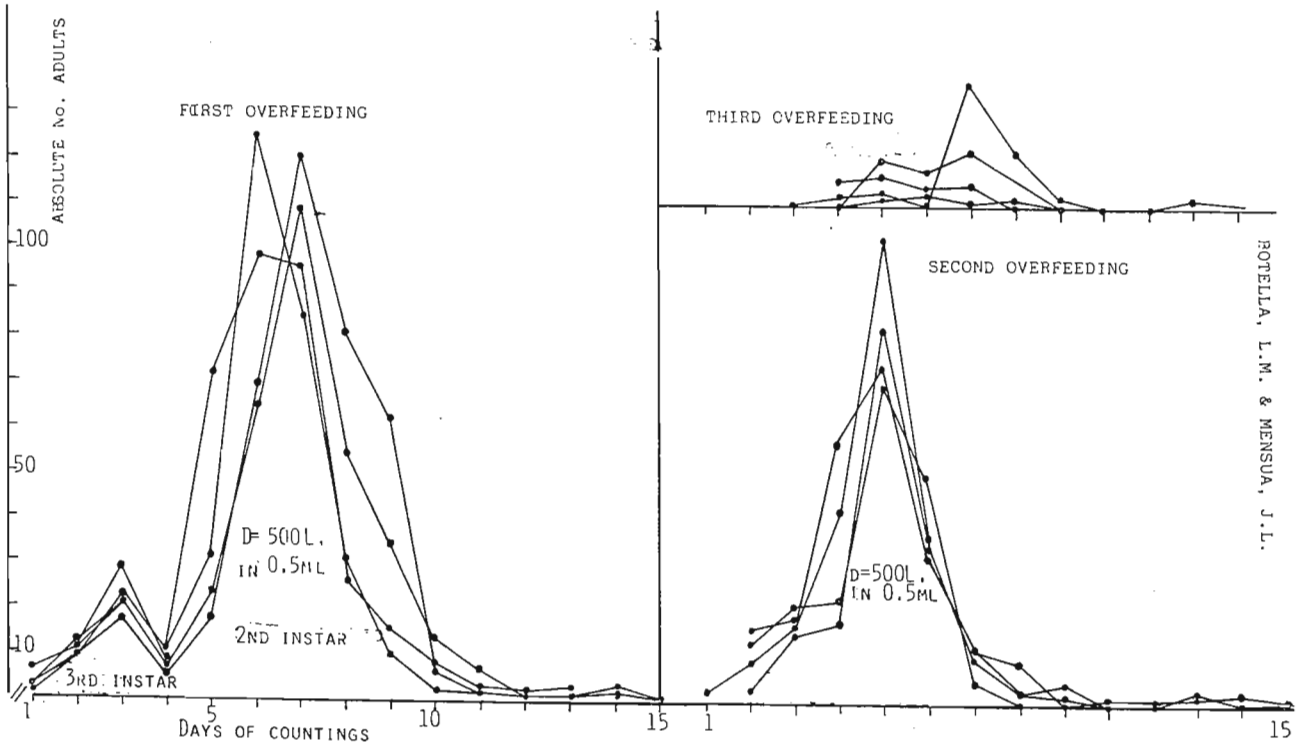


Figure 2. Absolute adult emergence in time in 3 earlier overfeedings at density of 500 larvae in 0.5 ml food.

In relation to mean developmental time, at a density of 150 larvae, the regression over the different overfeedings is similar to that found by Mensua & Moya (1983) working with 70 larvae in 0.5 ml (the slope close to 1), but the regresssion lines at higher densities (300 and 500) differ considerably.

Samples of 100 larvae were analyzed at the different overfeedings by jaw analysis under the light microscope to determine their stage of development. While at a density of 150 all larvae were stopped in third instar, it was found that at higher densities a great proportion (amounting to 60% at a density of 500 larvae) was stopped in 2nd instar. This result moved us to consider that perhaps at these high densities, the phenomenon detected in **D.hydei** was being reproduced. In an attempt to further study the development of larvae at the highest density (500 larvae in 0.5 ml), the number of adults emerged daily over the development of the cultures has been presented in Figure 2 for the three earlier overfeedings. It can be seen, above all when crowding is interrupted earlier (first overfeeding at the 8th day) that two peaks of adult emergence in time become apparent. The first peak (the sooner in emergence) is smaller and corresponds to larvae which have previously been stopped in 3rd instar, and the second and more numerous peak corresponds to larvae stopped in the second instar before the overfeeding took place. When the regression of mean developmental time was calculated for each peak over the three first overfeedings, the slope was equal to 1 for both peaks, which seems to support the existence of a larval stop in development in both instars: 2nd and 3rd.

Altogether, we may say that competition in densities within the chaos range (Hassell et al. 1976) gives rise to a double stop in larval development of **D.melanogaster**. Populations might have evolved to face highly unfavourable conditions through the acquisition of a "double brake" in their development. In the absence of this mechanism the final outcome of competition would be catastrophic, since the amount of food available in such crowded conditions is hardly enough for the development of a 2% of total population. However, the possibility of developmental arrests enables the larval population to wait for the eventual supply of new food resources.

References: Hassell, M.P., J.H. Lawton & M. May 1976, Ecology 45:471-486; Mensua, J.L., L.M. Botella & A. Moya 1983, VIIIth Europ. Drosophila Research Conf., Cambridge; Mensua, J.L. & A. Moya 1983, Heredity 51:347-352; Moya, A. & J.L. Mensua 1983, DIS 59:91-92; Perez Tome, J.M. 1980, Ph.D. Thesis Madrid.

Botella, L.M. & J.L. Mensua. Universidad de Valencia, Spain. Effect of the deficiencies in essential nutrients on the development of **D.melanogaster** stopped larvae.

The existence of a larval arrest in **D.melanogster** development (Mensua & Moya 1983) may be explained by means of two hypotheses which are not mutually exclusive. One of these hypotheses is based on the idea that an accumulation of residual metabolites, laid by the competing larvae in a limited space, would lead to a stopped development in the last larval instar. This hypothesis has been supported by the results obtained with urea and uric acid as residual compounds by Botella et al. (1985). The second possibility is that the nutritional scarcity in some essential food principle may interrupt also larval development.

This idea has been tested by the use of axenic synthetic media, either complete or deficient in each single essential food principle for Drosophila development. The complete synthetic medium employed was that proposed by Sparrow & Sang (1975). The experiments were performed as follows. Crowded cultures were established by seeding 70 recently emerged larvae from an isogenic Oregon-R strain in small vials containing 0.5 ml of a yeasted sterile medium. These cultures were reared at a temperature of 25°C, and crowding was interrupted at different days from the starting in different sets of 5 small vials by the technique of overfeeding (Moya & Mensua 1983). The media used in each overfeeding (in an amount of 10 ml in inclined disposition) were either complete or deficient in the following essential nutrients: cholesterol, choline, casein, inosine, and vitamins. A negative control medium only made out of water, agar, salts and sugar was also employed. Adults emerging every day in the overfeeding vials were recorded until the exhaustion of the cultures. All the experiments were carried out in sterile conditions.

The results obtained as regards viability in complete and deficient medium for the larvae recovered in the different overfeeding vials are shown in Figure 1. All the different media showed slightly lower viability than the complete medium, but the lowest viability was obtained in a casein deficient medium. Thus, proteins seem to be the main scarce principle in our crowded conditions. In Figure 2 mean developmental times over the different overfeedings in the different synthetic media are represented. No significant differences among the slopes of the regression lines were found. This result means that food deficiency cannot be the actual and only cause for the arrest in development. If a larva is stopped because it needs some particular nutrient requirement, it should remain in this stage for the deficient medium does not provide it. However, the adults emerged in time in the different deficient media following the same regularity as in the complete medium throughout the overfeedings. As a conclusion from this group of experiments, we can say that food scarcity may explain the decrease in viability operated in crowded

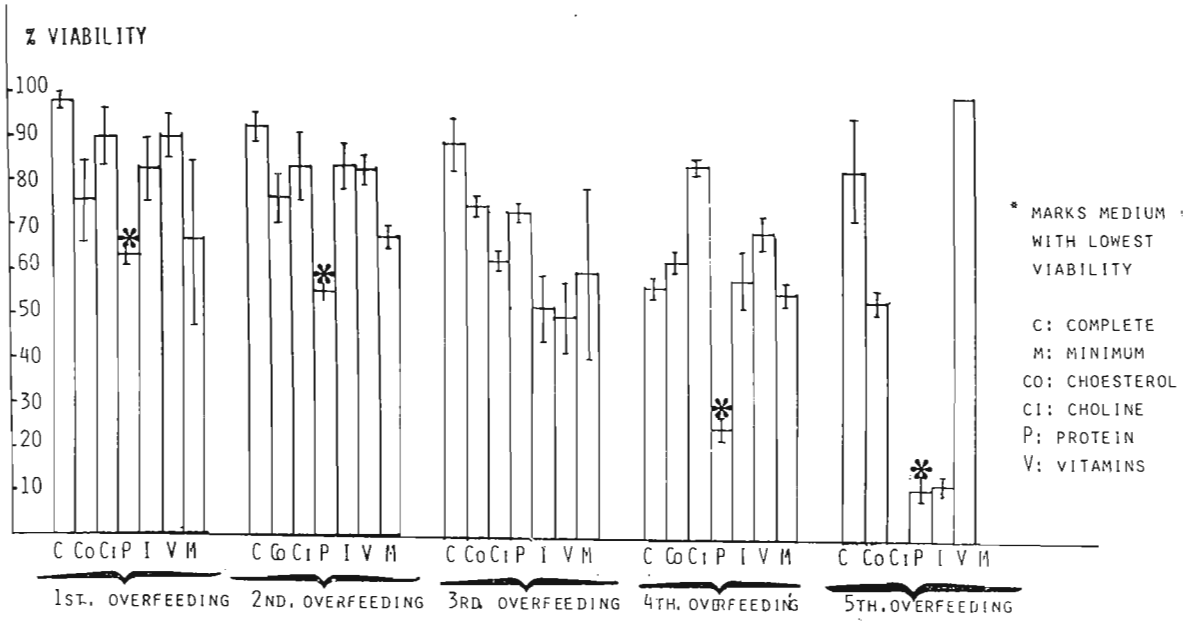


Figure 1. Larva-adult viability of the stopped larvae in different synthetic media in different overfeedings.

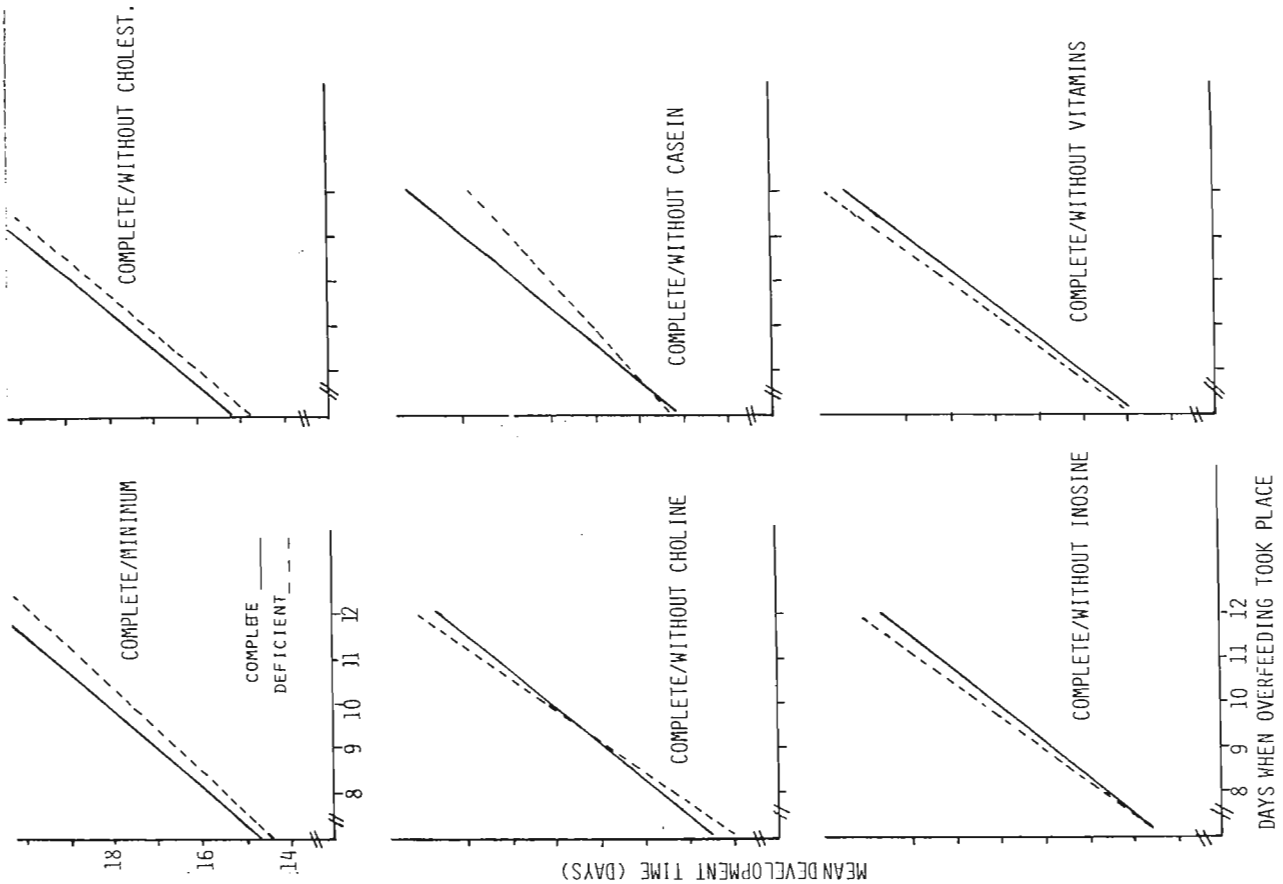


Figure 2.

conditions, but does not seem to be the direct and actual cause of the larval arrest in 3rd instar developmental stage of Drosophila.

References: Botella, L.M., A. Moya, C. Gonzalez & J.L. Mensua 1985, J. Insect Physiol. 31:179-185; Mensua, J.L. & A. Moya 1983, Heredity 51:347-352; Moya, A. & J.L. Mensua 1983, DIS 59:91-92; Sparrow, J.C. & J.H. Sang 1975, Genet. Res. Camb. 24:215-227.

Botella, L.M. and J.L. Mensua. University of Valenica, Spain. Selection for faster and slower mean developmental time in crowded cultures of **Drosophila melanogaster.**

The process of selection for development time in Drosophila has usually led authors to find an asymmetrical response with a slight or almost absent progress in the fast direction (Sang & Clayton 1957; Robertson 1963). Bakker & Nelissen (1963) explain this type of response to selection because developmental time is a selectively optimized character in those species, such as Drosophila, which depend on temporary food resources. On the other hand, it is well documented that mean developmental time may be considerably delayed by competition for food (Bakker 1961; Robertson 1963; Miller 1964; Barker & Podger 1970; Mensua & Moya 1983). Nevertheless, there are no references in literature on what occurs in a process of selection for developmental time when it is carried out under crowded conditions. We have focused our study on this point, since it is well known that many responses shown under suboptimal conditions, may be hidden in more favourable environments (Robertson 1963, 1964).

Selection for faster and slower developmental time was practised as follows. Two lines of selection were started from a stock of **Drosophila melanogaster** consisting of a mixture of three different wild stocks: one from laboratory origin (Or-R) and the other two from natural origin. In each line, a total of eight 5 x 0.8 cm vials with 0.5 ml of a boiled yeasted medium (Lewis medium) were seeded with 70 recently hatched larvae each. In the fast line, competition was interrupted at the 8th day from the seeding by means of the "overfeeding technique" (Moya & Mensua 1983). In this way the initial population was divided into two subpopulations: an "inner subpopulation" constituted by those larvae which have pupated inside the vials before the 8th day, and an "outer subpopulation" composed by those larvae recovered by overfeeding. The four earliest couples to emerge as adults from the "inner population" were selected as parents in successive generations. In the slow line, overfeeding was postponed until the 12th day. Larvae recovered by overfeeding constituted the "outer subpopulation". The four latest couples to emerge as adults in this latter subpopulation were chosen as parents of the next generation. Selection proceeded for 10 generations in the slow line and for 15 generations in the fast line. Two replications were run simultaneously for each line.

Figure 1 shows the mean developmental time obtained over the selection process in both directions. The straight lines are the best fit regressions. As can be seen, there is a slight significant trend to a decrease in mean developmental time in the course of selection in the fast direction, if one excludes the initial generation. This tendency is observed in both replications, F1 and F2. In the slow direction there is an increase during the early generations (from 0-5th in SL1, and from 0 to 4th in SL2). In the case of the SL2 line, two regressions have been represented which correspond from the 1st to the 15th generation and from the 2nd to the 15th generation, respectively. It is also worth mentioning that the drop in developmental time found between generations 5th and 6th in SL1, and between 4th and 5th generations in SL2 was due to a failure in the temperature regulation system. However, there was no significant increase in developmental time later. The results are altogether in concordance with others reported in literature by Sang & Clayton (1957) and Robertson (1963), in relation to the asymmetry and type of response obtained in uncrowded conditions. However, when selection in crowded cultures is further studied with the help of an "overfeeding technique", a new type of response becomes apparent.

Table 1. Differences between outer and inner mean developmental times in the fast and slow lines throughout the process of selection.

Generations	F1[a]	F2[b]	SL1[c]	SL2[d]
0	4.33	3.46	6.99	5.56
1	4.57	4.36	5.69	6.50
2	4.37	2.72	4.48	7.81
3	3.69	3.86	6.63	5.87
4	3.99	3.48	5.88	7.43
5	3.67	3.29	7.64	5.19
6	3.39	3.82	5.98	6.02
7	3.74	3.37	6.10	5.58
8	2.89	2.49	7.85	6.70
9	2.81	2.94	5.88	8.28
10	3.13	2.81	5.35	5.69
11	3.12	2.84		
12	2.17	3.29		
13	3.80	2.69		
14	2.99	4.10		
15	3.10	3.41		

a=Values of regression over generations: slope = -0.10, intercept at origin=4.23; p<0.001. **b**=slope= -0.03, intercept at origin=3.53; n.s. **c**=slope= 0.016, intercept at origin= 6.14; n.s. **d**=slope=0.02, intercept at origin=6.31; n.s.

Table 1 presents the differences between developmental times in outer and inner subpopulations in each generation over the process of selection in both directions. Two main points deserve attention: (i) Differences in the slow lines are larger than in the fast lines, which is not surprising since crowding was kept for a longer time in these latter (12 days in slow lines compared with 8 days in fast lines). (ii) In the fast direction, differences tend to decrease in time in a more or less regular way. This trend may be explained in terms of a response to selection pressure in both subpopulations "inner" and "outer", though more marked in this latter, where developmental rates would be further increased.

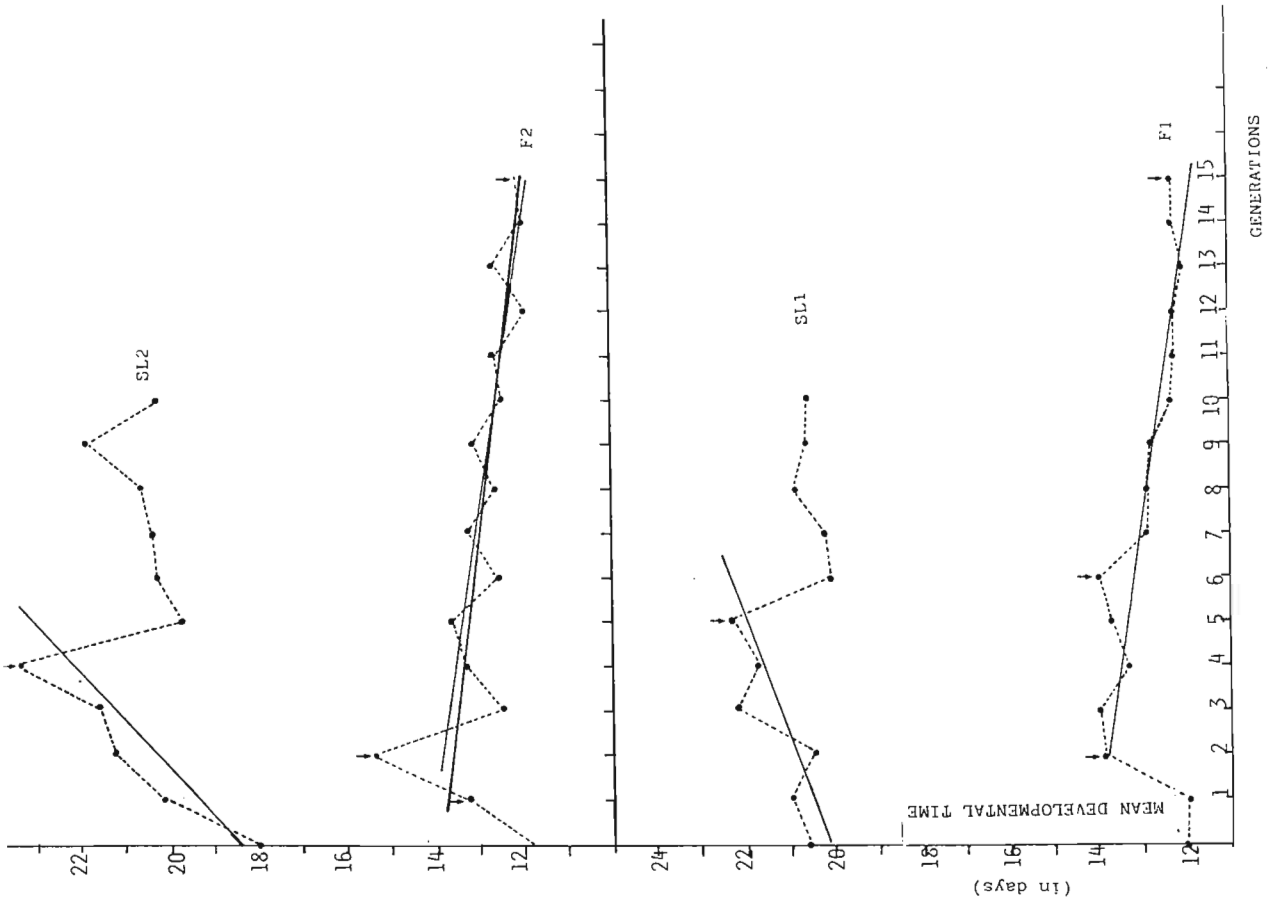


Figure 1.

On the contrary, the slow lines show a typical pattern where an increased difference is always followed by a decreased one over the process of selection. This alternating pattern between successive generations may be obtained as a consequence of the following "feed-back" or "relais" mechanism. When inner subpopulations take longer to develop, crowding is stronger, and hence the least adapted genotypes will be eliminated. The best adapted genotypes in crowding conditions (recovered by overfeeding) will develop faster in outer subpopulations. Thus, the intragenerational difference in outer and inner mean developmental times would be short. The best adapted genotypes will be selected as parents of the next generation, and will give rise to a progeny, part of which will take short to develop in crowded conditions. The remaining larvae, recovered by overfeeding (less adapted genotypes) will take longer to emerge as adults in outer subpopulation, and then, the intragenerational difference between outer and inner mean developmental times would be large.

References: Bakker, K. 1961, Arch. Neerl. Zool. 14:200; Bakker, K. & F.X. Nelissen 1963, Ent. exp. & appl. 6:37; Barker, J.S.F. & R.N. Podger 1970, Ecology 51:170; Mensua, J.L. & A. Moya 1983, Heredity 51:347; Miller, R.S. 1964, Ecology 45:132; Moya, A. & J.L. Mensua 1983, DIS 59:90; Robertson, F.W., Genet. Res. Camb. 4:74; _____ 1964, Genet. Res. Camb. 5:107; Sang, J.H. & G.A. Clayton 1957, J. of Heredity 48:265.

Capy, P. and J. Rouault. C.N.R.S., Gif-sur-Yvette, France. Sampling on decaying fruit of **Opuntia.**

In mediterranean countries, one of the most frequent breeding sites for Drosophila species is the fruit of **Opuntia ficus-indica.** Generally, fruit flies and their parasites are found in the cavity of decaying fruit. This cavity is closed to the outside except for a small hole. When fruit is touched insects fly away one by one because of the narrowness of the hole.

Using two fly aspirators, two consecutive samples have been collected from the same fruits. The first sample corresponds approximately to the first twenty seconds, and the second one to the last thirty seconds of the collection. This sampling method was carried out in November 1982 when a large number of fruits were available (Nasr'Allah, Tunisia).

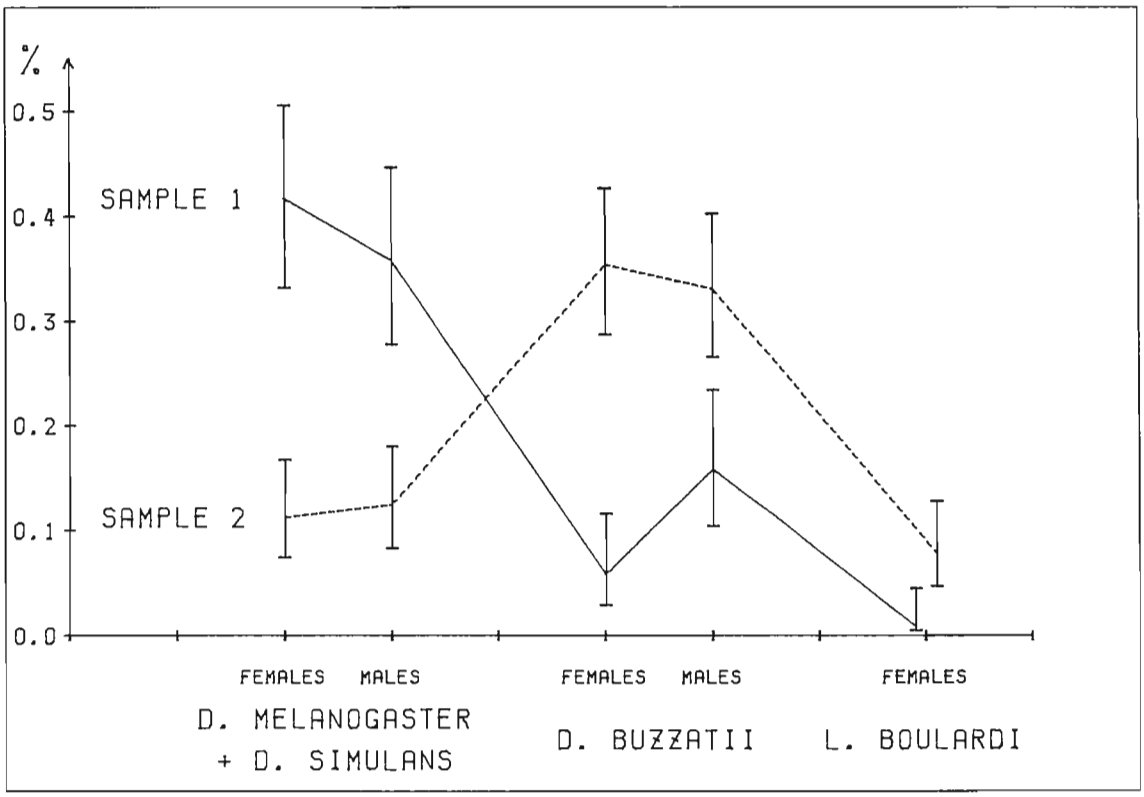


Figure 1. Ratio and confidence intervals (95%) of individuals caught in two consecutive samples.

Table 1. Number of individuals caught for each species in two consecutive samples.

	D.melanogaster					
	D.simulans		**D.buzzatii**		**L.boulardi**	Total
	0	0	0	0	0	
1st sample	50	43	7	19	1	120
2nd sample	20	22	63	59	14	178

Three Drosophila species were found: **D.melanogaster**, **D.simulans** and **D.buzzatii**; and one parasite: **Leptoplilina boulardi.** These results are similar to those previously reported by Rouault & David (1982) and David et al. (1983).

The number of individuals caught per species and per sample are given in Table 1. Owing to the determination error due to the likeness of **D.melanogaster** and **D.simulans** females, individuals of these species have been grouped.

The size of the two samples are similar. However, there is a significant difference between the two samples of Drosophila species. It appears that **D.melanogaster** and **D.simulans** fly away before **D.buzzatii.** Furthermore, within a species, both sexes have been caught in the same proportions. Finally, females of **L.boulardi** were caught almost exclusively in the second sample. All these results are presented in Figure 1. The 95% confidence intervals of the observed proportions exhibited in this figure show that intraspecific differences observed between the two samples are statistically significant for each species.

These preliminary observations lead to the conclusion that to get an accurate sample, it is necessary to collect all individuals living in the fruit cavity. All truncated samples would give a biased representation of a population.

References: David, J.R., Y. Carton, Y. Cohet, D. Lachaise, J. Louis, J. Rouault, L. Tsacas & J. Vouidibio 1983, Acta Oecologia, Oecol. Gener. 4/1:43-63; Rouault, J. & J.R. David 1982, Acta Oecologia, Oecol. Gener. 3/3:331-338.

Carracedo, M.C., E. San Miguel,* P. Casares and M.T. Alvarez.* University of Oviedo, Spain; *University of Leon, Spain. More on sexual isolation between **D.melanogaster** females and **D.simulans** males: Female receptivity and hybridization.

In a previous work about sexual isolation between **D.melanogaster** females and **D.simulans** males, we found a positive correlation between the speed at which females reached sexual maturity and the frequency of hybridization (Carracedo & Casares 1986). By following suggestions made then, we intend to prove now whether or not these two traits are related in some way to the females' level of receptivity. For this purpose we have chosen 8 isofemale lines of **D.melanogaster** which had previously shown different hybridization frequencies with **D.simulans** (Carracedo & Casares 1985), designated as M1, M2 ... M8; these lines were rechecked for hybridization with a single **simulans** line using the same method. The hybridization percentages calculated as the number of **melanogaster** females out of fifty that hybridized with **simulans** males were: 76, 42, 32, 28, 24, 18, 12 and 8%, for the M1 to M8 lines, respectively.

The maturity speed of **melanogaster** females was measured as follows. Five pairs of virgin flies from the same line, aged two hours, were kept in a vial for 30 hr. Then, females were individually placed in small vials with some food. Mating, which was inferred from the presence of larvae in the vials, was taken as evidence of female sexual maturity. The arc-sine transformed percentages of the number of fertile females out of five constituted the basic measure of the females' maturity speed. Ten replicates per line were carried out.

The level of the females' receptivity was measured by the time they needed to accept a standard **melanogaster** male. In order to make maturity speed and receptivity measurements independent factors, it was necessary to use sexually mature females. For this, we used 3-day-old virgin flies, the age at which females from the 8 lines had proven to be completely mature (Carracedo et al., submitted). The 8 lines were tested with two different lines of **melanogaster** males, the M1 and M4 lines. Females of each line were placed individually in a vial with two males from the same line. No anaesthesia was used. The basic measure was the time elapsed till the onset of copulation. Since a pilot experiment showed that over 99% of the females from these lines mated within a 15-min period, unmated females were discarded after this time. Each male x female combination was replicated 45 times giving a total of 8 x 2 x 45 females to be scored. The measurements were taken at 21.5°C between 9-11 a.m.

Table 1 shows the result of an analysis of variance for the time to copulation values in log. of secs., in which females and males were the sources of variation. Significant differences were only found between females. Because of this, the two male values were pooled, and the mean values of each **melanogaster** female line were calculated. These, after being retransformed to minutes, appear in Table 2 together with the values of the females' maturity speed.

Table 1. Analysis of variance of the female's "time to copulation" (in log of secs) for 8 lines of **D.melanogaster** females and two lines of **D.melanogaster** males.

sources of variation	df	MS	F
males	1	0.185	3.5
females	7	0.989	18.9*
males x females	7	0.070	1.3
error	704	0.052	

* P<0.001

Table 2. Sexual maturation speed, measured by percentages of number of females out of fifty that mated before reaching 30 hr of age, and level of female receptivity measured by mean values of "time to copulation" showed for single 3-day-old **D.melanogaster** females.

lines:	M1	M2	M3	M4	M5	M6	M7	M8
maturation speed:								
	90%	86%	44%	50%	56%	46%	16%	16%
receptivity level:								
	1'23''	1'08''	1'58''	1'23''	1'22''	2'02''	2'04''	2'05''

A negative correlation between maturity speed (in arc-sine percentages) and receptivity (in log of secs.) was found ($r=-0.82$; $P<0.05$) which means that the sexually mature **melanogaster** females that needed less time to mate, were those showing faster sexual maturity.

Our results not only show a close relationship between two important fitness components in Drosophila, sexual maturity and mating time, but also show connection between these homospecific traits and the hybridization frequency of **melanogaster** females and **simulans** males in the laboratory. As noted before, (Carracedo & Casares 1986), the success of such hybridization is mostly explained by the speed at which **melanogaster** females reach sexual maturity. Now, going a step further in the matter, our results suggest that hybridization is primarily determined by the level of **melanogaster** female receptivity: the greater this level, the greater the mating probability with **simulans** males. At least at the intrapopulational level, this suggestion does not support the existence of a species-specific discrimination key accounting for sexual isolation between these sibling species.

References: Carracedo, M.C. & P. Casares 1986, Genet. Sel. Evol., submitted; Casares, P. & M.C. Carracedo 1985, DIS 61:44.

Casares, P. and M.C. Carracedo. University of Oviedo, Spain. The influence of sex and duration of larval development on pupation behavior of Drosophila.

In this note we present an advance of some observations made about pupation behaviour in Drosophila. All the tests started with 75 newly hatched larvae seeded in a vial (18 x 200 mm) with 6 ml of standard baker's yeast food. Development was at 21°C and under 12:12 h L:D cycles. Two lines of **D.melanogaster** (M1 and M2) and one line of **D.simulans** (S1) were used in Test 1, with 10 replications per line. When all larvae had pupated in the vials, the pupae were classified according to their pupation height. Three groups per vial, named high, imtermediate and low pupation height, were made for **D.melanogaster**, approximately the same number of pupae in each; two groups, high and low, were made for **D.simulans**. The sex of the adults emerging from the pupae of each group was recorded, and the data, after pooling the replicates, appear in Table 1 for each line. Contingency chi-squares with 1 or 2 df, testing for sex differences between groups, were significantly different from zero. There were more males at the highest sites of the vials and more females at the lowest; that is, males pupated, on average, higher than females, for both species.

Table 1. Sex of the adults emerging from pupae located at the high, intermediate and low pupation sites in the culture vials. The chi-squares test for sex differences in pupation height.

	high pupae		intermediate pupae		low pupae		
line	m	f	m	f	m	f	X^2
M1	67	95	124	136	130	80	17.1 (2 df)***
M2	93	126	115	115	167	121	12.1 (2 df)***
S1	87	121	--	--	131	94	11.6 (1 df)***

*** $P < 0.001$

Test 2 was carried out with the M2 line of **D.melanogaster** and two new lines, S2 and S3, of **D.simulans**, with 11, 12 and 13 replicates, respectively, for each line. In each vial we recorded both the time at which larvae pupated and the height they attained. The pupae were classified, according to the duration of larval development (egg-to-pupa development time), as coming from early, intermediate or late pupating larvae, and the mean pupation height was calculated for each group. Thus, the relation between larval development time and larval pupation height was analyzed.

Table 2. Top: analyses of variance of the pupation height values shown by larvae pupating early, intermediate or late (3 groups) in the culture vials. Bottom: mean values of pupation height for each group and for each line. All the F-values were significant with $P<0.001$.

source of variation	line M2			line S3			line S4		
	df	MS	F	df	MS	F	df	MS	F
between groups	2	455.6	15.6	2	4031.8	66.4	2	895.0	18.1
error	30	29.2		33	60.7		36	49.4	
means of pupation height:									
early		61.7			59.6			42.2	
intermediate		58.6			32.8			29.2	
late		49.4			20.4			26.7	

Table 2 shows, together with the means of pupation height, the results of three analyses of variance in which groups of larvae with different pupation times were the sources of variation. In all cases, significant between-groups differences were found. Therefore, the larvae with a shorter development duration pupated, on average, higher than those with a longer development.

Sex differences in pupation height were first reported by Bauer (1984). Later, however, Bauer & Sokolowski (1985) did not find any sex difference, despite their use of the same material and method. Our results show that the male larvae of **D.melanogaster** and **D.simulans** pupate higher than females do. Also, a higher pupation site occurs for larvae with shorter development. These two conclusions might be interrelated in view of the general shorter development time for male larva than for female (see ref. in Ashburner & Thompson 1978). We realize that these facts could have important repercussions and be responsible for some of the failures reported on experiments selecting for pupation height (Mensua 1967; Ringo & Wood 1983).

References: Ashburner,M. & J.H. Thompson Jr 1978, in: Genetics and Biology of Drosophila, v.2a, Academic Pr. London; Bauer, J.S. 1984, DIS 60:48; Bauer, S.J. & M.B. Sokolowski 1985, Can.J. Genet. Cytol. 7:334-340; Mensua, J.L. 1967, DIS 42:76; Ringo, J. & D. Wood 1983, Behav. Genet. 13:17-27.

Casares, P., E. San Miguel* and M.C. Carracedo. University of Oviedo, Spain. *University of Leon, Leon, Spain. A preliminary study on the number of larval denticles in Drosophila.

The ventral surface of third instar larvae of **Drosophila simulans** and **D.melanogaster** shows thin, short, black spiculae or denticles, grouped in several rows forming denticle belts (Fig. 1). These belts appear at the boundaries of consecutive larval segments and because of this are referred to as thoracic or abdominal belts. The denticles display different size, shape and orientation. They help the larvae to move through the food which might be related with some described larval behaviours connected with food acquisition. **(1)**

Figure 1. Ventral view of a third instar **Drosophila simulans** larva showing denticle belts.

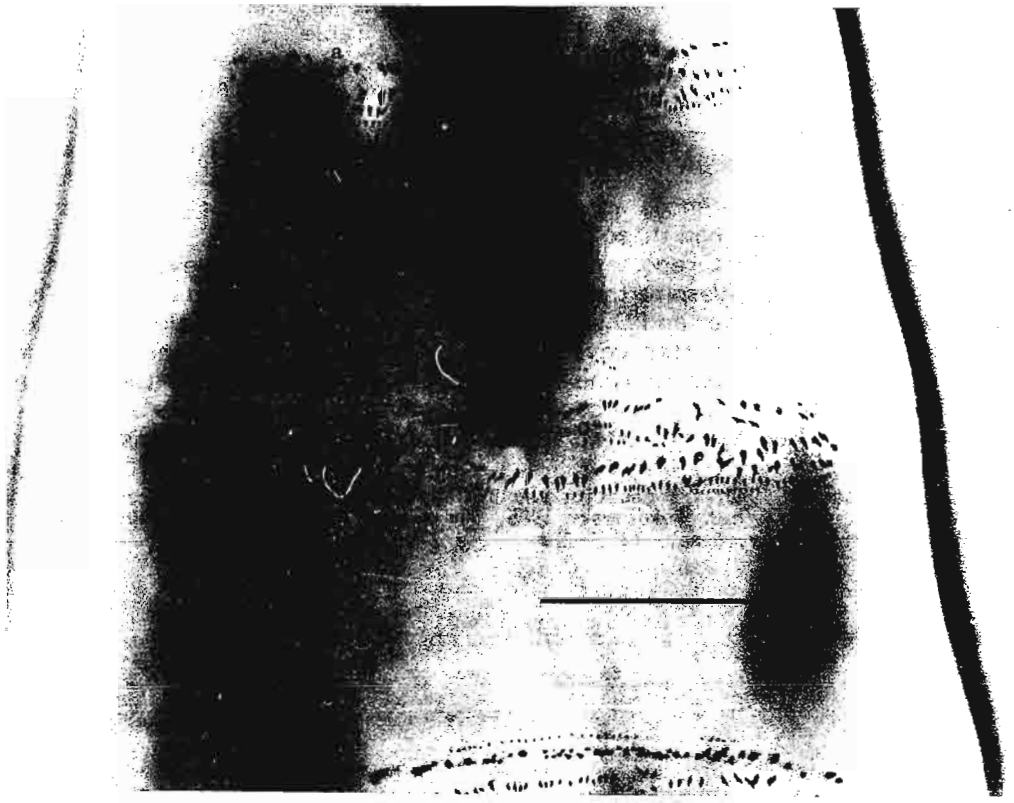


Figure 2. Denticle belts on the third thoracic (a) and first abdominal (b) segments of a **Drosophila simulans** larva. Bar represents 300 µ.

Table 1. Number of denticles (mean values ± s.e.) from 8 **D.simulans** isofemale lines developed either in medium S or in medium H.

Lines		thoracic segment		
medium S	L1	109.63 ± 3.98		
	L2	95.15 ± 3.02	t=3.41(18 df)	P<0.01
medium H	L3	99.53 ± 3.09		
	L4	94.08 ± 2.00	t=1.49(18 df)	P>0.05
		abdominal segment		
medium S	L5	163.82 ± 5.82		
	L6	188.56 ± 6.48	t=2.83(18 df)	P<0.05
medium H	L7	165.44 ± 6.26		
	L8	187.40 ± 3.70	t=3.02(18 df)	P<0.01

Table 2. Number of denticles (mean values ± s.e.) of **D.melanogaster** and comaprison with those of **D.simulans.** The mean values of **D.simulans** come from pooled data of two pairs of lines developed in medium S.

species	thoracic segment		
D.mel.	87.21 ± 2.24		
D.sim.(L1+L2)	102.39 ± 2.65	t=4.35(47 df)	P<0.001
	abdominal segment		
D.mel.	148.83 ± 2.99		
D.sim.(L5+L6)	176.18 ± 5.10	t=4.61(47 df)	P<0.001

We have analyzed the number of denticles on the third thoracic and first abdominal segments, two segments with different denticle distribution patterns (Fig. 2). Eight isofemale lines of **D.simulans** from the same population, four of them kept for two years in standard food (medium S, with 12% agar) and the other four in a thicker one (medium H, with 15% agar), and one strain of **D.melanogaster** reared in medium S were the biological material. Ten larvae from every **D.simulans** line and twenty-nine from **D.melanogaster** were fixed in Farmer's fluid for 6 hr and subsequently stored in 70% ethanol. Larvae were dorsally placed on a slide with a drop of lactic acid (20%), and a cover slide applied without pressure. The third thoracic and the first abdominal segments were photographed under a microscope. From each photograph we registered the number of denticles of the third thoracic (in a 200 x 200 μ^2 area) or first abdominal (in a 300 x 300 μ^2 area) segment in **D.simulans.** In **D.melanogaster** both segments of the same larva were counted.

In Table 1 the mean values of the number of denticles found in the **D.simulans** lines (L1 to L8) are given. Within each food class, the differences between some pairs of lines were checked by Student's t tests. All differences were significant except for L3 and L4 lines.

The mean values for the number of denticles corresponding to **D.melanogaster** are shown in Table 2. These values were contrasted with those of the **D.simulans** lines developed in medium S, and the differences were clearly significant for both larval segments. The number of denticles was higher for **D.simulans** than for **D.melanogaster**, which is in accordance with Agnew's results. **(2)** Following this author the above signifies that **D.simulans** might have some advantage over **D.melanogaster** when eating hard foods.

The differences found between the **D.simulans** isofemale lines and between both species suggests the existence of an extensive phenotypic variability for this character. The number of denticles could be in connection with the degradation of natural food and therefore with the fitness of Drosophila populations. At the present we are engaged in a wider investigation on the effect that genes and environment have on this character.

Literature cited: **(1)** Sewell, D., B. Burnett & K. Connolly 1975, Genet. Res. 24:163-173; Godoy-Herrera, R. 1977, Behav. Genet. 7:433-439; Sokolowski, M. 1980, Behav. Genet. 10:291-302. **(2)** Agnew, J.D. 1973, DIS 50:51.

Chandrashekaran, S. and R.P. Sharma. Indian Agricultural Research Institute, New Delhi, India. A new dominant temperature sensitive lethal induced on the SM5 balancer In(2LR) al^2Cy lt^v cn^2 sp^2 chromosome.

Dominant temperature sensitive (DTS) lethals are useful in crossing schemes where a large number of homozygous mutagenised lines are to be established for screening purposes. It is all the more advantageous if the DTS lethal is on a balancer chromosome, so that non-DTS recombinant-escapers are avoided. DTS lethals on the second chromosome balancers CyO and Pm were reported by Falke & Wright (1970) and third chromosome balancers with DTS lethals were constructed by Marsh (1978).

Reported here is a new dominant temperature sensitive lethal induced on the SM5 balancer.

The DTS lethal was induced by feeding 0.03 M Ethyl Methane sulphonate in 2% sucrose to 2 day old SM5/+ males. The mating and screening scheme is shown in Fig. 1.

The vials where no Curly winged progeny hatched at 28°C were maintained after retesting, by isolating SM5/+ female and male flies from the 19° vial.

Of the 3,293 cross II vials set up, 3,100 were scored at 28°C and 8 putative DTS lethals isolated. Seven were found to be leaky when reared in larger populations producing 2-10% Curly escapers.

Figure 1. Scheme for isolating DTS lethals on the SM5 balancer.

Cross I Canton-S ♀♀ x In(2LR) SM5 al^2Cy $lt^v cn^2 sp^2$/+ ♂ (Fed with 0.03 M EMS) (mass mated in 250 ml bottles)

Cross II Canton-S ♀♀ +/+ x SM5*/+ single ♂ (pair mated in vials)

Vial 1 →→→→→→→→→→→→ Vial 2 Flies from vial 1 transferred to fresh vial and reared at 28±2°C
reared at 19±2°C

SCREEN VIALS: Select vials which produce SM5-Curly flies only at 19° and not at 28°.

SM5*/+ ; +/+ 19°C SM5*/+ ; +/+ Dies 28°C

Table 1. Lethality at the embryonal, larval and pupal phases in F_1 individuals from a CS ♀ x SM5 448/+ ♂ mating at 19° and 28°C.

Growth temperature	Number of Eggs	Unhatched Eggs	% Embryonic Lethality	Expected Larvae	Pupae seen	% larval Lethality	Adults Cy	Adults Cy^+	% pupal lethality
19°C	1014	174	17.15	840	540	35.71	148	244	27.40
28°C	1167	720	61.69	447	153	65.77	0	87	43.13

Table 2.

Female parent	Male parent	Growth Temperature	Total Progeny	Cy Progeny ♀	Cy Progeny ♂	Cy^+ Progeny ♀	Cy^+ Progeny ♂	Proportion of Cy progeny*
SM5 DTS/+	+/+	19°C	484	128	120	128	108	0.512
SM5 DTS/+	+/+	25°C	685	141	126	224	194	0.389
SM5 DTS/+	+/+	28°C	438	0	0	220	218	0.0
+/+	SM5 DTS/+	19°C	280	26	18	154	82	0.157
+/+	SM5 DTS/+	25°C	523	10	18	270	225	0.053
+/+	SM5 DTS/+	28°C	462	0	0	226	236	0.0

* Number of Cy flies divided by total number of flies.

Table 3. Lethality at the embryonal, larval and pupal stages of individuals from a reciprocal mating of SM5 DTS/+ and +/+ flies at 25°C.

Female parent	Number of Eggs	Unhatched Eggs	% Embryonic Lethality	Expected Larvae	Pupae seen	% larval Lethality	Adults Cy	Adults Cy^+	% pupal lethality
+/+	1008	284	28.17	724	550	24.03	0	298	45.81
SM5 DTS/+	932	142	15.20	790	748	5.30	239	301	27.80

One fully penetrant line designated SM5 DTS 448 was analysed for its viability at various temperatures and its effective lethal phase at the non-permissive temperature.

Eggs laid by Canton-S virgin females mated to SM5 448/+ males were counted and allowed to develop at 19 and 28°C. The surviving individuals at the end of the embryonal, pupal and adult phases were counted.

Lethality at all the stages were calculated, the data for which is shown in Table 1.

It was concluded from the data in Table 1 that DTS 448 was a multiphasic lethal. However, it is evident that the non-DTS individuals were also surviving poorly at both growth temperatures, the survival frequency of Cy^+ being 0.4 at 19° and 0.09 at 28°C (calculated as observed Cy^+ individuals ÷ expected Cy^+ individuals).

To further assess the viability of the DTS carrying SM5 flies and non-DTS flies, two way crosses with SM5 DTS 448/+ either as the female or male parent with Canton S as the other parent were made. Twenty pairs of flies were mated for 5 days each in 250 ml bottles at each temperature, transferred for another 5 days to fresh bottles and then discarded. Progeny from each set of bottles were counted and sexed. Data presented in Table 2.

The recovery of SM5 DTS/+ flies is 3.3 times more at 19°C and 7.4 times more at 25°C when the female parent is SM5 DTS/+ than when the female parent is +/+. Because of this differential recovery of SM5 DTS progeny, the eggs from a SM5 DTS/+ ♀ x Canton S ♂ and Canton S ♀ x SM5 DTS/+ ♂ were allowed to complete development at 25°C and the number and percentage of individuals surviving at the end of each developmental phase calculated (Table 3).

The data shows that the survival frequency of DTS448 SM5/+ flies at 25°C is raised from 0 to 0.4 when the female is SM5 DTS/+ instead of being Canton S. The DTS448 gene appears to be a pupal lethal since nearly 50% of the individuals are pupal lethal. However, the lethal effect can be rescued when the $DTS448^+$ individuals are derived from a DTS 448/+ female rather than from a wild type female. In this respect DTS448 behaves as a recessive since it is rescuable prior to fertilization by the DTS448/+ gene product that may perdure in the egg cytoplasm. The rescue is not however possible at the non-permissive temperature probably because the mutant product is malformed beyond rescue.

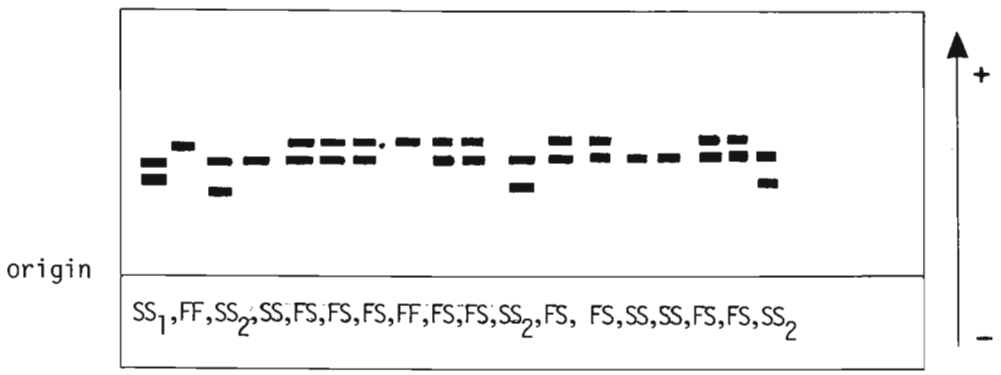
References: Falke, E.V. & T.R.F. Wright 1972, DIS 48:89; Marsh, J.L. 1978, DIS 53:155.

Charles-Palabost, L. Universite Francois Rabelais, Tours, France. Alleles found at six gene-enzyme systems in the French natural populations of **Drosophila melanogaster.**

Until now, we have analyzed more than 40 French natural populations of **Drosophila melanogaster** and it appears necessary to describe the different alleles found commonly at the Acph (acid phosphatase ; 3 - 101.4), Adh (alcohol dehydrogenase; 2 - 50.1), Est-C (esterase - C; 3 - 49), Est-6 (esterase - 6; 3 - 36.8) α-Gpdh (α-glycerophosphate dehydrogenase; 2 - 20.5) and Pgm (phosphoglucomutase; 3 - 43.4) loci. The technique used was horizontal starch-gel electrophoresis.

Gel preparation. The kind of starch used was Connaught (Toronto) hydrolysed starch. A 12.5% solution of starch in gel buffer (volum: 500 ml) was heated to near boiling point. The starch solution was degassed and poured in a plexiglass mold 27 x 16 x 1.5 cm. The gel was covered with saran-wrap.

Gel and electrode buffers. The buffer system used was Poulik's (1957); gel buffer: 76 mM TRIS and 5 mM citric acid, pH 8.65; electrode buffer: 300 mM boric acid and 60 mM NaOH, pH 8.1.



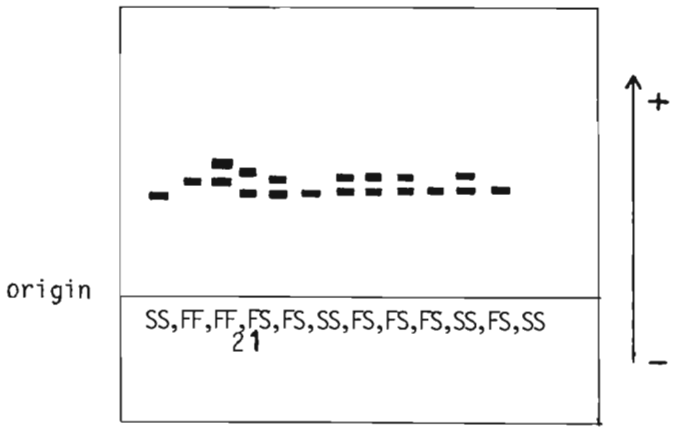


Figure 1. Alleles F, S, F_1, F_2, S_1 and S_2 at the Est-6 locues in the French populations of **Drosophila melanogaster.**

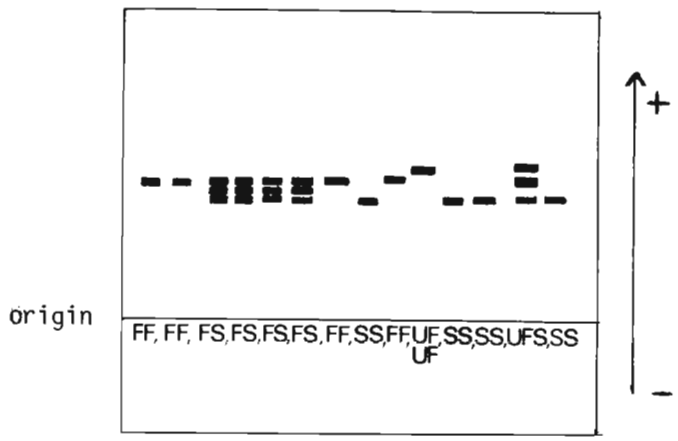


Figure 2. Alleles F, S and UF at the α-Gpdh locus in the French populations of **Drosophila melanogaster.**

Sample preparation. Single flies were ground in small wells in a plastic block with 30 µl of gel buffer or distilled water. This crude homogenate was absorbed with a piece of filter paper (Whatman n°3) 1.2 x 0.2 cm. The paper pieces containing the samples were inserted along a slot in the gel made about 2 cm from the edge. The gel was placed horizontally over two buffer trays with electrodes. Two bridges in Cofram paper were used to establish contact between the gel and the electrode buffer. This set-up was connected to a rectifier (Apelex 400 mA, 300V) and placed in a refrigerated room at 4°C. The time of run was approximately 2H30 under 260 volts and 175 mA per gel. After the run was completed, the four ends of the gel were cut off. The rest was divided into two parts. Each part was sliced horizontally into six 2.5 mm thick slices with the help of a taut wire and 2.5 mm thick guides. The top slices were discarded since surface effects made them unusable. The slices of each part were used for six different enzyme assays.

Enzyme assays (values are given for one slice). Acph: the stain used was

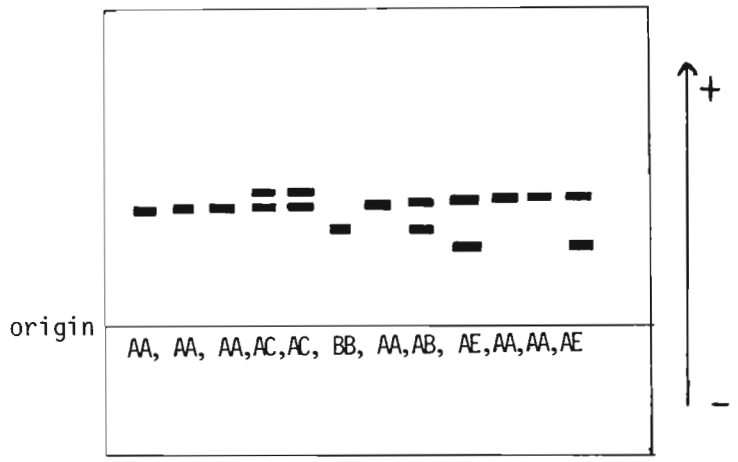


Figure 3. Alleles A, B, C and E at the Pgm locus in the French populations of **Drosophila melanogaster.**

50 mg Na-α-naphtyl acid phosphate, 25 mg Fast Garnet GBC salt in 75 ml 0.125 M acetate buffer pH 5.0. Adh and α-Gpdh were stained on the same slice of gel as follows: 4 ml EDTA 0.1 M, 125 mg α-glycerophospate, 12.5 mg β-NAD^+, 0.25 ml ethanol, 20.6 mg MTT, 3.75 mg PMS in 62.5 ml 0.05 M TRIS-HCL buffer, pH 8.5. Est-C: 1.25 ml 2% α-naphtyl-acetate solution (1:1 water: acetone was used as solvent), 75 mg Fast Blue RR salt, 8.25 ml propanol in 67.5 ml 0.1 M phosphate buffer pH 6.0. Est-6: 75 mg Fast Red TR salt, 3.75 ml propanol, 1.25 ml 2% α-naphtyl-acetate solution, 1.5 ml 2% β-naphtyl-acetate solution (1:1 water: acetone was used as solvent) in 75 ml 0.1 M phosphate buffer pH 6.0. Pgm: 66.5 mg glucose-1-phosphate (disodium salt), 7.5 ml $MgCl_2$ 0.1 M, 3.25 mg $NADP^+$, 4 units glucose-6-phosphate dehydrogenase, 25 mg MTT, 3.75 mg PMS in 62.5 ml 0.1 M TRIS-HCl buffer pH 8.0.

Alleles found at the different loci. In the case of Acph, Adh and Est-C, two alleles F (Fast) and S (Slow) are commonly present in the French populations and have been previously described (Acph: McIntyre 1966; Adh: Johnson & Denniston 1964; Est-C: Beckman & Johnson 1964). For esterase-6, six alleles are present: the two common F and S which produce, respectively, the Fast (migrating rapidly towards the anode) and the Slow (migrating more slowly towards the anode) variants, two alleles (F_1 and F_2) which give allozymes migrating more rapidly than the Fast variant, and two other (S_1 and S_2) which give allozymes migrating more slowly than the Slow variant (Fig. 1). At the α-Gpdh locus, three alleles are present: the two common F and S and a rare UF (ultra fast) which produces a variant migrating more rapidly than the Fast one (Fig. 2). For Pgm, according to the nomenclature of Trippa et al. (1977), four alleles are encountered in the French populations: A and B corresponding, respectively, to the Fast and Slow variants, C and E producing, respectively, variants which migrate more rapidly than Fast or more slowly than Slow (Fig. 3).

Note: enzyme substrates and dyes are Sigma reagents.

References: Beckman, L. & F.M. Johnson 1964, Hereditas 51:212-220; Johnson, F.M. & C. Denniston 1964, Nature 906-907; McIntyre, R. 1966, Genetics 53:461-474; Poulik, M.D. 1957, Nature 180:1477-1479; Trippa, G. et al. 1977, DIS 52:74.

Coyne, J.A. University of Maryland, College Park, Maryland USNA. Further evidence for X-Y interactions in the control of the Stellate locus.

XO males of **D.melanogaster** are sterile, and their primary spermatocytes contain crystals that are absent in XY males. These crystals are either needle- or star-shaped, depending on whether the allele at the Stellate locus (1-45.7) is Ste+ or Ste, respectively (Meyer et al. 1961). The appearance of the crystals depends on the absence of a particular region on the long arm of the Y chromosome (Hardy et al. 1984). Livak (1984) found that both X and Y chromosomes of **D.melanogaster** contain many tandem repeats of a 1250-base-pair DNA segment that may compose the Stellate locus. The tandemly-repeated DNA resides in the chromosome band containing the Stellate locus and in the segment of the Y chromosome whose deletion leads to crystal formation. This DNA codes for an 800-base-pair RNA that in turn produces a polypeptide much more abundant in XO than in XY testes; this may, in fact, be the polypeptide composing the spermatocyte crystals.

Based on these facts, Livak (1984) proposed that the Y-linked copies of the presumptive Stellate DNA serve to regulate those copies on the X. He posited that XO males, missing this regulatory segment, overproduce the product of the Stellate locus which then crystallizes in the spermatocytes.

I report here a test of this hypothesis in the sibling species **D.simulans.** Livak (1984) found that the putative Stellate DNA sequence is absent on the **D.simulans** X chromosome and is present on the Y but with reduced copy number. One would therefore predict that XO males of **D.simulans** would not have crystals in the primary spermatocyte because, although the Y-linked "regulatory" DNA is missing, there is no X-linked gene producing protein.

I obtained XO males of **D.simulans** as rare segregants from a Y-autosomal translocation stock provided by E.H. Grell. This stock, T(Y;2)3,+/net b, contains homozygous net, black (II) females and wild-type males, the latter heterozygous for a net b second chromosome and a wild-type chromosome attached to a Y. About one in five hundred males in this stock are net black XO males produced by nondisjunction.

Twenty-one four-day-old XO males from this stock were dissected in Ringer's solution and their testes examined under phase-contrast microscopy. Primary spermatocytes were compared to those present in XO **D.melanogaster** males. As reported previously, the **D.melanogaster** males had crystal-containing spermatocytes. None of the 21 **D.simulans** XO males, however, showed crystal formation. All of these had full-sized testes which contained sperm in various stages of development, but none had motile sperm. The lack of crystals in these males supports Livak's suggestion that Y-linked copies of the presumptive Stellate locus regulate the copies on the X, and militates against the idea that crystal formation in spermatocytes is an inherent property of XO males.

Acknowledgements: I thank Ken Livak for the idea of looking at XO **D.simulans** males. This work was partially supported by grants from the National Science Foundation (BSR-83-18558) and the National Institutes of Health (32221).

References: Hardy, R.W. et al. 1984, Genetics 107:591-610; Livak, K.J. 1984, Genetics 107:611-634; Meyer, G.F. et al. 1961, Chromosoma 12:676-716.

Di Pasquale Paladino, A., P. Cavolina and G. Romano. Universitá di Palermo, Italy.

Analysis of larval hemocytes in the melanotic tumor mutant tu-pb of **Drosophila melanogaster**.

As previous investigations have shown (Sparrow 1978), a common feature among melanotic tunor mutants of **Drosophila melanogaster** is the participation of larval blood cells in tumor formation. According to Rizki & Rizki (1980a), plasmatocytes, with their morphological variants podo- and lamellocytes, and the crystal cells (Fig. 1) are the cellular components of the larval hemolymph. They are produced in the lymph gland.

The hemolymph of tumorous 3rd instar larvae is characterized by a large number of lamellocytes owing to their precocious transformation from plasmatocytes (Sparrow 1978). The recently isolated melanotic tumor mutant, tu-pb, differs from the other tumor mutants since melanized masses appear only in the adult stage and are confined to the head. tu-pb tumors develop at 23.5°C (penetrance: ♀♀=35%, ♂♂=10%) but not at 18°C (Di Pasquale Paladino & Cavolina 1983). In order to elucidate the mechanism of tumor formation in tu-pb, we examined the patterns of blood cell types in the hemolymph of late 3rd instar male and female larvae (120 h aged at 23.5°C) of tu-pb and Oregon-R (as control) stocks.

Figure 1. Crystal cell from the larval hemolymph.

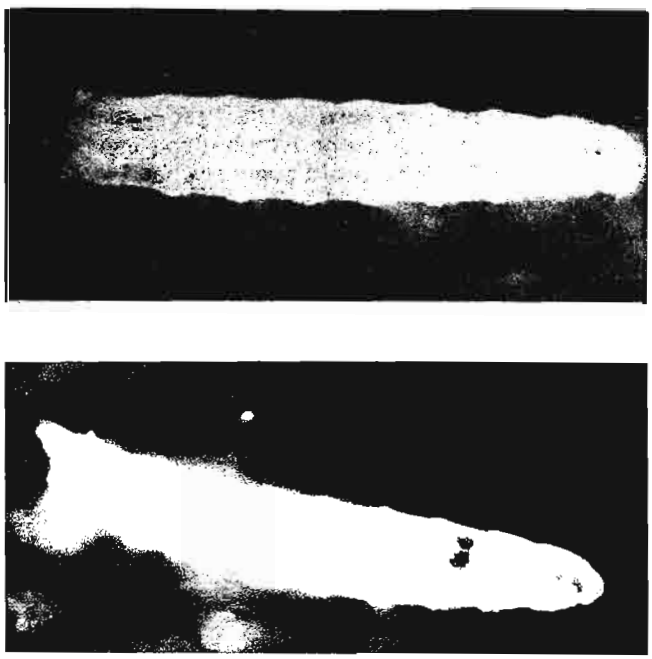


Figure 2. Heat treated larvae: (a) Oregon-R, (b) tu-pb.

Table 1. Mean cell numbers in hemolymph of Oregon-R and tu-pb 120h aged larvae.

Cell types	♂ Oregon-R	♂ tu-pb	♀ Oregon-R	♀ tu-pb
Plasmatocytes	3618.6±967.38	3122.2±300.54	3552.6±797.86	3505.4±624.91
	t=0.49		t=0.046	
Lamellocytes	3310.8±306.96	1880.0±384.97	2843.4±526.75	2591.6±305.70
	t=2.90 (sign.)		t=0.41	
Crystal cells	237.4±8.42	48.6±10.95	210.4±29.50	55.8±3.43
	t=13.63 (sign.)		t=5.19 (sign.)	
Cell totals	7166.8±1202.03	5050.8±340.77	6606.4±1393.75	6152.8±846.3
	t=1.69		t=0.28	

P=0.05

Table 2. Hot water (70°C, 15') treated larvae.

breeding temperature	age	stock	N	no melanization %	spread melanized cells %	melanized lymph gland %
23.5°C	96h	♀♀ Oregon-R	103	1.9	98.1	0.0
		♂♂ Oregon-R	104	2.9	97.1	0.0
		♀♀ tu-pb	111	98.2	0.0	1.8
		♂♂ tu-pb	97	97.9	0.0	2.1
23.5°C	120h	♀♀ Oregon-R	117	0.0	93.2	6.8
		♂♂ Oregon-R	109	0.0	95.4	4.6
		♀♀ tu-pb	141	75.2	0.0	24.8
		♂♂ tu-pb	127	70.1	0.0	29.9
18.0°C	240h	♀♀ Oregon-R	125	30.4	69.6	0.0
		♂♂ Oregon-R	100	16.0	84.0	0.0
		♀♀ tu-pb	113	88.5	0.0	11.5
		♂♂ tu-pb	105	78.1	0.0	21.9

Smears of fresh hemolymph from single individuals were stained with

Counts of cell types were made according to the classification into plasmatocytes, lamellocytes and crystal cells (Rizki 1978). Results and statistical analysis (t-Student) are presented in Table 1. Comparing tu-pb with Oregon-R, blood cells mean number patterns appear similar except for the crystal cells which are numerically fewer in tu-pb.

Therefore, we can conclude that tu-pb does not hold a larger amount of lamellocytes in its hemolymph. This fact seems to be in agreement with the later tumor manifestation in this stock. On the other hand, the reduced number of crystal cells might be significant. As Rizki has recently pointed out (Rizki 1980b), melanization of crystal cells can be induced by treatment of larvae with hot water. By using this functional criterion, we compared the distribution of heat-induced black cells in tu-pb and Oregon-R larvae. Results are presented in Table 2. In consequence of heating the two stocks manifest a different phenotype: blackened cyrstal cells appear spread in the hemocoel of Oregon-R larvae, while in tu-pb they are maximally confined into the lymph gland (Fig. 2).

The blackening of lymph gland is more frequent in late 3rd instar larvae (120 h aged) than in the younger ones (96 h aged) and in larvae reared at 18°C; the trait appears also to be recessive because etherozygous tu-pb/Oregon-R larvae manifest the Oregon phenotype. According to Shrestha & Gateff (1982), mature crystal cells are very rare in the first hematopoietic lobes and absent in the second lobes; therefore, their relative abundance in tu-pb lymph gland can be considered an unusual condition.

These findings could suggest that the releasing of mature crystal cells by the hematopoietic organs may be abnormal in tu-pb; they could also account for the low number of free crystal cells found in the hemolymph of the tumorous stock.

The hypothesis of a possible relation between the above mentioned facts and the manifestation of the melanotic tumor trait must be confirmed by further investigations. In this regard, we take into consideration the possibility of following the fate of the crystal cells in tu-pb making them marked with the Black-cells (Bc) mutant. In Bc/+ individuals naturally occurring pigmented crystal cells can be seen in larval, pupal and adult stages. We are now trying to combine tu-pb with Bc.

References: Di Pasquale Paladino, A. & P. Cavolina 1983, DIS 59:31; Rizki, T.M. 1978, in: Genetics and Biology of Drosophila, Academic Pr. London, 2b:397; Rizki, T.M. & R.M. Rizki 1980a, Experientia 36:1223; _____ 1980b, J. Exp. Zool. 212:323; Shrestha, R. & E. Gateff 1982, Devel. Growth & Diff. 24(1):65; Sparrow, J.C. 1978, in: Genetics and Biology of Drosophila, Academic Pr. London, 2b:277.

Dunkov, B.H. and K.H. Ralchev. University of Sofia, Bulgaria. Genetic localization of gene controlling one of diaphorases in **D.virilis.**

Diaphorases (NAD(P)H:(acceptor)oxidoreductases) in Drosophila are controlled by at least three structural genes (Ralchev et al. 1986). Using starch gel electrophoresis[1] followed by specific staining[2] and by means of recombination analysis, we localized the gene responsible for the diaphorase-3 enzyme (DIA-3).

Electrophoretic study of 20 wild and laboratory stocks of **D.virilis** revealed that stock No.112 is homozygous for an allele determining very low activity of diaphorase-3, i.e., Dia-3l (low activity) (Ralchev et al. 1986). Crosses of this stock with stock No. 147 possessing high activity of diaphorase-3, i.e., Dia-3h (high activity) showed sex-linked inheritance of this character (Fig. 1). For precise localization of Dia-3 gene on the X-chromosome genetic map stock No.127 carrying mutations cv - crossveinless, 1-25.0, and w - white, 1-105.0 (Alexander 1976) was used. The following crosses were carried out:

$$P\ ♀♀\ (127)\ \frac{cv\ w\ (Dia\text{-}3h)}{cv\ w\ (Dia\text{-}3h)} \times ♂♂\ (112)\ \frac{+\ +\ (Dia\text{-}3l)}{Y}$$

$$F_1\ ♀♀\ \frac{cv\ w\ (Dia\text{-}3h)}{+\ +\ (Dia\text{-}3l)} \times ♂♂\ \frac{cv\ w\ (Dia\text{-}3h)}{Y}$$

In F_2 progeny 733 male flies, recombinant by visible markers, were selected and analysed electrophoretically to determine the phenotype of diaphorase-3. The results of this analysis (Table 1) showed that Dia-3 gene is located at 65.3 ± 2.3 position.

Table 1. Genetic mapping of Dia-3 in **D.virilis.**

Recombinant males Phenotype of visible markers	Number	DIA-3 phenotype of the recombinants DIA-3h	DIA-3l
cv +	368	223	145
+ w	365	224	141

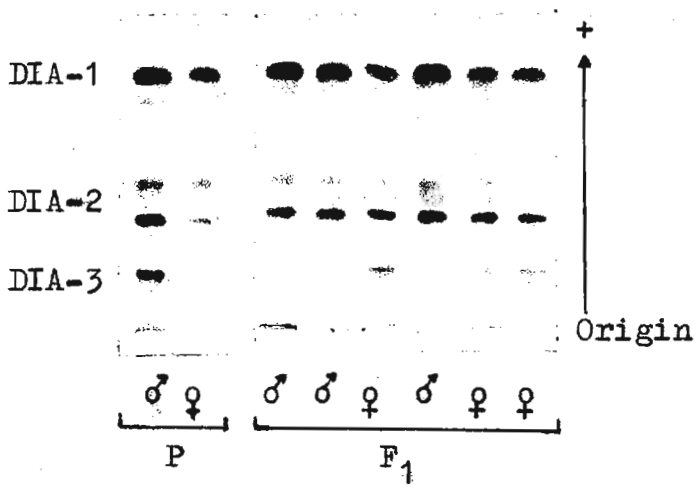


Figure 1. Electrophoregram showing DIA-3 phenotype in F_1 progeny of cross: ♀♀112 (DIA-3l) x ♂♂147 (DIA-3h).

[1]. 13% starch gel electrophoresis was used: gel buffer, 0.028 M Tris - citric acid, pH 8.6; electrode buffer, 0.3 M H_3BO_3 - NaOH, pH 7.6; 10 V/cm, 4 hours at 4°C.

[2]. On the basis of the Kaplan & Beutler (1967) staining system the gel was incubated for 2-4 hours at 37°C in solution containing: 100 ml of 0.05 M phosphate buffer, pH 7.4, 0.025 g NADH, 0.03 g dimetilthiazolyltetrazolium bromide (MTT), and 1 ml of 0.4% dichlorophenol indophenol (DCIP).

Acknowledgements: Authors are grateful to Dr. V.G. Mitrofanov, Inst. of Developmental Biology, Acad. Sci. of the USSR for kindly provided stocks of **D.virilis.**

References: Alexander, M.L. 1976, in: Genetics and Biology of Drosophila (Ashburner & Novitski, eds.), v.1c:1365-1427, Academic Pr NY; Kaplan, J.C. & E. Beutler 1967, Biochem. Biophys. Res. Commun. 29:605-610; Ralchev, K.H., B.H. Dunkov & A.S. Doichinov 1986, Genetics and Breeding (Bulg. Acad. Sci.), in press.

Duttagupta, A.K. and S. De. University of Calcutta, India. Transcriptive activity of X-chromosome in **D.virilis, D.americana** and in their hybrid.

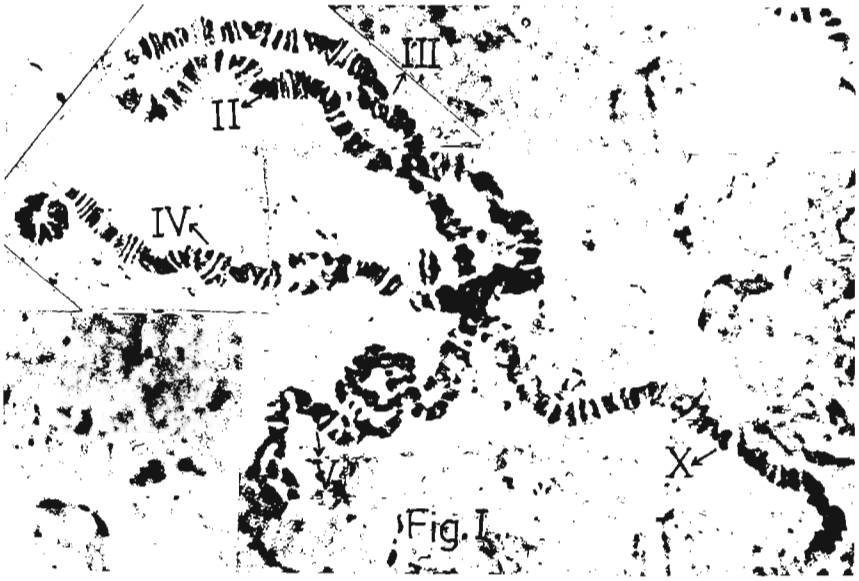
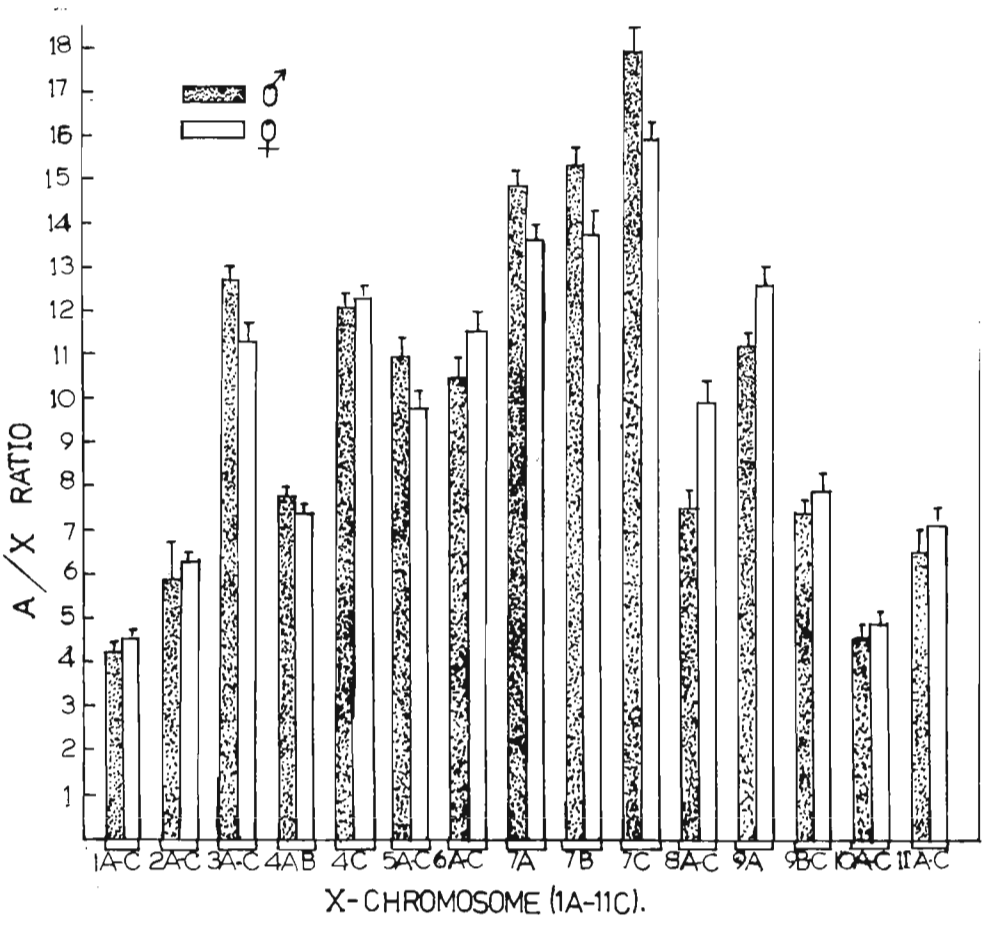


Figure 1. Morphology of hybrid (**D.americana** X **D.virilis**) male polytene chromosome.



Dosage compensation has been studied in 2 species: **D.virilis** and **D.americana** and in their hybrid (**D.americana** ♀ x **D.virilis** ♂). The chromosome complement of **D.virilis** is similar to that of the ancestral species with 5 acrocentric chromosomes (elements A to E) and a dot chromosome (element F). In **D.americana** fusion between B element and the X chromosome of the ancestral species has occurred. These two closely related species belonging to the same group (**virilis**) form hybrids (Patterson & Stone 1952) (Fig. 1).

We wanted to know whether **D.americana** (comparatively a younger species evolutionarily which evolved from **D.virilis** by X-B fusion) and **D.virilis** have the property of dosage - compensation and whether the hybrid shows any change from the parental species or not.

Results reveal that the total transcriptive activity of the X chromosome is equivalent in both female and male **D.virilis** (X/A ♀/X/A ♂ = 1.06). Sitewise transcriptional analysis shows that all the 15 subsegments of the distal part of the X chromosome (1A-11C) are dosage compensated (Fig. 2).

Same type of results have also been obtained for **D.americana** which showed a similar activity in male and female as revealed from the total grain count analysis (X/A ♀/X/A ♂ = 1.08). Sitewise analysis of the distal part (15 subsegments from 1A-10B) shows no dose-dependent sites. The slight deviation in the female/male ratio from 1 for the sites 4A and 4BC was found to be non-significant (P>0.7) (Fig. 3).

In the hybrid nuclei also, we fail to observe any significant deviation in the X chromosome/autosome ratio in male and female (female-/male ratio = 1.27) (Fig. 4).

Figure 2. Histogram showing the transcriptive activity of different X-chromsomal segments (1A-11C) of male and female **D.virilis.**

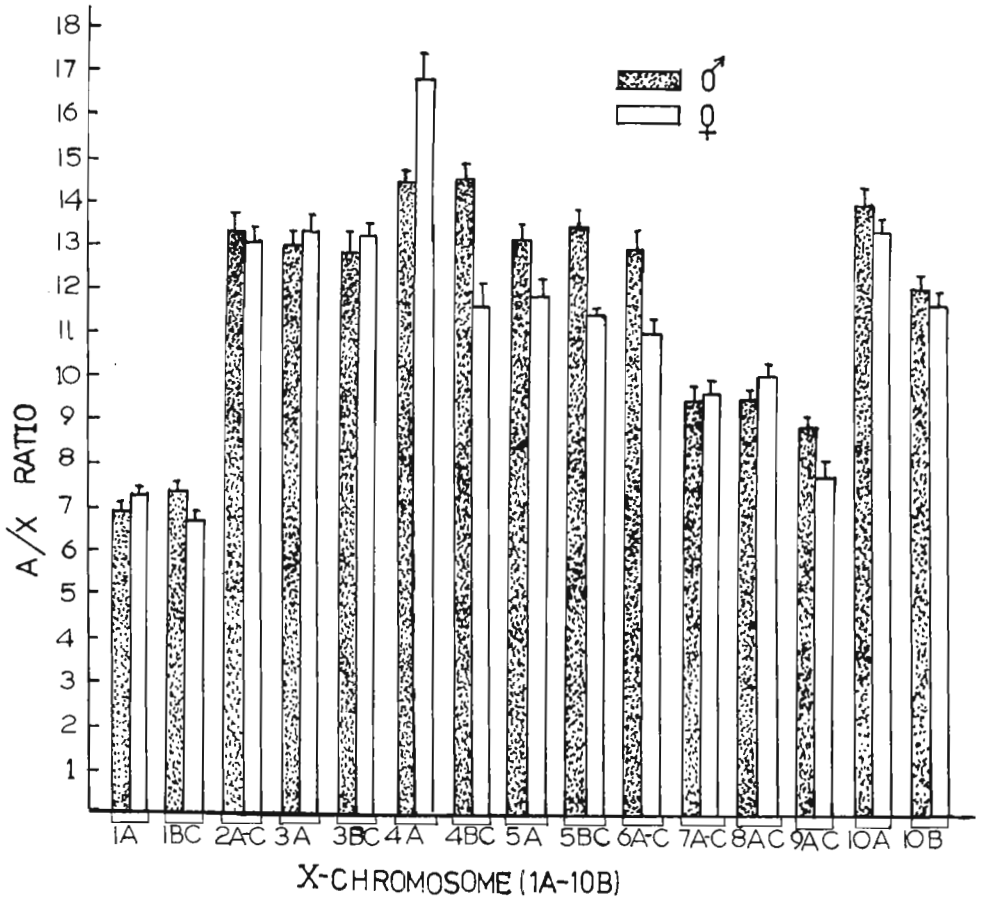


Figure 3. Histogram showing the transcriptive activity of different X-chromosomal segments (1At-10B) of male and female **D.americana.**

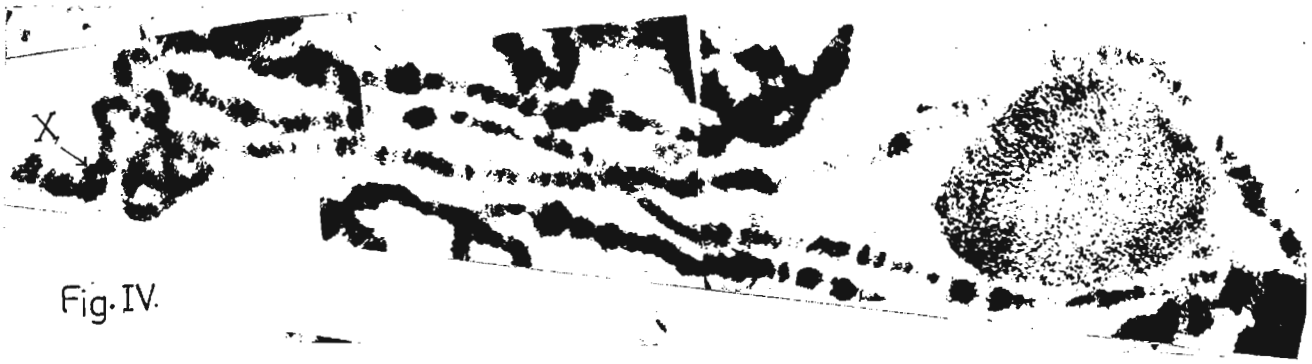
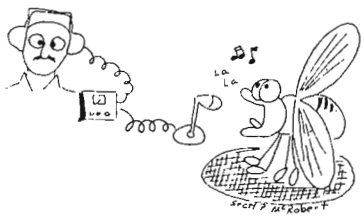


Figure 4. Photograph showing the ^{3}H-uridine incorporation in the hybrid (**D.americana** ♀ X **D.virilis** ♂) female X-chromosome.

We, therefore, conclude that the individual set of chromosomes maintain the property of compensation in the hybrid cytoplasm.

Acknowledgement: This work has been supported by a UGC scheme (F.23/167-83 (SR-II) of 17.1.84) to A.K.D.G.

Reference: Patterson, J.I. & W.S. Stone 1952, Evolution in the genus Drosophila.



Farmer, J.L. and D.J. Fairbanks. Brigham Young University, Provo, Utah USNA. Interaction of the bw and w loci in **D.melanogaster.**

The allele w^{co2} is suppressed by a gene on 2R (Farmer 1977). The experiments reported here show that the allele which was formerly called $Su(w^{co2})$ is bw^+ and the $Su^+(w^{co2})$ is a previously undescribed allele which we have named bw^6. This is the second reported interaction of these loci. Rifenburgh & Sutfin (1935) reported that w;bw flies were "slight light buff or cream", definitely darker than w alone. There have been reports of interaction between other eye color loci (e.g., Nolte 1958; Reedy & Cavalier 1971).

Materials and methods. Stocks were obtained from Mid-America Drosophila Stock Center, Bowling Green State University; the Division of Biology, California Institute of Technology; J.A. Beardmore, University College of Swansea; and P.T. Ives, Amherst College. Most of the w and bw alleles which were tested were in multiply marked stocks. Only the relevant alleles have been shown for simplicity. A full listing of genotypes is available on request. Wildtype flies from various populations were obtained from R.S. Singh, McMaster University.

Flies were maintained on a cornmeal-molasses-yeast-agar medium or on Carolina Instant Drosophila Medium.

The drosopterin content of single heads was measured using the acidic ethanol extraction method described previously (Farmer 1977).

Results and discussion. If bw and $Su(w^{co2})$are allelic, crossovers between them should be rare. In an attempt to find crossovers between bw and $Su(w^{co2})$, females of genotype w^{co2} v; px bw sp/+ + + were crossed with w^{co2} v;px bw sp males. The px bw sp chromosome which was used in this cross and the cross which follows did not suppress w^{co2} while the wildtype homologue did. The non-crossover-type progeny had white or suppressed (orange to vermilion) eye color. Crossover-type progeny should have had white (indistinguishable from one class of non-crossover-type progeny) or unsuppressed (pale gold, like zeste) eye color. No crossover-type progeny were observed among 33,305 progeny. This result corresponds to a maximum map distance of 0.023 between bw and $Su(w^{co2})$ at the 95% confidence level.

A second type of cross was performed to look for crossovers between bw and $Su(w^{co2})$. Females of genotype px bw sp/+ + + were crossed with px bw sp males. Male progeny which had crossed over between px and sp (557 of 7561) were testcrossed to unsuppressed w^{co2} v females. In every case, brown crossover-type males produced only unsuppressed male testcross progeny, while crossover-type males with wildtype eyes produced male testcross progeny half of which were suppressed and half of which were not. Thus there were no crossovers between bw and $Su(w^{co2})$. The nature of this cross made it possible to exclude the possibility that $Su(w^{co2})$ might be outside the px-sp interval.

Combining the data from the two crosses yielded the following map distances: px-bw = 4.65; bw-sp = 2.72; maximum bw-$Su(w^{co2})$ = 0.016 at the 95% confidence level.

The results of the mapping crosses were consistent with the hypothesis that $Su(w^{co2})$ was identical with bw^+. To test the hypothesis further, a large number of bw alleles were tested to see if any of them might be linked to a gene which could suppress w^{co2} in a male fly whose genotype was w^{co2} v;bw*/$Su^+(w^{co2})$, where bw* is any one of the bw alleles tested. None of the chromosomes carrying bw alleles which were tested acted as a suppressor of w^{co2}. The bw alleles which were tested were: bw^{2b}, bw^4, bw^5, bw^{38j}, bw^{45a}, bw^{49h}, bw^{75}, bw^{80j21}, bw^{81}, bw^D, and bw^{V1}.

We concluded that $Su(w^{co2})$ was identical with bw^+ since no crossing over was detected between them and since all chromosomes which carry bw^+ suppress w^{co2} while all chromosomes which carry a non-wild-type allele of bw do not suppress. The allele which was formerly called $Su^+(w^{co2})$ must therefore be a mutant allele of bw. We have named it bw^6.

No interaction between bw^6 and alleles of w other than w^{c02} had been noticed in previous experiments. We looked for enhancement of a number of w alleles by bw^6 in flies whose genotypes were $w^*;bw^6$. Flies whose genotypes were $w^*;bw^6/bw^+$ served as controls. The symbol w^* represents any one of the w alleles which were tested. None of the w alleles which were tested was enhanced by bw^6. The w alleles which were tested were w^a, w^{a3}, w^{bf}, w^{bf2}, w^{bl}, w^{Bwx}, w^{cf}, w^{ch}, w^{co}, w^{col}, w^e, w^{ec3}, w^h, $w^{mR7aH.1}$, w^{sat}, w^{sp}, and w^t.

The major properties of the bw^6 and w^{co2} alleles can be summarized as follows. Flies which were homozygous or hemizygous w^{co2} and homozygous bw^6 or heterozygous bw^6 with any other mutant bw allele had very dark eyes due to the near absence of drosopterins. Flies which were homozygous bw^6 and which had at least one w^+ allele were wildtype. Flies which were heterozygous bw^6 with any other recessive bw allele and which had at least one w^+ allele were wildtype in appearance, although the drosopterin level of some genotypes was above or below the control value (Table 1). The one exception to this was $w^{co2}/w^+;bw^5/bw^6$ whose eyes were noticeably darker than controls (Table 2). The bw^5 allele interacts with w^{co2} also, as shown by the very low drosopterin concentration in w^{co2} Y;bw^5/bw^+ flies. Flies which were homozygous or hemizygous w^{co2} and homozygous or heterozygous bw^+ were wildtype in appearance, although the heterozygotes had a lower concentration of drosopterins, perhaps due to gene dosage effect

Table 1. Drosopterin content of w^{co2} v/+ + flies heterozygous for bw^6 and other bw alleles.

genotype	mean absorbance at 480 nm	standard deviation	N[a]
bw^+/bw^6	0.050[b,e]	0.005	5
bw/bw^6	0.041	0.008	5
bw^4/bw^6	0.072[d]	0.009	5
bw^5/bw^6	0.030[d,e]	0.009	5
bw^{38j}/bw^4	0.048	0.007	5
bw^{45a}/bw^6	0.043[c]	0.002	5
bw^{49h}/bw^6	0.064[c]	0.010	5

[a] N is the number of measurements.
[b] control value used for t tests.
[c] $0.01 < p < 0.05$ using t test.
[d] $p < 0.005$ using t test.
[e] also in Table 2.

Table 2. Drosopterin content of flies containing the bw^5 and/or bw^6 alleles.

genotype	mean absorbance at 480 nm	standard deviation	N[a]
$w^{co2}/w^+;bw^+bw^+$	0.065	0.010	9
$w^{co2}/w^+;bw^5bw^+$	0.055	0.009	9
$w^{co2}/w^+;bw^6/w^+$	0.050[b]	0.009	5
$w^{co2}/w^+;bw^5/bw^6$	0.030[b]	0.006	5
$w^+/w^+;bw^5/bw^6$	0.062	0.008	4
w^{co2} Y;bw^+/bw^+	0.045	0.001	2
w^{co2} Y;bw^5/bw^+	0.014	0.000	5
w^{co2} Y;bw^6/bw^+	0.037[c]	0.004	10
w^+ Y;bw^+/bw^+	0.061	0.008	5
w^+ Y;bw^5/bw^+	0.058	0.006	2
w^+ Y;bw^6/bw^+	0.063	0.006	5
w^+ Y;bw^5/bw^6	0.057	0.014	8

[a] N is the number of measurements.
[b] also in Table 1. [c] from Farmer 1977.

(Farmer 1977). Flies which were heterozygous w^{co2} with any other recessive w allele and homozygous bw^6 had very dark eyes due to the near absence of drosopterins, while flies which had the same genotype except for being heterozygous bw^6/bw^+ had a nearly wildtype eye color, the drosopterins being reduced, perhaps due to gene dosage effect (Farmer 1977; this paper).

If one considers all of the possible combinations of w^{co2}, bw^6, and their wildtype alleles, only flies which were homozygous or hemizygous w^{co2} and homozygous bw^6 had the mutant (dark brown) eye color. Thus w^+ suppressed homozygous bw^6, and bw^+ suppressed homozygous or hemizygous w^{co2}. It is possible to construct at least two kinds of models which would account for this kind of interaction.

In the first model, it is presumed that gene products of w^+ and bw^+ join together to form a multimer which is the active protein. The alleles w^{co2} and bw^6 are assumed to be mutated at sites which prevent formation of the quaternary structure between their gene products. However, it is assumed that the mutations allow weak but effective bonding between the gene products of w^{co2} and bw^+ or between the gene products of w^+ and bw^6.

The second model assumes that the w and bw loci each code for at least two activities. This model requires that one of the activities coded for by the bw locus, deficient in bw^6, is identical to one of the activities coded for the w locus, deficient in w^{co2}. Then only the double mutant $w^{co2};bw^6$ would produce the phenotype which is caused by the loss of the common activity. This model predicts that most, if not all, previously described w and bw mutants would be deficient for one of the unique activities.

Like bw^6, the bw^4 allele is wildtype when homozygous. However, unlike bw^6, it produces dark eyes when heterozygous with the bw^5 allele. It is possible that bw^4 and bw^6 represent an unusual class of mutations at the bw locus.

A preliminary survey of wildtype stocks from various parts of the world indicates that some populations carry an allele which acts like bw^6 (fails to suppress hemizygous w^{co2}) (Fairbanks, unpubl. data). No frequency data are available. Several laboratory stocks also carry a non-suppressing allele (Farmer 1977). Although the suppressing allele was found to be more common in both laboratory and wildtype socks, the non-suppressing allele was not rare.

Acknowledgements: We thank M.M. Green for helpful suggestions and C.A. Istock for providing laboratory facilities to DJF. This research was supported by a research grant from Brigham Young University. DJF was supported by an undergraduate research fellowship from the Honors Program of Brigham Young University.

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Ford, S.C. Temple University, Philadelphia, Pennsylvania USNA. A comparison of the genitalia of **D.melanogaster** and **D.affinis** males and females.

Although **Drosophila melanogaster** females elicit vigorous courtship from **Drosophila affinis** males, the flies never mate (S.P. McRobert & L. Tompkins, in prep.). This observation suggested the possibility that the external genitalia of **D.affinis** males or **D.melanogaster** females might be structurally dissimilar to the corresponding parts of conspecific individuals, making intromission physically impossible. Accordingly, I examined the genitalia of males and females from these two species by scanning electron microscopy.

D.melanogaster males and females were collected from a Canton-S laboratory stock, while **D.affinis** males and females were collected from isofemale lines that had recently been established from local populations. Newly eclosed males and females were separated by sex and aged for five days, then anesthetized with CO_2 and affixed to pin mounts with SEM silver paint. The flies were then sputter coated with gold, examined with a Phillips 501B scanning electron microscope, and photographed.

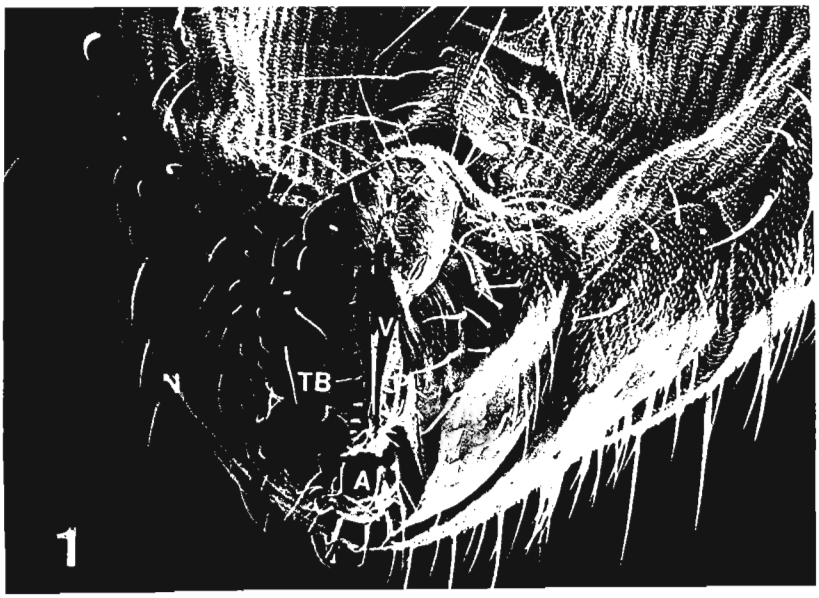

Figure 1. Posterior view of the external genitalia of a **D.affinis** female. **P**, vaginal plates; **TB**, thorn bristles; **V**, vulva; **A**, anus. Magnified 160X.

There are two vaginal plates on the ventral surface of the eighth segment of a Drosophila female. Each of the plates bears a row of thorn bristles, between which is located the vulva (Hodgkin & Bryant 1978). A comparison of the external genitalia of a **D.affinis** female (Fig. 1) and a **D.melanogaster** female (Fig. 2) reveals no conspicuous differences.

The penis of a Drosophila male is located between the posterior lobes of the genital arch, which is surrounded by the two halves of the eighth tergite (Hodgkin & Bryant 1978). A comparison of a **D.affinis** male (Fig. 3) and a **D.melanogaster** male (Fig. 4) reveals that the males' eighth tergites and genital arches are similar, but the **D.affinis** male's penis is much larger and is shaped differently from that of the **D.melanogaster** male. Ten males of each species were examined and these differences were consistently observed.

If living **D.affinis** males also have larger penises than living **D.melanogaster** males, this anatomical difference may be at least partially responsible for the fact that **D.melanogaster** females do not copulate with **D.affinis** males.

Figure 2. Posterior view of the external genitalia of a **D.melanogaster** female. See Fig. 1 for abbreviations. Magnified 160X.

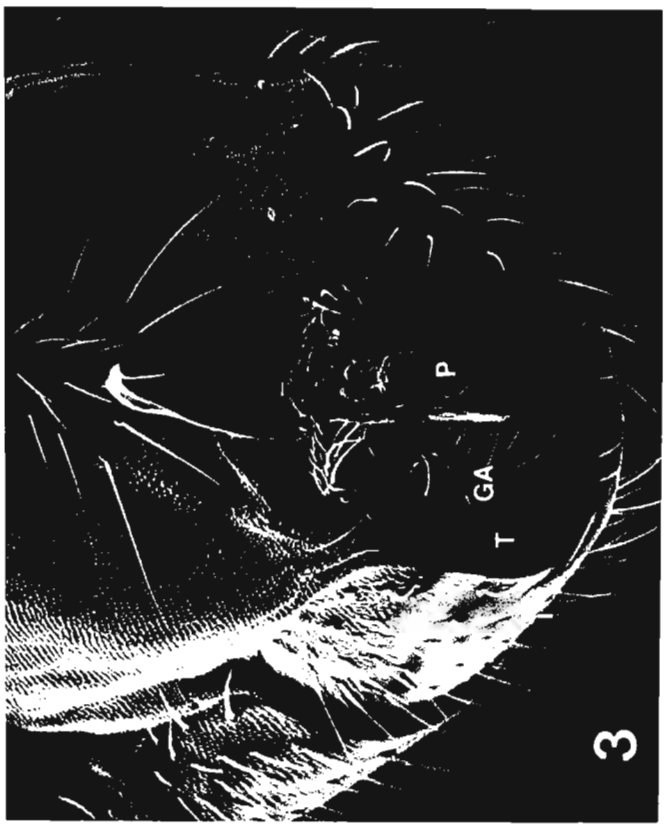

Figure 3. Posterior view of the external genitalia of a **D.affinis** male. **T**, eighth tergite; **GA**, genital arch; **P**, penis. Magnified 160X.

Figure 4. Posterior view of the external genitalia of a **D.melanogaster** male. See Fig. 3 for abbreviations. Magnified 160X.

Acknowledgements: I am grateful to Joel Sheffield for help with the SEM. I also thank Laurie Tompkins, Lisa Napolitano and Scott McRobert for advice, unpublished material, and Drosophila stocks. This research was supported by a Biomedical Research Support Grant from Temple University.

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Galissie, M., G. Abravanel and G. Vaysse.
Universite Paul Sabatier, Toulouse, France.

Consequences on the serotonin level in **Drosophila melanogaster** of an alimentary chronical pharmacological treatment.

We have previously described (Marenco et al. 1984) the fact that serotonin (5-HT) depletion only applied during larval instars, reduced learning performance of the seven-day imago. The same results were obtained when the depletion was carried out during the first week of the imago life. To obtain a noticeable effect on the learning performance, the usual amount of the inhibitor of serotonin synthesis: para-chlorophenylalanine (p-CPA), added to the synthetic chemically defined nutritive medium (S), has been carried out from 0.3g/l to 0.6g/l (Vaysse et al. 1983). Biochemical control of 5-HT level was performed on these flies treated for a week, the question is whether a seven day treatment gives the best depletion.

The present note concerns 5-HT amounts in imagos seven days old before the chemical analysis. They are fed on the S medium for seven, six, ... one or zero days; and, in each case, the seven day period is completed by a breeding on S medium added with a pharmacological agent:

- S + p-CPA medium with 0.6g/l of p-CPA,
- S + 5-HTP medium with 0.6g/l of 5-hydroxytryptophan (5-HTP), which is the immediate precursor of serotonin.

A rapid procedure of analysis by reversed phase high-performance liquid chromatography is applied to the estimation of serotonin from the flies's extracts, and allows us to follow, sample by sample, the consequences of a p-CPA or 5-HTP over-dose in the nutritive medium. The chromatographic system

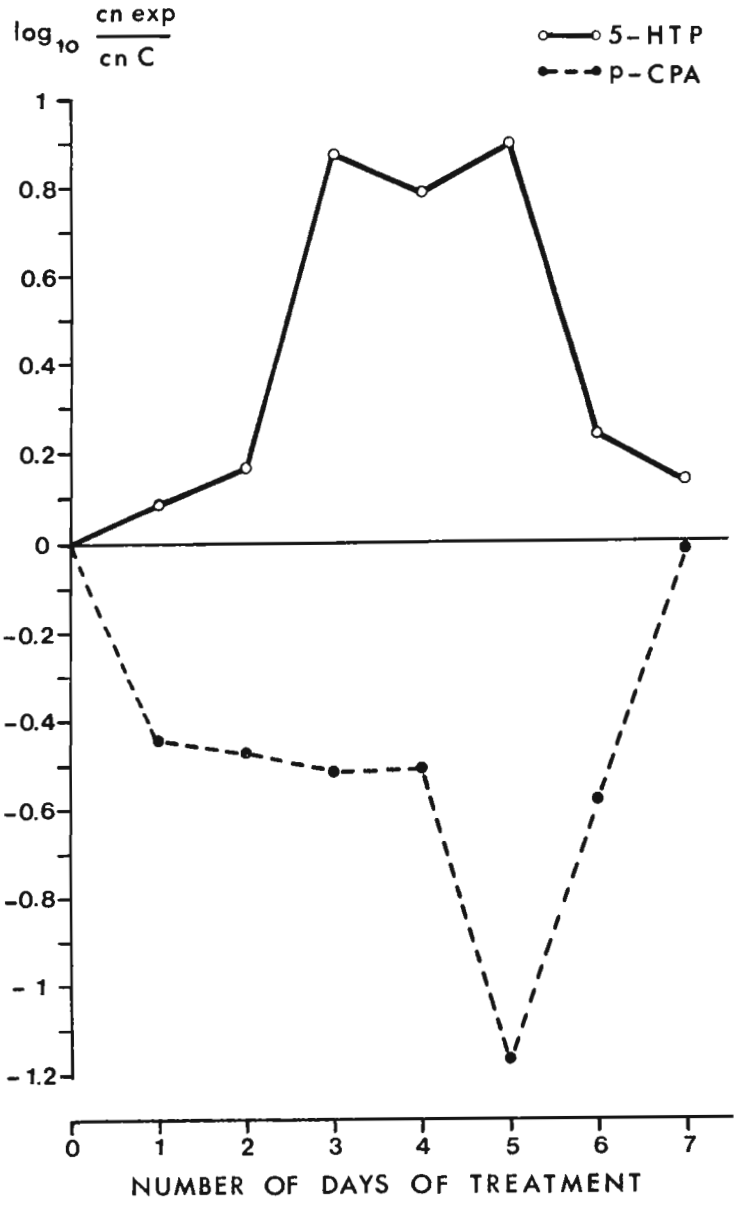

Figure 1. Level of 5-HT.

consists of LiChrosorb RP8 as stationary phase and methanol-sodium acetate gradient as mobile phase. Samples of twenty male flies are collected with an insect aspirator, weighed then frozen in liquid nitrogen at about 4 p.m. (accounting for the 5-HT circadian variation). Each one is lyophilysed for 24 hours, crushed in a micropotter crusher, then suspended in 1 ml HCl 0.1 N and centrifuged for two minutes at 6,000 g. The supernatant is injected directly into the chromatograph (two injections) and the eluted fraction is detected at 275 nm.

Figure 1 gives the results found, expressed as the decimal logarithm of the concentration of 5-HT (median values of the 5 samples used in each case), related to the concentration measured for the control group: for the experimental flies treated (a) by p-CPA; (b) by 5-HTP.

For the p-CPA group, 5-HT level falls from the first day of treatment; this tendency holds until the 5th day, when the strongest effect is seen. Following this, the control level is restored in this experimental group on the 7th day.

On the other hand, the 5-HTP level is markedly increased in the 5-HTP group on the third to the fifth day. If the treatment lasts longer, the curve returns towards the control level. In both cases, after a period of latency (one or two days), the level of the experimental groups differs from the control one, and in spite of the chronicity of the treatment returns towards the initial level. These results strongly suggest the existence of regulatory processes. It could be for each of these pharmacological agents: either an elimination under native form in the excreta; or an increase of the degradation processes by the normal metabolic paths of the 5-HT; or else an enzymatic induction (parametabolic for 5-HTP and xenometabolic for p-CPA).

References: Marenco, M.J., M. Galissie & G. Vaysse 1984, DIS 60:221; Vaysse, G., J.G. Sanz, M.J. Marenco, M. Galissie & G. Abravanel 1983, Bul. Soc. Hist. Nat. Toulouse 119:25-36.

García-Agustín, M.P., M.J. Martínez-Sebastián and J.L. Mensua. University of Valencia, Spain.

Possible bobbed alleles in a line of **Drosophila subobscura** selected for low abdominal bristle number.

In a selection experiment, involving four different lines (H1, H2, L1 and L2), for abdominal bristle number performed in **D.subobscura** (Martinez-Sebastian & Mensua 1986), different responses were noticed in one low selection line (L2). The differences included sudden increase of variability, disturbed abdominal bristle pattern and disturbed sclerotinization, which in **D.melanogster** have been shown to be due to the bobbed locus (Frankham, Briscoe & Nurthen 1978).

Table 1. Means ($\bar{X} \pm e_{\bar{X}}$) and coefficients of variability (C.V. $\pm e_{C.V.}$) with standard errors in each high (H1 and H2) and low (L1 and L2) selection lines, in the last generation of selection (A) and three years after the seelection was discontinued (B).

	$\bar{X} \pm e_{\bar{X}}$			C.V. $\pm e_{C.V.}$		
	A	B	A-B	A	B	A-B
H1	74.73±0.34	73.99±0.33	-0.74	6.98±0.32	6.34±0.32	-0.64
H2	72.08±0.36	69.81±0.38	-2.27	7.72±0.35	7.66±0.38	-0.06
L1	12.47±0.15	17.50±0.16	5.03	18.59±0.88	13.12±0.67	-5.47
L2	14.72±0.30	19.26±0.55	4.54	31.71±1.59	10.82±0.55	-20.89

When selection finished, the four lines were maintained under laboratory conditions without further selection for a three years period (30 generations approx.). At this time, the number of abdominal bristles was measured in each of the four lines.

Table 1 shows the means and coefficients of variability of abdominal bristle number of each line in the last generation of selection (A) and in each line three years after the selection was discontinued (B).

In the high selection lines (H1 and H2), the means as well as the coefficients of variability remained practically constant. These results support the hypothesis that abdominal bristle number is a neutral character for natural selection (Clayton, Morris & Robertson 1957).

However, the increase of the means occurring in the low selection lines (L1 and L2) suggests the existence of a minimum threshold for abdominal bristle number. The coefficient of variability decreased in both low selection lines, with the loss of variability being more extensive in the L2 than in the L1 line.

The behaviour of L2 line seems to suggest that part of the response to selection may be due, in this line, to the accumulation of bobbed alleles. During the three years period following selection the L2 line might then have tended to eliminate bobbed alleles in order to reach the normal level of rRNA. Consequently, the L2 line will have increased the number of abdominal bristles.

It was not possible to map the putative bobbed allele because there is no marker strain in **D.subobscura.** Work is in progress to demonstrate the presence of bobbed alleles using in situ hybridization with probes of **D.melanogaster.**

References: Clayton, G.A., J.A. Morris & A. Robertson 1957, J. Genet. 5:131-151; Frankham, R., D.A. Briscoe & R.K. Nurthen 1978, Nature 272:80-81; Martinez-Sebastian, M.J. & J.L. Mensua 1986, Genet. Selec. Evol., in press.

Garcia-Vazquez, E. and J. Rubio. University of Oviedo, Spain. Chromosomic analysis of two lines of selection.

We have canalized through selection the phenotype "2 anterior dorsocentral extra bristles", in two isofemale lines of **D.melanogaster:** ADC-4 and ADC-7 (Figure 1). The lines do not segregate individuals in classes higher than the selected class 2; their individuals do not have bristles in other positions than the selected one.

Ten males of ADC-7 were mated with 10 females of ADC-4, and vice versa, in two replicas. We analysed 100 individuals of each sex in the progeny. The two replicas of each crossing were homogeneous; by this reason, they were pooled.

After these crossings, we studied the influence of each chromosome in both lines obtained canalization. Experimental design is as the Kearsey & Kojima one (1967). Three independent replicas of each chromosomic combination have been obtained. They were homogeneous; by this reason they have been grouped. We counted 60 males and 60 females in each replica.

Eight homozygotic chromosomic combinations of the three main chromosomes were obtained. Each chromosome may be of the selected line (ADC-4 or ADC-7), or of the Oregon strain (normal phenotype, without extra bristles). We show female data; males behave in the same way.

The obtained distributions of both lines are very similar; but we have selected different genetic factors for extra bristle in each one: crossing both lines, the number of individuals with extra bristles decrease in F_1 and F_2 (Figure 2). There is not any significant difference among reciprocal crossings. We do not find any discanalization, since there are no individuals with more than 2 extra bristles, and extra bristles are only in the selected position.

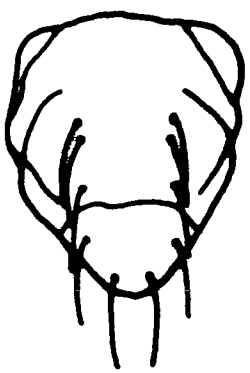

Figure 1. Canalized phenotype in ADC-4 and ADC-7.

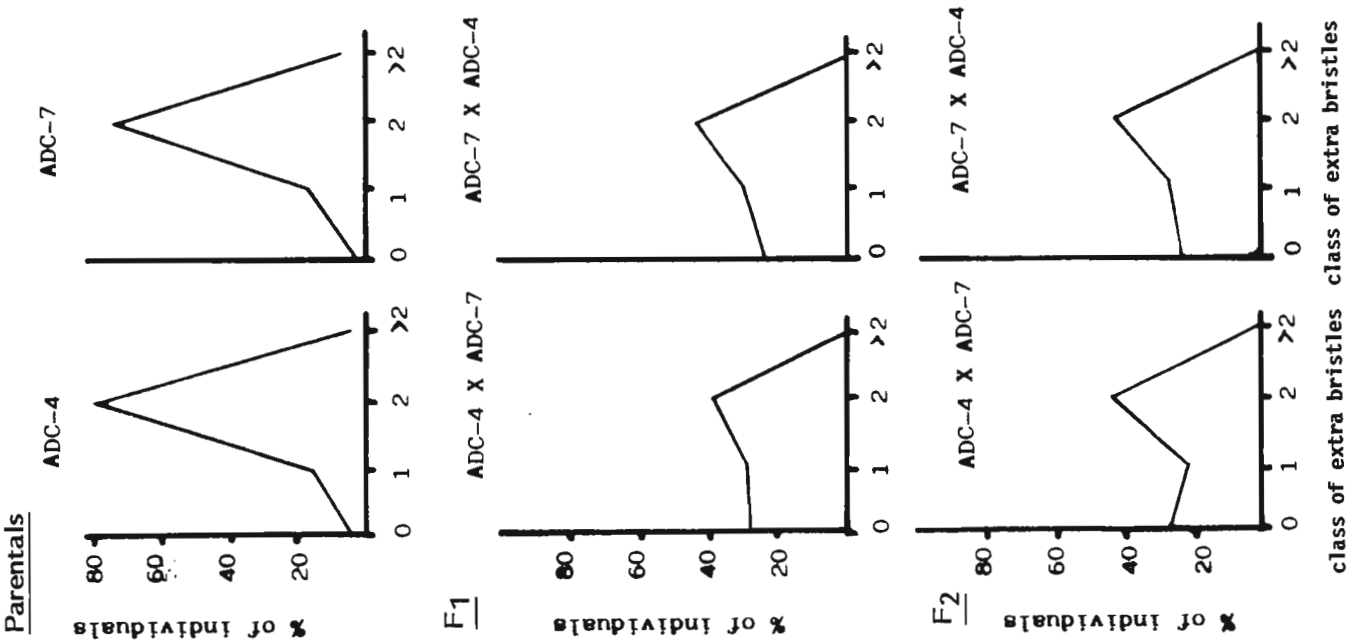

Figure 2. Extra bristles distribution (in percent of individuals of each class) of ADC-4 X ADC-7 and ADC-7 X ADC-4 crossings.

Table 1. Extra bristles distribution (in % of individuals) of each chromosomic combination, in ADC-4 and ADC-7.

Classes of	ADC-4								ADC-7							
Extra Bristles	BBB	BBA	BAB	ABB	BAA	ABA	AAB	AAA	BBB	BBA	BAB	ABB	BAA	ABA	AAB	AAA
Class 0	8.9	94.4	47.8	55.0	93.9	98.3	79.4	97.8	15.6	92.2	65.6	47.8	92.8	85.0	89.4	95.0
Class 1	33.9	4.4	24.4	23.9	4.4	1.7	10.0	2.2	33.3	7.2	16.1	20.5	7.2	10.6	8.3	3.3
Class 2. Sel.p.	57.2	1.1	20.0	19.4	1.7	0.0	8.9	0.0	45.0	0.6	15.0	15.0	0.0	4.4	2.2	0.6
Unsel.p.	0.0	0.0	4.4	0.0	0.0	0.0	0.6	0.0	0.6	0.0	2.8	6.7	0.0	0.0	0.0	0.6
Classes > 2	0.0	0.0	2.8	1.7	0.0	0.0	1.1	0.0	5.6	0.0	0.6	10.0	0.0	0.0	0.0	0.6
Numerical STP:	AAA	ABA	BBA	BAA	AAB	ABB	BAB	BBB	AAA	BAA	BBA	AAB	ABA	BAB	ABB	BBB
Positional STP:	BAB	AAB	BBA	BAA	ABB	BBB			AAA	ABB	BAB	BBB	BBA	AAB	ABA	

B = line chromosome; A = Oregon chromosome; sel.p. = selected phenotype; unsel.p. = unselected phenotype.

All the lines are carrying in all their chromosomes some genetic factors for bristle number increase (Table 1). The numerical STP analysis shows that the chromosome 3 is the main one responsible for this increase in both lines. AAB do not differ significantly from BBA: chromosome 3 increases the bristle number as chromosome 1 and chromosome 2 do. Chromosome 1 in ADC-4 and chromosome 2 in ADC-7 are the next chromosomes in importance to increase the number of bristles. There are strong interactions between the three chromosomes. These data confirm the results of other authors (i.e., Rubio & Albornoz 1982).

Some chromosomic combinations are discanalized: they have a high proportion of individuals in classes > 2, and they have two extra bristles in unselected positions (Table 1). Positional STP analysis shows that chromosome 2 is essential for the canalization keeping in ADC-4. Chromosome 1 is the main one responsible for this canalization in ADC-7, followed by chromosome 2. Obtained canalization of these lines have a polygenic or multifactorial basis.

As other authors say (Fraser 1967; Rendel 1976), lines have two systems determining the obtained phenotype. One system increases bristle number; another system maintains the canalization. First system is mainly in chromosome 3; the second one is mainly in chromosomes 1 and/or 2. Nevertheless, we cannot find any large effect gene in the two systems, as above cited authors do.

References: Fraser, A.S. 1967, Genetics 57:919-934; Kearsey, M.J. & K. Kojima 1967, Genetics 56:23-37; Rendel, J.M. 1976, Genetics 83:573-600; Rubio, J. & J. Albornoz 1982, Rev. Real Acad. Cien. Ex., Fis. y Nat., Madrid. Tomo LXXVI, Cuaderno 4:773-802.

Garcia-Vazquez, E. and J. Rubio. University of Oviedo, Spain. Phenotypic variation of the bristles between Asturian populations of **D.melanogaster.**

The character extra bristles is studied in this work. These extra bristles, both dorsocentral and scutellar, are normally canalized in nature for the phenotype that we can see in Figure 1. The hidden variation for this character is easily revealed using isofemale lines (Parsons & Hosgood 1967); some individuals with extra bristles appear in most of the lines.

Eleven Asturian natural populations have been examined, analysing the percent of individuals of each line which have extra bristles. Isofemale lines are jointed in three classes: Class I, well canalized (less than 5% of individuals have extra bristles); class II, (5-20% of individuals with extra bristles); and class III (more than 20%).

Likewise the isofemale lines are classified by their positional definition of extra bristles, as DC (more than 75% of extra bristles are dorsocentral), SC (more than 75% of extra bristles are scutellar), and DC/SC (extra bristles in both dorsocentral and scutellar positions).

The sites of capture are in the map of Figure 2. Four populations were captured along the river valley Paramo-Nalon (Proaza, Trubia, Sandiche and Soto del Barco); four along the Asturian coast (Aviles, Somio, Villaviciosa, Celorio); and three are central populations, captured near Oviedo city (Los Areneros A, Los Areneros B, La Granda). In each line we have examined 60 individuals of each sex.

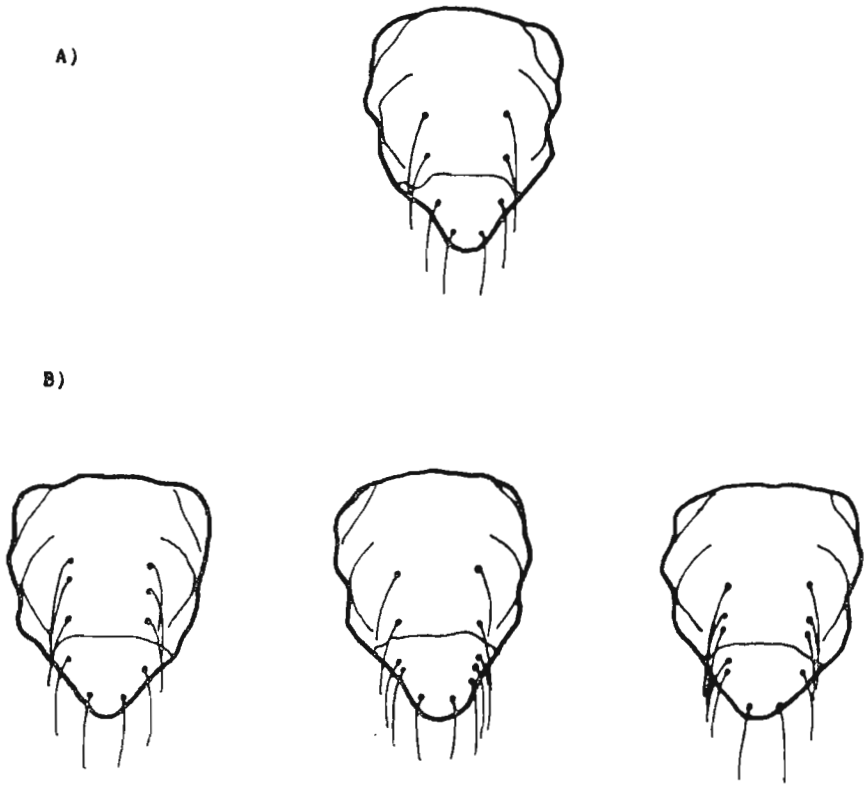

Figure 1. Normal phenotype of dorsocentral and scutellar bristles, canalized in natural populations.

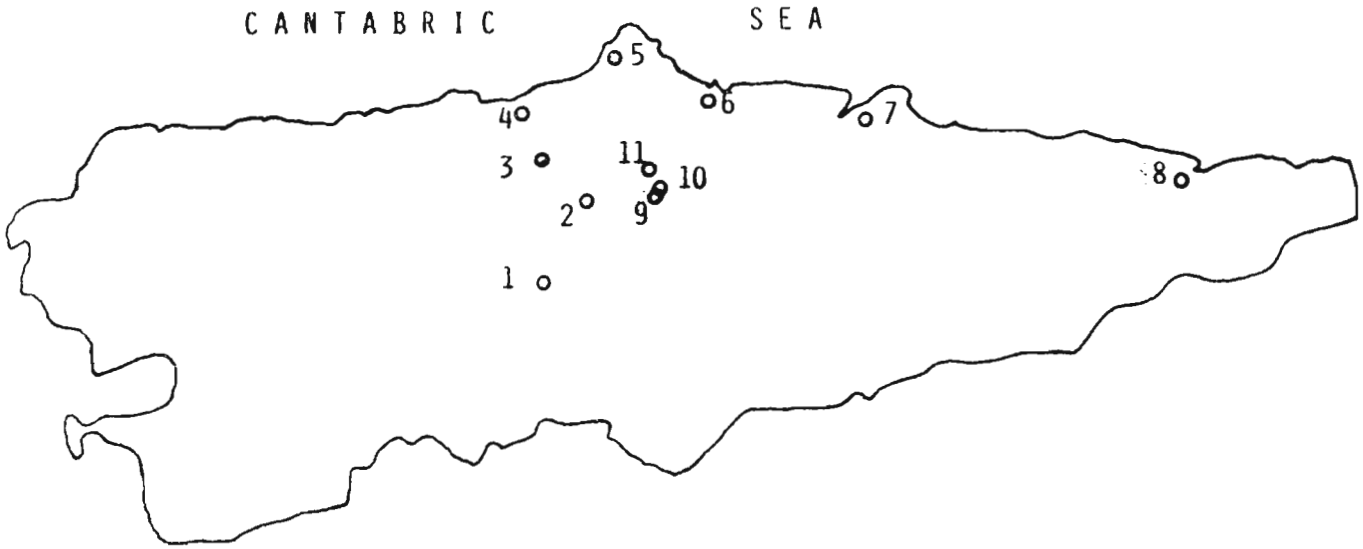

Figure 2. Capture sites in Asturias. VALLEY: 1-Proaza, 2-Trubia, 3-Sandiche, 4-Soto del Barco. COAST: 5-Aviles, 6-Somio, 7-Villaviciosa, 8-Celorio. CENTRAL: 9-Los Areneros A, 10-Los Areneros B, 11-La Granda.

Table 1. Percent of isofemale lines of each extra bristles positional class. NL = number of lines in classes II and III. Lines of class I are not positionally classifiables. $X^2_{20\ df}$(global)=23.710 N.S.

Populations	Class DC	Class SC	Class DC/SC	NL
Proaza	69.0	10.3	20.7	29
Trubia	73.7	17.8	10.5	19
Sandiche	70.2	14.9	14.9	47
Soto del Barco	71.4	25.0	3.6	28
Aviles	68.2	20.4	11.4	44
Somio	66.7	23.1	10.2	39
Villaviciosa	80.0	15.0	5.0	20
Celorio	80.0	3.3	16.7	30
Los Areneros A	74.0	5.2	20.8	77
Los Areneros B	71.4	12.1	16.5	91
La Granda	77.8	13.3	8.9	45

Table 2. Percent of isofemale lines of each extra bristles numerical class. NT = number of lines.

Populations	Class I	Class II	Class III	NL
Proaza	35.5	51.1	13.1	45
Trubia	26.9	57.7	15.4	26
Sandiche	36.5	47.3	16.2	74
Soto del Barco	42.9	40.8	16.3	49
Aviles	36.2	50.7	13.1	69
Somio	61.0	37.0	2.0	100
Villaviciosa	58.3	35.4	6.3	48
Celorio	28.6	66.7	4.7	42
Los Areneros A	29.3	54.1	16.5	109
Los Areneros B	29.4	62.8	7.8	129
La Granda	55.9	40.2	3.9	102

Between valley populations $X^2_{6\ df}$=2.521 N.S.
Between coast populations $X^2_{6\ df}$=25.211***
Between central populations $X^2_{4\ df}$=28.366***

$X^2_{20\ df}$(global) = 70.712***

Populations are not different for the extra bristles position (Table 1). Most of the lines have dorsocentral extra bristles; some lines of each population have only scutellar, and some of them, both scutellar and dorsocentral extra bristles. All populations have some variation in their isofemale lines for the extra bristles position, but this variation is the same in every populations, or at least there are not statistical differences.

Populations are significantly different for the extra bristle number of each line (Table 2). Classifying populations by their geographic situation, we find that both coastal and central ones differ significantly to their isofemale line variation; while, the valley populations are homogeneous, because their isofemale lines have the same distribution (X^2 = 2.52, N.S.).

There is a different rate of migration between populations, greater in the valley than in the coast or in the central belt of Asturias, because the dispersal along the river valley is favoured by the predominant N-S wind direction, and by the human agricultural transport, through main roads and railway. By this reason, we think that the main factor responsible for the homogeneity of the valley populations is the migration.

References: Parsons, P.A. & S.M.W. Hosgood 1967, Genetica 38:328-339.

Ghosh, A.K. and A.S. Mukherjee. University of Calcutta, India. Catalase activity in trisomy for the whole left arm of third chromosome in **Drosophila melanogaster.**

Previous studies on segmental aneuploids provided with some evidence toward autosomal dosage compensation in Drosophila which raised controversy over the significance of dosage compensation (Baker & Belote (1983). Autosomal structural genes which produce gene-dosage effect in eukaryotes have been interpreted to mean that "such eukaryotic loci are constitutive and the number of genes is a rate limiting factor in the total process of gene expression" (Carlson 1972). Segmental aneuploids of chromosomal arms were used to locate the structural loci of a particular gene by gene dosage effect. Devlin et al. (1982) showed that in trisomy for the whole left arm of 2nd chromosome in **Drosophila melanogaster**, a few autosomal genes are able to synthesize equal level of product with respect to euploid sib.

The present investigation aims at the assessment as to whether compensation of gene dosage occurs in case of catalase enzyme in trisomy for the whole left arm of 3rd chromosome in **Drosophila melanogaster.** Using segmental aneuploids, Lubinsky & Bewley (1979) established that the catalase structural gene is located in the region 75D-76A of 3L in **Drosophila melanogaster.** They showed that the catalase activity is 1.5 times higher in stock with a duplication for the region 75D-76A, in comparison with euploid level. We were interested to know the specific activity of catalase in trisomy for the whole left arm of third chromosome in **Drosophila melanogaster.** C(3L)VGI ru st;F(3R)VDI e^s/F(3R)VDI e^s/F(3R)VDI e^s and Oregon-R^+ stocks were used to generate the trisomy for 3L progeny (lethal in late pupal stage) following the protocol of Fitz-Earle and Holm (1977).

Table 1. Catalase activity in Trisomy-3L, Compound-3L and Oregon-R^+ stocks of **Drosophila melanogaster.**

Enzyme	Trisomy-3L(T) *Mean ± S.E.	Oregon-R^+(D) *Mean ± S.E.	Compound-3L(C) *Mean ± S.E.	T/D value	T/C value
Catalase E.C.1.11.1.6.	2.889 ± 0.226	2.440 ± 0.194	2.843 ± 0.491	1.18	1.02

*Specific activity: milimole H_2O_2 decomposed/minute/ug of total protein.

Catalase activity was measured in the 3rd instar larvae of trisomy-3L, Compound-3L and Oregon-R^+ stocks following the method used by Lubinsky & Bewley (1979). A unit of catalase activity is defined as 1 milimole H_2O_2 decomposed/minute assuming the molar extinction coefficient of 70.67 at 230 nm. Total protein was determined following the method of Lowry et al. (1951).

We have observed that the specific activity of catalase enzyme is almost same in trisomy for 3L, Compound-3L and Oregon-R^+ stocks of **Drosophila melanogaster** (see Table 1).

From this observation, it is clear that the catalase shows a compensatory level of activity in whole arm trisomic condition with the control Oregon-R^+ and compound-3L stocks. The specific activity of catalase in segmental aneuploids is 1.5 times higher than that of euploid level (Lubinsky & Bewley 1979). Whether duplication of the whole left arm of third chromosome is necessary for catalase gene to be compensated or not, awaits further investigation.

References: Baker, B. & J. Belote 1983, Ann. Rev. Genet. 17:345; Carlson, P.S. 1972, Mol. Gen. Genet. 114:273; Devlin, R.H., D.G. Holm & T.A. Griglitti 1982, PNAS USA 79:1200; Lubinsky, S. & G.C. Bewley 1979, Genetics 91:723; Fitz-Earle, M. & D.G. Holm 1977, Proc. 15th Int. Cong. Entom. Wash DC (White, ed.), Entomol. Soc. of Amer., p. 146; Lowry, O.H., N.J. Rosenbrough, A.L. Farr & R.J. Frandall 1951, J. Biol. Chem. 193:265.

Ghosh, A.K. and A.S. Mukherjee. University of Calcutta, India. Replication behaviour of trisomy for 2L and 3L arm in **Drosophila melanogaster.**

DNA replication in polytene chromosomes of different species of Drosophila has been examined autoradiographically by various groups of workers. In Drosophila polytene chromosomes a general pattern of synchronous replication among different sites of the same chromosome and between different chromosomes has been reported (Plaut et al. 1966), with the exception of the X-chromosome of the male. However, asynchronous relication is known to exist in Drosophila polytene nuclei, viz., under replication of the nucleolus organizer (Laird 1973) and faster replication of male-X (Lakhotia & Mukherjee 1970). In **Drosophila pseudoobscura** there is an asyncrhony in replication between the two autosomal arms (2nd and 3rd) though both the autosomes are in diploid condition. Nothing precisely is known about the regulation of these cases of asynchronous replication, although much work has been done on the significance of such asynchrony (Lakhotia & Mukherjee 1970; Laird 1973). In **Drosophila melanogaster** polytene chromosome, in which DNA replication has been studied in detail (Lakhotia & Mukherjee 1970), no asynchrony between nonhomologous chromosomes has been reported.

The aim of the present investigation on replication is to find out:

1. Whether asynchrony of replication exists between trisomic autosomal arms (2L and 3L) and other disomic chromosomal arms (2R and X ♀) where the trisomic arm contains 1.5 times more DNA in comparison with non-duplicated arms of the same nuclei?

2. If there is an asynchrony of replication, when does this occur, i.e., in the initial or terminal phase of the S?

C(2L)SHI$^+$; F(2R)bw, C(3L)VGI ru st; F(3R)VDI e^s, and Oregon R^+ stocks were used to generate the trisomy for 2L and trisomy for 3L progeny following the protocol of Fitz-Earle & Holm (1977).

For ^{3}H-thymidine autoradiography, salivary glands were dissected out from late third instar larvae of trisomy 2L and trisomy for 3L stocks and processed according to Lakhotia & Mukherjee (1970). To find out whether extra DNA content in the trisomic arms have any influence on its replication behaviour, we

Table 1a. ^{3}H-thymidine incroporation. Frequency of labelled sites on 2L arm, 2R and female-X chromosome in trisomy for 2L stock.

Chromosomal arms	>50%	>25%	<25%
Trisomy-2L	0.36	0.22	0.42
Disomic-2R	0.32	0.24	0.44
Female-X	0.29	0.23	0.48

Table 1b. Frequency of labelled sites on T3L arm, 2R, and X chromosome (female) in trisomy for 3L stock.

Chromosomal arms	>50%	>25%	<25%
Trisomy-3L	0.42	0.25	0.32
Disomic-2R	0.32	0.24	0.44
Female-X	0.33	0.24	0.43

have studied the patterns of replication (DD-1C-2C-3C-3D = 2D-1D), frequency of labelled sites on trisomic arms and compared with the reference segments of 2R (60A-56F) and female-X (1A-11A).

Data reveal that 36% of the total studied (69 subsegments) sites in T2L have labelling frequency greater than 50%, similarly 32% of the total studied sites (24 subsegments, 56F-60A) in 2R have labelling frequency greater than 50% and the labelling frequency greater than 25% and less than 25% have been presented in Table 1a. In trisomy for 3L nuclei, 42% of the total studied (68 subsegments 61A-80A) sites have labelling frequency greater than 50% whereas 33% of the sites in 2R have labelling frequency greater than 50% (Table 1b).

So there is no significant difference in the frequency of labelled sites in trisomic autosomal arms (2L and 3L) and disomic non-homologous chromosomal arms. Besides, it is evident from our observation that no asychrony exists in initial or in late phase of replication pattern between the T2L/T3L and disomic arms.

References: Fitz-Earle, M. & D.G. Holm 1977, Proc. 15th Int. Cong. Entom. Wash DC (White, ed.), Entom. Soc. of Amer. p. 146; Laird, C.D. 1973, Ann. Rev. Genet. 7:177; Lakhotia, S.C. & A.S. Mukherjee 1970, J. Cell. Biol. 47:18; Plaut, W., D. Nash & T. Fanning 1966, J. Mol. Biol. 16:85.

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Characterization of nucleolar chromatin thread (NCT) in salivary gland cells of **Drosophila melanogaster.**

In Drosophila, as also in various tissues of many eukaryote systems, the nucleolus manifests as a round or oval structure organized by a specific region of the genome commonly known as nucleolar organizer. In many systems, as in Drosophila larval salivary glands, the nucleolus is a very prominent body often attached to the chromocentric region through a bundle of thread-like structure. The thread-like connections seem to penetrate into the nucleolar mass and form different patterns of distribution in the nucleolar matrix.

Different morphological conformations of nucleolar chromatin threads in the form of granules, blocks and threads in the nucleoli of **Drosophila hydei** have been reported earlier (Ghosh & Mukherjee 1982). Such configurations are not unique to the species of **D.hydei** but are found in other species as well (Barr & Plaut 1966a, b; Rodman 1968, 1969; Ghosh 1984).

In the present investigation an attempt has been made to characterize the nature of the nucleolar chromatin thread (NCT) of **D.melanogaster.** For this purpose, aceto-carmine aceto-orcein staining, Feulgen staining, Acridine orange staining techniques as well as ^{3}H-thymidine and ^{3}H-uridine autoradiography have been performed.

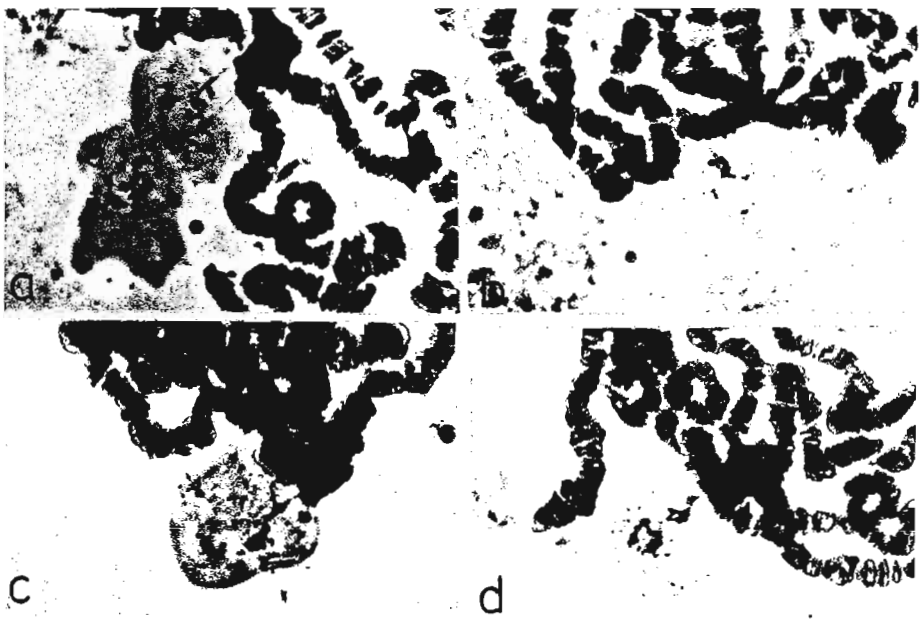


Figure 1. Photomicrographs showing four different types of nucleolar chromatin threads (NCTs) of **D.melanogaster.** Types 1, 2, 3 and 4 are represented by a, b, c and d, respectively.

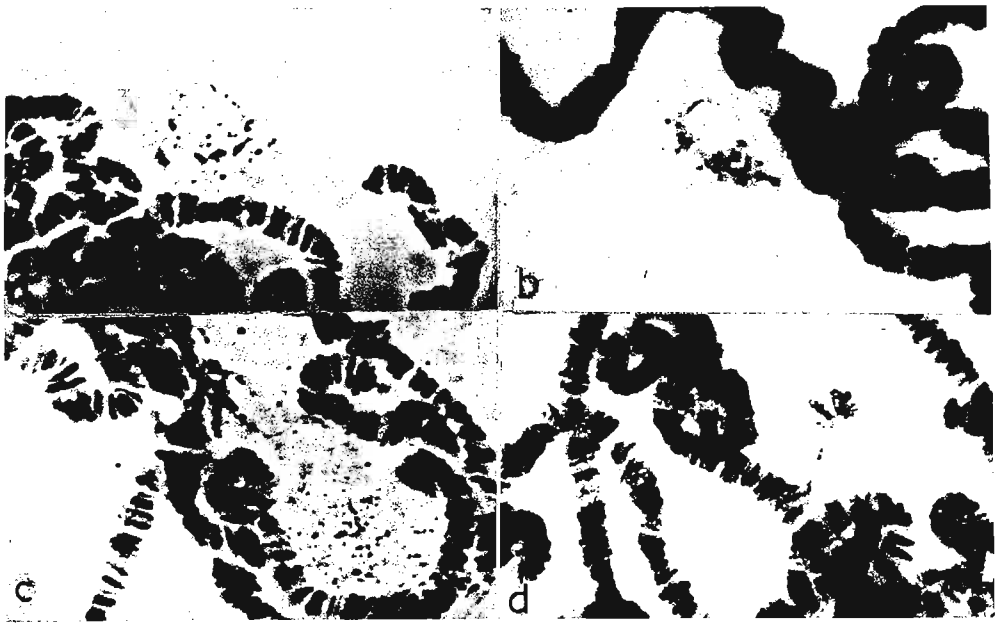


Figure 2. Photomicrographs displaying four different NCT types of **D.melanogaster** after Feulgen staining.

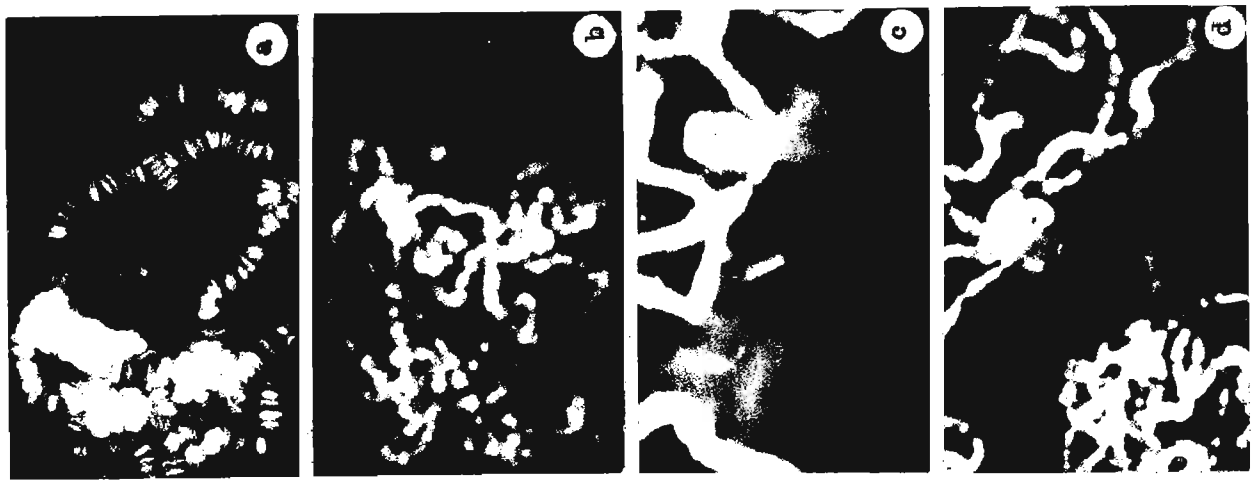


Figure 3. Photomicrographs of four different types ofNCTs of **D.melanogaster** stained with acridine orange.

Observations reveal that different configurations of NCTs as are found in **D.hydei** are also present in **D.melanogaster** (Fig. 1). However, the type 4 of **D.melanogaster** is not so much complex as they are in **D.hydei.** All the 4 NCT types of **D.melanogaster** are Feulgen positive (Fig. 2) which remain embedded in the Feulgen negative nucleolar matrix. Fluorescence staining with Acridine orange (AO) also reveals that all 4 NCT types exhibit bright yellow fluorescence while the nucleolar matrix shows red fluorescence (Fig. 3).

Data on ^{3}H-uridine and ^{3}H-thymidine labelling reveal that all the 4 different NCT types incorporate ^{3}H-uridine and ^{3}H-TdR. Therefore, all the 4 NCT types of **D.melanogaster** are transcriptionally and replicationally active. But the detailed analysis of the data suggest that the labelling density of ^{3}H-uridine and that of ^{3}H-thymidine as evident from the actual count of silver grains are not similar for 4 NCT types. This finding suggests that the 4 NCT types may be under different state of activity and the filamentous/branched network thread like nucleolar chromatin structures (that types 3 and 4) are metabolically most active.

References: Barr, H.J. & W. Plaut 1966a, J. Cell Biol. 31:10A; _____ 1966b, J. Cell Biol. 31:C17-C22; Ghosh, M. 1984, DIS 60:112-113; Ghosh, M. & A.S. Mukherjee 1982, Cell & Chrom. Res. 5(1):7-22; Rodman, T.C. 1968, Chromosoma 23:271-287; _____ 1969, J. Cell Ciol. 42:575-582.

Ghosh, M. and A.S. Mukherjee. University of Calcutta, India. Nucleolar chromatin thread in different mutant stocks of **Drosophila hydei.**

The nucleolus of larval salivary glands of Drosophila contains nucleolar chromatin structure (NCT) which have various conformations. It has been suggested that the various conformations are functionally related (Ghosh & Mukherjee 1982). The functional relation of these conformations have been further examined in different mutational forms in which the number of nucleolar genes is different.

The different stocks of **Drosophila hydei** used for this purpose were y m ch/Y (attached-X/Y), bb vg P, KOM^{FP} 290/2 and KOM^{TKS} 697/16. The number of nucleolar organizers and rDNA cistrons present in these stocks have been reported by Kunz & Schafer (1976) from their hybridization experiments.

The purpose of this investigation has been to find out whether the configurations of different chromatin thread morphotypes reported earlier by Ghosh & Mukherjee (1982) and their activity in terms of ^{3}H-uridine incorporation are related to the number and/or distribution of the nucleolar organizers or rDNA cistrons.

Results on the observations on the morphology of the nucleolar chromatin types in different mutant strains reveal that except y m ch/Y, all the 4 types of NCTs are present in the other three mutant stocks of **D.hydei.** Results are presented in Table 1. The females of y m ch/Y, KOM^{FP} 290/2 and KOM^{TKS} 697/16 possess attached-X/Y chromosomes (Kunz & Schafer 1976). As reported the X-chromosomal NORs are absent and only 2 Y-chromosomal NORs are present in the females of these strains. On the other hand, in the mutant strain, bb vg p, certain parts of NOR are deleted in both X and Y chromosomes.

Data on the morphology of NCT types in these mutant strains reveal that there is no direct correlation between the more active NCT types (i.e., types 3 and 4) and the nucleolar organizer(s) (Table 1). It is also evident from the data that the presence of the Y chromosome appears to induce considerably more activity than its absence as the frequencies of NCT types 3 and 4 are higher in both males and females of the mutant stocks possessing Y chromosome compared to the females of bb vg P and wild type where Y chromosome is absent. It is also interesting to note that the frequencies of NCT types 3 and 4 are comparatively lower where the intact Y is absent.

Data on the mean ^{3}H-uridine grain number in the nucleoli with different NCT types for the four different mutant stocks and wild type are presented in Table 2. Data show that there is a clear one-to-one relationship in the mean grain number in the corresponding NCT types of the two sexes, regardless of the dosage of the NOR(s).

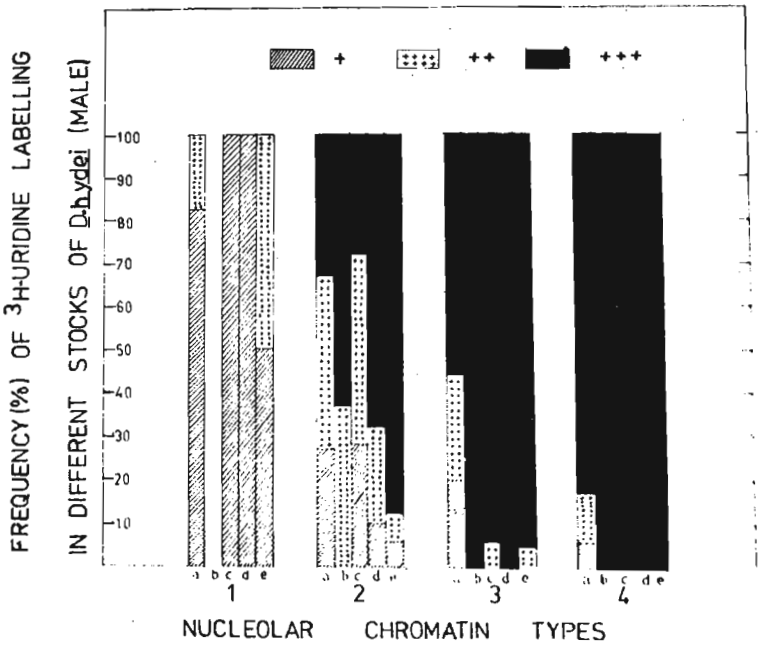
When the different intensity classes are plotted histographically, the results seem to indicate that in both male and female of all the genotypes, the frequency of low labelling intensity classes decreases from type 2 to 4 and that of the high labelling intensity classes which appear first in the type 2, increases from type 2 to 4. The pattern of labelling intensity appears to be similar in the two sexes. The pattern of distribution of the intensity classes presented in Figures 1a and b also indicate a differential labelling profile specific for a particular genotype.

It is evident from the results that the source of rDNA cistrons (X chromosomal or Y chromosomal) seems to be important for the frequency of different types of NCT. For example, it has been observed that in the presence of Y chromosome when the number of NOR is equal the differences in the intensity classes of labelling among them is minimum. This observation seems to reiterate the significance of the quality rather than the quantity of the rDNA cistrons. This interpretation is supported by Endow & Glover (1979) who proposed that in the diploid cells one of the two sets of NOR might be metabolically active. This is

Table 1. Nucleolar chromatin thread (NCT) types and their frequencies (in %) in wild type and different mutant stocks of **Drosophila hydei.**

	No. of NOR(s)		NCTs in males				NCTs in females			
Stocks	m	f	1	2	3	4	1	2	3	4
Wild type	3	2	90 (21.68)	242 (58.29)	45 (16.84)	38 (9.15)	144 (32.28)	129 (28.91)	151 (33.85)	22 (4.93)
Y m ch/Y (attached-X/Y)	3	2	--	224 (43.75)	267 (52.15)	21 (4.10)	--	215 (39.80)	330 (59.57)	9 (1.62)
bb vg P	3(-)	2(-)	117 (30.23)	125 (32.29)	141 (36.43)	4 (1.03)	75 (17.44)	246 (57.20)	55 (12.79)	54 (12.55)
KOM^{FP} 290/2	2	1	76 (17.16)	143 (32.27)	213 (48.08)	11 (2.48)	115 (30.10)	116 (30.37)	129 (36.33)	12 (3.14)
KOM^{TKS} 697/16	2	1	40 (12.08)	154 (46.53)	131 (39.58)	6 (1.81)	178 (36.63)	185 (38.10)	101 (20.78)	22 (4.53)

(-) indicates some portion deleted in the Nucleolar Organizer Region (NOR). m = male, f = female.



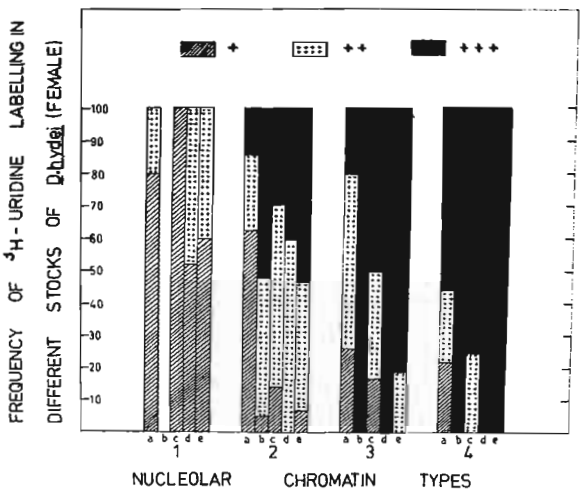


Figure 1 (A,B). Histogram showing frequency of ^{3}H-uridine labelling patterns (different intensity classes) in relation to different NCT types in **(A)** males and **(B)** females of wild type and mutant stocks of **Drosophila hydei** with different numbers of NOR(s). The numbers a, b, c, d and e represent stocks: wild type, y m ch/Y, bb vg P, KOM^{FP} 290/2 and KOM^{TKS} 697/16, respectively.

Table 2. ^{3}H-uridine induced grain number per unit square area over the NCT types in different stocks of **D.hydei.**

	NCTs in males				NCTs in females			
Stocks	1	2	3	4	1	2	3	4
Wild type	20	48	43	42	27	37	45	58
y m ch/Y (attached-X/Y)	--	67	61	75	--	75	57	62
bb vg P	8	75	71	93	9	82	93	131
KOM^{FP} 290/2	20	94	59	64	19	64	42	63
KOM^{TKS} 697/16	30	70	86	68	26	63	84	69

also revealed by the analysis of the relationship between the number of rDNA cistrons to the number of NOR in the wild type and different stock of **D.hydei.** Here, the number of actively transcribing rDNA cistrons are often under-represented with respect to the presumed number of rDNA cistrons reported by Schafer & Kunz (1976).

Therefore, from this observation it is suggested that the four NCT types are different forms of metabolic expression of the DNA sequences present in the rDNA cistrons.

References: Endow, S.A. & D.M. Glover 1979, Cell 17:597-605; Ghosh, M. & A.S. Mukherjee 1982, Cell & Chrom. Res. 5(1):7-22; Kunz, W. & U. Schafer 1976, Genetics 82:25-34; Schafer, U. &. W. Kunz 1976, Heredity 37(3):351-355.

Ghosh, M. and A.S. Mukherjee. University of Calcutta, India. Nucleolar chromatin thread (NCT) in different mutant strains of **Drosophila melanogaster.**

A large number of mutations have been found in **Drosophila melanogaster** which alter the structure and/or function of the nucleolar organizer (NOR). An investigation has been made to find out the relation between the structural conformations of nucleolar chromatin thread (NCT) and their function(s).

In the wild type strain of this species there are 2 NORs in the genome, one in the X chromosome and the other in the Y chromosome. Thus both male (XY;2A) and the female (XX;2A) possess 2 NORs. In both sexes, the nucleolar chromatin appears to show four morphological types.

In the present investigation wild type as well as several mutant strains, viz., y sn^3 bb (some portions from the NOR are deleted from both X and Y chromosome), y f :=/Y (attached-X/Y) and XY, T(X; y$^+$ Y^L) 11A and y f :=/Y, y w^a, w^a, y and normal [obtained from crosses between In(1) sc^4 and In(1) sc^8] have been used. The number of NOR(s) present in males and females of all these mutant strains are presented in Table 1. Genotypes bearing different number of NOR(s) have been generated using appropriate crosses (Fig. 1).

Cytological preparations reveal that all the four NCT types are present in the mutant strains studied. The four morphotypes have been grouped as Groups I and II, on the basis of low and high activity in transcription (Ghosh & Mukherjee 1982). From the data it appears that the frequency of Group I is

Table 1. Nucleolar chromatin thread and their frequencies (in percent) in wild type and different mutant stocks of **Drosophila melanogaster.**

Stocks	No. of NOR(s) m	No. of NOR(s) f	NCT types 1 and 2 (Broad Group I) m	NCT types 1 and 2 (Broad Group I) f	NCT types 3 and 4 (Broad Group II) m	NCT types 3 and 4 (Broad Group II) f
Oregon	2	2	66 (74.15)	76 (71.16)	23 (25.85)	30 (28.31)
y sn^3 bb	2(-)	2(-)	85 (75.88)	129 (73.71)	27 (24.12)	46 (26.29)
y w^a	1	1	184 (68.65)	172 (69.64)	84 (31.35)	75 (30.36)
y	2	2	180 (66.16)	240 (62.34)	82 (33.84)	145 (37.66)
w^a	2	2	144 (56.25)	190 (71.16)	112 (43.84)	7 (28.74)
Normal	3	3	240 (78.43)	273 (72.48)	6 (21.67)	106 (27.52)
yf:=/Y(attached-X/Y) and XY	2	3	132 (73.60)	116 (78.26)	47 (26.40)	35 (21.74)
T(X; y^+ Y^L) 11a and yf:=/Y	3	3	291 (76.53)	225 (82.79)	68 (23.47)	49 (17.82)

(-) indicates some portion deleted in the Nucleolar organizer region (NOR).
m = male, f = female.

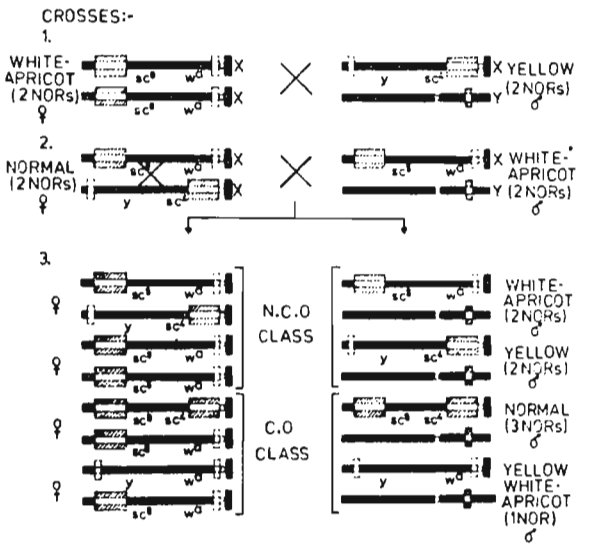


Figure 1. Diagrammatic representation of crosses designed for the synthesis of stocks of **D.melanogaster** containing variable number of NORs. The progenies are of four different types: two cross over classes and two non-cross over classes. The females are either phenotypically normal (with 2 NORs or 3 NORs and white apricot females with 1 or 2 NORs). The different types of males are white apricot (2 NORs), yellow (2 NORs), normal (3 NORs) and yellow white apricot (1 NOR).

always higher than Group II in both males and females of all the strains studied. Data reveal that Group I of both males and females forms 70-74% in wild type, 74-76% in y sn^3 bb, 73-78% in y f :=/Y (attached-X/Y), and itis 76-82% in T(X; y^+ Y^L) 11A andy f :=/Y (attached-X) (Table 1).

When the distribution of the frequencies for different NCT types in mutant strains of **D.melanogaster** containing different numbers of NOR(s) is compared, it is evident that the increase in the number of NOR is not concurrent with an increase in the frequency of the NCT types 1 and 2 (Group I) in either sex. However, there appears to be a threshold level in the activity of the NOR. This is borne out by the fact that the number of NOR is slightly less than 2, e.g., in y sn^3 bb which has a partial deletion of NOR, the frequency of NCT types is also not changed.

From the results it seems that the number of NOR does not have any direct influence on the variation of NCT types among the wild type and different mutant strains of **D.melanogaster.** The difference in the frequency of NCT morphotypes between male and female may be due to difference either in number of rDNA cistrons in the X and Y NOR, or may be due to differential activation of the cistrons.

The above mentioned findings on the activity of the rDNA lead to suggest that all the cistrons are not functionally active in the diploid cells. As pointed out by Endow & Glover (1979), only half the number of rDNA cistrons are active in producing the rRNA in the polytene nuclei. The data presented here on the distribution of the four morphotypes in genetically different strains of **D.melanogaster** also corroborate these findings. The data show that as long as a minimum of one complete set of rDNA cistrons is present, i.e., 130 cistrons in **D.melanogaster**, the activity is maintained at a normal level. This level is maintained and does not change considerably with the increase in the dosage of the NOR or rDNA cistrons. This implies, therefore, that the ribosomal genes maintain dosage compensation in both sexes.

References: Endow, S.A. & D.M. Glover 1979, Cell 17:597-605; Ghosh, M. & A.S. Mukherjee 1982, Cell & Chrom. Res. 5(1):7-22.

Graf, U. Swiss Federal Institute of Technology and University of Zurich, Schwerzenbach, Switzerland. Temperature effect on mwh expression in the wing somatic mutation and recombination test in **Drosophila melanogaster.**

The wing somatic mutation and recombination test (SMART) for the detection of genotoxic activity of chemicals makes use of the two wing cell markers mwh (multiple wing hairs, 3-0.0) and flr (flare, 3-39.0). The trans-heterozygous configuration of these markers allows the registration of somatic mutations (gene mutations, deletions, etc.) and of mitotic recombination. Mitotic recombination between the proximal marker and the centromere leads to the formation of twin spots on the wings of adult flies after treatment of larvae with a given compound. Single spots may be the consequence of somatic mutation or mitotic recombination between the two markers. Depending on the time of clone induction during larval development, smaller or larger spots are produced. The size of each spot can be determined by counting the number of cells expressing the mutant phenotype. The classification of small mwh spots consisting of only one or two cells poses some problems. In our routine scoring of wings only those spots are classified as "true" mwh spots in which at least one wing cell shows three or more hairs instead of only one hair as in wild type. However, quite frequently single cells are encountered with two hairs of more or less equal size. These "false" spots--which are probably mainly due to developmental disturbances in trichome pattern formation--are not included in our scoring. The terms "true" and "false" used here refer only to the phenotype of the mwh spots.

In the course of our investigations, we discovered that the frequency of the mwh spots, especially the false ones, depends on the temperature at which the larvae and pupae are reared. Therefore, an experiment was performed as follows. Two different crosses were set up: (1) Cross A: y; mwh jv females and y; Dp(1;3)sc^{J4}, flr/TM1, Méri sbd^{2} males, and (2) Cross B: mwh females and flr^{3}/TM3, Ser males.

From each cross eggs were collected for 8 h on standard cornmeal-sugar-agar-yeast medium in two bottles at 25°C. After the egg collection period one bottle from each cross was kept at 25°C, the other one at 29.5°C. No further treatment was given to the larvae. All the eclosing flies were collected and stored in 70% ethanol. Approximately 40 wings from each series were mounted and scored under a compound microscope at 400x magnification. All the spontaneous spots were counted and classified according to the two categories true and false as explained above. The true spots were mainly small mwh single spots (1 or 2 cells) with a few larger single and twin spots. The false spots were almost exclusively single cells with two hairs. The data are given in Table 1.

Table 1. Spontaneous mwh spots on wings of flies derived from two different crosses and reared at two different temperatures.

Cross	Progeny	Temp. (°C)	Wings (n)	True spots n	True spots f	False spots n	False spots f
A							
mwh x	mwh +/+ flr	25	40	19	0.48	398	9.95
flr/TM1		29.5	40	98	2.45	2347	58.68
	mwh/TM1	25	36	10	0.28	27	0.75
		29.5	44	10	0.23	49	1.11
B							
mwh x	mwh +/+ flr^{3}	25	40	10	0.25	7	0.18
flr^{3}/TM3		29.5	40	35	0.88	150	3.75

n = number; f = frequency.

The frequencies of both categories of spots are increased with the higher temperature in all three genotypes studied. This shows that higher temperature affects the expression of mwh spots quite drastically. However, in general this effect is far more pronounced for the false spots as compared with the true spots. More important, the increase is most pronounced in the trans-heterozygous mwh +/+ flr progeny of cross A. These flies carry the sc^{J4} duplication (which contains a y^{+} gene) at the tip of the flr chromosome in the immediate proximity of the mwh^{+} locus. This is not the case for the trans-heterozygous progeny of cross B. Apparently, the sc^{J4} duplication has an influence on mwh expression. This is further corroborated by the fact that no such influence is seen in the inversion-heterozygous mwh/TM1 flies. Furthermore, a comparison of the two crosses shows that flies derived from cross B have much lower frequencies of false spots. Although these false spots are not included in our scoring data in standard SMART experiments, they can be quite disturbing in the screening of the wings. Therefore we now prefer to use larvae derived from cross B for our experiments. In addition, the data presented above demonstrate clearly that strict temperature control is essential in wing SMART experiments in order to obtain reproducible and quantitatively comparable results.

Acknowledgement: Supported by Swiss National Science Foundation grant no. 3.657-0.84.

References: Graf, U., H. Juon, A.J. Katz, H.J. Frei & F.E. Wurgler 1983, Mutation Res. 120:233-239; Graf,U., F.E. Wurgler, A.J. Katz, H. Frei, H. Juon, C.B. Hall & P.G. Kale 1984, Envir. Mut. 6:153-188; Szabad, J., I. Soos, G. Polgar & G. Hejja 1983, Mutation Res. 113:117-133; Wurgler, F.E. & E.W. Vogel 1986, Chemical Mutagens (F. de Serres, ed.), Plenum, NY 10:1-72.

Holliday, M. and J. Hirsch. University of Illinois, Champaign-Urbana, Illinois USNA.
Individual differences in conditionability.

Recently we presented evidence for the classical conditioning of the proboscis extension reflex based on the average performance of a group of **D.melanogaster** (Holliday & Hirsch 1984, 1986). Now we describe an experiment to measure individual differences (IDs) in conditionability.

If, as is now widely believed, the contingency between the conditioned stimulus (CS) and the unconditioned stimulus (US) is necessary for the development of a conditioned response (CR), then a reduced CS-US contingency (CS-US presentation randomized or unpaired) will retard or prevent acquisition of the CR (Rescorla 1967; Ricker, Hirsch, Holliday & Vargo 1986). Traditional experiments in the contingency framework test this hypothesis by presenting to one group of subjects a conditioning procedure with a positive contingency, and comparing the results with those of a control group that received noncontingent pairings of the CS and US. By showing that the average CR is greater in the experimental group than in the control group, it can be inferred that: (1) Conditioning has occurred, and (2) the contingency is important for the development of the CR (e.g., Holliday & Hirsch 1984, 1986). Because no individual receives both contingent and noncontingent stimuli, however, this method precludes demonstrating the effectiveness of the contingency analysis to measure conditioning in identified individuals. To measure conditioning in individuals, we presented each subject with nine trials of an automated conditioning procedure (Vargo, Holliday & Hirsch 1983; Holliday, Vargo & Hirsch 1983) followed by six trials of unpaired stimuli. The conditioning procedure consisted of a 5 second tarsal stimulation of 0.5 M Nacl (the CS) followed 0.5 seconds later by 5 seconds of tarsal stimulation (available to the proboscis for 2-3 seconds) with 0.25 M sucrose solution (the US). In addition, to discharge sucrose-induced CES (Vargo & Hirsch 1982a, b), a 5 second tarsal stimulation of distilled water (the intertrial stimulus) was presented 2 minutes 55 seconds after the US (Figure 1). For the unpaired extinction procedure, the CS and US were separated by approximately 90 seconds.

Thirty-four subjects (18 females, 16 males) were tested with this procedure. Figure 2 depicts the average CR as a function of paired and unpaired trials. The characteristic conditioning curve increases over the first nine trials (Holliday & Hirsch 1984, 1986), followed by the hypothesized decrease in responding to the unpaired CS. The evidence thus supports our interpretation that the CS-US contingency is important for conditioning to occur in this species (Holliday & Hirsch 1984, 1986). And, the procedure allows us to look at the same effect in individuals.

Individual cumulative response patterns have been classified into four categories, with individual variation within categories. Examples appear in Figure 3: (a) acquisition followed by extinction, 24%; (b) acquisition without extinction, 15%; (c) responding, but failure to show either acquistion or extinction, 34%; and not shown is (d) no response to the CS, 26%.

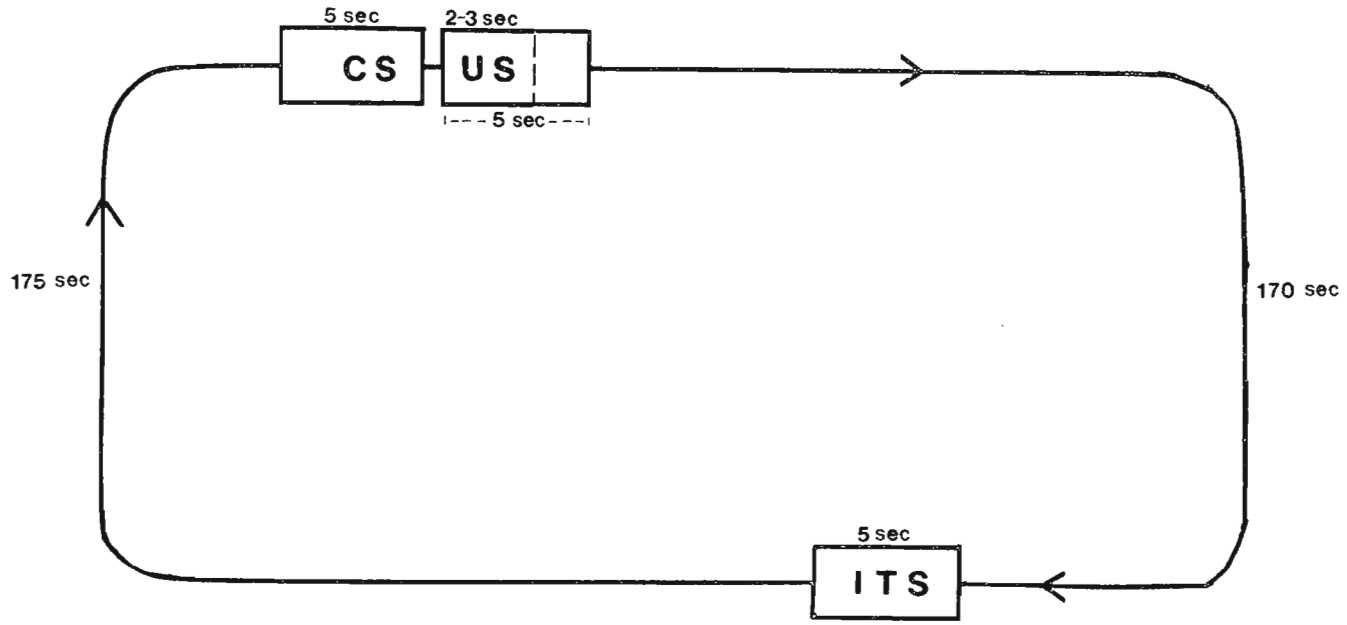


Figure 1. Diagram of the stimulus schedule (note the gap between the CS and US creating approximately 0.5 s trace [where the rubber band prevents contamination between stimuli]).

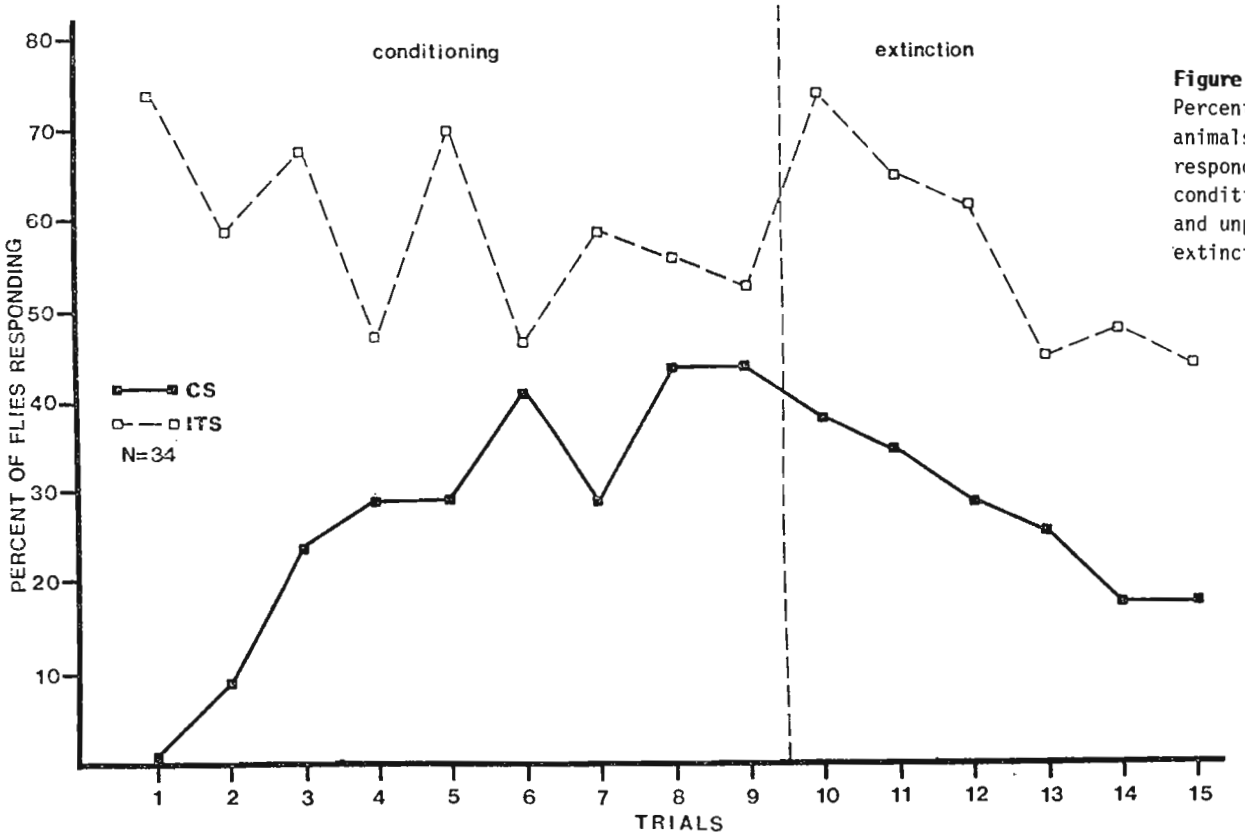


Figure 2. Percent of animals responding to conditioning and unpaired extinction.

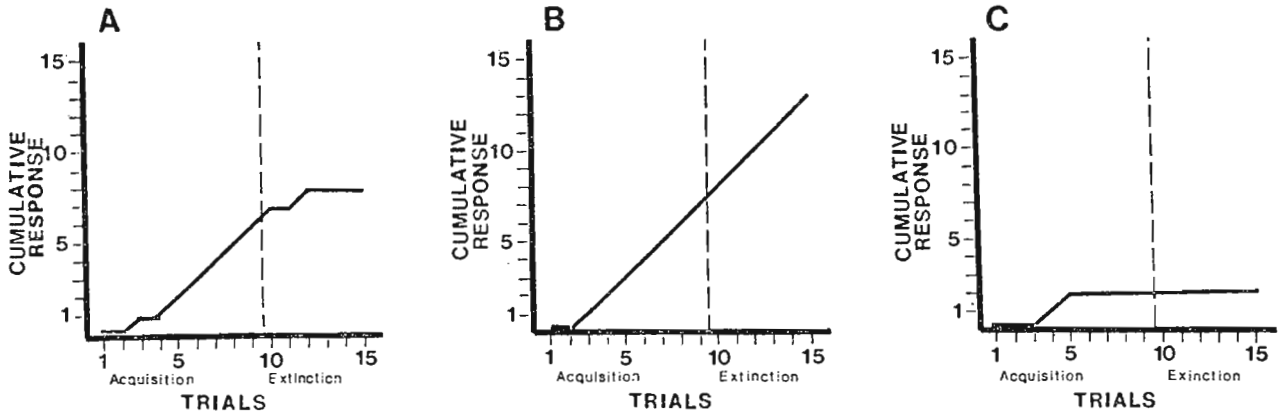


Figure 3. Cumulative responses to CS over nine trials of conditioning and six trials of unpaired extinction for: (A) an individual representing those flies (8 out of 34, 24%) showing both conditioning and extinction, (B) an individual representing those flies (5 out of 34, 15%) showing conditioning without extinction, and (C) an individual representing those flies (12 out of 34, 35%) responding, but showing neither conditioning nor extinction.

The ability reliably to identify IDs in conditionability now allows us to attempt divergent selection for high and low conditionability in **D.melanogaster.** Individuals in category (a) (above) would be classified as good conditioners, while those in category (c) would be poor or non-conditioners. It is not immediately certain why individuals in category (b) continue to respond when the CS and US are unpaired. Perhaps visual stimuli, in addition to the (NaCl) CS, serve as US predictors that are not affected by the unpaired procedure. Also, because sucrose is still present during extinction, CES may influence responding in these individuals.

It has been shown in the blow fly **Phormia regina** that breeding selectively for conditionability, using acquisition scores only to select individuals for breeding, resulted in lines divergent in fact for CS-induced sensitization, not conditioning (Ricker et al. 1986). Our ability now to distinguish between conditioned responding and that due to nonassociative excitation (e.g., between individuals in categories a and b) makes possible controls lacking in previous research.

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Islam, M.S., M.A.R. Khan, P.C. Barman and S.I. Ali. University of Rajshahi, Rajshahi, Bangladesh. Effect of copper and ferrous sulphates on offspring production in **Drosophila melanogaster.**

Attempts have been made to understand the developmental effect of gene action on Drosophila by subjecting the eggs, larvae or pupae of this fly to various stress conditions, such as chemicals, temperature-shock, radiations of various wave-lengths, ether-shocks and so forth. The present work deals with the effect of addition of copper and ferrous sulphates to food on the offspring production in **Drosophila melanogaster.** The wild type **D.melanogaster** was used in the present investigation. The flies were reared on the cornmeal-agar-molasses food medium. Traces of Nipagin were added as mould inhibitor. The doses of copper sulphate used were 0(control), 0.05, 0.10, 0.15, 0.20 and 0.25%, and those of ferrous sulphate were 0, 0.10, 0.15, 0.20, 0.25 and 0.30%. The experiments were started with 25 pairs of flies, equally shared by virgin females and unmated males, per population jar (size 15 cm x 6 cm). The experiments were replicated six times and five times for copper sulphate and ferrous sulphate, respectively, per dose per generation. Counting of flies on the control and treated foods was continued for five successive generations. Experiments were conducted at 25±0.5°C.

Table 1. Offspring production in **D.melanogaster** at different conc. of copper sulphate added to food.

	No. of flies at concentrations:					
Generations	0.00%	0.05%	0.10%	0.15%	0.20%	0.25%
0(parental)	50	50	50	50	50	50
1	517	321	267	162	97	62
2	892	394	265	153	81	52
3	1337	486	291	164	70	41
4	1772	531	334	186	62	38
5	1958	558	354	198	51	37

Table 2. Offspring production in **D.melanogaster** at different conc. of ferrous sulphate added to food.

	No. of flies at concentrations:					
Generations	0.00%	0.10%	0.15%	0.20%	0.25%	0.30%
0(parental)	50	50	50	50	50	50
1	438	314	198	112	98	78
2	807	529	367	286	198	108
3	1418	749	652	412	285	97
4	1852	1135	872	498	313	114
5	2022	1618	998	618	348	118

The results are given in Tables 1 and 2. Both copper and ferrous sulphates reduced the number of offspring of the fly ($P<0.001$). It was found that with the increase of doses the offspring production decreased. Copper sulphate produced a more drastic effect on Drosophila than did ferrous sulphate. Dobzhansky & Spassky (1967) reared **D.melanogaster** and **D.pseudoobscura** on sodium chloride added food medium and found that the latter was more sensitive to the salt than the former species. Islam (1981) reported that the Curly, an autosomal dominant mutant of **D.melanogaster,** had an advantage over its normal competitor in such an abnormal environment. Shakoori & Butt (1980) observed that at stronger doses of thioacetamide the larva of **Musca domestica** failed to metamorphose into adults. A recent study by Shakoori & Parveen (1983) showed that no adult emerged from the eggs of **M.domestica** placed in culture media containing 0.56 and 0.64% thioacetamide. These findings are similar to that observed in the present work. In the present study, it was also observed that flies reared at stronger doses of the salts showed certain morphological abnormalities, e.g., collapsed abdomen with blackened and stubby body. However, whether these characters were due to phenocopic effects or not was not investigated.

Both copper and ferrous sulphates was found to reduce the number of offspring of **D.melanogaster** significantly, and therefore every care should be taken against the contamination of food of **D.melanogaster** with these salts to avoid any shortage in the number of this fly for experimental purposes.

Acknowledgements: The authors express their sincere thanks to Dr. P.T. Ives, Dept. of Biology, Amherst College, Massachusetts, USA, for kindly supplying the stock of flies, and to Dr. M. Altaf Hossain, the then Chairman, Dept. of Zoology, Rajshahi University, for providing necessary laboratory facilities.

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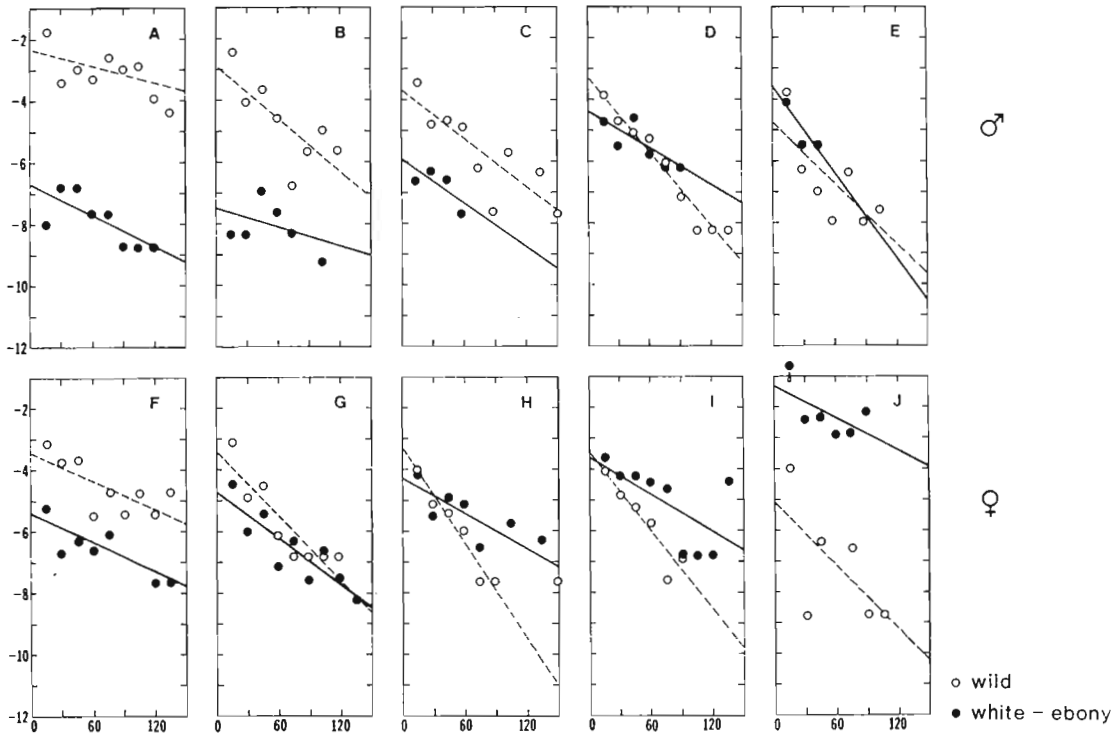
Jacquemin, F., M. Cereghetti, M. Lichtenberger and A. Elens. FNDP, Namur, Belgium. "Rare type advantage", hour, and intensity light.

An estimation of the "rare male advantage" can be done using the so called "Wattiaux's equation" (1964), which changes, by logarithmic transformation, the sigmoid curves of mating successes into straight lines. These straight lines, which can differ in slope or in mean elevation, can be compared by covariance analysis (Snedecor 1956). The "Wattiaux's formula" has been used, e.g., to study the competition between the mutant rucuca flies and the wild type Canton S or the blind mutant ora flies (Kaningini et al. 1986, this issue; Schuermans et al. 1986, this issue). The same formula is applied here with certain modifications. In the original formula, 1n dx/N - x, 1n is the naperian logarithm, dx the number of individuals of a given type (e.g., wild type males) mating during this time interval (15 min), N the total number of individuals of the same type, and x the number of these individuals having already mated before the considered interval of time. Wattiaux's equation was based on the assumption that males could mate only once in the total observation time (3 hr). Present experiments seem to indicate that they had oviously mated more than once, the original formula had to be modified by us. If x (the number of individuals having copulated before the considered interval of time) becomes greater than N (the total number of these types of individuals), the denominator of the equation dx/N - x becomes negative. Consequently, it seemed preferable to introduce "10. x/N" (a mating value) in place of x. The formula thus becomes:

$$1n\ dx.10/N\ /(N - x.10/N) \quad .$$

In the present experiments, we have studied the sexual activity of wild type Canton S flies when they are in the presence of mutant white-ebony flies, which are certainly less active (Grossfield 1975). The method of observation has been previously described (Elens 1957, 1958; Elens & Wattiaux 1964). A total of 30 pairs of virgin flies, 4-5 days old, were introduced together in the mating chamber, but the relative frequencies of both types were varied (for males and females simultaneously). All the observations were done at 25°C and 40-60% relative humidity, but at two different light intensities (50 and 1000 lux), and at two different hours (8 a.m. and 5 p.m.), with 10 repetitions for each relative frequency. The differences between homogamic and heterogamic mating frequencies were never significant. But significant differences were often found between the sexual activity of males or of females when they were "rare" (relative frequency = 5/30) than at other relative frequencies. Such differences were evident not only for the wild type flies but sometimes even for the mutant ones.

At a high intensity of light (1000 lux), in the morning, the wild type males were obviously more active when they were "rare" (Fig. 1, A) than at all the other relative frequencies that were tried ($P<0.001$). Similarly, the white-ebony males displayed their highest activity when they were rare (Fig. 1, D and E).



Figures: Relationships between the competing types wild Canton S and white-ebony, when their relative frequencies differ: columns, from left to right: 5+/25we; 10+/20we; 15+/15we; 20+/10we; 25+/5we. **Abscissa:** time in minutes. **Ordinate:** 1n 10 dx/N /(N - x). 1n is the naperian logarithm, N the total number of flies of one sex and one type, dx the number of these flies mating in the considered interval of time dt (15 min) and x the number of these flies having already mated before.

Figure 1.

morning, 1000 lux.

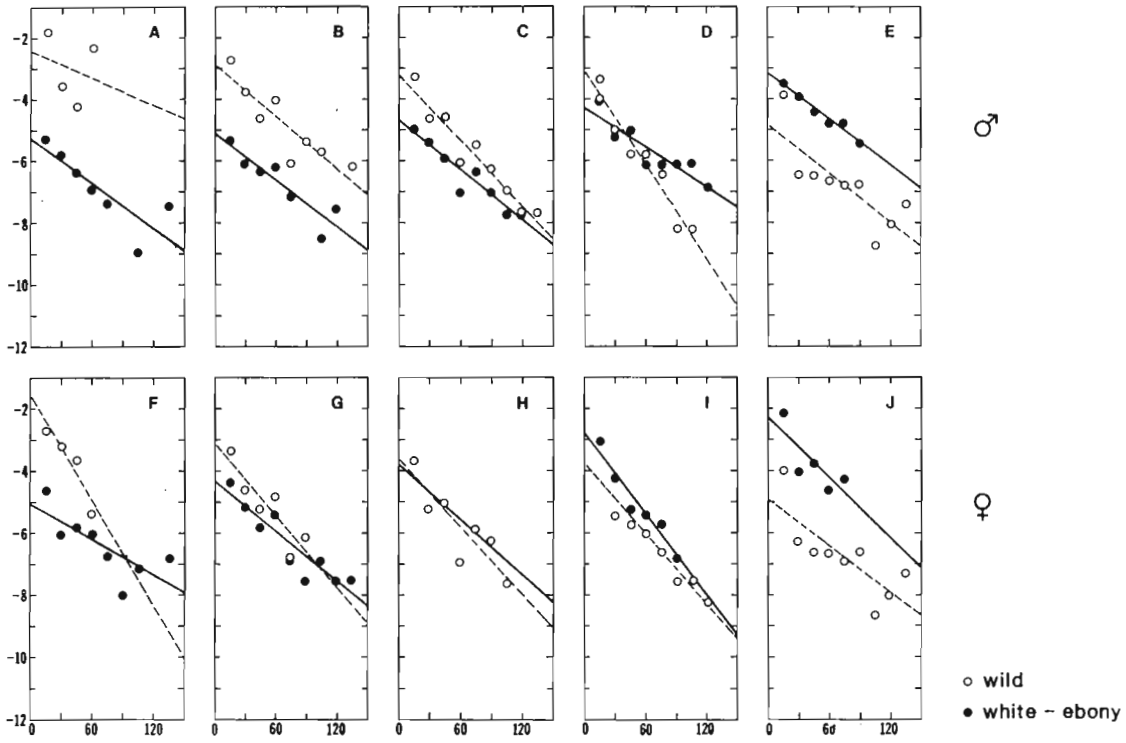


Figure 2. morning, 50 lux.

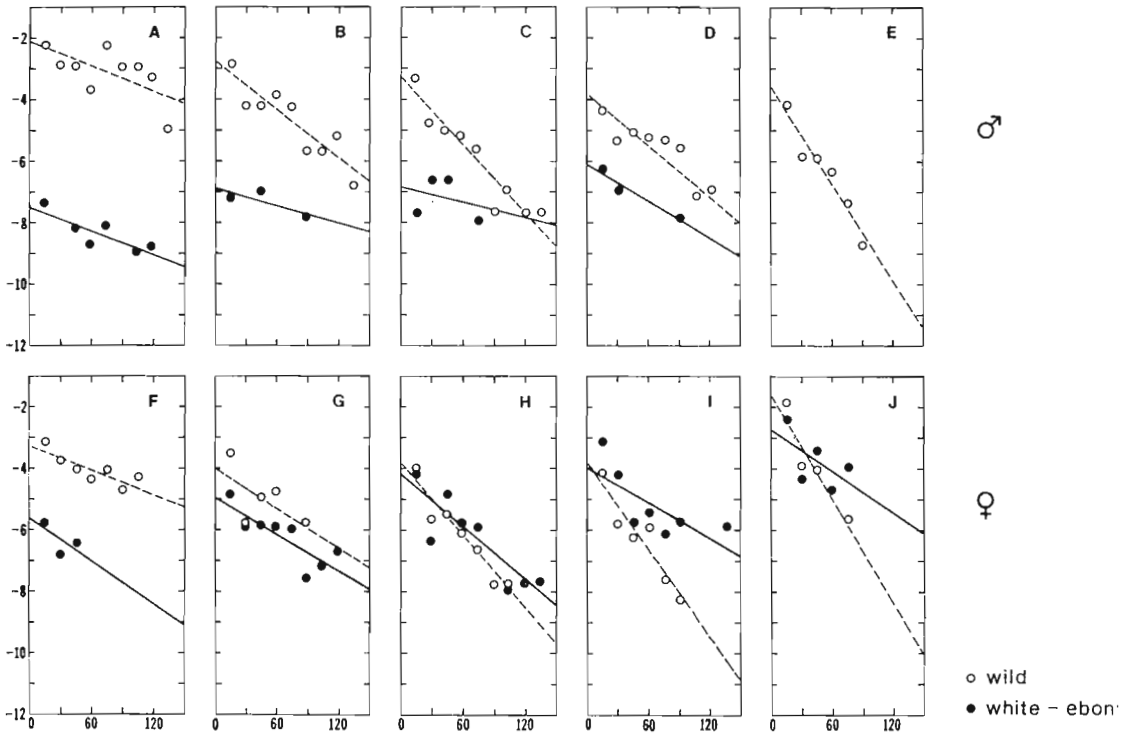


Figure 3. evening, 1000 lux.

In such conditions, they were even more active than the wild type males, which at other frequencies mated more often than the white-ebony ones ($P<0.001$). The activity level of the females of both types was also higher when they were "rare" (Fig. 1, F and J): at the frequency "5+/25we" the wild type females were more active at frequency "25+/5we" ($P<0.001$). In darkness (50 lux) the results were quite similar (Fig. 2). The white-ebony males were more active when rare ($P<0.001$), even more than the wild type ones (Fig. 1, E). At the relative frequencies "5+/25we", "10+/20we", "15+/15we", the wild type males activity was

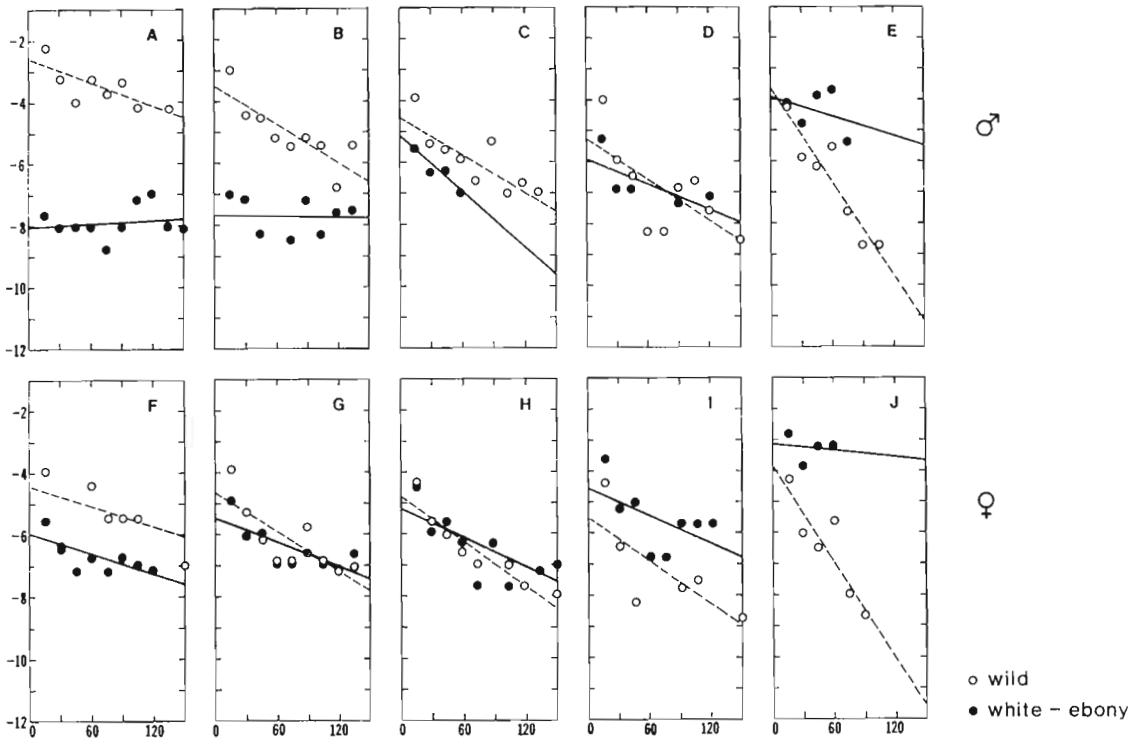


Figure 4. evening, 50 lux.

always higher. A "rare female advantage" was also observed, for the white-ebony flies (Fig. 2, J) as well as for the wild type ones (Fig. 2, F). At the intermediate relative frequencies, the differences between mutant and wild type females were not significant (Fig. 2, G, H, I).

In the evening, the white-ebony males activity level was lower than in the morning, at least in light (Fig. 3). They were always much less active than the wild males ($P<0.001$), and when they were the rarest they did not mate at all (Fig. 3, E). In darkness, one observed the same "rare male advantage" as in the morning experiments (Fig. 4, A and E). In light and in darkness, the females behaved as they did in the morning.

Knoppien (1985) has recently excellently reviewed the whole matter of the "rare male mating advantage", first observed by Petit (1951) and as a result often called "Petit effect". Its causes are discussed, even stronger its existence has sometimes been doubted. Knoppien concludes, however, that this phenomenon is very widespread in insects, at least under laboratory conditions, and perhaps even in nature (1985). Our present observations are in good agreement with this view. Besides, thus, we consider that equations such as the so called "Wattiaux's formula" are interesting tools for visualizing the differences in activity level of the competing types.

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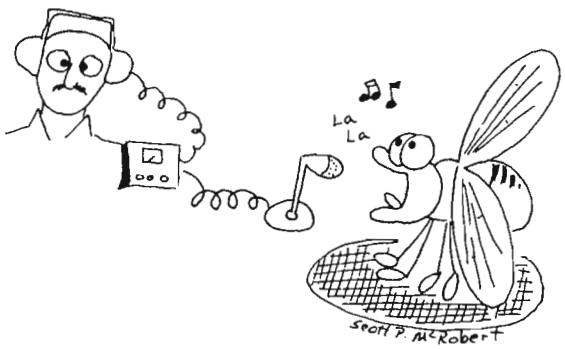

Kalisch, W.-E. Ruhr- Universität Bochum, FR Germany. The EM band-interband pattern of SSP chromosomes in **D.subobscura:** Division 100A-D.

The D (=Dot) chromosome of **D.subobscura** was observed electron microscopically (EM) using the surface-spread polytene (SSP) chromosome preparation technique (Kalisch et al. 1984, 1985a,b; Kalisch & Böhm 1985). Salivary glands were used from late 3rd instar larvae of a wild-type strain. Electron micrographs of SSP chromosomes were prepared with a final magnification of x 3,200 (Fig. 1C and D). Cytological data were calculated from eight electron micrographs and used for a computer plot (for methodological details, see Kalisch et al. 1984, 1986a,b) of an EM chromosome map (Fig. 1E). EM chromosome cytology was compared with the original chromosome map (Fig. 1A) based on LM analyses of squash preparations (Kunze-Mühl & Müller 1958) and with a single light micrograph of a chromosome squash preparation (Fig. 1B).

A total number of 27 chromosome bands was observed. This is about a 93% increase of additional bands compared with the 14 bands of the LM chromosome map (100C1 in Fig. 1A indicates a very tight doublet in the original map of Kunze-Mühl & Müller 1958). Earlier studies have already shown (Whitmore et al. 1984) that the dot chromosomes in Drosophila are very difficult to analyze, because they are usually attached to or embedded in the chromocenter along with the proximal ends of the other chromosomes. Furthermore, they are usually not well-extended in chromosome squash preparations or not well-spread in SSP chromosomes. The latter case is probably a methodological problem based on the two different spreading effects involved (Kalisch et al. 1985b). Due to this, it has not been possible to depict so far all of the polytene structures of this chromosome in an individual photo map. Therefore, two examples are shown of which Fig. 1C shows more structural details of the proximal and Fig. 1D more of the distal part of the dot chromosome.

Acknowledgements: The **D.subobscura** strain was provided by Prof. A. Prevosti, Barcelona. The assistance of V. Kohl, T. Whitmore and G. Schwitalla are kindly acknowledged. This study is part of a project which was supported by the Deutsche Forschungsgemeinschaft.

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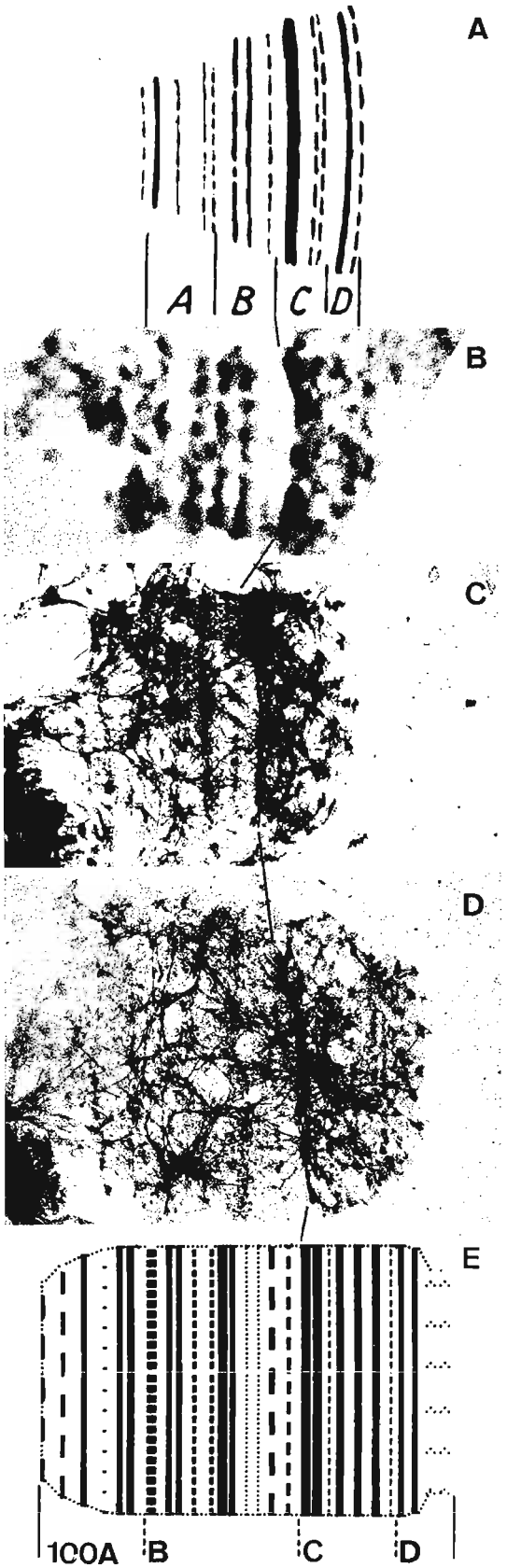

Figure 1. D (=Dot) chromosome of **D.subobscura.** (A) LM chromosome map based on squash preparations of salivary glands (Kunze-Mühl & Müller 1958). (B) Light micrograph, squash preparation, Orcein-staining, Neofluar 100/1.30 Ph objective, x3,200. (C-D) Electron micrographs of SSP chromosome preparations, x3,200. (E) EM chromosome map based on the cytological data of SSP chromosomes.

Kaningini, M., J. Lechien and A. Elens.
FNDP, Namur, Belgium. Blind flies and normal flies tested for phototactism.

Visual mutants with abnormal electroretinograms have been described by Hotta & Benzer (1969). Being blind, they are normally not attracted by light; and one may expect that their behavior in phototactical tests will differ from that of normal flies. Consequently, they would be a very good material for students' practical exercises in behavior genetics or ethology, comparing visual mutants and wild controls.

In the present experiments, blind ora flies (kindly given by L. Craymer, of the California Institute of Technology) have been compared with wild Canton S flies, using the Kekic test (1981), and the Benzer test (1967) modified by Tompkins et al. (1978). The flies had been raised and maintained at 25°C and 60-80% R.H. Males and females had been separated from birth, and the flies were 5 days old when tested. The phototactical tests were done at 25°C and 60-80% relative humidity, with 5 repetitions, and at least 100 flies of same sex and age for each test. As a preliminary, the locomotor activity of flies of both strains had been compared in a square arena (30 flies of each sex, 5 days old, for each strain) according to Manning (1961), at 25°C and 60-80% R.H.; the differences are not significant.

Figure 1 shows the results of the Kekic test for males (right) and females (left) of both strains. For the same strain, the differences between males and females are highly significant; and the differences between the males and between the females of the two strains are highly significant as well (as shown by a chi-square test). Both Figure 2 and Figure 3 show the results of the Benzer test, but in the second case the experiment has been done "inside out": the flies have been tested first for negative phototactism (from light), and after that for positive phototactism (toward light). The differences between sexes are always highly significant for the wild Canton S flies, but never for the ora flies. In both methods (right side out and inside out), the differences between ora and wild Canton S flies are always highly significant ($p < 0.001$), but the behavioral differences are more evident with the "inside out" method, especially for the male flies.

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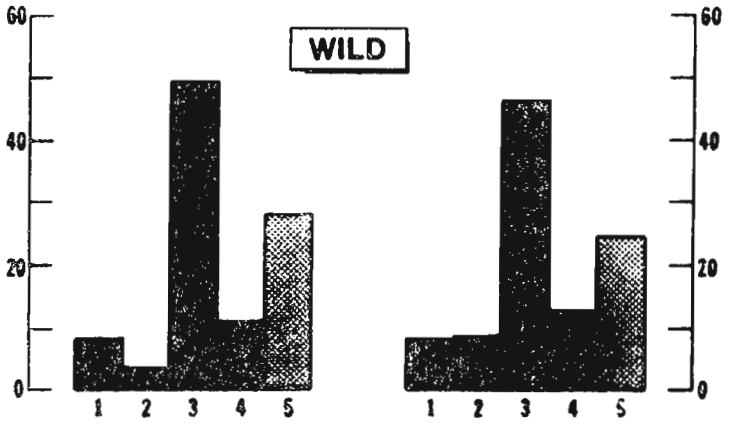

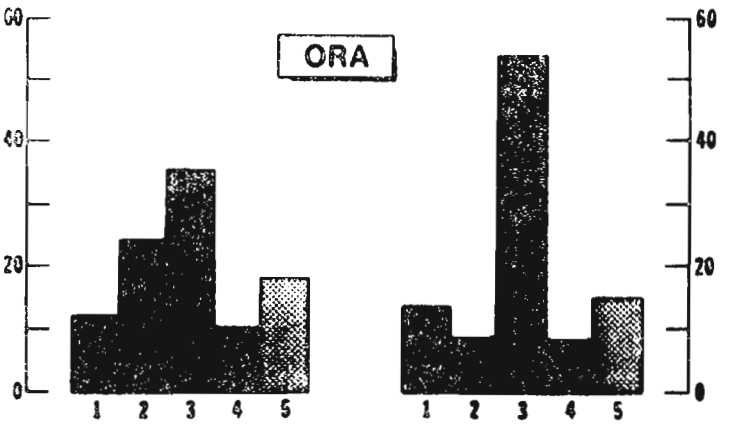

Figure 1. Distribution of the flies after 1 hr tests in the Kekic maze (in %): left: females; right: males.
Chambers: 1 (500 lux), 2 (800 lux), 3 (2,000 lux), 4 (4,500 lux), 5 (6,500 lux).

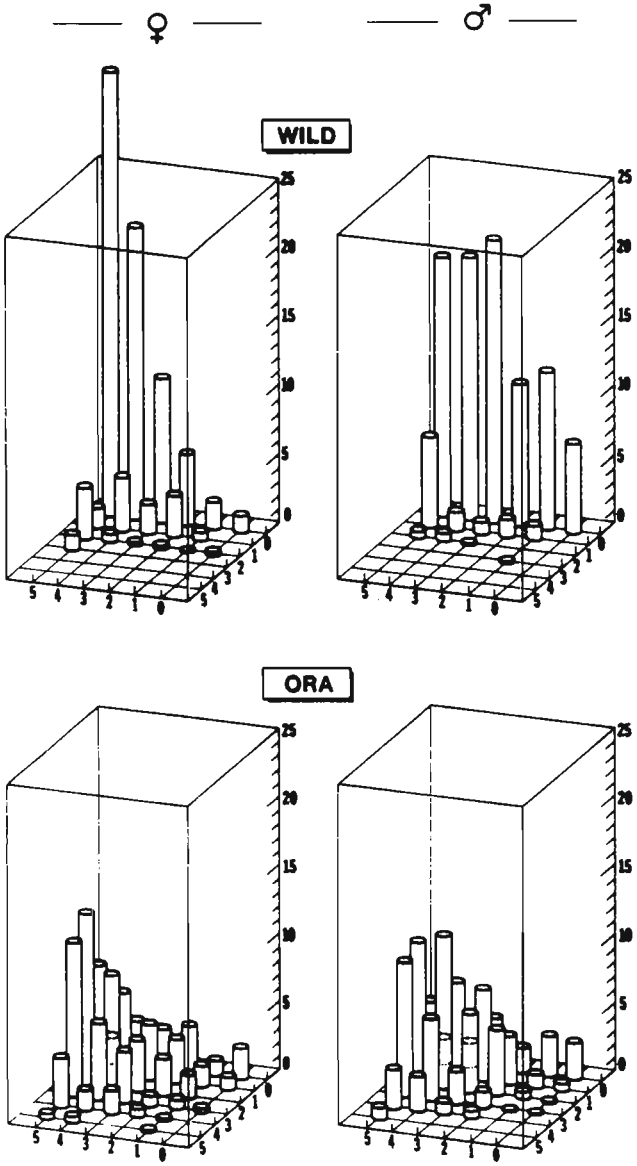

Figure 2. Distribution of the flies after a test for positive and negative phototactism according to Benzer (one minute runs). Ordinate: number of flies in the test tube. Front view abscissa: no. of positive responses (toward light). Side view abscissa: no. of negative responses (from light).

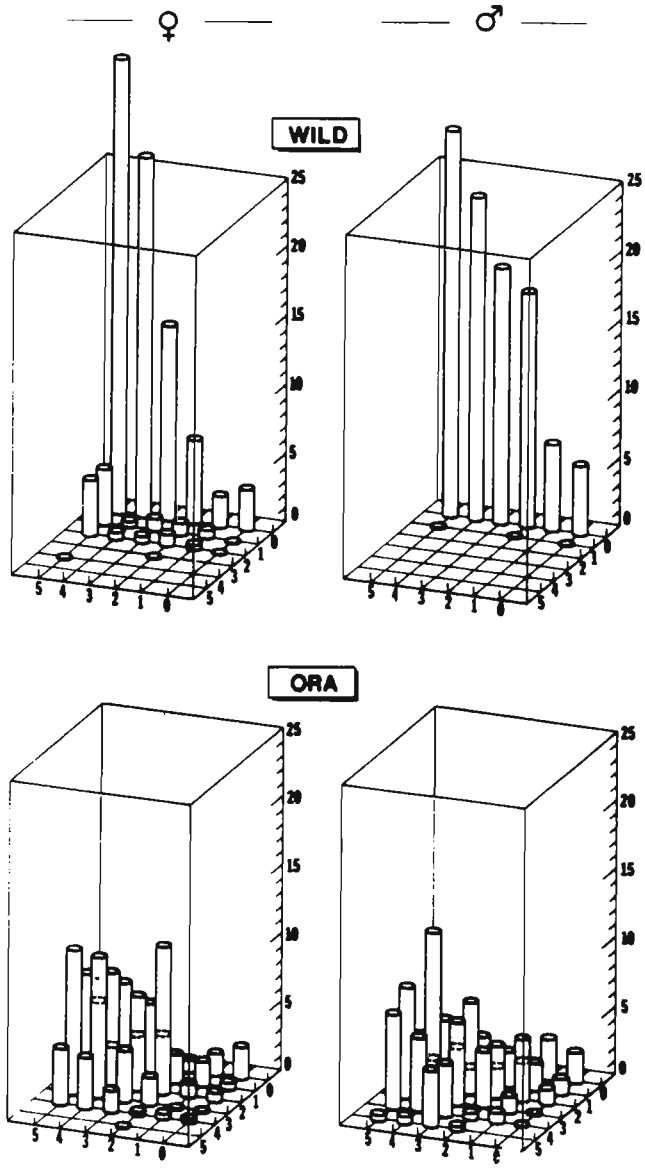

Figure 3. Same as fig. 1, but the test has been done "inside out".

Kaningini, M., J. Lechien, M. Lichtenberger and A. Elens. FNDP, Namur, Belgium. The measure of the "rare male mating advantage".

It appears that the "rare male mating advantage" was first observed by Petit (1951). This phenomenon has held the fascination of population geneticists for a long time and has given rise to a tremendous amount of controversy, with regards to its causes and even to its mere existence. Various approaches have been attempted to find its best mathematical expression, as seen in the recent, excellent review by Knoppien (1985). As a global expression of the phenomenon, the first formula, proposed by Petit, seems the best one. Petit's equation, also known as the "cross product ratio" (Knoppien 1985), can be expressed as

$$K_M = (q_M/p_M)(P_M/Q_M)$$

where p_M and q_M are the respective frequencies of both types among males (p_F and q_F being the

frequencies among females), and P_M and Q_M are the probabilities for the performance of any given mating by an **A** type male or by a **B** type male, respectively (Knoppien 1985). This equation expresses the male sexual fitness of one type relative to the other.

However, K_M is only a global expression of the rare male advantage; and does not allow one to follow its effect during the course of time. It must be remembered that the course of mating successes with time in a sexual competition experiment has been expressed as a composite curve starting as an exponential but ending as a sigmoid. Different competing genotypes are characterized by a variety of curves in which different "plateaux" are reached at different rates. Accordingly, comparisons between various competing types may be biased when the length of the observation period is kept constant from one experiment to the other. In the case of sexual competitions where both types are in equal number, Wattiaux (1964) suggested the use of a logarithmic transformation which changes the curves of mating successes into straight lines. In Wattiaux's formula

$$dx_A/(n_A - x_A)\,dt$$

n_A is the total number of individuals of one sex and one type, dx_A the number of these flies mating in the interval of time dt, and x_A the number of these individuals having already mated before. After logarithmic transformation the equation can be written as:

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

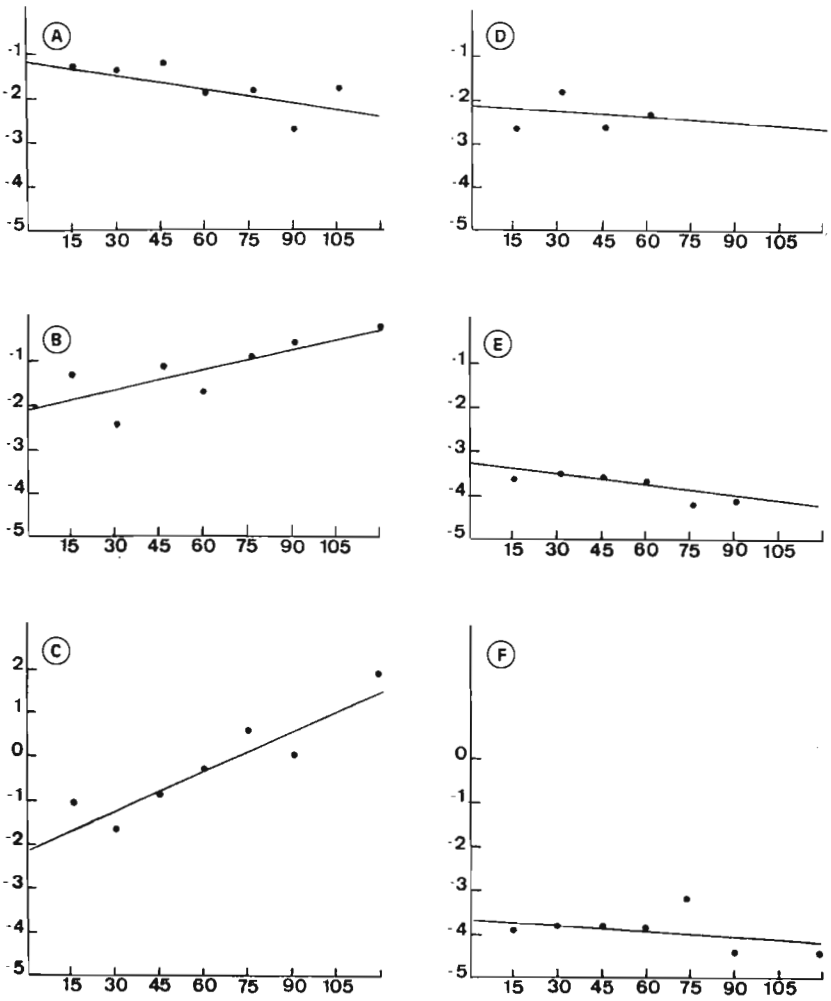

Figure 1. Sexual activity of the males; left: wild Canton S; right rucuca. Abscissa: time, in minutes. Ordinate: $\ln dx/N - x$. Relative frequencies "rucuca"/wild Canton S: A and D = 10/20; B and E = 15/15, C and F = 20/10.

The two constants **a** and **k** are easily estimated from the regression line $Y = A - k\,(X - X)$ where **Y**, (the value of Y is estimated from regression), is the naperian logarithm of x_A and **X** is the mean of the independent variable X (i.e., the time in minutes). The value of the method is based on the biological meaning of A and **k**. The first coefficient provides information on the general level of sexual activity, while the second gives an insight on the variation of sexual activity with time. For sexual competitions where both types are in equal number, Wattiaux suggested comparing the following regression lines: (1) homogamic and heterogamic matings, (2) matings of the males of both strains in competition, (3) matings of the females of both strains in competition, analogous to the "isolation estimate", "female mating ratio", and "male mating ratio" of Merrell (1950). This, indeed, has already been done for a sexual competition experiment where both types were in equal number (Elens et al. 1974).

The purpose of the present paper is to apply the same formula to the male sexual activities of the two types in competition, when their respective frequencies differ. For each interval of time (15 min), one has to calculate the quantity $\ln dx(N - x)$, dx being the number of males of a given type having mated during this interval, N the total number of males of the same type, and x the number of males which have already mated before the considered interval of time.

The laboratory strains used are the well known mutant strain "rucuca" and the Canton S "wild" strain. The sexual activity was determined by our "multiple choice direct observation method" fully described elsewhere (Elens 1958; Elens & Wattiaux 1964), and which has been widely used by Ehrman and others (Ehrman 1964, 1966, 1967, 1968; Ehrman et al. 1965; Petit & Ehrman 1968). Matings were directly observed. Individual males may mate more than once. Frequencies of types are varied among males and females simultaneously. In the present experiments, a total of 30 pairs of virgin flies, 4-5 days old, are used for each test. But the relative frequencies of both genotypes differed: 10/30, 15/30,, or 20/30 of the pairs were wild, the other flies being "rucuca". The three "copulating chambers" were observed simultaneously. All the tests have been done at 25°C and 40-60% relative humidity. The copulating pairs were observed at time intervals less than the duration of copulation and the observations were recorded every 15 minutes. Five repetitions have been made.

The results are shown in Figure 1. The "wild" males are obviously advantaged when rare: the regression line at q_M/p_M = 20/10 (C) differs in slope (probability lower than 1/1000) and in elevation (probability lower than 1/100) from the regression line at frequency q_M/p_M = 10/20 (A); p_M and q_M being the relative frequencies of "wild" and "rucuca". The regression line at q_{M^+}/p_M = 20/10 (C) even differs in elevation (probability lower than 1%) from the regression line at equal frequencies (B). Obviously, many "wild" males have mated more than once, when they were rare. For the "rucuca" males, the regression lines are never significantly different. It seems that the sexual activity of the "wild" males is really stimulated by the presence of many "rucuca" flies. The factors acting in such a stimulation remain to be determined more precisely.

It has been emphasized, in one of our previous publications (Elens et al. 1964), that such a method may throw more light on the causes of the rare male advantage. It has been suggested, by Faugeres et al. (1964), that one has to distinguish between the "vigor factors" --or "metabolic factors"--, and the "behavioral factors" responsible for the rare male advantage. It is possible that a genotype is always sexually more active than the other one (his sexual vigor is higher); but it could be attributed to the fact that the presence of the second type is actually a stimulant for the first one (as previously said, it appears to be applicable to the "wild" males in the present case).

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Knoppien, P. University of Groningen, Haren, Netherlands. Low density storage enhances mating speed in **Drosophila melanogaster.**

It has been known for some time that virgin Drosophila males stored singly are superior in mating compared to males stored in groups (Ellis & Kessler 1975; Knoppien 1985a; Knoppien, in press). It is conceivable that this effect results in rare male mating advantage in nature, since it may confer a mating advantage to males derived from a low-density population at the time that flies of different populations meet. Rare male mating advantage may in turn play a role in maintaining genetic variability (Knoppien 1985b). However, flies of both sexes are found together in nature, providing a situation different from the storage of virgin males at different densities in the laboratory. Therefore it was asked whether group-housing has also a negative effect on subsequent male mating success when males and females are stored together.

The experimental approach to answer this question was as follows. Flies of **D.melanogaster** from an outbred Bogota strain were reared on standard agar medium (Knoppien, in press). They were etherized once within 8 hr from eclosion to collect virgin males and females. These flies were stored in vials, both sexes combined, at two different densities: at low density (L): 1♀ and 1♂ per vial, and at high density (H): 5♀♀ and 5♂♂ per vial, and then aged for 3-5 days. Low-density and high-density males were alternately marked by wing clipping for identification. Probably most of the females had mated after that time (this prediction was easily tested for the low-density vials by scoring the presence of progeny: only one of the fifty low-density females failed to produce progeny). Additionally, in order to test male mating success, virgin tester (T) females were stored without males (5♀♀ per vial), and aged for 3-5 days. These females were marked throughout the experiment by wing clipping. To test male mating success, 25 flies (5 L ♀♀, 5 L ♂♂, 5 H ♀♀, 5 H ♂♂ and 5 T ♀♀) were combined in a mating chamber of the Elens-Wattiaux (1964) type of reduced size (all dimensions halved; Knoppien, in press), and mating was observed for one hour. An excess of males over non-fertilized females was used here, since this has been shown to intensify inter-male competition (Sharp 1982). Ten replicates of this experiment were made.

Only T females were found to mate in this experiment, which does not surprise, because probably nearly all L and H females had mated before. Mating success of low-density and high-density males was found to be strikingly diffeent: 38 L ♂♂ and only 2 H ♂♂ mated (X^2=30.63, P<0.001). Such a result is not entirely unexpected, since it is known that males can be discouraged to mate as a result of encounters with fertilized females (Siegel et al. 1984). These encounters will be more numerous at high density. The present results suggest that the density adult flies have experienced is an important agent in determining subsequent male mating speed. It is conceivable that this effect may also play a role in nature.

References: Elens, A.A. & J.M. Wattiaux 1964, DIS 39:118-119; Ellis, L.B. & S. Kessler 1975, Anim. Behav. 23:949-952; Knoppien, P. 1985a, DIS 61:101; Knoppien, P. 1985b, Biol. Rev. 60:81-117; Knoppien, P., submitted to Behav. Genet., in press; Sharp, P.M. 1982, Genet. Res. 40:201-205; Siegel, R.W. et al. 1984, Behav. Genet. 14:383-410.

Kortier, M.G. and M.H. Gromko. Bowling Green State University, Ohio USNA. The effect of esterase-6 genotype on productivity.

The esterase-6 (EST-6) enzyme of **D.melanogaster** is polymorphic for two forms, S and F, in all natural populations studied (Oakeshott et al. 1981). Considerable effort has been devoted to the study of the possible adaptive significance of the two allozymes. Because the enzyme is concentrated in the male reproductive system and is transferred in the seminal fluid to females during copulation (Richmond et al. 1980), the focus of most investigations has been on the effects of the male enzyme type on reproduction by females. Here we present data that suggest the female EST-6 allozymes may be having effects on progeny production.

Lines homozygous at the locus coding for EST-6 were derived by pair matings of virgin flies from a wild stock of **D.melanogaster** (the same stock used by Gromko & Pyle 1978). After completion of copulation, males were removed from the vials and individual females allowed to oviposit for six days on cornmeal-molasses agar. Starch gel electrophoresis of the males and females was used to determine if a mating was homogametic or heterogametic, with the progeny from homogametic matings used to start homozygous lines. Since the S allele was more common in the wild stock, 10 SS lines were derived within a month but six more months were required to derive 10 FF lines. The lines were maintained in 8 dram food vials for approximately seven months before all lines of the same genotype were chain-crossed to start a large cage population. Flies from the two large cages created were used to start small one-generation cages from which virgin flies were collected under ether anesthesia for use in this experiment.

To collect the productivity data, SS and FF virgins were pair-mated with SS or FF virgin males in individual food vials, resulting in four groups of 30 mated females each. Males were removed within 30 minutes of completion of copulation. Females were transferred to fresh food every other day for eighteen days. All progeny emerging by 20 days at 23°C were counted.

Table 1. Average number of progeny produced by each Est-6 genotype mating combination (± s.e.). Sample sizes are reported in square brackets.

genotypes	Male SS	Male FF
Female		
SS	58.6 (± 20.8) [25]	18.6 (± 5.1) [26]
FF	243.4 (± 38.8) [29]	261.0 (± 34.9) [29]

Two-way analysis of variance revealed that FF females produced many more progeny than did SS females (F=52.12, p<0.0001, Table 1). There was no effect of male genotype (F=0.14, p=0.7066) nor a significant interaction between male and female genotypes (F=0.95, p=0.3321). Thus the female genotype appears to be responsible for the large differences in productivity. Although the direction of this difference agrees with Kojima & Yarbrough's (1967) finding of a slight but insignificant superiority of FF over SS females, the magnitude of the difference reported here is much greater. Our data do not agree with Birley & Beardmore's (1977) finding of no difference in fecundity of Est-6 homozygotes. We acknowledge the limitations of the chain cross as a means to eliminate linkage disequilibrium. Thus, within the framework of the inherent limitations of this sort of approach, we conclude that the region of chromosome marked by Est-6 has an effect on female function.

References: Birley & Beardmore 1977, Heredity 39:133-144; Gromko & Pyle 1978, Evolution 32:588-593; Kojima & Yarbrough 1967, PNAS 57:645-649; Oakeshott, Chambers, Gibson & Willcocks 1981, Heredity 47:385-396; Richmond, Gilbert, Sheehan, Gromko & Butterworth 1980, Science 207:1483-1485.

Kumar, A. and J.P. Gupta. Banaras Hindu University, Varanasi, India. Inversion polymorphism in **Drosophila nasuta.**

Drosophila nasuta, a member of the **nasuta** subgroup of the **immigrans** species group, was originally described by Lamb (1914) from the Seychelles Islands in the Indian Ocean. Besides this type locality, it has been also recorded from Kenya, Madagascar, Mauritius, Sri Lanka and India (Kitagawa et al. 1982). In India, the species is widespread and commonly seen particularly during colder months of the year.

In recent years, some attempts have been made to analyse naturally occurring inversion polymorphism in the Indian populations (Sajjan & Krishnamurthy 1974; Ranganath & Krishnamurthy 1975, 1978a, b; Rajasekarasetty et al. 1979) as well as in the populations obtained from Sri Lanka, Mauritius, Kenya and Madagascar (Wakahama & Kitagawa 1980), but no efforts have been made to correlate their findings. During the last few years our studies on several populations of this species inhabiting the northern parts of the Indian subcontinent, in particular, have yielded altogether 22 paracentric inversions. In this report, we wish to provide a comprehensive review of the information on inversion polymorphism in this species. The final picture emerged from the results of these studies suggests this species to be highly polymorphic, carrying 85 distinct inversions (Table 1).

Acknowledgements: The financial assistance from the DST (Govt. of India) is thankfully acknowledged.

Table 1. Summary of naturally occurring inversions detected in **Drosophila nasuta** Lamb.

Inversion	Photographic Ref.	Description	Breakpoints	Locality
[Note: for column 2 references, see **References** at end of this note.]				
X-Chromosome:				
X-1	[7] as X-A; [8] as X-A	subterminal	2-4	Ind, Sey
X-2	[7] as X-B; [8] as X-C	subterminal, overlaps with X-1	4-6	Ind, Sey
X-3	[4] as X-C	median	9-11	Ind
X-4	[8] as X-D	submedian	17-19	Sey
X-5*	Present study	median	11-14	Ind
X-6	[8] as X-B	subterminal	4-8	Sey
X-7	[8] as LX-A	subterminal	4-9	SL
X-8	_____ as HX-A	subterminal	3-9	Mrt
IIL-Chromosome:				
IIL-1	[7] as 2L-A; [8] as 2L-B	submedian	33-45	Ind, Sey
IIL-2*	[4] as 2L-B	median	34-49	Ind
IIL-3	_____ as 2L-C	subterminal, overlaps with 2L-4	31-39	Ind
IIL-4	_____ as 2L-D	subterminal, overlaps with 2L-3	34-43	Ind
IIL-5	[8] as 2L-A	subterminal	30-34	Sey
IIL-6	[8] as 2L-C	submedian	34-39	Sey, SL
IIL-7	_____ as 2L-D	median	38-43	Sey
IIL-8	_____ as 2L-E	median	39-43	Sey, SL
IIL-9	_____ as 2L-F	submedian	39	Sey
IIL-10	_____ as 2L-G	submedian	40-49	Sey
IIL-11*	Present study	Complex	--	Ind
IIR-Chromosome:				
IIR-1	[8] as 2R-A; [4] as 2R-G	subterminal	82-77	Ind, Sey
IIR-2*	[8] as 2R-B	submedian	74-71	Sey
IIR-3	_____ as 2R-C	submedian	74-68	Sey
IIR-4	[7] as 2R-A	subterminal	82-74	Ind
IIR-5	_____ as 2R-B	subterminal	81-76	Ind
IIR-6	_____ as 2R-C	subterminal	81-74	Ind
IIR-7	_____ as 2R-D	submedian	75-61	Ind
IIR-8	[4] as 2R-E	submedian, overlaps with 2R-7	73-59	Ind
IIR-9	_____ as 2R-F	submedian, included within 2R-7	71-69	Ind
IIR-10*	Present study	subterminal	81-73	Ind
IIR-11*	_____	submedian, included within 2R-12	77-60	Ind
IIR-12*	_____	subterminal, included within 2R-11	77-70	Ind
(IIL-IIR)-1	[7] as (2L-2R)-A	pericentric inversion between basal regions of 2L & 2R		Ind

Inversion	Photographic Ref.	Description	Breakpoints	Locality
III-Chromosome:				
III-1A	[8] as 3A	terminal	84-85	Sey, SL
III-1B	_____ as 3B	subterminal	84-85	Sey
III-2*	[4] as 3V	subterminal	85-87	Ind
III-3	[8] as 3C	submedian	86-89	Sey, SL
III-4	_____ as 3D; [7] as 3B	subterminal	89-94	Ind, Sey
III-5	[8] as 3E	median	97-112	Sey
III-6	_____ as 3F; [7] as 3D	submedian	97-106	Ind, Sey, SL
III-7	[8] as 3G	submedian, included within III-6	97-99	Sey, SL
III-8	_____ as 3H	median	104-106	Sey
III-9	[8] as 3I	median	109-118	Sey
III-10	_____ as 3J; [7] as 3P	basal, overlaps with III-23	118-134	Ind, Sey, SL, Mad
III-11	[8] as 3K	subterminal	126-130	Sey, Mad
III-12	[7] as 3A	subterminal	85-87	Ind
III-13	_____ as 3C	subterminal	91-96	Ind
III-14*	_____ as 3E	basal	112-132	Ind
III-15*	_____ as 3H	basal, overlaps with III-14 & III-19	121-133	Ind
III-16	_____ as 3I	subterminal	132-135	Ind
III-17	_____ as 3J	basal overlaps with III-14 & III-17	128-136	Ind
III-18	_____ as 3G	subterminal, tandem inversion with III-33	132-133	Ind
III-19	_____ as 3K	basal, overlaps with III-15	127-134	Ind
III-20	_____ as 3L	basal, overlaps with III-14	119-137	Ind
III-21	_____ as 3M	basal, overlaps with III-14	122-136	Ind
III-22	_____ as 3N	basal, overlaps with III-10	122-134	Ind
III-23	_____ as 3O	basal, overlaps with III-10	119-136	Ind
III-24	[4] as 3S	basal	127-131	Ind
III-25	_____ as 3T	basal	132-134	Ind
III-26	_____ as 3U	submedian, associated with III-13	90-91	Ind
III-27	_____ as 3W	submedian, included within III-28	121-124	Ind
III-28	_____ as 3X	basal	120-134	Ind
III-29	_____ as 3Q	submedian, included within overlapping inversions III-14 + III-15	116-121	Ind
III-30*	_____ as 3R	submedian, included within overlapping inversions III-14 + III-15	119-123	Ind
III-31	[3] as 3Y	submedian, overlaps with III-32	112-127	Ind
III-32	_____ as 3Z	submedian, overlaps with III-31	119-136	Ind
III-33	[7] as 3F	basal, tandem inversion with III-18	126-132	Ind
III-34*	Present study	submedian	97-110	Ind
III-35*	_____	median	98-123	Ind
III-36*	_____	basal	124-133	Ind
III-37*	_____	basal, includes III-30	119-133	Ind
III-38*	_____	basal	117-133	Ind
III-39*	_____	submedian	105-118	Ind
III-40*	_____	submedian	114-122	Ind
III-41*	_____	median	98-127	Ind
III-42*	_____	submedian, tandem inversion with III-43	112-122	Ind
III-43*	_____	submedian, tandem inversion with III-42	122-132	Ind
III-44	[4] as Fig. 5B	basal, complex	--	Ind
III-45	_____ as Fig. 5C	basal, complex	--	Ind
III-46	_____ as Fig. 5D	basal, complex	--	Ind
III-47	_____ as Fig. 5E	basal, complex	--	Ind
III-48	_____ as Fig. 5F	basal, complex	--	Ind
III-49*	Present study	basal, complex	--	Ind
III-50	[8] as M3-A	subterminal	86-88	KY
III-51	_____ as M3-B	submedian	94-96	KY
III-52	_____ as L3-A	basal	129-131	SL

*Inversions encountered during present study. Ind=India; Sey=Seychelles; Mrt=Mauritius; SL=Sri Lanka; KY=Kenya; Mad=Madagascar.

References: [1] Kitagawa, O., K.I. Wakahama, Y. Fuyama, Y. Shimada, E. Takanashi, M. Matsumi, M. Uwabo & Y. Mita 1982, Jap. J. Genet. 57:113-141; [2] Lamb, C.G. 1914, Trans. Linn. Soc. London 16:307-372; [3] Rajasekarasetty, M.R., S.R. Ramesh & N.B. Krishnamurthy 1979, the nucleus 22(2):92-95; [4] Ranganath, H.A. & N.B. Krishnamurthy 1975, J. Hered. 66:90-96; [5] _____ 1978a, Genetica 48:215-221; [6] _____ 1978b, the nucleus 21:158-161; [7] Sajjan, N.S. & N.B. Krishnamurthy 1974, Egypt. J. Genet. & Cytol. 3(2):211-228; [8] Wakahama, K.I. & O. Kitagawa 1980, Mem. Fac. Sci. Shimane Univ. 1:103-126.

Lamooza, S.B. and S.R. Ali. Nuclear Research Center, Baghdad, Iraq. Enzyme variability in natural populations of **Drosophila melanogaster** in Iraq.

Table 1. Est-6 allele frequencies in Tuwaitha and Basrah populations of **D.melanogaster**, where the results for the faster (F) allele (out of two alleles) are shown.[a]

Year of collection	F allele frequency ± S.D. Tuwaitha	Basrah
1981	0.282±0.033 (181)	0.34 ±0.033*** (200)
1982	0.358±0.053 (81)	0.693±0.043*** (114)
1983	0.271±0.347 (164)	0.289±0.046*** (79)

[a] figures in () show sample size.
*** H.S. difference from Tuwaitha data.

Table 2. PGM allele frequencies for Tuwaitha population.[a]

Year of collection	F allele frequency ± S.D.
1981	0.968±0.019 (79)
1982	0.849±0.039 (83)
1983	1.000 (164)

[a]Figures in () show sample size.

Two populations of **D.melanogaster** from Iraq were analyzed to determine the maintenance of genetic variation at Esterase-6 (Est-6 map position 36.8) and Phosphoglucomutase (PGM map position 43.6), both located on chromosome 3. This work is a continuation of our previous investigation (Lamooza et al. 1985). Flies were collected from the two localities: Basrah (B) and Tuwaitha (T); the nature of collections and collecting procedures were the same as that described in our previous investigation. Collections were continued over a period of 3 years for about the same time each year. Electrophoretic separations were performed as specified for each reaction mixture for the two enzymes, where phenotypes are similar to those which were observed in our previous studies. Chi-square tests for heterogeneity were performed to demonstrate the variation between populations. Allozyme allele frequency estimates are given in Table 1. The fast allele in all collections ranged from 0.271 to 0.693. The allele frequencies were significantly different between T & B populations among collections at PC 0.001. The slow allele in T population was the most dominant allele through 1981-1983. Previous results indicated that the fast allele acquired the most adaptive value 0.72, 0.732 for 1979, 1980, respectively, and this pattern of genetic variation may be due to chance and/or to the result of external causative forces. In B population the fast allele frequency fluctuated and showed a dominant value in 1982. The results show that configuration of allelic frequencies at Est-6 locus was different between the two localities during the years of collection. Variation in allele frequencies could be ascribed to the geographic and environmental variability (Rockwood-Sluss 1973; DE Albuquerque et al. 1981). On the other hand, Bryant (1974) considers that the genetic variation seems to be associated with temporal variation in the environment.

As PGM locus the fast allele in T population shows the most dominant values as shown in Table 2, confirming the previous results in 1979, 1980 (0.910, 0.912, respectively). The PGM^f allele shows an apparent increase and seems to become monomorphic in 1983 (frequency of fast allele: 1.000). Although it is impossible to predict whether PGM^s allele would be eventually eliminated, this requires further and more extensive sampling of this population to have a clear picture of this situation.

Acknowledgements: We wish to thank Dr. Mohammed S.H. Ahmed for critical reading of the manuscript.

References: Bryant, E.H. 1974, Am.Naturalist 108:1-19; Lamooza, S.B. et al. 1985, Biochem. Genet. 23(3/4):321-328; Poulik, M.D. 1957, Nature 180:1477-1479; Rockwood-Sluss, E.S. et al. 1973, Genetics 73:135-146.

Lee, T.J. and J.H. Pak. Chung-ang University, Seoul, Korea. Biochemical phylogeny of the **D.auraria** complex.

Phylogenic relationships among five sibling species, **D.auraria, D.biauraria, D.triauraria, D.quadraria** and **D.subauraria** were investigated by SDS polyacrylamide gel electrophoresis (SDS PAGE) and two-dimensional electrophoresis (TDE).

Water soluble protein patterns of the five species were comared by SDS PAGE. Similarities among **D.auraria, D.triauraria** and **D.quadraria** were found though there was a slight difference in density, while the pattern of **D.biauraria** was similar to **D.subauraria.**

Table 1. Estimates of genetic distance between **D.auraria** complex obtained by TDE.

	D.auraria	D.biauraria	D.triauraria	D.quadraria
D.biauraria	0.204			
D.triauraria	0.130	0.124		
D.quadraria	0.110	0.163	0.070	
D.subauraria	0.213	0.052	0.090	0.170

About 100 protein-spots were detected in TDE slab gel. In the comparison between gels, the genetic distance of five species were calculated according to Aquadro & Avises' (1981) equation. The genetic distance between **D.biauraria** and **D.subauraria** was 0.052, the lowest of all, and the next 0.070, between **D.triauraria** and **D.quadraria.** In contrast with these, the genetic distance between **D.auraria** and **D.subauraria** was 0.213, the highest of all, and the next 0.204, between **D.auraria** and **D.biauraria** (Table 1).

In consequence, it could be found that **D.biauraria** was more closely related with **D.subauraria,** and **D.auraria** was distantly related with **D.subauraria.** The direction of speciation in these sibling species appeared to be made from **D.quadraria** to **D.auraria** and **D.triauraria,** and from **D.triauraria** to **D.biauraria** and **D.subauraria,** respectively.

References: Lee, T.J. 1981, Tech. & Sci. (Chung-ang Univ) 8:17-24; _____ 1983, Thesis Collection, Chung-ang Univ. 27:105-130; Ohnish, S., K.W. Kim & T.K. Watanabe 1983, Jpn. J. Genet. 58:141-151.

Liebrich, W. Institut fur Genetik, Universitat Dusseldorf, FR Germany. In vitro spermatogenesis in Drosophila: A comparative study of different species.

Pupal, larval or adult testes were isolated from seven different species of Drosophila representing the four species groups **melanogaster, repleta, immigrans** and **virilis.** To isolate single cysts the testes were punctured with fine tungsten needles. Routinely around 50 cysts would flow undamaged through the punctured testis membrane. They were cultured as described elsewhere (Liebrich 1981), except that 1 mm thick (rather than 2.2 mm) culture chambers were used.

In general, cysts were isolated from the testes of late pupae and newly emerged adults where a wide range of stages in spermatogenesis is present. Exceptions were **D.melanogaster** where cysts isolated from late larval or young pupal testes have already commenced to differentiate and are relatively easy to isolate and **D.bifurca** where only cysts of adult tests could be successfully cultured.

The cysts of all six species (**D.melanogaster, D.neohydei, D.bifurca, D.fulvimacula, D.virilis, D.simulans**) continue to differentiate in culture and pass through meiosis and early spermiogenesis. A time-table of duration of phases of meiosis and spermiogenesis was constructed for five species. Although in all 5 species investigated the differentiation processes and the duration of the different stages are similar, there are particular species-specific differences in morphological details.

Especially in **D.melanogaster** during elongation phase a few spermatid nuclei can often be detected in the "tail region" of the bundle (Fig. 1d, arrows). Since this phenomenon is also seen in freshly explanted cysts, it cannot be caused by any culturing effect. The lagging behind of the nuclei may be a genuine failure of the nuclei to migrate to the head, but could also be due to an improper orientation of the whole spermatid.

In detail there are differences between the cysts of all investigated species of Drosophila such as the morphology of the Y-chromosomal loops appearing in prophase I (Hess 1967), the number of germ cells per cyst (Hanna et al. 1982; Liebrich et al. 1982) or the number of Nebenkern derivatives (Hess & Meyer 1968). On the other hand, differentiating cysts of species of the same group or subgroup (according to Patterson & Stone 1952) may have the same morphological appearance during meiosis and spermiogenesis in vitro: cysts isolated from **D.melanogaster** and **D.simulans** (melanogaster group) are of very similar appearance. It is nearly impossible to discriminate between cysts of **D.hydei, D.neohydei,** and **D.eohydei** (repleta group, hydei subgroup). Surprisingly, males of **D.virilis,** though this species belongs to a different group, have cysts with a morphology similar to cysts of **D.hydei. D.bifurca** and **D.fulvimacula,** on the other hand, are an exception in that they belong to the same subgroup (melanopalpa of repleta group), but their

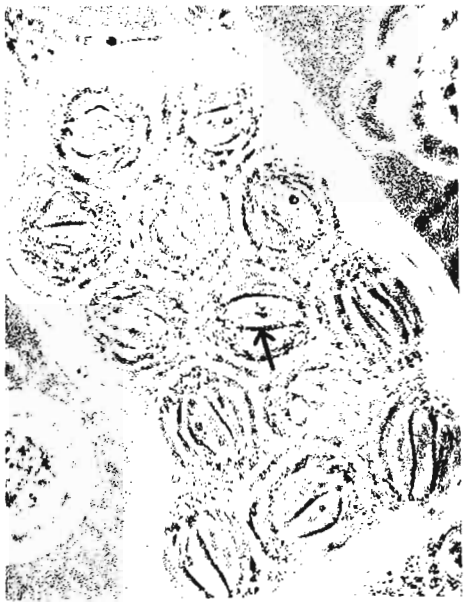


Figure 1a-d. Single isolated cyst of different species of Drosophila differentiating, in vitro.
1d. D.melanogaster. Spermatid bundle elongation. Spermatid nuclei (n) aggregate at the head end, a few at the "wrong" tail end of the bundle (Aarrows). DIC, x480.
1a. D.fulvimacula. Meiosis I. Arrow indicates chromosomes that can be seen between the mitochondria of a spermatocyte. Phaco, x770.
1c. D.bifurca. Meiosis I (m: mitochondria of a spermatocyte). DIC, x480.
1b. D.immigrans. Interphase between meiosis I and II (m: aggregated mitochondria of a spermatocyte). Phaco x480.

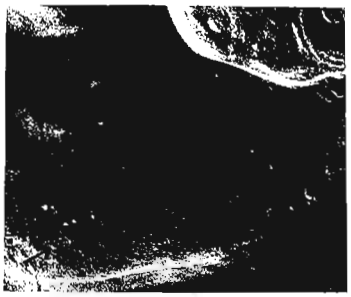
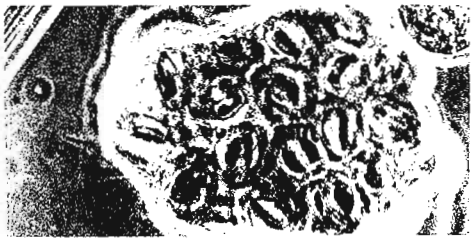


Figure 1a ↑

↓ Figure 1c

Figure 1d ↓

↑ Figure 1b



cysts can be readily distinguished. Cysts of **D.fulvimacula** have a very characteristic, long pear-shaped nucleus (Hess 1967) and are unique in that it is possible to observe chromosome movement during meiosis (Fig. 1a). **D.bifurca** is unique in having cysts with very short germ cell mitochondria. During meiosis they are arranged in the equatorial plate, while in the cysts of all species they extend from one pole of the spindle to the other (cf. Fig. 1c with 1a).

In cysts of **D.immigrans** (subgroup melanogaster) the germ cell mitochondria aggregate by the end of the first meiotic division (Fig. 1b). During the second meiotic division they remain more or less aggregated. Thus, it is difficult to discriminate between metaphase I and the phase of Nebenkern formation at the end of meiosis II.

In cysts of **D.melanogaster** during prophase I the Y-chromosomal loops first disintegrate into granular material after which the spindle apparatus is formed. In cysts of **D.hydei** the reverse occurs. Spermatids

Table 1. Time of duration of the single phases of meiosis and spermiogenesis of 5 Drosophila species at (22-23)°C. Time in hours, + = not exactly determined: in all cases more than 7 hr.

phase	D.melano-gaster	D.hydei	D.fulvi-macula	D.vir-ilis	D.immi-grans
metaphase I to telophase I	0.7-1.5	2	1.5-2	2	2-2.5
interphase	0.8-1	0.3-5	1	1	1.5
meiosis II	0.7-2	2	1-1.5	2.5	1.5
Nebenkern formation	0.8-2	2-3	1.7	1	1-1.5
onion phase	6.5-7	8	+	+	+
Spermatid elongation and nuclear movement	2-3	5	5	4	5

of **D.melanogaster** and **D.simulans** possess only a minute nucleolus-like body, while in cysts of **D.hydei** this body persists until the late elongation phase. In **D.melanogaster** it disappears earlier.

The results show that the general course of meiosis and spermiogenesis is the same in all Drosophila species investigated so far. However, the events within the single phases are not strictly determined. It seems unimportant, e.g., whether there exist one or two Nebenkern derivatives and 16 or 21 spermatogonia. The spindle apparatus may appear earlier or later in prophase I, or the germ cell mitochondria aggregate during meiosis or after meiosis. For the correct course of spermatogenesis, it is only important that these events take place. The findings may serve as an additional example that general norms exist for distinct differentiation processes. The way, however, to reach this norm is not determined exactly.

Acknowledgement. The author wishes to thank Mrs. Ursula Glos-Mettbach for her helpful technical assistance. The work was supported by the Deutsche Forschungsgemeinschaft.

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Loukas, M. and Y. Vergini. Agricultural College of Athens, Greece. Mannose-phosphate isomerase: A new highly polymorphic locus in **D.subobscura**; location and mapping data; genetic differentiation of MPI-locus in Drosophila species of the obscura group.

A new highly polymorphic locus coding for the enzyme mannose-phosphate isomerase was electrophoretically detected in adults of **Drosophila subobscura.** Electrode buffer: 0.25 M TRIS-HCl, pH 8.5. Gel buffer: 1 part of electrode buffer + 4 parts of distilled water. Electrophoresis: Horizontal starch gels were run for 3.5 hr at 200 mAs. Staining: It was performed in agar overlay consisting of 0.1 M TRIS-HCl, pH 8.5 and containing 10 mg NADP, 20 mg $MgCl_2$, 10 mg mannose-6-phosphate, 40 units glucose-6-phosphate dehydrogenase, 30 units phosphoexose isomerase, 2 mg Nitro Blue Tetrazolium and 1 mg Phenazine Methosulfate.

To localize MPI we used the net strain (net: a recessive mutant located on chromosome U), the ch cu strain (cherry eyes and curled wings: both recessive mutants located on chromosome O), the p pl strain (poppy eyes and plexus wings: both recessive mutants located on chromosome E), the enzyme locus PGM (phosphoglucomutase located on chromosome J), and finally the enzyme locus XDH (xanthine dehydrogenase located on chromosome O). The following crosses demonstrated that MPI is located on chromosome O.

For chromosome U: Males of the net strain, homozygous for the allele $MPI^{1.00}$ were crossed with females of a wild type strain homozygous for the allele $MPI^{1.07}$. F_1 males were then crossed with females of the net strain. Half of the wild and half of the net progeny of this backcross were heterozygous ($MPI^{1.07}/MPI^{1.00}$). Those of the other half of both phenotypes were homozygous ($MPI^{1.00}/MPI^{1.00}$).

For chromosome J: Males of a strain homozygous for allele $PGM^{1.00}$ and for allele $MPI^{1.07}$ were crossed with females homozygous for the alleles $PGM^{1.10}$ and $MPI^{1.00}$. F_1 males were then backcrossed with one of the parents. Half of the homozygous and half of the heterozygous for the PGM locus progeny were homozygous for the MPI locus. Those of the other half of both electrophoretic phenotypes for the PGM locus were heterozygous for MPI.

For chromosomes E and O: Males of the ch cu strain, homozygous for allele $MPI^{1.11}$, were crossed with females of the p pl strain, homozygous for the allele $MPI^{1.07}$. Some of the F_1 males were crossed with females of the p pl strain and some others with females of the ch cu strain. Half of the wild and half of the p pl progeny of the first backcross were heterozygous ($MPI^{1.11}/MPI^{1.07}$) while those of the other half of both phenotypes were homozygous ($MPI^{1.07}/MPI^{1.07}$). All the wild type progeny of the second backcross were heterozygous ($MPI^{1.11}/MPI^{1.07}$) and all the ch cu homozygous ($MPI^{1.11}/MPI^{1.11}$).

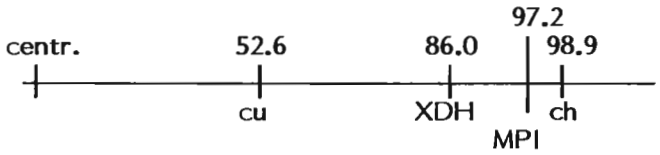
Table 1.

Genotype of female parent	Markers	Cross-overs	Sample size	Recombination value	Map distance
1. O_{3+4}, cu, $MPI^{1.11}$, ch	cu-MPI	102	276	0.370±0.029	0.461
O_{3+4}, + $MPI^{1.00}$, +	MPI-ch	3	276	0.011±0.006	0.011
2. O_{3+4}, cu, $XDH^{1.00}$, $MPI^{1.11}$, ch	cu-XDH	53	185	0.286±0.033	0.327
O_{3+4}, +, $XDH^{0.94}$, $MPI^{1.00}$, +	XDH-MPI	20	185	0.108±0.023	0.112
	MPI-ch	5	185	0.027±0.012	0.027

Therefore, the enzyme locus MPI is located on chromosome O.

In order to construct a genetic map of the region of chromosome O in which the MPI is located, we analyzed the progeny of two relative crosses: For each cross we give all the data pertaining to the estimation of the recombination distances (shown in Table 1).

The male parent of each cross was cytologically and genically homozygous. The map distance was corrected for multiple crossovers using the formula proposed by Haldane (1919): $x=0.7y-0.15\log_e(1-2y)$, where y is the percentage of crossovers. Pooling the data of crosses 1 and 2 (that gave homogenous results) and taking into account map distances from previous reports (Loukas et al. 1979; Zouros & Krimbas 1973), we have for the chromosome O of **D.subobscura:**



In order to study the genetic differentiation of MPI-locus in Drosophila species of the obscura group, we used eight different isofemale strains for **D.subobscura;** one for **D.madeirensis;** one for **D.guanche;** eleven for **D.obscura;** three for **D.tristis;** seven for **D.ambigua;** two for **D.subsilvestris;** three for **D.bifasciata;** one for **D.pseudoobscura** and one for **D.helvetica.** MPI classified the ten species into six different electrophoretic phenotypes. **D.madeirensis, D.obscura** and **D.subsilvestris** displayed the allele 1.00. Four strains of **D.subobscura** were homozygous for allele 1.00, two for allele 1.07 and two for allele 1.11. **D.guanche** and **D.tristis** displayed the allele 1.03. Four strains of **D.ambigua** were homozygous for allele 1.03 and three for allele 0.98. Finally, **D.bifasciata, D.pseudoobscura** and **D.helvetica** displayed the alleles 1.09, 1.11 and 1.16, respectively.

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Lovering, R. Sheffield University, Sheffield, England. Morphology of the proximal region of 3R in salivary gland polytene nuclei of **D.melanogaster.**

In the polytene chromosome 3R of **D.melanogaster** the region proximal to 81F was described by Bridges (1935-1940) and by Lefevre (1976) as heterochromatic. Heterochromatin is characteristically unbanded and granular in appearance, but the Bridges's maps, and electronmicrographs by Saura (pers. comm. 1979), suggest that some heterochromatic bands can be identified. In Lefevre's photographic representation of chromosome 3R the most proximal band illustrated is the doublet 81F1-2. However, Lefevre also suggests that heterochromatic material is present between 81F and 80F.

Electron micrographs produced by Saura suggest that bands proximal to 81F do exist, and these they have labelled 80B-F. Nevertheless, the bands are heterochromatic, maintaining some of the granular appearance characteristic of this region. The morphology of 81F in the electron micrographs suggests that it may contain heterochromatin, as the bands are more dense than euchromatic bands and, like the chromocentre, it is strongly fluorescent when stained with quinacrine; this suggests that it contains a high frequency of A-T base pairs (Barr & Ellison 1972). This region cannot, therefore, be considered as strictly euchromatic.

I have been investigating the DNA content of the proximal region of 3R by Feulgen staining of salivary gland nuclei, and have observed that the morphology of the region proximal to 81F varies from cell to cell. Four different morphological categories can be detected (see Fig. 1).

(a) The doublet in 81F is closely associated with the heterochromatic chromocentre; it is not possible to define the proximal boundary of the band.

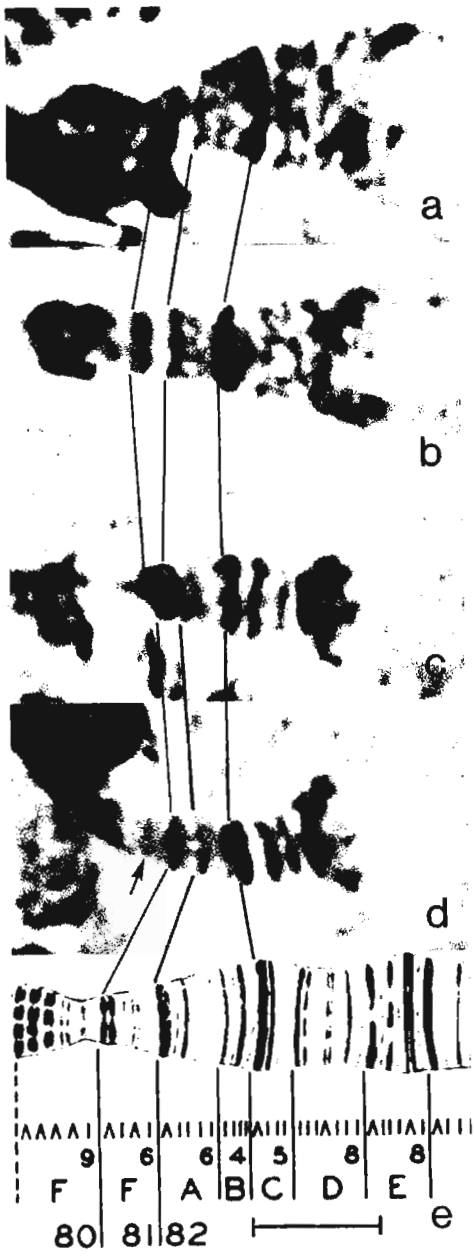

Figure 1. The proximal region of 3R; (e) Bridges's map 1941; the arrow indicates an extra band proximal to 81F1-2. The bar corresponds to 5μ.

Table 1. The effect of larval incubation temperature on the morphology of the region proximal to 81F.

temp.	Morphology of the proximal region of 3R a	b	c	d	total nuclei	No. slides
15°C	23	12	11	4	50	5
25°C	10	6	18	16	50	5
Total	33	18	29	20	100	10

$X^2 = 16.01$; D.O.F. = 3; $p < 0.01$.

(b) An interband is visible between the doublet and the heterochromatin; the proximal boundary of the band can be defined.

(c) 81F1-2 is attached to the chromocentre by a thin thread-like structure, as seen in Lefevre's photographs (1976). The doublet itself is not usually distorted by this condition.

(d) One or two bands are visible proximal to 81F1-2 and distinct from the heterochromatin. It is also possible that these bands are 81F4-5 and that 81F1-2 is very closely associated with the chromocentre.

The morphology of the proximal region of 3R was studied in larvae reared at different incubation temperatures. The distribution of nuclei among the morphological classes was significantly affected by the temperature at which the larvae were reared ($p<0.01$, see Table 1). In larvae reared at 15°C the double band 81F1-2 was closely associated with the chromocenter (type a) in almost half of the nuclei, whereas only a fifth of the nuclei from 25°C-reared larvae fell into this class. In addition at 25°C four times more class d nuclei were observed, with bands proximal to 81F, than at 15°C.

Using the light microscope it is not possible to determine whether the extra bands observed proximal to 81F originate from 81F or from the chromocentre. As the larval incubation temperature has a significant effect on the morphology of this region, it seems unlikely that the presence or absence of bands proximal to 81F is an artifact.

The heterochromatic chromocentre usually replicates twice, without cell division (Rudkin 1969), whereas the euchromatin may initiate between 8 and 11 S-phases. In chromosome 3R the bands 81F1-2 are in, or very close to, a region where the transition from euchromatin to heterochromatin occurs. At the junction between the two different types of chromatin, a large number of replication forks are thought to be present (Laird 1973) because of the substantial change in polyteny.

Studies on the Feulgen-DNA content of the polytene nuclei (Hartmann-Goldstein & Goldstein 1979, and unpubl.) have shown that an increase in larval incubation temperature causes a decrease in polyteny. In larvae reared at 15°C the euchromatin is usually at a higher polytene level than in 25°C reared larvae. Thus the effect of temperature observed on the morphology of 3R may be due to the effect of temperature on the polytene levels in the nucleus. If the increase in the DNA content occurred only in the euchromatin, and not the heterochromatin, then at the proximal regions of the chromosomes there would be an increase in the number of replication forks. The alignment of chromomeres might be disrupted in a region where many replication forks occur, so causing the banding pattern to be disorganised; these bands might then appear granular.

In nuclei of 25°C-reared larvae, bands are observed proximal to 81F more frequently than in 15°C larvae. An increase in euchromatin polyteny, due to a decrease in the larval incubation temperature, could cause the loss of banding pattern proximal to 81F by increasing the stress imposed on the structure by the large numbers of replication forks. Alternatively, the structural proteins themselves might be affected by the temperature.

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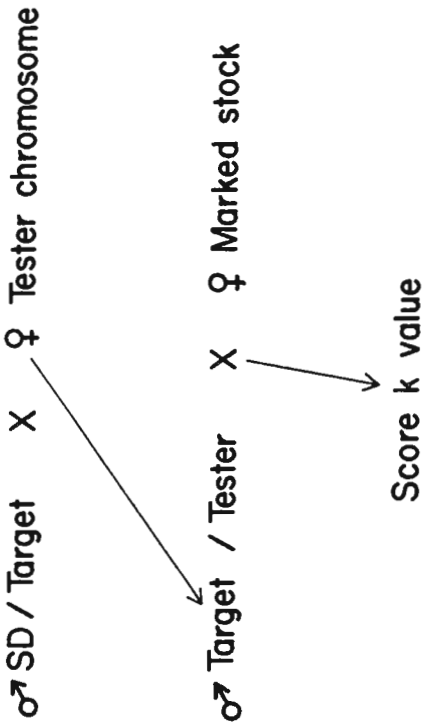
Loverre, A.,[1] D.A. Hickey* & G. Carmody.§
Universita di Roma, Italy; *University of Ottawa, Canada; §Carleton University, Ottawa, Canada. [1Present address: Biology Dept., University of Ottawa, Ont. K1N 6N5 Canada] A test of the hypothesis that the Segregation Distortion phenomenon in Drosophila is due to recurrent active genetic transposition.

The Segregation Distorter phenomenon in **Drosophila melanogaster** is characterized by the preferential recovery of Distorter (SD) second chromosomes in the progeny of heterozygous males (Sandler, Hiraizumi & Sandler 1959; see also Hartl & Hiraizumi 1976; Sandler & Golic 1985, for reviews).

The Distortion system consists essentially of two interacting sites, Sd and Rsp, which are closely linked and located near the centromere of chromosome II. Alleles at the Responder locus may be Responder-sensitive (Rsp^S) or Responder-insensitive (Rsp^i); SD chromosomes normally carry the Rsp^i allele at their responder site. Distortion occurs when an SD chromosome is made heterozygous with a chromosome of the genotype Sd^+ Rsp^S. Gametes carrying the Rsp^S allele from an Sd/Sd^+ male are rendered dysfunctional. A molecular model to explain the interaction between the Sd and Rsp loci was proposed by Hartl (1973) and some modifications have been suggested by Ganetsky (1977), Brittnacher & Ganetsky (1983) and Hiraizumi, Martin & Eckstrand (1980).

A new model of Segregation Distortion in Drosophila has been proposed (Hickey, Loverre & Carmody 1986) and experimental results are presented here as a test of the proposed model. According to the model, the Segregation Distorter (Sd) locus is the residence site of a transposable element, while the responder (Rsp) locus is a target site for insertion of copies of this element. Insertion of the transposon causes disruption of the sensitive chromosomes which, in turn, leads to the dysfunction of non-SD-bearing sperm. The experimental work is aimed at finding evidence of chromosomal contamination by the Sd element of non-Sd chromosomes which had been combined with SD chromosomes in heterozygotes. The non-SD chromosomes were then tested for the acquired ability to induce distortion, a phenotype which would indicate the presence of an acquired Sd element.



The one major complication in this test is the following. According to the model, the non-SD homologue is normally contaminated at the Rsp site and this event leads to sperm dysfunction. Therefore we can only hope to pick up transposition events at secondary sites. Secondly, because we use SD-activity as a measure of chromosome contamination, any transposition events which involved rearrangement of transposon sequences or insertion in chromosomal regions which did not allow further high-frequency transposition, would not be detected. Moreover, Sd elements on naturally-occurring SD chromosomes are surrounded by a co-adapted series of elements that allow a high degree of distortion against sensitive chromosomes. Thus, although a positive result would strongly support the genetic transposition model, a negative result would not rule it out.

Most experiments followed the scheme outlined in Figure 1. SD chromosomes were made heterozygous with non-SD "target" chromosomes. These non-SD chromosomes, which had been exposed to SD activity, were then made heterozygous with other non-SD chromosomes ("tester" chromosomes) and the heterozygous males mated to appropriately marked females to calculate a k-value, i.e., the ratio of target chromosome-bearing progeny to total progeny.

The SD chromosomes used in this study were SD-5, SD-72 and SD-Madison. All three chromosomes display high levels of distortion ($k \geq 0.98$) when heterozygous with sensitive chromosomes.

Figure 1. Scheme of crosses to recover SD+ chromosomes after exposure to SD, and for testing them for distorting ability.

Table 1. Distribution of k-values for potentially contaminated and control chromosomes. Each cross in this case was between one bw/lt bw male and two lt bw/lt bw females.

SD/Target	Target/Tester	no. males tested	Total progeny	k±st.dev.	Males with k>0.70
SD-5/bw	bw/lt bw	8	629	0.65±0.10	3
SD-72/bw	bw/lt bw	9	877	0.59±0.12	2
Control (bw/bw)	bw/lt bw	14	948	0.58±0.08	1

Table 2. Average k-values for insensitive target chromosomes when tested in heterozygosis with sensitive tester chromosomes. For estimating k values, males heterozygous for cn bw were crossed with cn bw/cn bw females, while males heterozygous for lt bw were crossed with lt bw /lt bw females.

Target/Tester Combination	Average k-value ±st.dev.	Range (min-max)	No. Chromosomes tested	Total progeny scored	Males with k>0.65
pr cn/cn bw	0.49±0.06	0.36-0.63	37	2558	0
Df(2R)14C/lt bw	0.50±0.05	0.37-0.61	20	1372	0
Df(2R)2J/ lt bw	0.53±0.07	0.38-0.76	40	2543	3
R(Cy)-40/cn bw	0.52±0.07	0.37-0.75	122	7833	4
Control*					
[R(Cy-40/cn bw]	0.49±0.08	0.33-0.63	19	1752	0

*The "control" genotype is identical to the preceding R(Cy)-40/cn bw combination. In the case of the control experiments, however, the R(Cy)-40 chromosome was not heterozygous with an SD chromosome in the previous generation (see Fig. 1).

Single crosses (1 male X 2 females) were carried out for measuring k values.

All stocks and crosses were maintained at 25±1°C.

For a full description of the chromosomes and markers used, see Lindsley & Grell (1968).

I. Testing for chromosomal contamination in rare escaper males. The first set of experiments tested for contamination of Sd^+ Rsp^s chromosomes in SD/SD^+ males showing a k value greater than 0.9. The bw chromosome, isolated from a bw st laboratory stock, allows a high degree of distortion by SD-5 or SD-72 ($k \geq 0.98$). Aged males of the genotypes SD-5/bw (k=0.96) and SD-72/bw (k=0.93) were crossed to lt bw homozygous tester females. The use of older males results in an increased recovery of bw chromosomes. From among the progeny, bw/lt bw males were recovered and examined. In this case the bw chromosome represents the target chromosome and the lt bw is a more sensitive tester chromosome. The results are presented in Table 1. The prediction of the model is that contaminated target chromosomes might distort ratios in their own favor when heterozygous with the very sensitive lt bw chromosome. As can be seen from Table 1, some bw chromosomes do indeed show evidence of an ability to distort against lt bw (k>0.7) and this is reflected in the average k value of 0.61 which is slightly higher than the average for the control cross (k=0.58). The control in this case is an identical cross using bw chromosomes which had not been previously exposed to an SD chromosome. At first glance, the results indicate that some but not all of the bw chromosomes show some evidence in favor of contamination by Sd and that the distorting ability of these chromosomes is low relative to that of the original SD-5 or SD-72. The potentially contaminated chromosomes were retested for the heritability of this effect in subsequent generations. Three bw chromosomes showing high k-values (k>0.7) and one with a low k-value ($k \leq 0.45$) were tested in subsequent generations. There was a significant heritability of low and high k-values among the F1 progeny but the difference between the high and low lines tended to disappear in subsequent generations. The results indicate that the abnormal k-values, although heritable are not stably inherited.

A parallel set of experiments which tested the ability of target cn bw chromosomes, descended from SD-72/cn bw aged males, to induce self-distortion when heterozygous with the pr cn insensitive chromosome resulted in k-values that were close to 0.5 (data not shown). Contaminated chromosomes would show suicide behaviour when heterozygous with the insensitive pr cn tester chromosome. This result might reflect a lack of chromosomal contamination or it might be due to the presence of suppressors of SD on the pr cn tester chromosome.

II. Testing for chromosomal contamination in females. There is the formal possibility that the Sd-Rsp interaction occurs in heterozygous females as well as in heterozygous males, but that there is no detectable phenotypic effect in the case of females. If this were the case, the chromosomes which were heritably changed in the heterozygous females might show the phenotypic effects of this change in the male offspring of these females. From SD-72/cn bw females, cn bw (Rsp sensitive) chromosomes were recovered and tested for the ability to induce self-distortion when heterozygous, in progeny males, with an Sd+ insensitive chromosome (pr cn or Df(2R)14C). Among a total of 36 chromosomes examined, no unusually low k-values were observed (data not shown). The lack of self-distortion in this case is best explained by the assumption that the lack of observed phenotypic effect in female gametes does reflect the absence of Sd activity in heterozygous females.

III. Testing for chromosomal contamination using insensitive target chromosomes. Sd+ second chromosomes insensitive to SD were tested for the acquired ability to distort a sensitive homolog in males after having been in heterozygosis with an SD chromosome. Our prediction was that if transposition of

Sd occurred in the heterozygous parental males, this could be expressed phenotypically as a distorting ability of the contaminated chromosome in the offspring males. The use of insensitive target chromosomes has two advantages. First, since the Rsp site is "blocked" all of the non-SD chromosomes can be recovered. Secondly, if these chromosomes acquire an Sd element at another site, they will have the genotype Sd Rsp^i and so can be used directly to test for distortion against sensitive tester chromosomes.

The insensitive target chromosomes used were pr cn, a laboratory insensitive chromosome, and two deficiencies for the heterochromatic 2R centric region where Rsp has been localized. In addition, the R(Cy)-40 chromosome was used because it carries a Rsp^i allele which originated from an SD chromosome, and it also includes E(SD) and M(SD), two major enhancers of SD activity. The results are summarized in Table 2. Of the 37 pr cn chromosomes tested, after having been in heterozygosis with SD-5, none showed k-values greater than 0.65. Generally, the values were normally distributed about a mean value of 0.49. Those chromosomes which gave the most extreme values (highest or lowest) were re-tested in the following generation; there was no evidence for heritability of the more extreme values. These negative results could again be explained by postulating the presence of suppressors of SD on the pr cn chromosome. The problem of possible suppressors on the target chromosome can be circumvented by using the deficiencies of the Rsp region, Df(2R)14C, bw and Df(2R)2J, bw. The two deficiencies used have been shown to induce a significant degree of suicide behaviour in Sd Rsp^s chromosomes (Ganetsky 1977; Sharp et al. 1985). Target Df(2R) chromosomes were recovered from Df(2R)/SD-5 heterozygotes and tested for their ability to distort the sensitive lt bw tester chromosome. Again, there is no evidence that the majority of these chromosomes acquired a distorting ability subsequent to the exposure to SD chromosomes. There were however three chromosomes among the sixty tested which gave k-values greater than 0.65. When these three chromosomes, which gave an average k-value of 0.72 were re-tested in the following generation the average k-value for eight progeny lines was 0.57 and the range was from 0.50 to 0.67. This shows that there is some degree of heritability of these elevated k-values. The final test for chromosomal contamination in heterozygous males was performed using the R(Cy)-40 chromosome (Hartl 1980). This is an Sd+ chromosome that through recombination has acquired the centromeric region and right arm from the SD-36 chromosome. The presence of E(SD) and M(SD) on this chromosome should enhance their distorting ability if they acquire an Sd element from the SD chromosome. The mean k-value, after exposure to SD-5 and SD-MAD, of the 122 chromosomes tested, 0.52±0.57 was not significantly higher than the controls. Among this set, however, were several chromosomes with k-values greater than 0.65. The maximum k-value in the control is 0.63 whereas three experimental chromosomes showed values greater than 0.65. The chromosome which showed the highest k-value (0.75) was tested again in nine heterozygous F1 offspring males; there was a large variation in k-values among these progeny (range: 0.32-0.71) and one of the nine had a k-value that was again greater than 0.7. One possible explanation for this proportion of partially heritable high k-values would be the presence of an acquired but unstable Sd element. Because of the possibility that E(SD) might show some residual SD (or meiotic drive) activity (Sharp et al. 1985), we compared our results to a control carrying an identical R(Cy)-40 E(SD) chromosome, which had not been exposed to SD activity in the previous generation.

Our proposal for an underlying mechanism for segregation distortion suggests that the Sd element was originally inserted into SD chromosomes by a genetic transposition event in the past, and, moreover, that the distortion we observe is caused by high-frequency site-specific transposition of copies of this element into sensitive homologous chromosomes.

In our experiments, a few combinations gave evidence of having acquired distorting ability. In no case did we succeed in isolating a line which repeatedly, over many generations, gave high levels of distortion. These results indicate to us that the chromosome contamination effect we were looking for may occur, but at low frequency and that the altered chromosomes are genetically unstable.

The major difficulty in demonstrating chromosomal contamination by copies of an Sd element is analogous to the problem of isolating dominant lethal mutations. That is, the event of interest may take place at detectable frequencies but one cannot recover the mutant products. In the case of the SD system, if normal transposition is into the Rsp site, we can only hope, in these experiments, to recover secondary transpositions into other chromosomal sites. Although the model predicts very high levels of transposition and insertion into the Rsp^s site, we have no way of predicting the rate of insertion at other sites. Secondly, because of the nature of our biological assay for the presence of Sd, many possible insertions would go undetected. Sd insertions which did not subsequently lead to high levels of transposition into the Rsp site of the tester chromosome would go undetected. In addition, it is not unreasonable to suspect that the levels of SD activity may be subject to position effects. Given these considerations, the results we obtained here are encouraging.

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Maiti, A.K. and A.K. Ghosh. University of Calcutta, India. Hybrid dysgenesis in **Drosophila ananassae.**

Hybrid dysgenesis is a syndrome of correlated genetic traits that is spontaneously induced in hybrids between certain mutually interacting strains, usually in one direction only (Kidwell 1979). Dysgenic traits are: mutation, chromosomal aberration, distorted segregation, sterility, etc. Using sterility as an indicator of hybrid dysgenesis, we have tested some long established laboratory strains and some newly caught wild type strains of **D.ananassae** to see whether or not similar type of dysgenesis occurs in the hybrids of these strains like that of **D.melanogaster.** Twelve newly collected strains (from different regions of India) were tested within which two strains behaved like inducer or P-strain.

Table 1. Hybrid sterility test.

F_1-Hybrid	Total ♀ tested	Total no. of eggs	Total no. of adults	% of hatching	Complete sterile	% complete sterile
0085 ♀ X Belur ♂	65	2462	1209	49.11%	13	20%
0085 ♂ X Belur ♀	54	3124	2334	74.71%	1	1.8%
a^{cal} ♀ X Garia ♂	55	1862	862	46.28%	11	20%
a^{cal} ♂ X Garia ♀	42	1435	1176	81.8%	0	0%

Strain Belur and Garia (West Bengal) were used as P-strain and 0085 and a^{cal} stocks are maintained in our laboratory, used as long established laboratory stocks.

When the females of a^{cal} or 0085 were crossed with Garia or Belur male, then some F_1 females showed reduced or absence of fertility. But in the hybrid females of the reciprocal cross, no such trait was found (see Table 1).

On examining the gonads of the complete sterile female were observed that they were of dysgenic form. If such dysgenesis is due to the presence of some P-element like factors, like that of **D.melanogaster**, then it can be concluded that **D.ananassae** must conserve the P-element in its genome in the course of evolution.

Reference: Kidwell 1979, Genet. Res. Camb. 33:205-217.

Manousis, T.H. and C.D. Kastritsis. University of Thessaloniki, Greece. Electrophoretic analysis of polypeptide and mucopolysaccharide content of several tissues and organs of **Drosophila auraria** larvae.

In the course of an investigation dealing with gene activities in **Drosophila auraria**, we have included the study of the polypeptides, and PAS-positive polypeptides of several late 3rd instar and prepupal tissues and organs. The study was carried out by SDS polyacrylamide gel electrophoresis followed by PAS and C.B. staining. PAS-positive polypeptides are tissue specific and are different from those of the salivary glands of the same animal at the developmental stages studied (Manousis & Kastritsis, submitted). Some quantitative and qualitative changes of the polypeptide content can be observed during stages expected to coincide with a high titre of ecdysterone in the hemolymph. Hemolymph contains a group of polypeptides (Fig. 1, Ha) present in the fat tissue (Fig. 1, FBa) which in the case of other diptera produces the dominant polypeptides of the hemolymph.

Acknowledgements: This work was supported by a grant from Volkswagenwerk Stiftung to C.D.K.

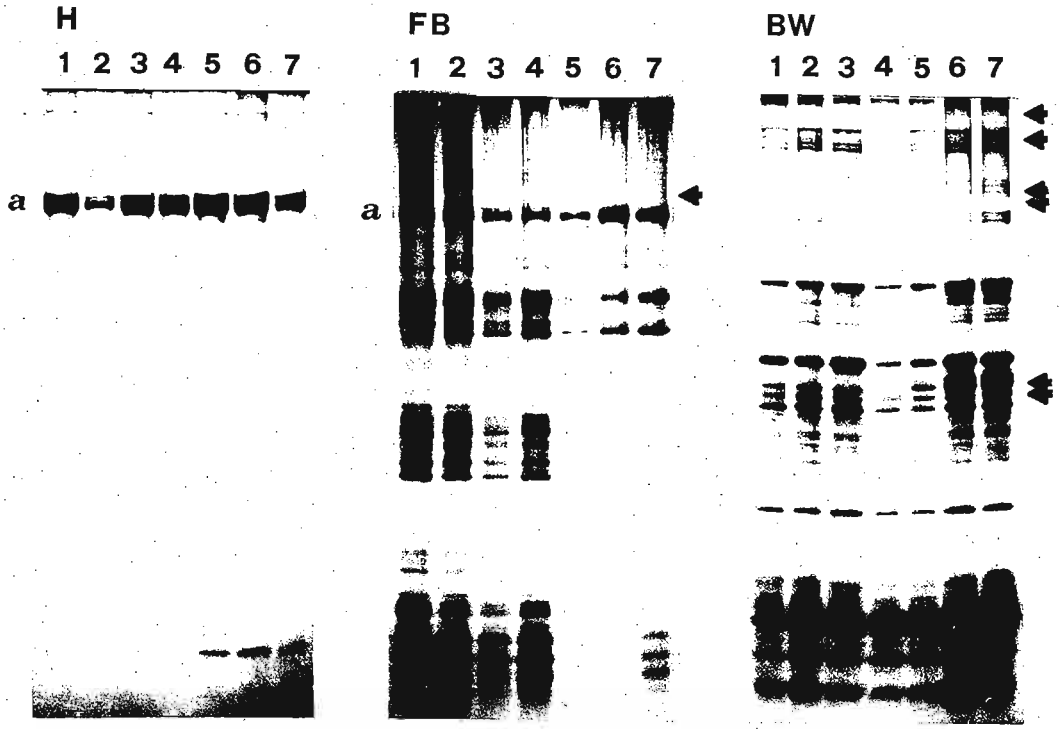


FIgure 1a

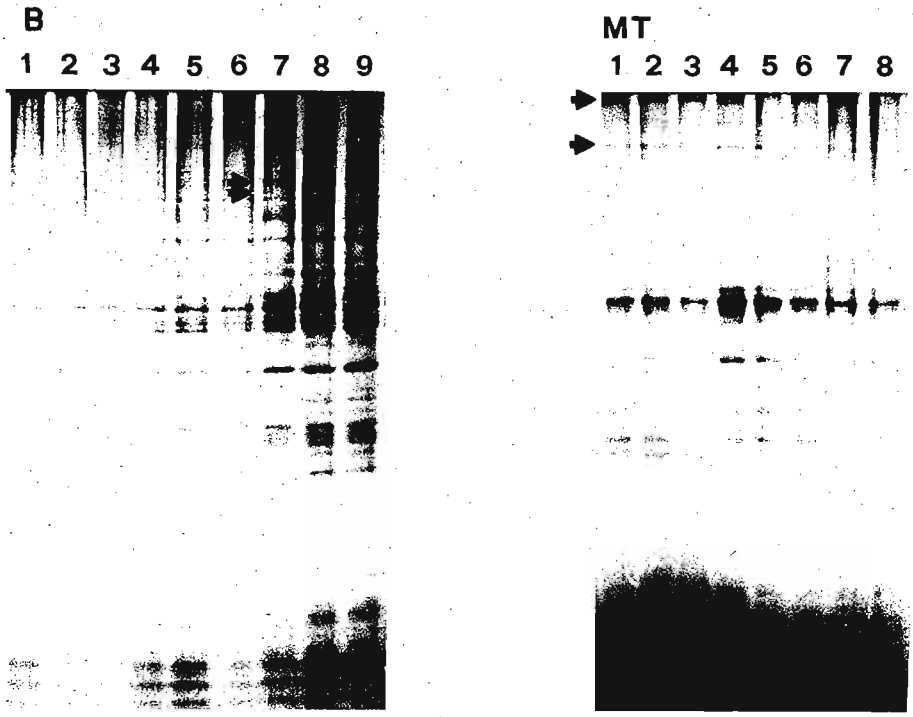
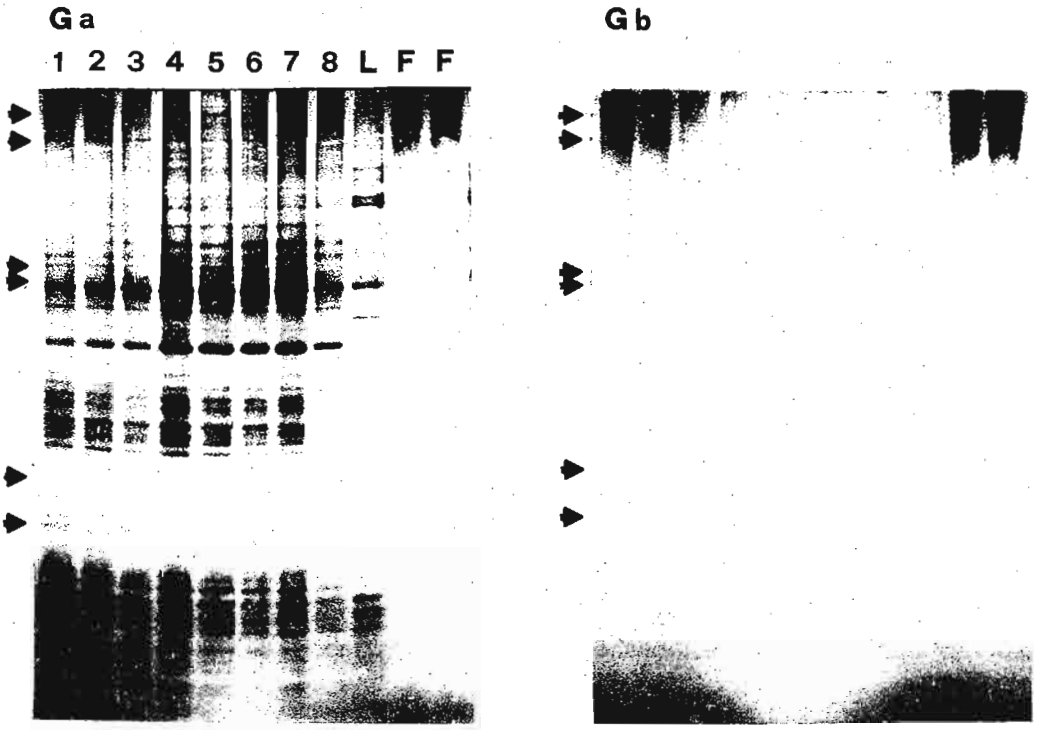


Figure 1b

Figure 1. Electrophoretic profiles of polypeptides of tissues and organs of 20 individuals per sample, after PAS-staining followed by C.B. staining. The arrows indicate the position of the PAS-positive polypeptides which in the case of the guts are shown, as an example, on the PAS stained gel (Gb). The numbers indicate the developmental stages as defined by Scouras & Kastritsis, **L.** whole larva, **F.** food extract, **H.** Hemolymph, **FB.** Fat Body, **BW.** Body Wall, **B.** Brains, **MT.** Malpigian Tubules, **Ga.** Guts after C.B. staining, **Gb.** Guts after PAS-staining of the same gel.

References: Kilby, B.A. 1963, Adv. Insect Physiol. 1:111-174; Manousis, T.H. & C.D. Kastritsis, submitted; Scouras, Z.G. & C.D. Kastritsis 1984, Chromosoma (Berl) 89:96-106; Tissieres, A. et al. 1974, J. Mol. Biol. 84:389-398; Zacharius, J.J. et al. 1969,Anal. Biochem. 30:148-152.



Manousis & Kastritsis:

Figure 1c

Mather, W.B. and R. Casu. University of Queensland, St. Lucia, Australia. Inversions from Phuket, Thailand. 8th Report.

Table.

Inversion	Chromosome	Simple	Complex	Het. Freq. %
A5	II L	x		48
C5	II R	x		64
C1	III	x		48
F3	IIi	x		12
P5	III	x		4
W2	III	x		10
X2	III		x	4

In January 1984 twenty-five isolines of **D.s.albostrigata** and two isolines and two isolines of **D.albomicans** were established from Phuket, Thailand. The inversions from the region were last reported on from a collection made in July 1984 (Mather & Pope, DIS 60:143).

(a) **D.s.albostrigata.** Six simple and one complex inversion were detected (Table). All inversions had previously been detected from Southeast Asia but X2 was new to Phuket. The heterozygosity frequency of all inversions detected is given in the Table.

(b) **D.albomicans.** Five simple inversions, E' on chromosome II L and B6, C1, L3 on chromosome III were detected. All had previously been recorded from Phuket.

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by R.C.

Mather, W.B. and R. Casu. University of Queensland, St. Lucia, Australia. Inversions from Phuket, Thailand. 9th Report.

In July 1984 twenty-seven isolines of **D.s.albostrigata,** eight isolines of **D.albomicans** and one isoline of **D.kohkoa** were established from Phuket, Thailand. The inversions from this region were last reported on from a collection made in January 1984 (Mather & Casu, DIS this issue).

(a) **D.s.albostrigata.** Five simple and two complex inversions were detected (Table 1).

Six of the seven inversions had previously been detected from Phuket. A photograph of a new inversion (D7) is presented with breakpoints 26.7-38.0 assigned in relation to the standard photographic map (Thongmeearkom 1977, DIS 52:154).

The heterozygosity frequency of all inversions detected is given in Table 1.

Table 1.

Inversion	Chromosome	Simple	Complex	Het. Freq. %
A5	II L	x		66.7
D5	II L		x	3.7
C5	II R	x		66.7
C1	III	x		51.5
F3	III	x		3.7
W2	III	x		63.0
D7	III		x	3.7

Table 2.

Inversion	Chromosome	Simple	Complex
E'	II L	x	
C1	III	x	
E3	III		x
E6	III		x
L3	III	x	
T4	III	x	

Figure. The free end of the chromosome is to the right.

(b) **D.albomicans.** Four simple and two complex inversions were detected (Table 2). All had been detected from southeast asia but F3 was new to Phuket.

(c) **D.kohkoa.** The one isoline analyzed proved to be inversion free.

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by R.C.

Mather, W.B. and R. Casu. University of Queensland, St. Lucia, Australia. Inversions from Phuket, Thailand. 10th Report.

Table 1.

Inversion	Chromosome	Simple	Complex	Het.Freq. %
G	I	x		5.0
A5	II L	x		50.0
D5	II L		x	10.0
Q5	II L	x		5.0
C5	II R	x		85.0
C1	III	x		56.0
F3	III	x		5.0
W2	III	x		35.0
X2	III		x	5.0

Table 2.

Inversion	Chromosome	Simple	Complex	Het.Freq. %
R5	I	x		21.1
E'	II L	x	x	31.6
J6	II L			5.3
B6	III	x		36.8
C1	III	x		68.4
E6	III		x	73.7
L3	III	x		26.3
W5	III	x		5.3
E7	III	x		5.3

Figure. The free end of the chromosome is to the right.

In January 1985 twenty isolines of **D.s.albostrigata** and nineteen isolines of **D.albomicans** were established from Phuket, Thailand. The inversions from this region were last reported on from a collection made in July 1984 (Mather & Casu, DIS 63 this issue).

(a) **D.s.albostrigata.** Seven simple and two complex inversions were detected (Table 1). All the inversions had previously been detected from Southeast Asia but G and Q5 were new to Phuket. The heterozygosity frequency of all inversions detected is given in Table 1.

(b) **D.albomicans.** Seven simple and two complex inversions were detected (Table 2). Of these J6 and W5 were new to Phuket. A photograph of a new inversion (E7) is presented with breakouts 19.7-24.2 assigned in relation to the standard photographic map (Mather & Thongmeearkom 1980, DIS 55:101). The heterozygosity frequency of all inversions detected is given in Table 2.

Mather, W.B. and A.K. Pope. University of Queensland, St. Lucia, Australia. Inversions from Chiang Mai, Thailand. 6th Report

Table.

Inversion	Chromosome	Simple	Complex	Het.Freq. %
A5	II L	x		48.1
E	II L	x		14.8
C7	II L		x	3.7
D5	II L		x	3.7
C5	II R	x		7.4
C1	III	x		3.7
B5	III	x		14.8

In July 1985 twenty-seven isolines of **D.s.albostrigata** and three isolines of **D.albomicans** were established from Chiang Mai, Thailand. Inversions in these species were last reported on from Chiang Mai in July 1984 (Mather & Pope, DIS 61:116).

(a) **D.s.albostrigata.** Five simple and two complex inversions were detected (Table). All inversions had previously been detected from Chiang Mai. The heterozygosity frequency of all inversions detected is given in the Table.

(b) **D.albomicans.** Three simple inversion (E', S5 and A7) on chromosome II L were detected. All had previously been detected from Chiang Mai.

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by A.K.P.

Mather, W.B. and A.K. Pope. University of Queensland, St. Lucia, Australia. Inversions from the River Kwai, Thailand. 8th Report.

Table 1.

Inversion	Chromosome	Simple	Complex	Het.Freq. %
A5	II L	x		73.7
E'	II L	x		15.8
D5	II L		x	10.5
C5	II R	x		63.2
C1	III	x		10.5
B5	III	x		5.3

Table 2.

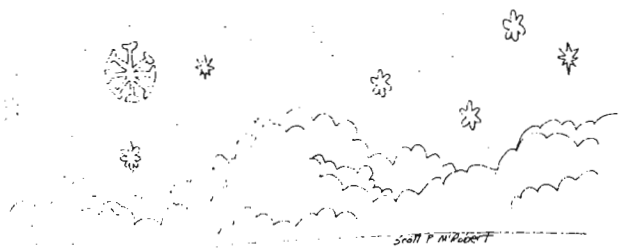
Inversion	Chromosome	Simple	Complex	Het.Freq. %
R5	I	x		8.7
E'	II L	x		8.7
S5	II L	x		39.1
C1	III	x		17.4
L3	III	x		13.0
E6	III		x	67.6

In July 1985 nineteen isolines of **D.s.albostrigrata** and twenty-three isolines of **D.albomicans** were established from the River Kwai region of Thailand. The inversions from the region were last reported on from a collection made in July 1983 (Mather & Pope, DIS 60:143).

(a) **D.s.albostrigata.** Five simple and one complex inversion were detected. All inversions have been previously found at the collection site. The heterozygosity frequency of the inversions detected is given in Table 1.

(b) **D.albomicans.** Five simple and one complex inversion were detected. All of the inversions had previously been found at the River Kwai. The heterozygosity frequency of the inversions detected is given in Table 2.

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by A.K.P.



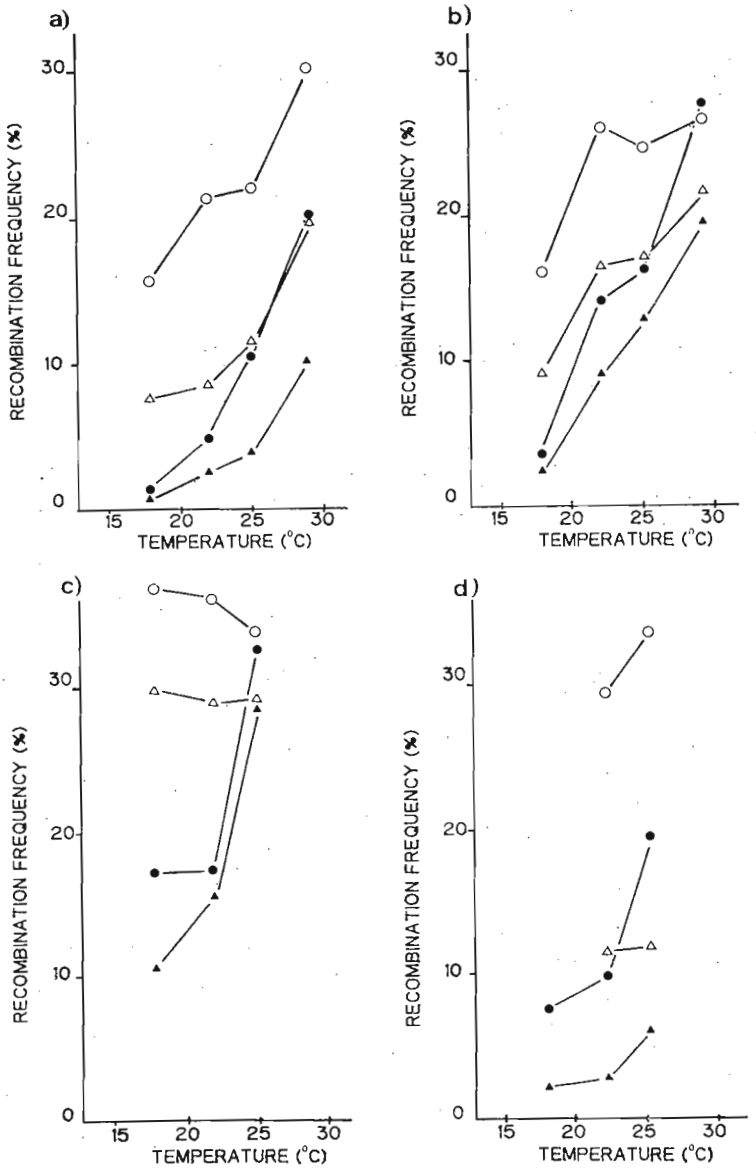


Figure 1. Recombination frequencies for chromosomes 2 and 3 in F_1 males derived from 8 crosses. Δ: Average recombination frequency in chromosome 2 in markers/wild F_1 males. ▲: Average recombination frequency in chromosome 2 in wild/markers F_1 males. 0: Average recombination frequency in chromosome 3 in markers/wild F_1 males. ●: Average recombination frequency in chromosome 3 in wild/markers F_1 males. (**a**) b se/+(TNG); bri ru/+(TNG) ♂ & +(TNG)/b se; +(TNG)/bri ru ♂. (**b**) b pea/+(L8); bri ru/+(L8) ♂ & +(L8)/b pea; +(L8)/bri ru ♂. (**c**) b pea/+(HW); bri ru/+(HW) ♂ & +(HW)/b pea; +(HW)/bri ru ♂. (**d**) b pea/+(TNG); bri ru/+(TNG) ♂ & +(TNG)/b pea; +(TNG/bri ru ♂.

References: Hinton, H. 1970, Genetics 66:663-676; 1974, in: Mechanisms in Recombination (Plenum); 1983, Genetics 104:95-112; Matsuda, M. & Y.N. Tobari 1983, Jpn. J. Genet. 58:181-191; Matsuda, M., H.T. Imai & Y.N. Tobari 1983, Chromosoma 88:286-292; Moriwaki, D., Y.N. Tobari & Y. Oguma 1970, Jpn. J. Genet. 45:411-420; Moriwaki, D. & Y.N. Tobari 1975, in: Handbook of Genetics (Plenum); Tobari, Y.N., M. Matsuda, Y. Tomimura & D. Moriwaki 1983, Jpn. J. Genet. 58:173-179.

Matsuda, M. Tokyo Metropolitan University, Tokyo, Japan. The effect of temperature on recombination in males of **D.ananassae.**

Recombination in males of **D.ananassae** has been proved to be meiotic in origin (Matsuda et al. 1983) and to be controlled by a variety of genetic factors (Moriwaki et al. 1970; Hinton 1970, 1974, 1983; Moriwaki & Tobari 1975; Matsuda & Tobari 1983; Tobari et al. 1983). Two marker stocks, b pea; bri ru and b se; bri ru, and 3 wild stocks, TNG, L8 and HW, were used in the present experiment, because each stock carries different genetic factors controlling recombination in males (Matsuda & Tobari 1983). The F_1 heterozygous males of 8 different genotypes were obtained by crossing marker females with wild males or vice versa, as shown in Fig. 1 legend. F_1 heterozygotes were denoted as markers/wild or wild/markers according to whether the marked chromosomes were derived from their mothers or fathers, respectively. F_1 males exposed to four different temperatures, 18°C, 22°C, 25°C and 29°C during the entire life cycle were obtained to test the effect of temperature on recombination in males.

The 10-25 F_1 males from each cross at each temperature were mated to 3-day old marker females individually at 22°C, and, after 4 days, the flies were transferred to new vials to let them lay eggs for another 4 days. Progeny from each vial were scored for recombination up to 20 days after egg laying.

Average recombination frequency in a given genotype is plotted at a given temperature in Fig. 1. Map distances for b-pea and b-se in chromosome 2 and bri-ru in chromosome 3 are about 15, 60, and 100, respectively. Only a few of F_1 males emerged from the cross b pea; bri ru ♀ x TNG ♂ at 18°C, and very rare progeny were obtained from F_1 males of b pea/+(HW); bri ru/+(HW), +(HW)/b pea; +(HW)/bri ru and +(TNG)/b pea; +(TNG)/bri ru at 29°C.

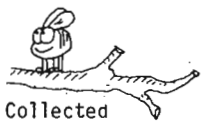
As shown in Fig. 1, elevated temperature enhanced meiotic crossing-over in males of **D.ananassae.** However, the effects of temperature were quite different between reciprocal F_1 males.

These results have led us to the experiment to localize the time of crossing-over in spermatocytes.

Acknowledgements: This study, supported by The Yamada Science Foundation, was done largely in the laboratory of Prof. James F. Crow whose generosity in giving help to the author is sincerely appreciated.

McRobert, S.P. and L. Tompkins. Temple University, Philadelphia, Pennsylvania USNA.

Drosophila species in the Philadelphia area.



As part of an ongoing study of the reproductive behavior of Drosophila, we have collected flies at various locations in the Philadelphia area. These surveys were conducted during the spring and summer of 1983 and the fall of 1985, in several surburban locations approximately 17 miles west of Philadelphia.

Our traps were clear plastic cups, baited with banana and live yeast or pumpkin and live yeast, that were suspended from trees or, in one case, an apartment balcony (see McRobert & Tompkins 1983, for a description of the trapping procedure). Flies were identified immediately after capture except for some females that appeared to be from species that could only be identified by keying males. In these cases, the females were maintained individually on cornmeal-molasses medium until their offspring emerged and the males could be identified. After identification, the flies were either killed, used to start laboratory cultures or preserved in alcohol.

Table 1. Species trapped in the Philadelphia area.

Species	No. of Individuals Collected Spring-Summer	Fall	Total
D.affinis	203	3	206
D.melanogaster	71	0	71
D.immigrans	1	69	70
D.duncani	8	32	40
D.robusta	19	6	25
D.tripunctata	14	2	16
D.quinaria	14	0	14
D.melanica	11	0	11
D.busckii	0	1	1

Two aspects of these data are of interest. First, for all species except **D.busckii**, in which only a single male was caught, the numbers of males and females collected were approximately equal. Second, the distribution of species was seasonal. **D.affinis** and **D.melanogaster** were the most common species in the spring and summer months, while **D.immigrans** was the most common species in the fall.

Acknowledgement: This research was supported by NIH Grant GM 33511 awarded to L.T.

Reference: McRobert & Tompkins 1983, DIS 59:143.

McRobert, S.P. and L. Tompkins. Temple University, Philadelphia, Pennsylvania USNA.

Drosophila species in Woods Hole, Massachusetts.

Although biological research has been conducted in Woods Hole for many years, we know of no descriptions of Drosophila species that are indigenous to this area. Therefore, during a recent stay, we surveyed the drosophilids at various sites on the grounds of the Marine Biological Laboratory (MBL).

Flies were trapped in plastic cups that were baited with banana and live yeast, then suspended from trees (McRobert & Tompkins 1983). Twenty collections were made between July 22 and August 7, 1985. In most cases, the flies were immediately keyed. However, since **D.affinis** and **D.algonquin** females cannot be distinguished, females that were from one of these two species were maintained individually on Carolina Instant Medium until their offspring emerged, at which time the males were keyed.

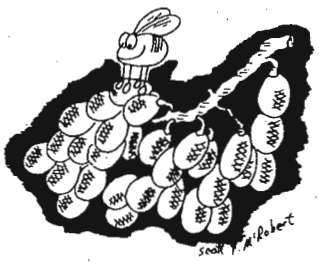
Table 1. Species trapped in Woods Hole.

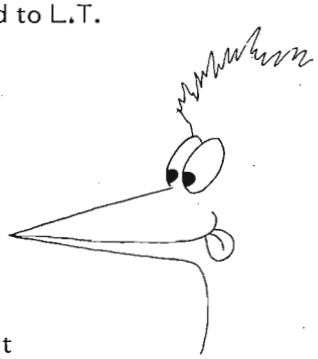
Species	No. Individuals Collected
D.affinis	68
D.busckii	5
D.immigrans	4
D.algonquin	3
D.robusta	3
D.quinaria	1
D.melanica	1

Surprisingly, in light of the fact that all of the traps were in windy areas within 300 yards of the ocean, all but one of the seven traps that we set attracted flies. Most of the flies that we collected from these traps are listed in Table 1. We also caught approximately 50 **D.melanogaster** males and females. However, since students in one of the MBL courses released laboratory flies into the environment, we do not know whether this species is indigenous.

Acknowledgement: This research was supported by NIH Grant GM33511 awarded to L.T.

Reference: McRobert & Tompkins 1983, DIS 59:143.





Graphics by Scott P. McRobert

Miklos, G.L.G.,[1] P.G.N. Kramers[2] and A.P. Schalet.[3] 1-Australian National University, Canberra, Australia; 2-Natl. Inst. of Public Hlth. & Env. Hygiene, Bilthoven, Netherlands; 3-State University of Leiden, Netherlands.

The proximal-distal orientation of two lethal complementation groups A112 and LB20 in region 19F at the base of the X chromosome.

The base of the X chromosome from maroonlike to suppressor of forked has been particularly well characterized in terms of contiguous lethal complementation groups, most of which have been unambiguously ordered (Schalet & Lefevre 1976; Lefevre 1981; Kramers et al. 1983). There are, however, some pairs of complementation groups in region 19F in which the proximal-distal orientation of lethals or visibles remains unresolved. These are pairs [lfl/B214], [sol/slg] and [A112/LB20] (Fig. 1). Small optic lobes (sol) and sluggish (slg) are two visible complementation groups (Fischbach & Heisenberg 1981; Ghysen & Benzer, unpubl.) which have recently been mapped into this region (Miklos, Kelly, Coombe, Leeds & Lefevre, unpubl.).

The major part of the mal-su(f) region has now also been microcloned, and a large number of clones have been assigned to single complementation groups (Miklos, Davies, Yamamoto & Pirrotta, unpubl.). However, those clones which have fallen into the pairs of unordered complementation groups remain unassigned at present. Finer localization of such clones would be possible if the order of complementation groups within a pair could be determined by chromosomal rearrangements.

During their studies on hycanthone methanesulphonate (HMS)-induced recessive lethal mutations, Kramers et al. (1983) found that HMS was very effective in inducing multilocus deficiencies in the proximal part of the X chromosome.

We have tested a number of these HMS-induced deficiencies to further refine their genetic breakpoints and report that a large deficiency, Df(1)HM44, has a proximal breakpoint between lethal complementation groups A112 and LB20, thus unambiguously orienting these two loci. The previous tentative order of LB20-A112 was based on deficiencies whose breakpoints required additional characterization (Lefevre 1981). When tested for allelism with various deficiency-bearing chromosomes as well as reference lethals and visibles, Df(1)HM44 yielded the following results (Fig. 1). It was allelic to Df(1)mal^{6}, Df(1)mal^{8}, Df(1)mal^{10}, Df(1)16-3-35, and Df(1)T2-14A. It complemented with Df(1)JC4, Df(1)su(f)4B, Df(1)JA27, and was covered by both y^{+}Ymal^{+} and y^{+}Ymal^{106}. Furthermore, Df(1)HM44 did not complement alleles at the following loci: l(1)17-234, l(1)17-457, mel, l(1)16-398, mal, leg, l(1)R-9-29, l(1)R-9-28, lfl, l(1)B214, l(1)W-2, sol and l(1)A112. It did however complement alleles of l(1)34, l(1)LB20, eo and l(1)114. Thus its proximal breakpoint separates l(1)A112 and l(1)LB20, whereas its distal breakpoint has yet to be more precisely localized.

We have also examined a number of alleles of the complementation groups near to the proximal breakpoint and these have behaved as expected. lethals 11P1 and 17-62, which are alleles of l(1)A112, were included within Df(1)HM44, whereas lethal DA618, an allele of l(1)LB20, was excluded from Df(1)HM44. Df(1)HM44 has already been most useful to us in mapping clones deep in region 19F. Its proximal

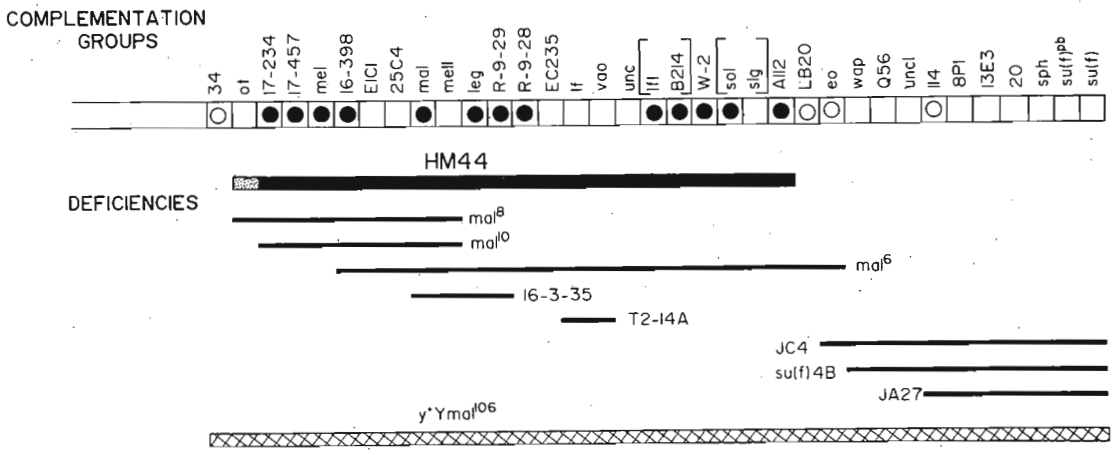


Figure 1. The genetic boundaries of Df(1)HM44 as determined by complementation tests involving the y^{+}mal^{106} duplication, and reference lethals and visibles in divisions 19 and 20. Alleles of the tested lethal and/or visible complementation groups which fall into Df(1)HM44 are indicated by closed circles, those which complement this deficiency are shown as open circles. It should be noted that Df(1)JA27 is a double deficiency, with another deletion in the 18A5; 18D1-2 area.

breakpoint should also provide a landmark for future chromosomal walks in this area.

Finally, two further points need to be made. First, some additional visible and lethal complementation groups (or complex loci) may still need to be added to the 19-20 region as more data become available. l(1)EC235 is a new lethal complementation group in 19E (Fig. 1) and there may well be an additional lethal between EC235 and lf (Miklos, unpubl.). Tumorous head (Pyati 1976) and l(1)EA41 (Lefevre, unpubl.) are two further mutants awaiting accurate assignations. Second, it should be remembered that su(f)^{pb} and su(f) are not a pair of complementation groups. While l(1)R-9-18 and l(1)3DES are indeed two lethal complementation groups in the su(f) complex, their situation is very probably an example of allelic complementation.

Acknowledgements: We thank Professors G. Lefevre, E. Munoz, S. Benzer and K. Fischbach for the provision of stocks.

References: Fischbach, K.F. & M. Heisenberg 1981, PNAS 78:1105; Kramers, P.G.N., A.P. Schalet, E. Paradi & L. Huiser-Hoogteyling 1983, Mutation Res. 107:187; Lefevre, G. 1981, Genetics 99:461; Pyati, J. 1976, Mol. gen. Genet. 146:189; Schalet, A.P. & G. Lefevre 1976, in: Genetics and Biology of Drosophila (Ashburner & Novitski, eds.), Academic Pr. 1b:848.

Moltó, M.D. and M.J. Martínez-Sebastián.
University of Valencia, Spain. Gene arrangements in polytene chromosomes of **Drosophila guanche** differing from standard arrangements in **Drosophila subobscura.**

Drosophila guanche, which is endemic to Laurel forest of the Canary Island, is a member of the Obscura group. The karyotype of **Drosophila guanche** resembles that of **Drosophila subobscura** which is thought to have the karyotype most closely

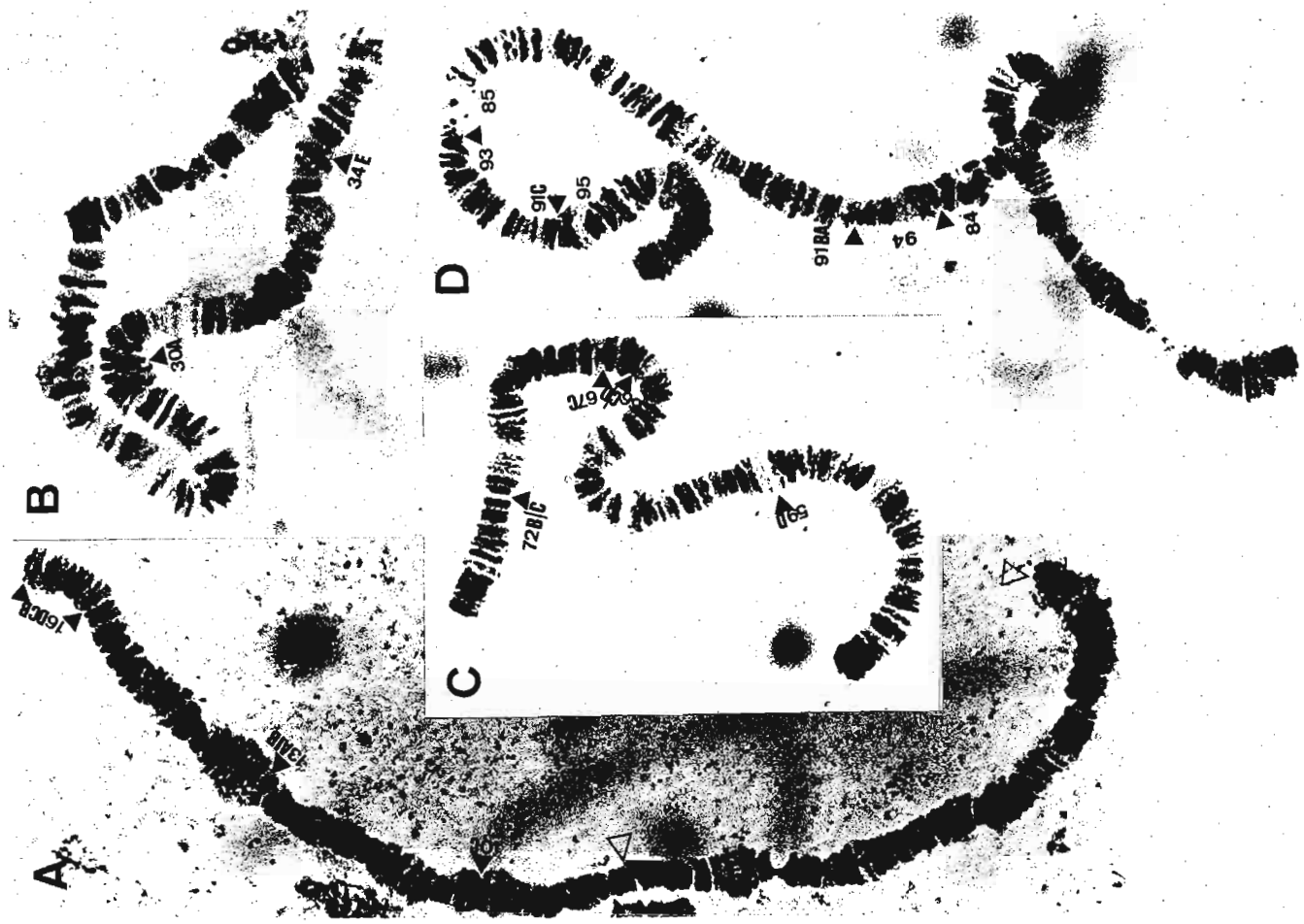


Figure 1. Chromosomes of **Drosophila guanche** showing characteristic inversions. A = chromosome A; B = chromosome J; C = chromosome E; D = chromosome O.

resembling the ancestral form of the genus Drosophila. Both have five pairs of acrocentric or subacrocentric chromosomes (Prevosti 1976) and a pair of point chromosomes (Krimbas & Loukas 1984).

Compared to the standard gene arrangements in **D.subobscura**, **D.guanche** shows characteristic inversions in most of its chromosomes.

CHROMOSOME A (Fig. 1A). In the distal part of this chromosome, **D.guanche** has two inversions. The first is a small and is located in the section 16BCD (Krimbas & Loukas 1984). The second is larger and involves the region between 10C and 13A/B subsections.

The proximal part of this sex chromosome (see Fig. 1A labeled by), also shows many differences with that in **D.subobscura**. Because there are so many differences in this region, it is difficult to make correlations relative to this area between the two species. One of the changes which probably occurred between the two species is the inversion A1, an inversion characteristic of **D.subobscura**.

CHROMOSOME J (Fig. 1B). This chromosome shows one inversion involving the region between 30A and 34E subsections. Krimbas & Loukas (1984) also indicate the existence of an inversion in this part of the J chromosome but give the boundaries as subsections 31 and 34A.

CHROMOSOME U. In agreement with Krimbas & Loukas (1984), the U chromosome of **D.guanche** carries the U1+2 gene arrangement of **D.subobscura**.

CHROMOSOME E (Fig. 1C). Figure 1C shows that the E chromosome of **D.guanche** has two inversions. The first one consists of the region between 59D and 66C/D subdivisions. The second one includes the region between 67C and 72B/C.

CHROMOSOME O (Fig. 1D). This chromosome carries two overlapping inversions: the O3 arrangement, also present in **D.subobscura**, and another which includes the region between 84D/85A and 91AB+94. These inversions have been detected by Krimbas & Loukas (1984), though they give different boundaries for this second inversion.

In conclusion, **D.guanche** has the following altered gene arrangements compared to those of the standard in **D.subobscura**: A(A1 + several specific inversions), (inv. 10C-13A/B) and (inv. 16BCD); J (inv. 30A-34E); U(U1+2); E (inv. 59D-66C/D) and (inv. 67C-72B/C); O (O3) and (inv. 84D/85A-91AB+94).

References: Krimbas, C.B. & M. Loukas 1984, Heredity 53:469-482; Prevosti, A. 1976, Bol. R. Soc. Esp. Hist. Nat. (Biol.) 74:215-217.

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The polymorphism of Aldox in Mediterranean populations of **Drosophila simulans**.

Polymorphism at the Aldehyde oxydase locus (Aldox, III 75.4) was investigated in three Mediterranean populations of **Drosophila simulans** from France (La Sirole - 43°7 N), Spain (Barcelona (41°9 N) and Tunisia (Nasr-allah 35°6 N). Electrophoresis of wild caught flies was carried out in starch gel, using the Tris-citrate II buffer system of Selander (1971), thus allowing us to separate more segregating alleles than the two previously observed in European and African populations by Cabrera et al. (1982) and Hyytia et al. (1985). The staining procedure was adapted from Ayala et al. (1972).

Figure 1 shows zymograms of single-fly homogenates. Alleles are numbered according to their relative mobility referring to that of the more common allele (100).

The allelic frequency pattern is rather close in our three samples (Table 1): three common alleles (100, 112, 118) are present in the three populations; the 106 and nul alleles are rare but detected in all

Figure 1. Aldox variants: **A:** 118-118; **B:** 100-124; **C:** 118-124; **D:** 100-100; **E:** 76-100; **F,G:** 100-100; **H:** 106-106; **I:** 112-112; **J:** 118-118; **K:** 100-118; **L:** 100-106; **M:** 106-118; **N:** 106-112; **O:** 100-112; **P:** 112-118; **Q:** 100-118.

Table 1. Allelic frequencies at the Aldox locus in the investigated populations (± 1.96 s.e.). (n = no. of gametes assayed, for rare alleles limits of the 5% confidence interval are given in brackets.)

allele	La Sirole 82	Barcelone 82	Nasrallah 83
	n = 674	n = 856	n = 644
76	0.001(0-0.009)	0.000	0.000
100	0.499 ± 0.038	0.572 ± 0.033	0.601 ± 0.038
106	0.022 ± 0.011	0.053 ± 0.015	0.008(0.003-0.018)
112	0.233 ± 0.32	0.121 ± 0.022	0.168 ± 0.029
118	0.239 ± 0.032	0.252 ± 0.029	0.220 ± 0.032
124	0.003(0-0.012)	0.000	0.000
NUL	0.003(0-0.012)	0.001(0-0.007)	0.003(0-0.012)

Table 2. Chi 2 tests (3 d.f.) for allelic frequency homogeneity between populations.

	La Sirole 82/ Barcelone 82	La Sirole 82/ Nasrallah 83	Barcelone 82/ Nasrallah 83
Chi 2	40.383***	18.309***	29.758***

*** sign. 0.1%

samples; the 76 and 124 rare alleles were only detected in the French sample. However, Chi-squared tests performed between pairs of samples show that allelic frequencies are significantly different between populations (Table 2).

Unfortunately, a direct comparison with the Aldox variants, described by Steiner et al. (1976) in Hawaiian populations and by Kojima et al. (1970) in a Texas population, is impossible at the moment; nevertheless, the frequency distribution pattern of alleles in these populations is very different from the Mediterranean pattern.

References: Ayala, F.J., J.R. Powell, M.L. Tracey, C.A. Mourad & S. Perez-Salas 1972, Genetica 70:113-139; Cabrera, V.M., A.M. Gonzalez, J.M. Larruga & A. Gullon 1972, Genetics 59:191-202; Hyttia, P., P. Capy, J.R. David & R.S. Singh 1985, Heredity (in press); Kojima, K., J. Gillespie & Y.N. Tobari 1970; Bioch. Genet. 4:627-637; Selander, R.K., M.H. Smith, S.Y. Yang, W.E. Johnson & J.B. Gentry 1971, Univ. Tex. Publ. 7103-49; Steiner, W.M., K. Chang Sung & Y.K. Paik 1976, Bioch. Genet. 14:495-506.

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Study of eye colour mutant variability in natural populations of **D.melanogaster**. III. Pine-Wood.

Two samples of **D.melanogaster** were captured in a pine-wood at La Canada (Valencia, Spain), in two different seasons of the year: autumn and spring. The purpose was to search for eye colour mutations by inbreeding through F_1 pair matings from the collected females, as in previous works made in cellar (Najera & Mensua 1985) and vineyard (Najera 1985) populations.

The number of heterozygous females for eye colour mutation was 17/45 (37.7%) in autumn and 24/67 (35.82%) in spring. The mean number of mutations per fly was 0.40 in both populations. Adding the results of both captures, 36.61% of the females were heterozygous and there were 0.40 mutations per fly in the pine-wood.

The distribution of mutations was the following: females with 1 mutation (autumn: 16, spring: 21), and females with 2 mutations (autumn: 1, spring: 3). Both fit a Poisson distribution (X^2 = 0.469 ns; X^2 = 0.319 ns).

The percentage of heterozygous loci for eye colour mutants was 12.50 (autumn population) and 18.75 (spring population).

The overall frequency of allelism was 10.9±3.9 (7.64) for the autumn population; 6.0±2.0 (9/151) for the spring population and 9.2±2.0 (20/217) interpopulational.

The distribution of alleles in both populations was at random (X^2 = 0.895 ns; X^2 = 0.052 ns).

It seems that the pine-wood population is more stable since the proportion of eye colour mutations as well as the proportion of sexes (Najera 1985) found in both captures (autumn and spring) were similar.

References: Najera, C. 1985, DIS 61:129-130; ____ 1985, DIS 61:130; Najera, C. & J.L. Mensua 1985, DIS 61:131.

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Study of eye colour mutant variability in natural populations of **D.melanogaster**. IV. Comparison between populations and allelisms.

The percentages of heterozygous females for eye colour mutations and the total number of mutations found in the populations subject of preceding works were compared by means of the ANOVA test. Table 1 shows that the number of mutations and of heterozygous females are higher in cellar populations than in the others; with significant differences between the habitats, but on the contrary, no significant differences appear between the two seasons of the year.

Table 1. Percentage of heterozygous females and number of mutations for eye colour in natural populations.

	CELLAR		VINEYARD		PINE-WOOD	
	Autumn	Spring	Autumn	Spring	Autumn	Spring
ANALYZED FEMALES	68	80	51	70	45	67
Heterozygous females	36 (52.94%)	42 (52.50%)	13 (25.49%)	23 (32.85%)	17 (37.77%)	24 (35.82%)
Mutations	42 (62%)	52 (65%)	13 (25%)	27 (39%)	18 (40%)	27 (40%)

		HETEROZYGOUS FEMALES				MUTATIONS			
Sources of Variation	FD	SS	MS	F	P	SS	MS	F	P
Season of Year	1	1.6	1.6	0.34	ns	16.53	16.53	2.00	ns
Habitat	2	194.90	97.45	29.76	*	354.03	177.02	21.42	*
Error	2	9.39	4.69			16.52	8.26		
TOTAL	5	205.89				307.09			

Table 2. Percentage of heterozygous females and number of morphological eye mutations in natural populations.

	CELLAR		VINEYARD		PINE-WOOD	
	Autumn	Spring	Autumn	Spring	Autumn	Spring
ANALYZED FEMALES	68	80	51	70	45	67
Heterozygous females	6 (8.82%)	7 (8.75%)	3 (5.88%)	3 (4.29%)	1 (2.22%)	4 (5.97%)
Total mutations	48	59	16	30	19	31
Morphol. eye mut.	6 (12.50%)	7 (11.86%)	3 (18.75%)	3 (10.00%)	1 (5.26%)	4 (12.90%)

		HETEROZYGOUS FEMALES				MUTATIONS			
Sources of Variation	FD	SS	MS	F	P	SS	MS	F	P
Season of Year	1	0.98	0.98	0.15	ns	0.35	0.35	0.01	ns
Habitat	2	30.43	15.22	2.41	ns	19.95	9.98	0.42	ns
Error	2	12.64	6.32			47.69	23.85		
TOTAL	5	44.05				68.00			

Table 3. Frequency of intra- and interpopulational allelic crosses.

	CA	CS	VA	VS	PA	PS
CA	29/381 0.076±0.014	90/902 0.100±0.010	18/230 0.078±0.018	59/558 0.106½0.014	34/342 0.099±0.016	32/504 0.063±0.010
	CS	66/497 0.133±0.014	26/284 0.091±0.017	88/610 0.144±0.014	53/386 0.137±0.018	57/577 0.099±0.014
		VA	2/28 0.071±0.049	18/156 0.115±0.026	10/104 0.096±0.028	9/163 0.055±0.017
			VS	30/184 0.163±0.026	36/236 0.152±0.030	38/343 0.108±0.017
				PA	7/64 0.109±0.039	20/217 0.092±0.020
					PS	9/151 0.060±0.020

Both, from a Student-Newman-Keuls multiple range test and from a "t" test (cellar-vineyard t = 3.95***; cellar-pine-wood t = 2.57**; vineyard-pine-wood t = 1.24 ns), it can be concluded that the differences between habitats are due to a higher number of heterozygous females in the cellar populations.

Morphological eye mutations were also detected, and there are no significant differences as regards habitats or seasons for this type of mutation (Table 2).

To carry out inter- and intrapopulational allelism tests, the crosses were made using phenotypes (so that the total number of crosses is reduced from 15931 to 6909) on the basis that dark eyes and light eyes possibly block different metabolic pathways and that it is difficult to find alleles between them, and on the other hand that caramel eyes normally affect both pathways at the same time and even the depossition of pigment granules.

Although the estimated frequency of allelic crosses is not the actual populational frequency but a higher value, the results are useful for comparing the different populations, because in all cases the same method was used (Table 3). The populations showing the highest number of allelic crosses between them

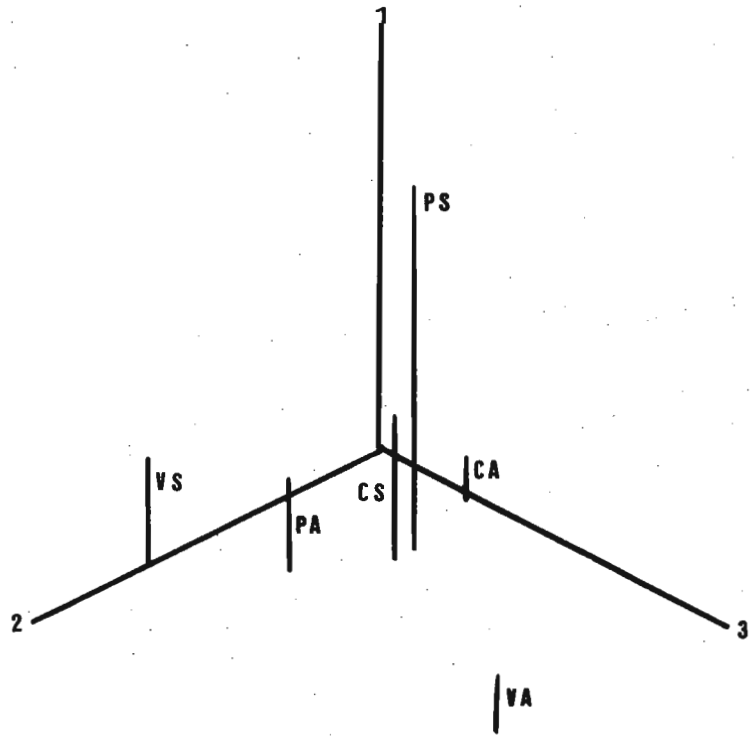

Figure 1. Tridimensional graph corresponding to the six natural populations.

were CS (cellar spring), VS (vineyard spring) and PA (pine-wood autumn). These populations also showed the highest intrapopulational allelisms. On the contrary, CA (cellar autumn) and VA (vineyard autumn), compared to PS (pine-wood spring), were the populations which showed the lowest allelic crosses and intrapopulational allelisms. Both inter- and intrapopulational allelic crosses had the same range.

Considering the total number of different mutations in each population in relation to the total number of analyzed females in such populations as a more exact reflect of the variability, and performing an ANOVA test, differences between habitats, although not significant, can be observed. It can be suggested, therefore, that the allelism rate in the cellar is higher than in the other habitats, so that the variability decreases in the population in relation to different mutations. Altogether, these results seem to suggest that either we deal with a more endogamic and closer related population or selection is favouring a greater accumulation of mutations in this population.

By means of a factorial analysis of correspondances which reflects the 65% of the total variance, and drawing a tridimensional graph of the points corresponding to the six populations (Fig. 1), it can be observed that the closest populations are the cellar ones, the vineyard and the pine-wood populations remaining approximately at the same distance.

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Study of eye colour mutant variability in natural populations of **D.melanogaster.** V. Effective sizes and distances.

From a total of the six populations belonging to captures performed in three different habitats (cellar, vineyard and pine-wood), and in two seasons of the year (autumn and spring) subject of preceding works, a series of parameters were estimated.

The effective sizes of these populations were estimated, using the "temporarl method" of Krimbas & Tsakas (1971) and applying the estimator of Pollack (1983). The population sizes are rather big in the three populations, but slightly smaller in the cellar population (cellar-Ne = 12000; vineyard-Ne = 15000; pine-wood-Ne = 175000) which was predictable from the peculiar characteristics of this habitat.

Table 1. Average heterozygosity in the six natural populations.

	Lewontin & Hubby	Nei
CELLAR AUTUMN	0.010553 ± 0.004179	0.010500 ± 0.000003
CELLAR SPRING	0.011479 ± 0.003973	0.011505 ± 0.000004
VINEYARD AUTUMN	0.011512 ± 0.005267	0.011602 ± 0.000001
VINEYARD SPRING	0.010590 ± 0.004112	0.010615 ± 0.000007
PINE-WOOD AUTUMN	0.014143 ± 0.006115	0.014272 ± 0.000006
PINE-WOOD SPRING	0.009539 ± 0.004137	0.009187 ± 0.000001

Table 2. Genetic distances between natural populations.

	Cavalli	Sneath	Nei	Rogers	Prevosti
CA-CS	0.2674	0.0066	0.00002	0.0043	0.0043
VA-VS	0.2113	0.0061	0.00003	0.0049	0.0049
PA-PS	0.2435	0.0061	0.00002	0.0047	0.0047
CA-VA	0.2567	0.0059	0.00003	0.0048	0.0048
CA-PA	0.2571	0.0067	0.00002	0.0046	0.0046
VA-PA	0.2145	0.0074	0.00004	0.0057	0.0057
CS-VS	0.2603	0.0061	0.00002	0.0043	0.0043
CS-PS	0.2516	0.0062	0.00002	0.0044	0.0044
VS-PS	0.2305	0.0056	0.00002	0.0042	0.0042

The average heterozygosity in the six populations was calculated following the estimations of Lewontin & Hubby (1966) and Nei (1978). The results are reflected in Table 1. The values are low, identical in both estimations and rather similar in all populations. The pine-wood populations were the most different; the autumn pine-wood registered the higher heterozygosity and the spring pine-wood populations the lowest.

To quantify the variation of the gene frequencies, the genetic distances between these populations were calculated using five indexes (Table 2). The Cavalli-Sforza index gave the largest distances. In general distances between populations are small and a few are larger in autumn than in spring. Nevertheless, this kind of variability does not seem to be adequate to calculate genetic distances between populations.

References: Krimbas, C.B. & S. Tsakas 1971, Evolution 25:454-462; Lewontin, R.C. & J.L. Hubby 1966, Genetics 54:595-609; Nei, M. 1978, Genetics 89:583-590; Pollak, E. 1983, Genetics 104:531-548.

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Effect of environmental factors on glutamate dehydrogenase activity in **D.melanogaster** larvae.

Since in some invertebrates, including insects, glutamate dehydrogenase (GDH) seems to be involved in the energy-linked metabolism (Bursell et al. 1976; Hansford et al. 1975; Storey et al. 1978), **D.melanogaster** larvae were subjected to such environmental stress conditions impairing this metabolism within the cells as: anaerobiosis, recovery from anaerobiosis, heat-shock, and starvation.

Materials and Methods. D.melanogaster, wild strain, was reared on the medium of Mitchell et al. (1968) supplemented with dried yeast (214 g per 1 liter of water), at 25°C, in a diurnal cycle of 12 hr light - 12 hr night photoperiod. Egg-laying periods of 4 hr were used to synchronize of larvae. Larvae of the third instar, i.e., at the stage when GDH activity is the highest (Nowak & Piechowska 1980), were used for experiments.

The larvae were subjected to the following treatments: (1) anaerobiosis under N_2, for up to 2 hr; (2) recovery from 2 hr anaerobiosis for up to 60 min; (3) heat-shock at 37°C for up to 2 hr; (4) starvation for up to 3 hr. Larvae reared under standard conditions served as controls.

Mitochondria were isolated in 10 mM Tris buffer, pH 7.4, containing 60 mM sucrose, 240 mM mannitol, and 0.2 mM EGTA (Storey et al. 1978a), supplemented with 0.1% 2-mercaptoethanol. Nuclei and cell debris were removed by centrifugation of the homogenate at 2200 g for 6 min. The supernatant obtained was centrifuged at 7000 g for 10 min. The sedimented mitochondria were washed with the isolation buffer and 0.1 M potassium phosphate buffer, pH 7.8. Mitochondria were solubilized with 1% Lubrol PX to liberate the enzyme (GDH) from the matrix. Mitochondrial membranes were removed by centrifugation at 100,000 g for 30 min, and the supernatant obtained was used for the assays. All the isolation procedure was performed at 4°C.

GDH activity was detemined both for the deamination and amination reactions at room temperature, by automatic recording of absorbance at 340 nm on a Varian-Cary 118C spectrophotometer, in 1 ml quartz cuvettes, at 1 cm light path. The reaction mixture contained substrates and coenzymes at optimum concentrations (for deamination 6 mM NAD, 10 mM L-glutamate; for amination 0.2 mM NADH, 6 mM 2-oxoglutarate, 300 mM NH_4Cl) in 0.1 M potassium phosphate buffer, pH 7.8, and 50 µl of the enzyme preparation, corresponding to 50-70 µg protein (as determined according to Lowry et al. 1951).

Specific activity of the enzyme was expressed in micromoles of NAD or NADH transformed during 1 min of the reaction by 1 mg of the extract protein. $K_{m\ glu}$ was estimated from the Lineweaver-Burke plot (Segel 1975) for that range of the substrate concentration (1-10 mM) at which the plot was linear. The results were analyzed by Student's t test, assuming that the difference is statistically significant when $p \leq 0.05$.

Results and Discussion. In the reaction catalysed by glutamate dehydrogenase isolated from the **D.melanogaster** larvae subjected to anaerobiosis, i.e., when cellular metabolism is shifted to the glycolytic pathway, the level of deamination was lowered by 30% as compared with controls. This statistically significant decrease was first observed after 1 hr of anaerobiosis and remained at a practically unchanged level also after 2 hr (Table 1).

The decrease in GDH activity could be due to lowered level of L-glutamate in the cell as observed by Meyer (1980) for **Callitroga macellaria** larvae subjected to 4 hr anaerobiosis. Moreover, under conditions of anaerobiosis, there is an increase in the NADH:NAD ratio in the cell (Meyer 1980), and the appearing excess of NADH could inhibit GDH, an enzyme the activity of which is known to be regulated by reaction products (Bursell 1975; Storey et al. 1978).

Under these conditions, accumulation of the anaerobic metabolism products (alanine, pyruvate, lactate) observed in insects (Price 1963; Meyer 1978) could also lead to lowering of GDH activity.

Table 1. Effect of anaerobiosis on GDH activity in the deamination reaction.

time of anaerobiosis (hours)	GDH activity (µmoles/min/ mg/protein)	number of exp.	p
control	0.356 ± 0.082	10	--
1/2	0.348 ± 0.067	5	>0.05
1	0.234 ± 0.050	4	<0.05
2	0.242 ± 0.069	12	<0.05

Table 2. Effect of recovery from anaerobiosis on GDH activity in deamination reaction.

time of release from anaerobiosis (minutes)	GDH activity (µmoles/min/ mg/protein)	number of exp.	p
control	0.356 ± 0.082	10	--
0	0.242 ± 0.069	10	<0.05
15	0.349 ± 0.051	3	>0.05
30	0.271 ± 0.089	4	>0.05
60	0.291 ± 0.033	4	>0.05

Table 3. Effect of heat-shock on GDH activity in the deamination reaction.

time of heat-shock (hours)	GDH activity (µmoles/min/ mg/protein)	number of exp.	p
control	0.292 ± 0.069	31	--
1/2	0.218 ± 0.050	8	<0.05
1	0.282 ± 0.086	14	>0.05
2	0.247 ± 0.086	9	>0.05

Table 4. Effect of heat-shock on K_m glu value of GDH.

time of heat-shock (hours)	kind of probe	K_m glu (mM)	number of exp.	p
1/2	control	1.34 ± 0.078	2	>0.05
	exp.	1.07 ± 0.064	2	
1	control	1.27 ± 0.230	4	>0.05
	exp.	1.11 ± 0.025	3	
2	control	1.33 ± 0.071	2	>0.05
	exp.	1.24 ± 0.430	3	

Table 5. Effect of starvation on the GDH activity in the deamination reaction.

time of starvation (hours)	GDH activity (µmoles/min/ mg/protein)	number of exp.	p
control	0.280 ± 0.055	29	--
1/2	0.276 ± 0.049	8	>0.05
1	0.276 ± 0.064	9	>0.05
2	0.322 ± 0.087	8	>0.05
3	0.296 ± 0.025	6	>0.05

Table 6. Effect of starvation on the K_m glu value of the GDH.

time of starvation (hours)	K_m glu (mM)	number of exp.	p
control	1.35 ± 0.130	4	--
1/2	1.34 ± 0.078	2	>0.05
1	1.27 ± 0.030	4	>0.05
2	1.33 ± 0.071	2	>0.05

Francesconi et al. (1969) demonstrated that pyruvate and alanine inhibited GDH activity in rat liver slices. Thus it can be expected that GDH from **D.melanogaster**, which in many respects resembles the enzyme of higher organisms (Bond & Sang 1968) could also be inhibited by those metabolites.

Thus, the pattern of anaerobiosis-induced changes in the enzyme activity indicates that the reactivity of GDH in the deamination reaction is dependent on functioning of the aerobic respiratory metabolism.

This dependence was confirmed in experiments on the activity of GDH isolated from larvae recovering after 2 hr anaerobiosis. As early as 15 min after the larvae had been brought back to aerobic conditions the level of deamination returned to control values (Table 2). Thus the resumed activity of the respiratory chain and the Kreb's cycle resulted in the return of the normal capacity of GDH to catalyse of 2-oxoglutarate, NADH and NH_4^+.

Such a relationship between the level of deamination and functioning of cellular oxidative processes suggests that GDH could cooperate with this metabolic pathway, e.g., by supplying 2-oxoglutarate and/or NADH.

Studies on the activity of GDH isolated from larvae subjected to another type of stress conditions probably also disturbing the functioning of respiratory metabolism - the heat shock, demonstrated that after 30 minutes at 37°C the level of deamination reaction was statistically significantly, by about 25%, decreased as compared with control. In our experiments, on further duration of the shock (1 and 2 hr) the GDH activity ceased to be affected and returned to control value (Table 3). Leenders et al. (1974) suggested that heat-shock activated the respiratory metabolism. However, Meyer (1978) demonstrated that, in **Callitroga macellaria** larvae, both the increased temperature and anaerobiosis led to the appearance in the cells of the same set of compounds. Since in various organisms studied the response to heat-shock is very similar (Ashburner et al. 1979), it can be concluded that the increased temperature inhibits through a still unknown mechanism the aerobic respiratory metabolism. This would lead, in turn, to lowering of GDH activity in the first phase of heat-shock. On further duration of the shock, the energy providing metabolism may become unblocked, resulting in the recovery of the GDH activity observed after 1 and 2 hr of thermal stress.

The changes observed in GDH activity in the deamination reaction were not due to changed enzyme structure, as the K_m glu remained at the control level throughout the time of the experiment (Table 4).

Starvation is another factor known to affect the respiratory activity of the cell. As demonstrated by Bosquet (1976) the respiratory metabolism in **Bombyx mori** larvae starved for 2 hr was decreased by 10%.

Table 7. Effect of various environmental factors on the GDH activity in the deamination reaction.

A. Anaerobiosis

time of anaerobiosis (hours)	GDH activity (μmoles/min/ mg/protein)	number of exp.	p
control	1.356 ± 0.331	14	--
1/2	1.410 ± 0.377	5	>0.05
1	1.134 ± 0.373	4	>0.05
2	1.294 ± 0.277	12	>0.05

B. Recovery from anaerobiosis

time of release from anaerob. (minutes)			
control	1.356 ± 0.331	14	--
0	1.294 ± 0.277	12	>0.05
15	1.521 ± 0.562	5	>0.05
30	1.272 ± 0.453	6	>0.05
60	1.099 ± 0.324	5	>0.05

C. Heat-shock

time of heat-shock (hr)			
control	1.283 ± 0.315	17	--
1/2	0.933 ± 0.346	4	>0.05
1	1.212 ± 0.344	7	>0.05
2	1.082 ± 0.326	6	>0.05

D. Starvation

time of starvation (hr)			
control	1.242 ± 0.351	28	--
1/2	1.417 ± 0.149	6	0.05
1	1.303 ± 0.112	6	>0.05
2	1.455 ± 0.299	5	>0.05
3	1.368 ± 0.102	5	>0.05

Such a small effect of starvation on mitochondrial metabolism, was not reflected in changes in the activity of GDH in the deamination reaction (Table 5) or in the $K_{m\ glu}$ value (Table 6). On the basis of these experiments as well as the effect of anaerobiosis on GDH activity (a 30% decrease of deamination despite complete blocking of the aerobic metabolism; cf Table 1), it could be concluded that products of glutamate deamination can be utilized not only in respiratory metabolism (by the Krebs cycle or the respiratory chain(but also in other important metabolic pathways.

Simultaneously it should be noted that amination by GDH was not affected by any of the environmental factors applied (Table 7 A,B,C,D) which evidences its independence of the respiratory metabolism, while the lack of its activation during starvation suggests that amination is not involved in the processes supply L-glutamate under conditions when the inflow of the energetic substrates is disturbed.

Summing up, the results presented suggest that, in **D.melanogaster** larvae GDH is involved in aerobic respiratory metabolism of the cell, most probably supporting it by supplying 2-oxoglutarate and/or NADH; however, the products of the deamination reaction could also be utilized by other metabolic pathways.

References: Ashburner, M. 1979, Cell 17:241; Bosquet, G. 1976, J. Insect Physiol. 22:541; Bursell, E. 1975, Insect Biochem. 5:289; Bursell, E., E. Slack 1976, Insect Biochem. 6:159; Francesconi, R.P. & C.A. Villee 1969, Arch. Biochem. Biophys. 129:509; Hansford, R.G. & R.N. Johnson 1975, Biochem. J. 148:389; Leenders, H.J., H.D. Berendes, P.J. Helmsing, J. Derksen & J.F.J.G. Koninx 1974, Sub-cell. Biochem. 3:119; Lowry, O.H., N.J. Rosebrough, A.L. Farr & R.J. Randall 1951, J. Biol. Chem. 193:265; Meyer, S.G.E. 1978, Insect Biochem. 6:471; 1980, Insect Biochem. 10:449; Mitchell, H.K. & A. Mitchell 1968, DIS 39:135; Nowak, J. & M.J. Piechowska 1982, DIS 58:117; Price, G.R. 1963, Biochem. J. 86:372; Segel, I.H. 1975, in: Enzyme kinetics: Behaviour and analysis of rapid equilibrium and steady-state enzyme systems, Wiley & Sons, New York-London-Toronto, pp. 377-382; Storey, K.B. & E. Bailey 1978, Insect Biochem. 8:75; Storey, K.B., J.H.A. Fields & P.W. Hochachka 1978a, J. Exp. Zool. 205:111; Tissieres, A., H.K. Mitchell & U.M. Tracy 1974, J. Mol. Biol. 84:389.

Nowak, J. and M.J. Piechowska. Institute of Biochemistry and Biophysics, Warsaw, Poland.

Kinetic analysis of glutamate dehydrogenase from **D.melanogaster** larvae.

The smallest, catalytically active subunit of glutamate dehydrogenase (GDH) of **D.melanogaster** is an oligomer composed of six subunits (Caggese et al. 1982). In proteins composed of subunits, mutual interactions between subunits can take place (Levitzki 1978). The studies of Engel & Dalziel (1969) point to occurrence of negative cooperativity between the subunits of bovine GDH hexamer. The aim of the present work was to make an initial assessment of the nature of cooperativity between the subunits of GDH from **D.melanogaster** larvae.

Materials and Methods. Biological material, methods of enzyme isolation, and determination of its activity were the same as in the accompanying paper (Nowak & Piechowska 1986). To assess the type of cooperativity between the subunits of **D.melanogaster** GDH, kinetic data on the dependence of the deamination rate on coenzyme (NAD) concentration at a fixed, 10 mM L-glutamate, as well as on substrate concentration at fixed, 6 mM NAD, were subjected to graphical analysis. Michaelis-Menten, Lineweaver-Burke & Scatchard plots (Segel 1975) are presented.

Results and Discussion. The Michaelis-Menten plot (Fig. 1) representing the dependence of GDH activity on NAD concentrations is of hyperbolic character. On the other hand, in the Lineweaver-Burke plot (Fig. 2) three linear regions can be discerned, their slope becoming steeper with respect to the ordinate

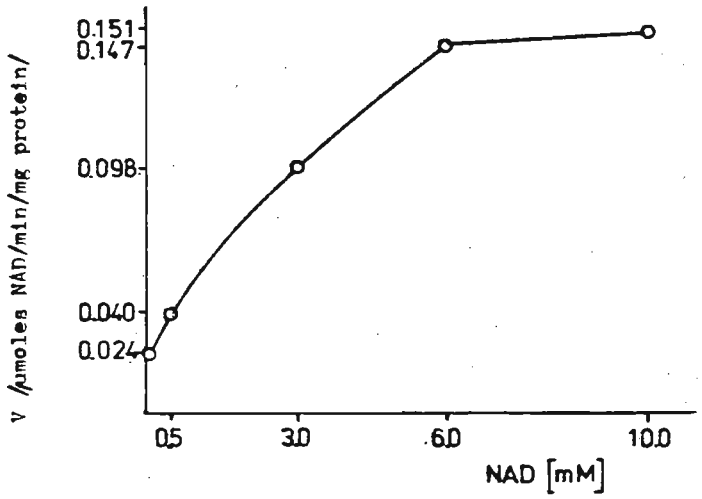

Figure 1. The activity of **D.melanogaster** GDH on NAD concentration: the Michaelis-Menten plot.

Table 1. Dependence of $K_{m\ NAD}$ values of glutamate dehydrogenase on NAD concentration.

region number	concentration range of NAD (mM)	$K_{m\ NAD}$ (mM)
I	0.05 - 0.50	0.06 ± 0.02
II	0.50 - 3.00	1.22 ± 0.17
III	3.00 - 10.0	3.31 ± 0.41

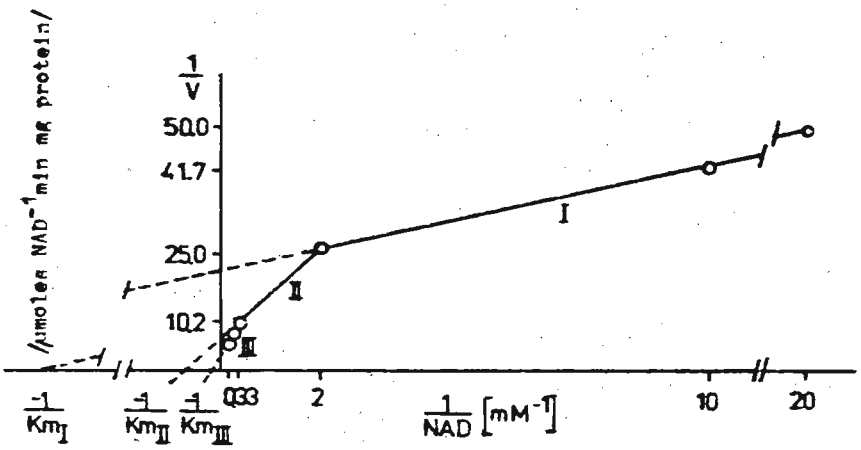

Figure 2. Variation of the reciprocal of the activity of **D.melanogaster** GDH with the reciprocal of NAD concentration: the Lineweaver-Burke plot.

with increasing NAD concentration. The $K_{m\ NAD}$ values determined from the particular regions rose with coenzyme concentration (Table 1). Thus the plot was not linear but became bent downward with increasing NAD concentration.

A similar course of these plots was reported by Engel & Dalziel (1969) for GDH isolated from bovine liver. They found four linear regions, with slopes consistently increasing with respect to the ordinate; the values of the Michaelis constants calculated from these regions also increased with NAD concentration.

The authors suggest that this behaviour of GDH in the presence of NAD could be explained by negative cooperativity between the active sites of the enzyme because, as they have demonstrated in an earlier study (Dalziel & Engel 1968), the concave downward Lineweaver-Burke plot corresponds to the Michaelis-Menten plot which apparently does not differ from hyperbola. In fact, it is steeper below the half-saturation point and less steep above this point. Thus, this is a different kind of deviation from michaelian kinetics, indicating occurrence of negative cooperativity, as opposed to the sigmoidal Michaelis-Menten plot corresponding to the Lineweaver-Burke plot concave upward which points to the existence of positive cooperativity.

In view of the facts, analysis of the Lineweaver-Burke plot representing the dependence of the activity of **D.melanogaster** GDH on NAD concentration suggests that cooperativity between enzyme subunits on binding of coenzyme is of negative character.

Data on dependence of the reaction rate on L-glutamate concentration indicate that binding of this substrate is also accompanied by negative cooperativity between the subunits: the Michaelis-Menten plot is hyperbolic (Fig. 3) and the corresponding Lineweaver-Burke plot is concave downward (Fig. 4). $K_{m\ glu}$ value determined from the linear regions of the plot increase with substrate concentration (Table2). Moreover, deamination is inhibited by L-glutamate over 10 mM. A similar Lineweaver-Burke plot for GDH from bovine liver was reported by Barton & Fisher (1971).

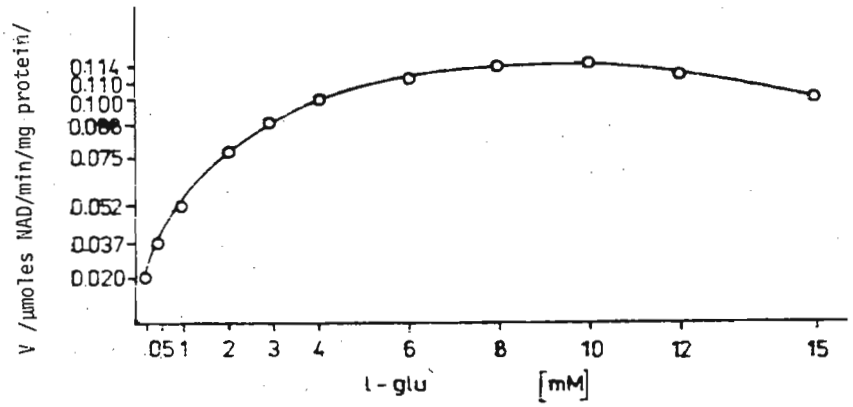

Figure 3. The activity of **D.melanogaster** GDH on L-glutamate concentration: the Michaelis-Menten plot.

Table 2. Dependence of K_m glu values of glutamate dehydrogenase on L-glutamate concentration.

region number	concentration range of L-glu (mM)	K_m glu (mM)
I	0.2 - 1.0	0.68 ± 0.08
II	1.0 - 10.0	1.35 ± 0.13

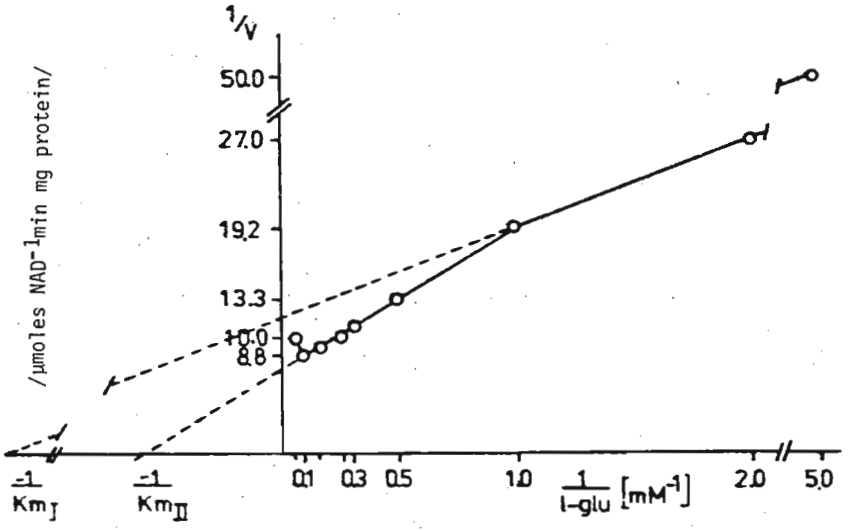

Figure 4. Variation of the reciprocal of the activity of **D.melanogaster** GDH with the reciprocal of the L-glutamate concentration: the Lineweaver-Burke plot.

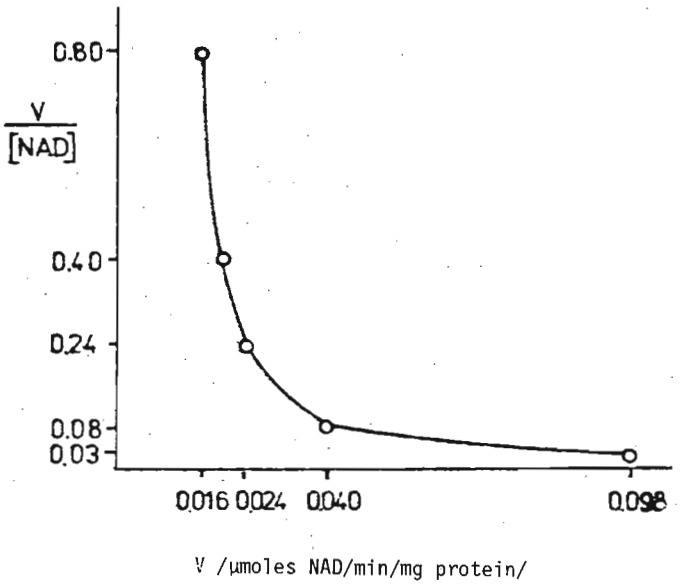

Figure 5. Variation of the ratio of **D.melanogaster** GDH activity to NAD concentration with GDH activity: the Scatchard plot.

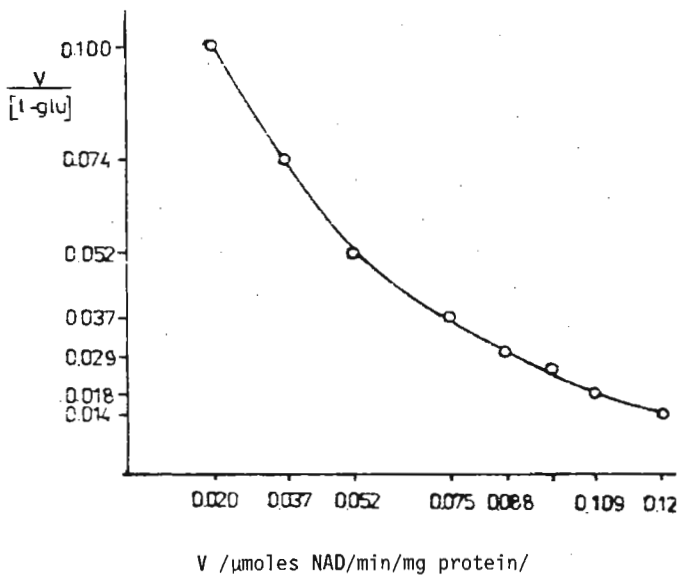

Figure 6. Variation of the ratio of **D.melanogaster** GDH activity to L-glutamate concentration with GDH activity: the Scatchard plot.

To determine with greater certainty the model of cooperativity between subunits of **D.melanogaster** GDH, the data obtained on the dependence of the reaction rate on concentration of NAD or L-glutamate were presented also as a Scatchard plot (Fig. 5 and 6). The course of the curves, concave upwards, was consistent with negative interactions between subunits of **D.melanogaster** GDH.

Thus, the kinetic data presented permit us to suggest that the subunits of glutamate dehydrogenase from **D.melanogaster** larvae exhibit negative cooperativity. However, for final assessment of the actual type of subunit interaction, the purified enzyme should be analyzed by isotope exchange experiments or by X-ray crystallography.

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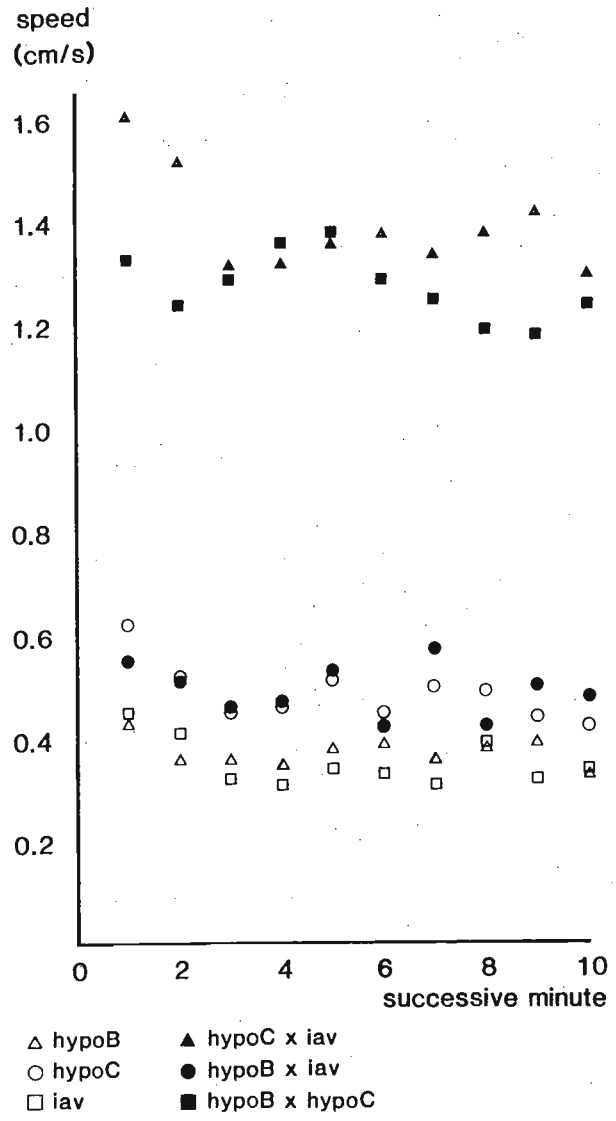
O'Dell, K.M.C. and B. Burnet. The University, Sheffield, England. Allelism of the behavioural mutants hypoactive B^1 and inactive in **D.melanogaster.**



Hall (1982) and Lindsley & Zimm (1985) have suggested that the two X-linked behavioural mutants hypoactive B^1 (hypo B^1, Homyk & Sheppard 1977, Homyk 1977) and inactive (iav, Kaplan 1977) are alleles. They are currently mapped at 20.7 and 18.8, respectively (Lindsley & Zimm 1985).

To discover whether hypo B^1 and iav are indeed allelic, we have crossed the two stocks to observe the behaviour of the F_1 heterozygous hypo B^1/iav females. As a control, a third and independent gene, hypoactive C^1 (hypo C^1, Homyk & Sheppard 1977) mapping at X:c42 was also crossed to hypo B^1 and iav and the behaviour of the F_1 female progeny scored. The mutant hypo C^1 shows a similar phenotype to hypo B^1 and iav, but at more than twenty map units away hypo C^1 is unlikely to be allelic to either hypo B^1 or iav.

The mutants hypo B^1, hypo C^1 and iav are all recessive. Hence the crosses hypo B^1 x hypo C^1 and iav x hypo C^1 should (and do) produce behaviourally wildtype female progeny. The female F_1 progeny of the cross hypo B^1 x iav should show normal locomotor activities if the mutans are at separate loci, but will show marked reductions in locomotor activities if the mutants are allelic.

The technique used to measure locomotor activity of F_1 females was a modified version of that of Connolly (1966). Single flies were observed under a 10 x 10 cm open field grid for a period for 10 minutes and the frequency and duration of key presses made by the observer was recorded using an Apple II Microcomputer as a data logger. In this manner scores for amount and speed of movement, distance travelled, time spent preening and frequencies of falling and jumping could immediately be tabulated for each successive minute observed. By this method we can show that these three mutants are characterized by showing similar and extreme reductions in both amount and speed of movement as well as showing deficits in other locomotor activities.

The average scores per minute for the three hybrid classes were calculated using ten replicate individuals per class. The results for speed of movement are shown below.

Clearly the hybrid F_1 females from the cross hypo B^1 x iav show a marked reduction in speed of movement with respect to the hybrid female offspring from the other crosses. In the other behavioural tests a similar result is obtained.

Given the additional infomation that a stock derived from these hypo B^1/iav hybrids still retains this marked reduction in all activities over twenty generations later, this must lead us to the conclusion that hypo B^1 and iav are allelic.

References: Connolly, K. 1966, Anim. Behav. 14:444-449; Hall, J.C. 1982, Quart. Rev. Biophys. 15:223-479; Homyk, T. 1977, Genetics 87:105-128; Homyk, T. & D.E. Sheppard 1977, Genetics 87:95-104; Kaplan, W.D. 1977, DIS 52:1; Lindsley, D. & G. Zimm 1985, DIS 62:165.

Paik, Y.K. and M.S. Kim. Hanyang University School of Medicine, Seoul, Korea. Temporal changes of gene arrangement frequencies in a wild Korean **Drosophila melanogaster** population.

The frequency of inversion-carrying chromosomes in natural populations of **Drosophila melanogaster** has recently been reported rather high even in this cosmopolitan species (e.g., Stalker 1976; Mettler et al. 1977; Paik 1979; Knibb et al. 1981), but information on inversion polymorphism of this species is not yet effectively collated. This study was therefore undertaken to measure the year-to-year changes in the frequencies of these polymorphic inversions in a locality. This preliminary communication covers the first four years (1978 - 1981) of the study extending to 1985.

The flies were taken in a large pear-orchard district called Taenung about 16 kilometers N.E. of central Seoul. The population samples were collected once every year in late October when the orchard populations reach their peak size. Haploid sets of chromosomes carried by the wild males were analyzed for their gene arrangements by mating them individually with virgin Oregon-R females homozygous for the standard gene arrangement, and then by examining preparation of smears of the salivary gland cells of one F_1 larva from each mating. The breakpoints of inverted gene arrangements were identified on the basis of the salivary gland chromosome maps provided by Bridges & Breheme (1944) and Lefevre (1976).

A total of 49 types of inversions were detected from the four year collections, of which only six belong to the category of cosmopolitan and polymorphic type, and the rest to that of the type unique to the area. The frequencies of these polymorphic inversions throughout the period of study are given in Table 1 and illustrated in Fig. 1. These table and figure show some points of interest:

(1) Directional frequency changes took place at least in three of the cosmopolitan inversions: In(2L)t, In(2R)NS, In(3R)C. This suggests they are genetically flexible and sensitive to environmental changes whose factor(s) remain to be clarified in the future.

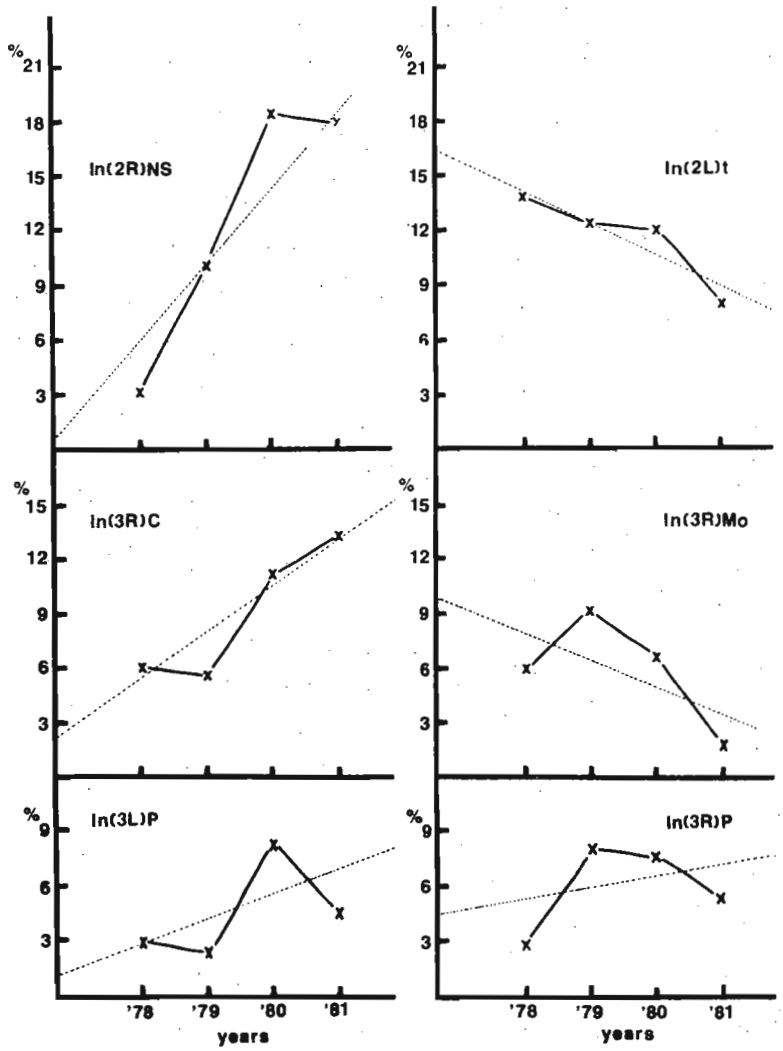


Figure 1. Frequencies of arrangements 2Lt, 2RNS, 3LP, 3RC, 3RP, 3RMo, plotted against date of collection at Taenung.

Table 1. Relative frequencies of standard and inverted gene arrangements for a given chromosome arm recovered from the Taenung populations of **Drosophila melanogaster.**

Chrom. arm	Gene arrangement	Year 1978	1979	1980	1981	Average	X^2
2L	St	0.8377	0.8688	0.8681	0.8796	0.8663	
	t	0.1361	0.1208	0.1172	0.0817	0.1168	3.4504
	end	0.0262	0.0104	0.0147	0.0385	0.0169	
2R	St	0.9529	0.8941	0.8059	0.8077	0.8741	
	NS	0.0366	0.1013	0.1831	0.1779	0.1181	32.7588***
	end	0.0105	0.0046	0.0110	0.0144	0.0078	
3L	St	0.9581	0.9724	0.9084	0.9519	0.9565	
	p	0.0262	0.0230	0.0806	0.0433	0.0363	20.5161***
	end	0.0157	0.0046	0.0110	0.0048	0.0071	
3R	St	0.8482	0.7618	0.7399	0.8077	0.7748	
	p	0.0314	0.0829	0.0733	0.0433	0.0694	8.3050*
	C	0.0576	0.0564	0.1026	0.1298	0.0746	17.9249**
	Mo	0.0576	0.0863	0.0659	0.0192	0.0701	11.9037*
	end	0.0052	0.0127	0.0183	0.0000	0.0110	
	N:	191	869	273	208		

The letters, St and end are abbreviations for standard and endemic gene arrangements, respectively. N, number of male gametes tested.
* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

(2) In(2L)t shows a downward frequency change from 1978 through 1981, while In(2R)NS reveals the opposite trend, i.e., an upward trend from 1978 through 1981. Thus, in the 1970's In(2L)t > In(2R)NS. But in the 1980's, In(2R)NS > In(2R)t. In(2L)t > In(2R)NS had been observed up to the beginning or the middle part of the 1970's in the Old World, Australia, and Japan (Mourad & Mallah 1960; Ashburner & Lemeunier 1976; Waters 1944; Xachalopourou 1974; Watanabe 1967; Inoue & Watanabe 1979); In(2R)NS ≥ In(2L)t was observed thereafter in Australia and Japan (Inoue & Watanabe 1980; Knibb et al. 1981). In North America, this trend was found as early as the latter part of the 1960's (Yang & Kojima 1972; Stalker 1976; Mettler et al. 1977; Mukai et al. 1974).

References: Ashburner, M. & F. Lemeunier 1976, Proc. Roy. Soc. Lond. B 193:137-157; Bridges, C.B. & K.S. Brehme 1944, Carn. Inst. Wash. Pub. 552; Inoue, Y. & T.K. Watanabe 1979, Jap. J. Genet. 54:69-82; Inoue, Y. & T.K. Watanabe 1980, Ann. Rep. Nat. Inst. Genet. Japan 31:92-93; Knibb, W.R., J.G. Oakeshott & J.B. Gibson 1981, Genetics 98:833-847; Lefevre, G. 1976,in: Genetics and Biology of Drosophila, v.1a, Academic Pr.; Mettler, L.E., R.A. Voelker & T. Mukai 1977, Genetics 87:169-176; Mourad, A.M. & G.S. Mallah 1960, Evolution 14:166-170; Mukai, T., T.K. Watanabe & O. Yamaguchi 1974, Genetics 77:771-793; Paik, Y.K. 1979, Korean J. Genet. 1:18-27; Stalker, H.D. 1976, Genetics 82:323-347; Watanabe, T. 1967, Mem. Fac. Sci. Kyushu Univ. 4:159-182; Waters, M. 1944, Univ. Tex. Pub. 4445:129-174; Yang, H.Y. & K.I. Kojima 1972, Univ. Tex. Pub. 7213:229-236; Zacharopourou, A. 1974, DIS 51:110.

Ramachandra, N.B. and H.A. Ranganath. University of Mysore, India. Estimation of fecundity in the parent and hybrid populations of **Drosophila nasuta nasuta** and **Drosophila nasuta albomicana.**

Drosophila nasuta nasuta (2n=8) and **D.n.albomicana** (2n=6) are cross fertile chromosomal races of the **nasuta** subgroup of **immigrans** species group of Drosophila (Nirmala & Krishnamurthy 1972; Ranganath & Hagele 1981, 1982; Ramachandra & Ranganath 1986). Interracial hybridization between these races and followed by maintaining the hybrid population for over 20 generations has resulted in the evolution of two new cytological populations called as Cytoraces I and II. The reconstituted karyotypes of these Cytoraces are new in composition, the chromosomes being drawn from both the parental races. In brief, the Cytorace I is a product of the hybridization between **D.n.nasuta** male and **D.n.albomicana** female. The males and females of Cytorace I have 2n=7 and 2n=6, respectively. Of these 13 chromosomes, 8 chromosomes belong to **nasuta** parent while 5 are of **albomicana** parent. Similarly, the Cytorace II has emerged out of the cross between **D.n.nasuta** female and **D.n.albomicana** male. The karyotype of the Cytorace II is 2n=6 in both males and females. Of these 12 chromosomes of the Cytorace II, 2 are of **nasuta** parent, while 10 belong to the **albomicana** parent. In response to this karyotypic repatterning, the amount of heterochromatin present in these Cytoraces differ within them and between parents (Ramachandra & Ranganath 1986).

With the evolution of these new stabilized Cytoraces, we have four cytologically distinct populations having differences and similarities in the constitution of the karyotypes.

The estimation of fitness is the first step in understanding the adaptive evolution of any evolutionary lineage. Egg laying capacity or fecundity of a population is one of the important parameters of fitness which can be used as a yardstick for comparisons.

In the present study, fecundity of the parental races (**D.n.nasuta** and **D.n.albomicana**), the Cytoraces I and II as well of the F_1 hybrids has been estimated. The aim of this experiment is to know the impact of hybridization and karyotype restructuring on the fecundity of individuals.

After aging the isolated flies of the above said subjects for five days, pair matings were made. These pairs were transferred to fresh vials without etherization once in 24 hr. Then, the number of eggs

Table 1. Fecundity of **Drosophila nasuta nasuta, D.n.albomicana,** F_1 hybrids and of Cytorace I and II. 60 females of each category was screened for a period of ten days.

Strain	Total no. eggs laid	Eggs/ individual
1. **D.n.nasuta** (2n=8)	12,058	200.96
2. **D.n.albomicana** (2n=6)	10,749	179.15
3. F_1 of the Cross: (2n=7) **D.n.nasuta** ♂ x **D.n.albomicana** ♀	14,008	233.46
4. F_1 of the cross: (2n=7) **D.n.nasuta** ♀ x **D.n.albomicana** ♂	15,315	255.25
5. Cytorace I*: (♂ 2n=7; ♀ 2n=6) a product of hybridization between **D.n.nasuta** ♂ and **D.n.albomicana** ♀	13,232	220.52
6. Cytorace II* (♂ and ♀ 2n=6) a product of hybridization between **D.n.nasuta** ♀ and **D.n.albomicana** ♂	12,030	200.50

* A continuous inbreeding of the respective hybrid populations for over 20 generations has resulted in the evolution and stabilization of these Cytoraces.

laid in each vial was recorded. Likewise egg counts for 60 pairs of flies were made for each category for over a period of ten days. The findings of the fecundity is presented in Table 1. The statistical comparison of these has revealed the following: (1) **D.n.nasuta** is more fecund than that of **D.n.albomicana**, (2) the F_1 hybrids of both the reciprocal crosses are heterotic by exceeding the performance of their parents as well as those of Cytoraces, (3) the Cytorace I is more fecund than the Cytorace II and as well as the parental races. The Cytorace II is more fecund than **D.n.albomicana** and it does not significantly differ from that of **D.n.nasuta.**

Thus, the hybridizations between **D.n.nasuta** and **D.n.albomicana** has resulted in the formation of new Cytoraces which differ from their parents in their karyotypic constitution, quantity of heterochromatin and in their ability to lay different number of eggs.

Acknowledgements: Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Zoology, for his help and encouragement; to the University Grants Commission and the Indian National Science Academy, New Delhi, for financial assistance.

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Ramachandra, N.B. and H.A. Ranganath.
University of Mysore, India. Resource utilization divergence in two cosmopolitan and sympatric species of Drosophila.

Drosophila melanogaster and **D.ananassae** of the **melanogaster** species group of the subgenus **Sophophora** of the genus Drosophila are a pair of cosmopolitan and sympatric species. They cohabit in the domestic localities and occur in varying frequencies throughout the year. Preliminary studies made by Nirmala & Krishnamurthy (1973) have revealed that under laboratory conditions, **D.melanogaster** is competitively superior to **D.ananassae.** Mechanisms of their apparent "coexistence" in nature are not known. Different dimensions of their ecological relationship and their requirements or preferences have not been worked out. Our preliminary report (Ramachandra & Ranganath 1984) on the nutritional requirements of these two species revealed the existence of 'subtle' differences. In view of this, experiments have been initiated in this direction to understand the impact of different ecological regimes on certain aspects of the parameters of fitness in these two ecologically closely placed forms of Drosophila.

Drosophila utilize a variety of sugar sources (Bamberger 1919; Hassett 1948; Taylor & Condra 1983; Jaenike & Grimaldi 1983; Powel & Andgelkovic 1983). Nutritional methodology is a sharper instrument for the dissection of the genotype, particularly through the use of variety of sugars (Sang 1972). In the light of this, we have used four different types of wheat cream agar medium containing either molasses or sucrose or glucose or fructose to study the differences in the ability of **D.melanogaster** and **D.ananassae** to utilize these different resources. The impact of differences in the media was assessed by estimating certain parameters of fitness, namely egg to adult rate of development, viability and adaptedness.

The egg to adult rate of development and viability were estimated by adapting the procedure of Ranganath & Krishnamurthy (1974) with little modifications, while the adaptedness was assessed by following the serial transfer technique of Ayala (1965). The present findings are as follows:

(1) The egg to adult rate of development is defined as the time between egg laying and emergence of the imago. The statistical comparison revealed that **D.melanogaster** has the fastest rate of development in the media with molasses and the slowest in the media with glucose, while **D.ananassae** has achieved the fastest rate of development in the media with sucrose and slowest in the media with fructose. **D.melanogaster** has better speed of development than **D.ananassae** in the media with molasses and fructose, while **D.ananassae** has experienced better rate of development than **D.melanogaster** in the media with sucrose and glucose. For **D.melanogaster** the mean rate of developmental time in days in four different media, ranges from 13.57 to 15.53 days and for **D.ananassae**, the range is 13.79 to 15.34 days.

(2) The viability is defined as the proportion of eggs reaching the adulthood. Maximum viability for **D.melanogaster** was in the media with molasses (85%) and the least was in the media with sucrose (69%). For **D.ananassae**, the media with molasses has given the highest viability of 64%, while the least was recorded in the media with fructose (51%).

(3) The adaptedness of a population is defined as "the ability of the carriers of a genotype or a class of genotypes to survive and reproduce in a given environment" (Dobzhansky 1968). Population size, productivity, mortality and flies per bottle were used as four different facets of adaptedness. The analysis of variance test revealed that only in the media with fructose **D.ananassae** has more of adaptedness than **D.melanogaster**. While in all the other three types of media, **D.melanogaster** has significantly more values than that of **D.ananassae**. Both for **D.melanogaster** and **D.ananassae**, the media with molasses is more suitable than other media and the sequence is molasses > sucrose > glucose > fructose.

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Cytogenetic analysis of lethal X chromosomes derived from dysgenic hybrids of *Drosophila melanogaster*.

Simmons et al. (1985) measured the frequencies of recessive X-linked lethal mutations occurring in various dysgenic males derived from crosses between pure M females and P-element-containing males. The latter were collected from two pairs of related stocks: π_2, a strong P strain, and C(1)DX, y f/Y/+; π_2, an attached-X strain with the π_2 genetic background; and ν_6, a Q or weak P strain, and C(1)DX, y f/Y/+; ν_6, an attached-X strain with the ν_6 genetic background. Through genetic manipulations, Simmons et al. (1985) obtained dysgenesis-induced lethal mutations on both the π_2 and ν_6 X chromosomes and on X chromosomes derived from a pure M strain but having been exposed to the autosomes of either of the attached-X stocks. See their paper for experimental details.

We have analyzed 378 of the 1,030 lethal X chromosomes recovered from these P element mutagenesis experiments. In this analysis, a single ℓ/FM7 female from each lethal stock was mated to Df(1)Basc/sc^{8}Y males. The ℓ/Df(1)Basc daughters were then crossed either to y sn^3 v males to produce ℓ /y sn^3 v larvae for cytology, or to duplication/deficiency males in an attempt to locate the lethal mutation through complementation tests. For the latter, we employed six duplication/deficiency stocks; these included the first five stocks listed in Table 1 of Simmons et al. (1984), plus C(1)DX, y w f/Df(1)y^{75e}/y^{2}Y^{67g}. The deficiency in this last stock spans 1A1-2; 1B6-9 and the duplication spans 1A1; 2B17-18 + 20A3;20F4. Cytological data on the other five stocks are given in Table 1 of Simmons et al. (1984). Altogether, the deficiencies in these six stocks allowed us to screen approximately 16 percent of the euchromatic portion of the X chromosome for the location of the lethal mutations. The chromosomes having these lethals were maintained in stocks for 20-30 generations before the cytogenetic analysis began. During this time no effort was made to control the cytotype, so secondary dysgenesis-induced lethal mutations and chromosome rearrangements might have occurred.

A summary of complementation tests with the six deficiencies is given in Table 1. In total, 73.5% of the chromosomes tested had a lethal mutation in at least one of the regions uncovered by the six deficiencies. Before discussing the distribution of these lethals in detail, it is important to note that in many instances where a lethal mutation was uncovered by a deficiency, the corresponding duplication did not cover it; this indicated that the tested chromosome had more than one lethal mutation. Sometimes another deficiency uncovered the extra lethal, permitting its localization. Altogether, 109 (39%) of the 278 chromosomes with localized lethals had extra lethal mutations.

The first category of lethal X chromosomes analyzed came from the weak P strain ν_6. The vast majority (81.8%) of these had recessive lethal mutations in one of the regions uncovered by the y, r, and mal deficiencies. The other deficiencies (w, ct, and m) uncovered lethals on only four chromosomes--less than 3% of those analyzed. The mal deficiency uncovered lethals on over half the ν_6 X chromosomes, the r deficiency on nearly a third and the y deficiency on approximately a sixth. Clearly, these deficiencies reveal hotspots for the occurrence of dysgenesis-induced lethal mutations on the ν_6 X chromosome.

Table 1. Complementation analysis of dysgenesis-induced lethal X chromosomes.

Deficiency	Breakpoints	ν_6 dysgenic hybrids ν_6-X	ν_6 dysgenic hybrids M-X	π_2 dysgenic hybrids π_2-X	π_2 dysgenic hybrids M-X	Total
Df(1)y^{75e} (y)	1A1-2;1B6-9	28 (16.9)	5 (15.2)	22 (19.6)	25 (36.8)	80 (21.1)
Df(1)TEM-1 (w)	2E2-F1;3C1-2	2 (1.2)	0 (0.0)	13 (11.6)	2 (2.9)	17 (4.5)
Df(1)ct^{78} (ct)	6F1-2;7C1-2	1 (0.6)	1 (3.0)	9 (8.0)	3 (4.4)	14 (3.7)
Df(1)m^{259-4} (m)	10C1-2;10E1-2	1 (0.6)	0 (0.0)	2 (1.8)	0 (0.0)	3 (0.8)
Df(1)r^{+75e} (r)	14B13;15A9	48 (29.0)	20 (60.6)	28 (25.0)	25 (36.8)	121 (32.0)
Df(1)mal^3 (mal)	19A1-2;20A	84 (50.9)	1 (3.0)	20 (17.8)	5 (7.4)	110 (29.1)
Total chromosomes with at least one lethal mutation localized		139 (84.2)	24 (72.7)	67 (59.8)	48 (70.6)	278 (73.5)
with additional mapped lethal(s)		24 (14.5)	2 (6.1)	24 (21.4)	11 (16.2)	61 (16.1)
with additional unmapped lethal(s)		5 (3.0)	6 (18.2)	16 (14.3)	21 (30.9)	48 (12.7)
Total chromosomes analyzed		165	33	112	68	378
Clusters		3 of 2 1 of 3	1 of 2 1 of 5 1 of 8	4 of 2 2 of 3	3 of 2	

Entries are numbers of chromosomes, with percentages in parentheses.

Simmons et al. (1984) had detected the r and mal hotspots in an earlier study; however, because they did not use the y deficiency, they failed to notice the hotspot it uncovers. Each of the three mutable regions encompasses a P element site, as judged by in situ hybridization (Simmons et al. 1984), which is probably the major reason for their pronounced mutability. Another P element, located at 11A on the ν_6 X chromosome, lies in a region that was not uncovered by any of the deficiencies used in this analysis, so its contribution to the production of lethals could not be assessed. It is noteworthy that the w, ct, and m regions on the ν_6 X chromosome do not contain any P elements, and neither do they give rise to appreciable numbers of lethal mutations. The few lethals that were detected may have been due to P element insertions in essential loci, or to other events unconnected with dysgenesis. Significantly, only 29 (21.0%) of the 139 ν_6 X chromosomes with locatable lethals had more than one lethal mutation. This is the lowest such frequency among the four sets of X chromosomes that we analyzed. Thus, despite the significant amount of time that elapsed between the recovery of these lethal chromosomes and their analysis by complementation testing, few secondary lethal mutations had accumulated.

Only 33 lethal M X chromosomes derived from ν_6 dysgenic hybrids were analyzed. Nearly half of these were recovered in three different clusters, so the data probably do not represent independent mutational events. This must be taken into consideration when the distribution of the lethal mutations is evaluated. Although 20 (60.6%) of these M X chromosomes had lethals uncovered by the r deficiency, seven came from a cluster of eight and two came from another cluster of five. Counting each of these clusters as two mutational events and the other cluster as one, there were at least 23 independent mutational events in this sample of chromosomes. By comparison, there were at least 13 independent lethal mutations in the r region, suggesting a rather high frequency of mutation therein. Likewise, five independent lethals were uncovered by the y deficiency, pointing to another potential hotspot. Since the M X chromosomes were initially devoid of P elements, these two hotspots could represent sites for preferential P element insertion. These hotspots on the M X chromosome coincide with two of the three hotspots on the ν_6 X chromosome, suggesting that on the latter chromosome, some of the y and r region mutations might have been caused by P element insertions rather than by local P element action.

Lethal mutations were localized on 67 of the 112 π_2 X chromosomes that were analyzed. Nearly 60% of these had more than one lethal mutation, suggesting that many secondary lethal mutations had occurred. The r deficiency uncovered the most lethal mutations, followed by the y, mal, w, ct, and m deficiencies in that order. From in situ hybridization it is clear that the π_2 X chromosome possesses P elements in the regions uncovered by the w and mal deficiencies, but not in those uncovered by the y, ct, m and r deficiencies (Engels & Preston 1981). Therefore, although the high frequencies of lethal mutations in the w and mal regions are explicable in terms of local P element action, the high frequencies in the y and r regions are not. It is possible that the abundance of lethals in these two regions is due to frequent P element insertions. Significantly, the m region and, to a lesser extent, the ct region on the π_2 X chromosome were not unusually mutable; this is consistent with the absence of P elements in these parts of the chromosome.

In addition to the P elements in the w and mal regions, the π_2 X chromosome possesses P elements at 5E, 11A and 17C (Engels & Preston 1981); however, none of these was uncovered by the deficiencies used in this analysis. In this connection it is notable that the π_2 X chromosomes had the highest frequency of unlocalized lethal mutations; 45 of the 112 chromosomes did not have a lethal uncovered by any of the deficiencies. Perhaps some of the lethal mutations on these chromosomes were caused by the 5E, 11A or 17C P elements.

The M X chromosomes that were derived from the π_2 dysgenic hybrids had a concentration of lethal mutations in the regions uncovered by the y and r deficiencies; there were 25 in each case, compared to a total of 68 chromosomes analyzed. This is reminiscent of the distribution of lethals on the M X chromosomes derived from the ν_6 hybrids. Evidently, these two regions are unusually receptive to P element insertions. The receptivity of the y region may reflect a general tendency for P elements to insert near the ends of chromosomes (W.R. Engels, pers. comm.), but the receptivity of the r region would have to be explained on other grounds. Perhaps, along with other frequently mutated loci, some part of the r region has an ideal sequence for P element insertion. Only a direct molecular analysis can settle this issue.

The M X chromosomes derived from the π_2 dysgenic hybrids had a high frequency of multiple lethal mutations; 32 out of 48 chromosomes with localized lethals had at least one extra lethal mutation. This suggests that secondary lethals occurred on these chromosomes, most likely while they were kept in stock.

From this complementation analysis, it appears that dysgenesis-induced lethal mutations are frequently found in chromosomal regions containing P elements. As has been speculated previously (Simmons et al. 1984), these lethals probably arise from local P element action. There is, however, an additional group of lethals that is not associated with local P element activity. The lethals on the M X chromosomes belong to this group, as do the lethals in the y and r regions on the π_2 X chromosome. Curiously, the lethals on the M X chromosomes are concentrated in the y and r regions. The concentration in the y region is all the more striking because the effective size of that region is quite small. Although the y deficiency spans bands 1A1-2; 1B6-9, the bands spanned by the deficiency in Df(1)Basc must be excluded; the reason is that on account of our methods, any lethal exposed by the latter deficiency could not have been tested against the y deficiency. This means that the lethals that were mapped to the y region in fact map to only a portion of that region, revealing a pronounced mutational hotspot. Together with the r region, this hotspot may reflect a tendency of P elements to transpose to particular sites. Eeken et al. (1984) performed very detailed genetic analyses of P-element-induced lethals on M X chromosomes and found quite a scattered distribution. There was no tendency for lethals to map in the y and r regions, as found here. This discrepancy might indicate that X chromosomes differ in the positions of their favored P element insertion sites, or that P elements from different strains have different target specificities.

In addition to the complementation analysis, 255 of the lethal X chromosomes were examined cytologically. Eighty-five (33.3%) contained structural abnormalities, consisting primarily of deficiencies and simple two-point inversions. Excluding the deficiencies, there were 116 breaks in this sample of chromosomes, 32 (26.9%) of which appeared to be associated with a lethal effect. Another 26 (22.6%) may have been lethal, but the remaining 58 (50.4%) definitely were not.

Breakage on the ν_6 X chromosomes was concentrated in bands 14C7-E1 (in the r region) and in bands 18F-20A (in the mal region); 62, or nearly half of the 129 chromosomes that were analyzed, had some sort of structural abnormality. The distribution of breakpoints, which is shown in Figure 1, corresponded closely to the distribution of P elements on the ν_6 X chromosome. Of the 62 chromosomes with rearrangements, 27 had a break in bands 14C7-E1; although none of these breaks was associated with a cytologically detectable deficiency, 26 appeared to have a lethal effect (the viability effect of the remaining break could not be determined). It should be noted, however, that many ν_6 X chromosomes without breakage at this point had lethals in the r region, so breakage is not the only cause of this kind of lethality. The other breakage hotspot on the ν_6 X chromosome was in bands 18F-20A. All 62 of the rearranged chromosomes had breakage in these bands, with the preponderance (40) of the chromosomes having breaks in 19C2-D1. Many of the breaks in 19C2-D1 were associated with lethal deficiencies, but the 14 that were not had no lethal effect. Some of the lethals in the mal region were clearly not associated with any breakage there. Simmons et al. (1984) studied over 30 such lethals and found that they were nearly all allelic to HF326, a lethal mutation obtained from George Lefevre. Since the publication of their paper, HF326 has been reassigned to bands 19B3-C1, rather than to 19C4 as Simmons et al. (1984) reported (George Lefevre, pers. comm.). Significantly, there is a P element in 19C on the ν_6 X chromosome; presumably, the lethals allelic to HF326 arise from the activity of this element, as do the breaks in 19C2-D1.

Only one of the 23 lethal M X chromosomes derived from the ν_6 hybrids which were examined cytologically was structually abnormal. This chromosome had an inversion (breakpoints 11A3-9; 12E-F) which was not associated with any lethal effect. Thirteen of the chromosomes with a lethal in the r region were examined and none had a break in 14C7-E1, the breakage hotspot in the r region on the ν_6 X chromosome.

Sixty-one of the lethal X chromosomes derived from π_2 were examined cytologically and 19 were found to be abnormal. Nine had simple inversions, six had deficiencies and four had more complicated rearrangements. Excluding the deficiencies, there were 36 breakpoints altogether, 3 being lethal, 14 nonlethal and 19 with unknown effects on viability. The distribution of the breakpoints is shown in Figure 1. Except for some clustering near the base, the breakpoints are distributed rather uniformly over the chromosome map. There is no pronounced concentration in the vicinity of resident P elements, as was the

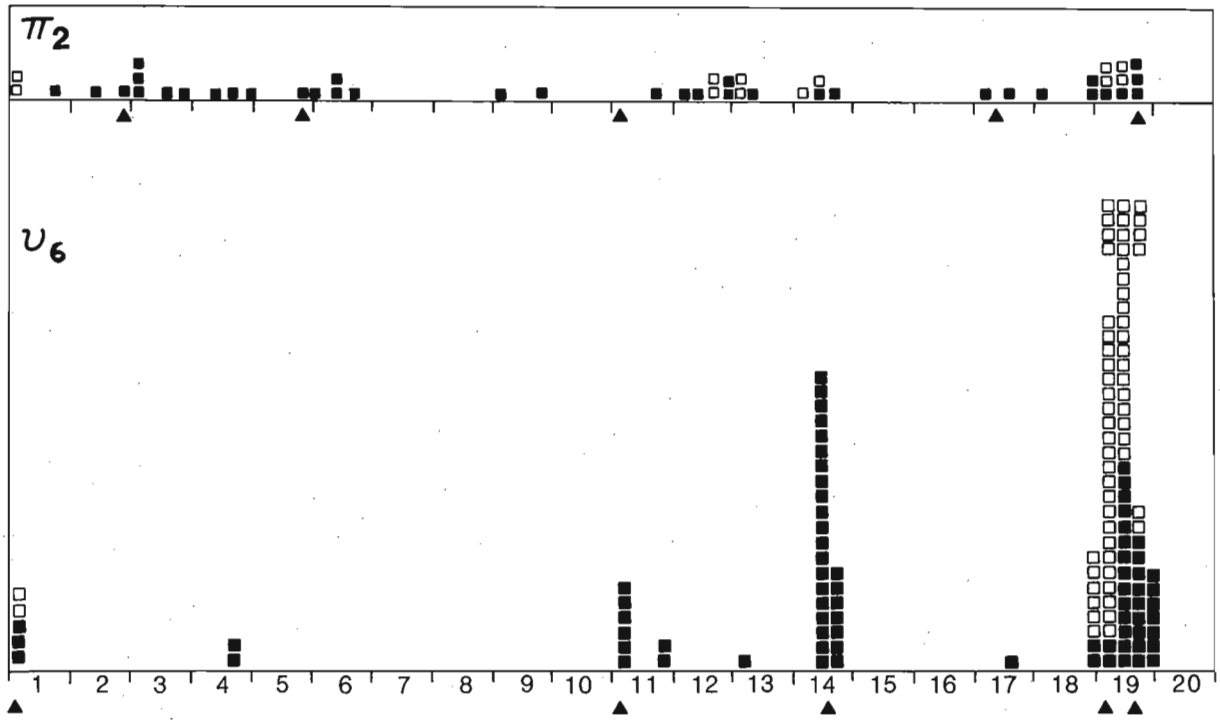

Figure 1. Distribution of breakpoints on π_2 and ν_6 lethal X chromosomes from dysgenic hybrids. Open squares, deficiency breakpoints; closed squares, inversion breakpoints; triangles, P element sites in the chromosomes from which these lethal chromosomes were derived.

case for the ν_6 X chromosome. This contrasts with the results of Berg et al. (1980) and Engels & Preston (1981), who found that the breakpoints of dysgenesis-induced rearrangements on the π_2 X chromosome preferentially involved P element sites. The discrepancy between our findings and theirs might have at least two explanations. First, we selected lethal X chromosomes whereas Berg et al. (1980) selected chromosomes which suppressed recombination and Engels & Preston (1981) selected chromosomes with a rearrangement-associated visible mutation; the latter were all nonlethal. In both instances, chromosomes with primary rearrangements were selected, whereas the chromosomes that we analyzed were selected because they had primary lethal mutations, which may or may not have been associated with rearrangements. Second, the chromosomes we analyzed were kept in stocks for 20-30 generations without controlling the cytotype. In the other studies cited, the rearranged chromosomes were analyzed immediately, or, if this was impossible, after they had been maintained in stocks with the P cytotype, which severely limits P element transposition and P-element-mediated chromosome breakage. Therefore, the scatter of breakpoints on the π_2 map in Figure 1 is probably due to an accumulation of secondary transpositions and rearrangements, which, evidently, did not occur in the sample of ν_6 X chromosomes that we studied. This explanation is supported by the much higher frequency of chromosomes known to have multiple lethal mutations in the sample derived from π_2 (59.7% vs 19.6% from ν_6).

The M X chromosomes derived from the π_2 dysgenic hybrids showed very few rearrangements. Two of the 42 chromosomes that were analyzed had small deficiencies and one had a simple inversion. One of the breakpoints of the inversion was nonlethal, but the viability effect of the other could not be determined. Both of the deficiencies were in the mal region and had lethal effects.

It is evident that the lethal X chromosomes derived from ν_6 and π_2 had numerous rearrangements. In the case of the ν_6 X chromosomes, the breakpoints coincided closely with P element sites. In the case of the π_2 X chromosomes, a coincident distribution of breaks and P elements was not observed. For the lethal M X chromosomes which had been exposed to ν_6 or π_2 autosomes in dysgenic hybrids, few rearrangements were detected. Evidently P-element-mediated rearrangements are rare on these chromosomes, even after they have been maintained for many generations in the absence of the P cytotype.

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Rim, N.R., B.S. Lee and T.H. Lee. Jeonbuk National University, Jeonju, Korea. Inversion polymorphisms in a Korean natural population of **Drosophila melanogaster.**

Chromosome studies of **Drosophila melanogaster** collected five times with a month apart from mid-August to mid-December in 1982 from a large vineyard near Jeonju, South Korea, discovered seventeen inversions among a total of 1056 males tested. For analyzing the pairs of homologous chromosomes seven F1 larvae per male strain were selected randomly. All of the inversions found were paracentric and on two major autosomes only, and none on X- and fourth chromosomes. Among them, six were common cosmopolitan inversions, and the other eleven were new, rare and endemic inversions. The inversions found in this study are listed in Table 1. And the frequencies of each common cosmopolitan inversion are as shown in Table 2. Therefore the frequency of wild males carrying one or more cosmopolitan inversions and the mean number of cosmopolitan inversions per individual is 0.540 and 0.743, respectively. Although linkage disequilibrium appeared significantly within the third chromosome when all of the third chromosomal combinations were pooled, the data indicate a random distribution of inversions on the whole (Table 3). And, in the present population of **D.melanogster** as shown in Table 4, there was no differential mortality for inversion homozygotes and heterozygotes by natural selection. Particularly, one of the interesting results in this study was the discovery of a cis-coupled overlapping inversion which was identified to be the cosmopolitan In(3R)C and In(3R)P in approximate breakage points. Therefore, if cosmopolitan inversions were originally produced by site-specific transposable elements, and also, if the cis-coupled overlapping inversion was in fact composed of the two cosmopolitans, the possibility that the origin of cosmopolitan inversions may be from each geographical population just as endemics cannot be excluded.

Table 1. Polymorphic inversions from a Jeonju natural population of **Drosophila melanogaster.**

Inversion	Approximate Breakpoints	Month collected (%)
Second, Left		
In(2L) A	22B - 26A	A(0.5),S(0.9),O(1.4),N(0.5),D(0.5)
In(2L) B	22E - 34A	All samples (see Table 2)
In(2L) C	26A - 33E	D(0.5)
In(2L) D	30E - 33B	A(0.5)
Second, Right		
In(2R) E	42E - 50B	D(0.5)
In(2R) F	42C - 56E	D(0.5)
In(2R) G	42C - 58B	S(0.4)
In(2R) H	53A - 56F	All slamples (see Table 2)
Third, Left		
In(3L) I	63C - 72E	All samples (see Table 2)
In(3L) J	64D - 67A	S (0.9)
In(3L) K	66C - 71B	N(0.5)
In(3L) L	67A - 71B	N(0.5),D(1.0)
Third, Right		
In(3R) M	89C - 96A	All samples (see Table 2)
In(3R) N	91D - 96F	N(0.5)
In(3R) O	92D -100F	All samples (see Table 2)
In(3R) P	93D - 98F	All samples (see Table 2)
In(3R) Q	96A - 98D	A(1.0)
Overlapping Inversion		
In(3R) PC	89C - 96A	S(0.4),O(0.9): repulsion phase
	92D -100F	N(0.4): coupling phase
In(3R) PMo	89C - 96A	N(0.5): repulsion phase
	93D - 98F	
Included Inversion		
In(3R) CMo	92D -100F	S(0.4): repulsion phase
	93D - 98F	

Table 2. Frequencies of sperms carrying each of six cosmopolitan inversions from five samples of **D.melanogaster.**

Sample	N	(2L)t	(2R)NS	(3L)P	(3R)C	(3R)Mo	(3R)P	FI	MI
Aug.	400	0.135	0.103	0.013	0.010	0.013	0.025	0.500	0.62
Sep.	462	0.071	0.102	0.052	0.033	0.013	0.096	0.524	0.74
Oct.	416	0.041	0.142	0.031	0.051	0.010	0.099	0.567	0.75
Nov.	428	0.094	0.140	0.056	0.051	0.019	0.110	0.617	0.94
Dec.	406	0.084	0.079	0.039	0.030	0.017	0.086	0.493	0.67
Pool	2,112	0.084	0.113	0.039	0.035	0.014	0.085	0.540	0.74

N=no. of wild sperms treated; FI=frequency of males carrying one or more cosmopolitan inversions; MI=mean no. of cosmopolitan inversions per male.

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Table 3. Correlation coefficients from chromosomal inversion associations between chromosome arms and between 2nd and 3rd chromosome.

Association	Aug.	Sep.	Oct.	Nov.	Dec.	Mean ± Se
2L - 2R	-0.109	-0.065	-0.049	0.055	-0.088	0.073 ± 0.011
3L - 3R	-0.027	-0.041	-0.078	0.415**	0.219**	0.098 ± 0.095***
2L : 3L	-0.044	-0.011	0.033	0.201**	0.076	0.051 ± 0.043
2L : 3R	0.137	0.077	0.043	0.142*	0.013	0.082 ± 0.025
2R : 3L	-0.038	0.050	0.085	0.106	0.035	0.048 ± 0.025
2R : 3R	0.031	0.065	0.163*	0.039	0.176**	0.095 ± 0.031**
2 : 3	0.022	0.045	0.081	0.122	0.075	0.069 ± 0.031

* P<0.05; ** P<0.01; *** P<0.001.

Table 4. Homozygosity test to determine deficiency of homozygotes by differential mortality.

Sample		No.Obs.	No.Exp.	t	P
Aug.	In/In	4	5.99	18.956	-1.366	0.086
	St/St	99	102.96		
Sep.	In/In	4	6.76	22.971	0.814	0.792
	St/St	110	103.33		
Oct.	In/In	3	7.30	22.350	-1.668	0.048*
	St/St	92	94.58		
Nov.	In/In	5	9.77	27.529	0.394	0.653
	St/St	83	75.96		
Dec.	In/In	5	4.75	17.391	-0.284	0.388
	St/St	100	101.44		
TOTAL	In/In	21	34.77	106.624	-0.398	0.345
	St/St	484	479.27		

* P, probability.

Rypstra, A.L. and T.G. Gregg. Miami University, Oxford, Ohio USNA. Facultative carnivory in **Drosophila hydei.**

Members of the family Drosophilidae are characterized by their tendency to forage on yeasts, other fungi, and bacteria associated with fermenting and decaying vegetable matter. The few known exceptions have been reviewed by Ashburner (1981), and Lachaise & Tsacas (1983). In the genus Drosophila itself, reports of facultative utilization of other substrates, as opposed to permanent shifts (such as the **simulivora** subgroup which has evolved predatory aquatic larvae, or **D.carcinophila** and **D.endobranchia** which live and reproduce in the excretory canals of land crabs), have been restricted to **D.melanogaster, D.buskii,** and **D.latifaciaeformis.** These species have been reported breeding successfully in the wild on dead Lepidoptera caterpillars. Whether this ability is a species characteristic in each case or has only evolved in populations in certain habitats in particular geographical areas where there is strong selection for alternative substrates has not been addressed.

Apparently no account exists, anecdotal or otherwise, of **Drosophila hydei** breeding on alternative substrates. Here we report examples of this species reproducing on a dead cicada (**Tibecen chloromera**), a dead spider (**Nuctenea cornuta**), the carcasses of other adult **hydei**, other **hydei** pupae, and even ground beef (**Bos taurus**). The species identification of the cicada was not absolutely certain because of its deteriorated condition when the identification was made. The first example of facultative entomophagy came to our attention in an experiment in which adult **hydei** were to serve as prey for spiders. These adults were placed in one pint specimen jars containing single female spiders. Moisture was supplied by a small piece of damp sponge (2 cm x 2 cm x 1 cm) but no food source was provided for the Drosophila. After one week a cicada was added to one jar as additional prey for the spider. Twenty-four hours later the cicada was dead. It was not, however, killed by the spider nor did the spider feed on it. Six days after the death of the cicada, what appeared to be Drosophila puparia were noticed on the side of the jar. Eleven days after the cicada died and 12 days after it had been placed in the bottle, adult **D.hydei** began to emerge. This is decidedly faster than they emerge on the cornmeal-molasses-agar laboratory medium (17 days) or on Carolina instant medium (15 days). A total of 30 adults emerged over a four-day period. The possibility that the cicada had been previously parasitized by a Drosophila-like fly was ruled out by a careful comparison of the emerging adults with laboratory **hydei** stock.

Once our attention had been focused on this instance of entomophagy we noticed larvae and puparia of **D.hydei** in several other jars. In some cases the only food available to the larvae were the carcasses of dead adults. In other cases the spider had molted adding its exoskeleton as a potential food source. In one instance the spider itself died and was thus available for consumption by the **hydei** larvae. There is no evidence that the larvae attempted to utilize the sponge as a food source.

When feeding on adult **hydei** carcasses the larvae were able, either by themselves or with the help of bacteria, to completely solubilize the adult exoskeletons except for the wings and the heads. In instances in which the spiders had molted, their exoskeletons were worked over by the larvae and partially solubilized. The jar which contained the dead spider had only a few living Drosophila remaining at the time the spider died. Consequently, only a few eggs were laid. Nonetheless three larvae developed, were able to solubilize the spider's abdomen, pupariate, and emerge as adults. These larvae appeared fully robust and vigorous. We cannot exclude the possiblity of bacterial hydrolysis of the tissues and exoskeletons in these examples. However, the fact that they do not solubilize in the absence of larval activity leads us to conclude that the larvae themselves are secreting digestive enzymes onto the carcasses. It has been observed that larvae in the **cardini** group cannibalize their own pupae in laboratory cultures by producing entry holes into the puparia of other individuals (Heed & Krishnamurthy 1959). In that instance, too, it seems plausible that those larvae were secreting enzymes extra-somatically in order to gain entry. The larvae of **hydei** will also cannibalize puparia, sometimes pupariating within the original puparium. Larvae

provided with puparia as the only food source appear superficially to gain entry by a combination of chemical and mechanical attack.

Given **hydei**'s quite surprising success in breeding on insects and spiders, we attempted to culture them on ground beef as a substrate. A small amount of raw ground beef was placed in a jar with some breeding adults. Females appeared reluctant to oviposit on this substrate, but nonetheless some eggs were laid and larvae hatched. After two days the adults were removed so that dead adults could not serve as substrate. For the first 10 days the larvae spent much time wandering about the jar apparently looking for a more appropriate food source. They seemed to be both attracted to, and repelled by, the ground beef. They did not grow well and were not robust and vigorous. In the length of time it took for adults to emerge from the cicada (11 days) these larvae had only developed to the second and early third instar stages. However, after the 10th day the substate seemed to become much more palatable and some of the larvae began to flourish. By the 26th day adults began to emerge, taking more than twice as long to develop as the flies developing on the cicada. On the 20th day a second batch of breeding adults was added to the jar and removed two days later. These females found the ground beef, which by now had a thin film covering it, a very attractive oviposition site and littered it with eggs. The thin film presumably was the result of bacterial action. The larvae immediately took to this aged ground beef, but the length of time required before adults began to emerge was still much longer than for the cicada or the lab media. Nonetheless, the developmental time was shortened from 26 days to 20 days.

The behavior of **hydei** on ground beef is consistent with the widely held belief that Drosophila actually feed on the yeast and/or bacteria that are growing on the fermenting and decaying substrate. However, in the case of the cicada the larvae apparently were feeding on the insect tissues directly. Six of the adults that emerged from the cicada were weighed and averaged 2 mg. If we assume that the puparium and larval sheaths weigh another 0.5 mg, an emerging adult accounts for 2.5 mg of biomass. Thus 30 adults would account for 75 mg of Drosophila biomass. The nine third instar larve that failed to pupariate properly probably account for another 10 or 15 mg for a grand total of 90 mg. The cicada exoskeleton, after it had been quite thoroughly cleaned out by the **hydei** larvae weighed 420 mg. The mean weight of 20 living cicadas was 1330 mg. Thus, a reasonable estimate is that the cicada contained from 900 to 1000 mg of consumable biomass. Based on the standard assumption that there is only a 10% efficiency in converting the biomass of one trophic level to that of a higher one, 900 mg of cicada could only have supported the production of 90 mg of Drosophila if the larvae consumed most of the cicada tissues directly, with very little of it having been first converted into the biomass of microorganisms. If the cicada had been first converted into microorganisms which were then consumed by the larvae, 9000 mg (9 grams) of consumable cicada biomass would have been required to produce 900 mg of microorganisms to be converted into 90 mg of Drosophila, and a 9 gram cicada (or really 13.3 grams including the exoskeleton) is out of the question. Even these rough calculations strongly suggest that the **hydei** consumed the cicada tissues directly.

Accepting this evidence that most of the tisses were consumed directly, questions arise concerning the role of **hydei** digestive enzymes. Do these enzymes accomplish the initial breakdown of Arthropod tissues, or is the initial solubilization of tissues accomplished by cellular autolysis and/or microbial digestion? If solubilization and utilization depend on **hydei** enzymes are these enzymes secreted onto the substrates to be digested? We conclude that not only are these larvae producing their own digestive enzymes, but that they are indeed secreting them extra-somatically onto the substrates being consumed. The evidence for these conclusions is based on (1) our calculation that there was not much excess cicada biomass to spare on microorganisms, (2) our observation that adult **hydei** carcasses and spider exoskeletons were not solubilized in the absence of larval activity, and (3) the fact that the larvae turned the viscera of the cicada, the spider, and the adult flies into juicy messes, much as they do the culture medium. In addition, extra-somatic secretion of digestive enzymes is consistent with the very large size of the salivary glands relative to the rest of the digestive system, a characteristic of the entire order, Diptera. Unfortunately, little is known about the role of salivary glands in the production of digestive enzymes. Extra-somatic secretion of digestive enzymes, whether by the salivary glands or not, is also consistent with the observation that Drosophila larvae, as well as those of other Dipterans, frequently cluster together in writhing masses as though there is a collective effort to solubilize the substrate. A third question is, if the larvae do produce enzymes for the initial solubilization of arthropod and mammalian substrates, are these the same enzymes used to digest microorganisms and plant material, or a different set specifically produced for this purpose?

The ability of the **hydei** to breed on unusual substrates in the laboratory does not insure their ability to compete for these resources in the wild. Nonetheless, it is plausible that this ability may enable **hydei** to opportunistically exploit unusual niches and thereby be part of the reason that **hydei** is a cosmopolitan species. These results also suggest that the ability of Drosophila in general to use a variety of resources has been underestimated, and their dependence on yeast and microorganisms exaggerated.

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Salam, M.A.* Moscow State University, Moscow, USSR. The inhibiting effect of actinomycin-D on the yield of N-nitroso-N-ethylurea-induced second chromosome recessive lethal mutation in **D.melanogaster.**

Table.

Mating pairs male	female	Total no. of chromosomes tested	% of lethals
Control	Control	1009	0.19±0.09
Control	NEU	1245	18.88±0.35
Control	ACM-D	1171	1.37±0.17
ACM-D	Control	1141	1.13±0.09
ACM-D	NEU	1087	12.12±0.84
Control	ACM-D + NEU	1156	9.83±0.36

Actinomycin-D (ACM-D) was found capable of reducing the frequency of X-ray induced recessive lethal mutations and translocations (Burdette 1961, Mukherjee 1965; Proust et al. 1972), and one of the methods offered for explanation was the inhibition of protein synthesis. The genetic effect of ACM-D is contrasting on different parameters. On one hand it causes chromosome aberrations, but, on the other, inhibits the occurrence of spontaneous mutations (Puglisi 1968). The biological effects of this substance include: (i) inhibition of protein synthesis, and also (ii) inhibition of DNA mediated RNA synthesis.

The present work was undertaken with a view to find out whether ACM-D would be able to reduce the N-nitroso-N-ethylurea (NEU) induced second chromosome recessive lethal mutation.

Domodedovskaya-32 (D-32) males and the Curly Lobe/Plum (CyL/Pm) females were used for the experiment. For the treatment with ACM-D flies were starved for 24 hr, and then a properly drenched filter paper in ACM-D solution (1.5 mg ACM-D + 2 mg sugar + 100 ml distilled water) was kept in the vials of flies. Treatment with NEU (30 mg NEU in 1 ml distilled water) was made by exposing flies to NEU-vapour in the air tight dessicator. Duration of treatment was 24 hr at 25°C. For the detection of the second chromosome recessive lethal mutation the classical CyL/Pm method was used. Results pooled for experiments are tabulated in the Table below.

The present study indicates that ACM-D holds the ability to reduce the NEU-induced second chromosome recessive lethal mutation.

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Sanchez-Refusta, F. and E. Garcia-Vazquez. University of Oviedo, Spain. Correlation between inversion polymorphism and extra bristles variation in isofemale lines of **D.melanogaster.**

There are several studies which relate inversion polymorphism with morphometrical variation for different characters and species: White & Andrew (1960, 1962) and White et al. (1963) for body size in **Moraba scurra**; Prevosti (1965, 1967) and Aguade & Serra (1980) for wing length in **D.subobscura**; Butlin et al. (1982) for wing length in **Coelopa frigida** males.

In this work we perform the correlation in **D.melanogaster** between inversion polymorphism and the polygenic character: "extra bristles" both dorsocentrals and scutellars.

Wild flies were captured in banana-yeasted traps and belong to ten natural populations geographically apart from each other by at least 10 Km. Their offspring were analysed in first generation in the lab. This analysis reveals the variation for extra bristles that generally underlies hidden in natural populations, where individuals usually present a normal phenotype (Figure 1). Isofemale lines are classed by counting 60 males and 60 females into three groups (classes) according to the rate of individuals carrying extra chaetae: Class I gathers those lines with less than 5% of individuals with extra bristles (Low variability); Class II from 5% to 20%; and Class III more than 20% (High variability). Inversions are determined through direct observation of the chromosomes of one single larva from each

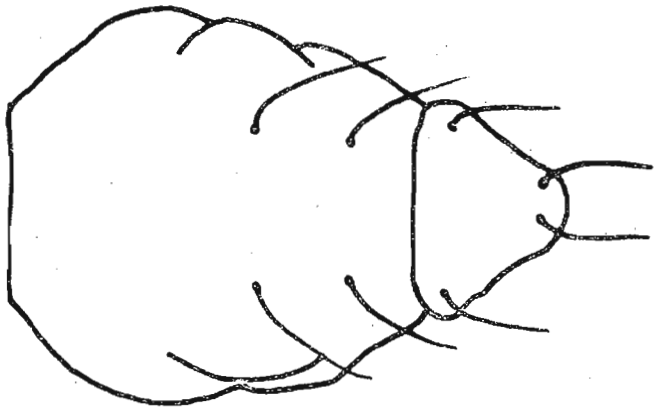

Figure 1. Normal phenotype of bristles in natural populations (A), and some phenotypes of extra bristles (B).

Table 1. Correlation between the two levels of variability. L.E.= % of lines with extra bristles (classes II and III). I.I.=% of individuals with inversions. N.L.=number of lines. r=0.8593 ** 8 df.

Sample	L.E.	I.I.	N.L.
Villaviciosa	39.02	46.34	41
Somio	42.86	70.33	91
Soto del Barco	58.33	66.67	48
Proaza	64.44	75.55	45
Sandiche	65.63	82.81	64
Trubia c.	66.18	83.82	68
Aviles c.	69.05	92.86	42
Aviles nc.	72.73	87.27	55
Trubia nc.	73.08	88.46	26
Celorio	77.78	83.33	36
Average:	62.91	77.74	

Table 2. Distribution (in percent of chromosomes in each class) of each inversion in the three classes of lines. N.C. = number of chromosomes. [a] = N.S.

Classes	2Lt	2RNS	3LP	3RP	3RC	NC
I	17.91	11.19	14.43	19.65	9.20	402
II	20.08	10.24	18.11	15.75	16.14	508
III	22.95	17.21	15.57	16.39	20.49	122
$X^2_{2\ df}$=	1.67[a]	4.77[a]	2.29[a]	2.47[a]	13.92**	

line using as pattern the standard map of Lefevre (1976). We take into consideration for study the cosmopolitan inversions 2Lt,2RNS, 3LP, 3RP and 3RC.

Table 1 shows the correlation between the two levels of variability measured as percent of lines with extra bristles (Classes I and II) as well as percent of individuals carrying at least one inversion. This correlation is proved to be positive and significant ($p<0.01$). Population variation for both levels undergoes increases and decreases of the same sign.

Table 1 shows, too, the high variability of these populations for both characters: particularly the frequencies of inversions, which are the highest reported from Europe until present.

When we class the isofemale lines by their range of extra bristles frequency, we find that only inversion 3RC is unequally distributed among classes I, II and III: the higher rate of flies with extra chaetae; the higher frequency for this inversion is found (Table 2). Conversely this effect is not found for any of the other cosmopolitan inversions.

This 3RC effect can be related to the general chromosome 3 influence reported by other researchers in experiments dealing with selection for increase of the number of extra bristles (Scowcroft 1966; Whittle 1969). Rubio & Albornoz (1982) find in chromosome arm 3R a segment carrier of polygenic loci that enhances the expression of extra bristles in a selected line. This segment lies from 70.0 to 100.7 cM on 3R arm and overlaps 3RC inversion which lies from 84.7 to 110.0 cM. This fact suggests that in the natural populations analysed, In(3R)C carries as a polygenic complex, some factors that favour the expression of extra macrochaetae.

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Frequency dependent sexual activity.

Preliminary experiments have shown that the so-called "rare male mating advantage" was particularly evident when wild Canton S flies are in competition with flies from the well known mutant strain rucuca, whose curled wings could perhaps be a visual stimulus for the sexual activity of the wild males (Kaningini et al. 1986, this issue). The estimation of the "rare male advantage" was done using Wattiaux's equation (1964): a logarithmic transformation changes the sigmoid curves of mating successes into straight lines, which differ in slope or in elevation, and can be compared by covariance analysis (Snedecor 1956). Wattiaux suggested comparing the following regression lines: (1) homogamic and heterogamic matings, (2) matings of the males of both strains in competition, (3) matings of the females of both strains in competition. This formula has already been applied to sexual competition experiments where both types were in equal number (Elens et al. 1974). In the present experiment, the same method is applied to the sexual activities of the two types in competition, when their relative frequencies differ.

Besides the wild Canton S and rucuca strains, the blind flies of the ora strain have been used, in competition with the rucuca ones; as they are blind, the possibility of any visual stimulus can be excluded. The method of observation has been previously described: matings are observed in a small mating chamber, and individuals may mate more than once (Elens 1958; Elens & Wattiaux 1964; Ehrman 1966; Petit 1968). 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: 5/25, 10/20, 15/15/, 20/10, 25/5 (the frequencies being the same for both sexes of the

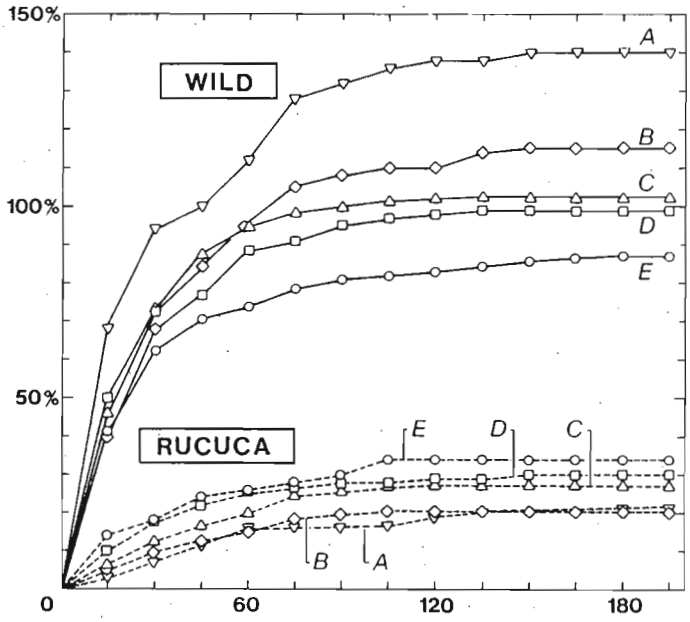
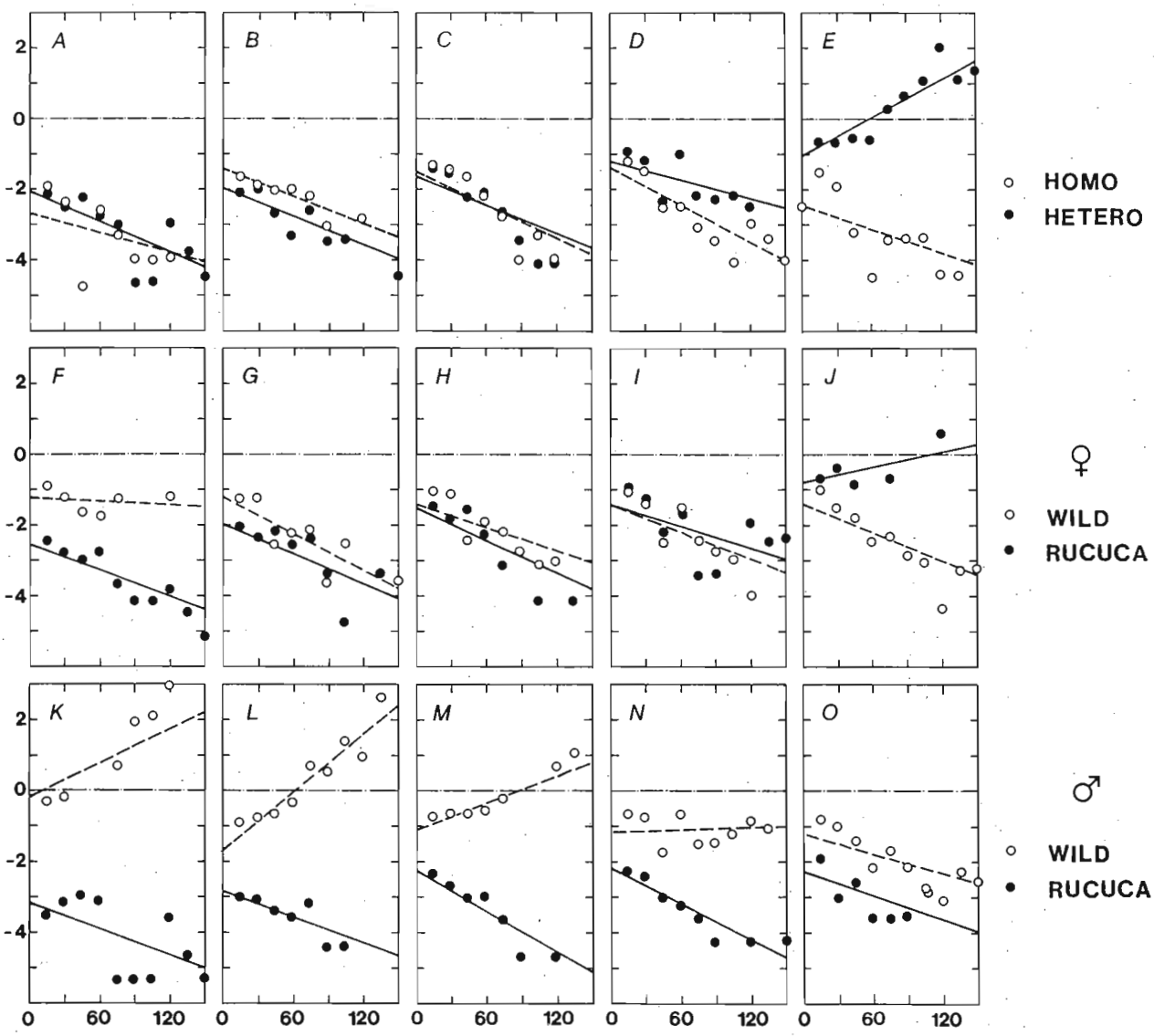


Figure 1. Cumulated percentage of the "male sexual activity" of wild Canton S and rucuca flies: the wildtype males copulate more than once, when "rare". Relative frequencies: A = 5 +/25 ru; B = 10 +/20 ru; C = 15 +/15 ru; D = 20 +/10 ru; E = 25 +/5 ru.

Figure 2. Relationships between the competing types wild Canton S and rucuca, when their relative frequencies differ: columns, from left to right = 5 +/25 ru; 10 +/20 ru; 15 +/15 ru; 20 +/10 ru; 25 +/5 ru. **Abscissa:** time in minutes. **Ordinate:** **ln** dx/N - X. **ln** is the naperian logarithm, N is the total number of flies of one sex and one type, dx the number of these flies mating in the considered interval of time dt (15 min), and X the number of these flies having already mated before.



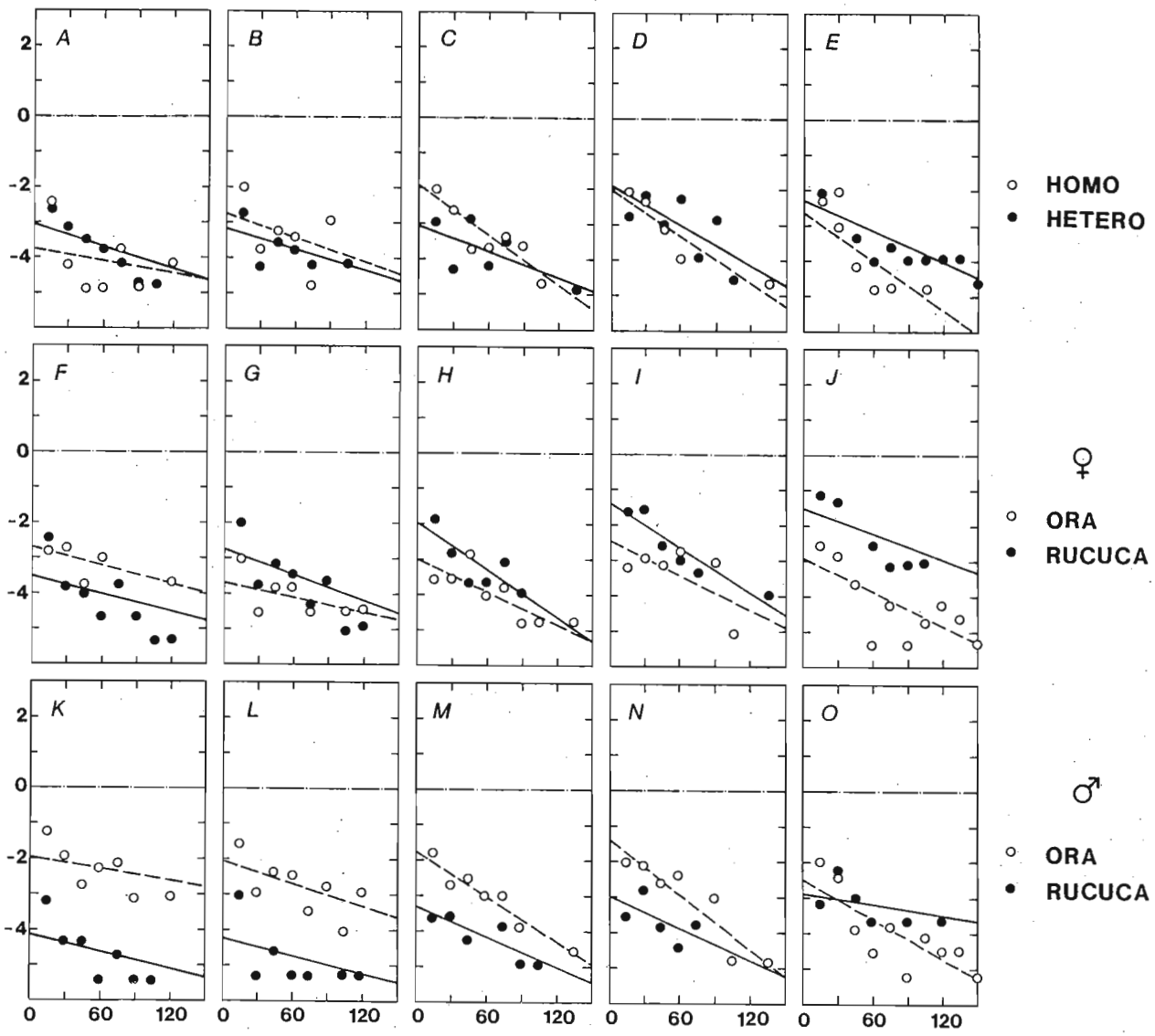


Figure 3. Relationships between the competing types ora and rucuca, when their relative frequencies differ: columns, from left to right = 5 ora/25 ru; 10 ora/20 ru; 15 ora/15 ru; 20 ora/10 ru; 25 ora/5 ru. **Abscissa:** time in minutes. **Ordinate:** **ln** dx/N - X. **ln** is the naperian logarithm, N is the total number of flies of one sex and one type, dx the number of these flies mating in the considered interval of time dt (15 min), and X the number of these flies having already mated before.

same type). 24 hours 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. For the test, the females of both types were introduced together in the mating chamber. Afterwards, the male of both types were similarly introduced, in the same frequency as the females. All the observations were done simultaneously for the five relative frequencies, at 25°C, 1000 lux, and 40-60% relative humidity, with 10 repetitions for each test.

As in previous experiments, the greatest differences concern the "male sexual activities": the wild type males are always more active. When they are "rare", they evidently copulate more than once (Fig. 1). The regression lines characterizing the competition with the mutant rucuca of the wild type flies and of the blind flies ora are given in Fig. 2 and 3. The most intersting observation is that the "frequency dependence" of both the male and the female activities is a much more general phenomenon than was expected. A "rare male advantage" is generally observed, not only for the wild or the ora flies but even for the rucuca ones. The difference is highly significant ($P<0.001$) between the male activity of the wildtype flies at the frequency 5 +/25 ru and at the other frequencies ($P<0.001$). The difference is significant (P at least <0.05 and sometimes <0.01 or even <0.001) between the activity of the ora males at

the frequency 5 ora/25 ru and the other ones. Even the rucuca males can be significantly more active when rare (Fig. 2, 0 and Fig. 3 0), although they are never "advantaged" in the competition. The wild females are significantly more active, when rare, than the rucuca ones (Fig. 2, F); they are less active when the rucuca flies are rare (Fig. 2, J). For the intermediate frequencies, the differences between the two types are never significant (Fig. 2). In the competition between ora and rucuca, the rucuca females are more active than the ora ones only when they are "rare" (Fig. 3, J); at the other frequencies, the activity of the ora females is always higher (Fig. 3). A "rare female advantage" is observed for the rucuca flies as well as for the wild ones (Fig. 2, F and J), and for the ora as well as for the rucuca ones when they are in competition (Fig. 3, F and J). If one compares the relative frequencies of "homogamic" and "heterogamic" matings, one sees that the frequency of "homogamic" mating is significantly higher only when the rucuca flies are "rare" (Fig. 2, D & E; Fig. 3, E). The "heterogamic" matings are more frequent ($P<0.05$) only when the relative frequency between wild and rucuca flies is: 20 rucuca for 10 wild (Fig. 2, B). As a general conclusion, one could say that in our experiment the sexual activity appears as "frequency dependent" (for females as well as for males) in almost every condition, whatever the causes of such a phenomenon.

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Sharma, A.K. and K.S. Gill. Punjab Agricultural University, Ludhiana, India. Heterosis, homeostasis and genotype x environment interaction for acid phosphatase in **D.malerkotliana.**

Acid phosphatase (ACPH), a dimeric enzyme in **D.malerkotliana**, is controlled by a single locus Acph-1. We prepared homozygous stocks of two alleles Acph-$1^{1.05}$ and Acph-$1^{0.90}$, hereafter referred to as fast (F) and slow (S) depending upon their mobility towards anode. Biochemical properties of various enzyme preparations were studied at 25±1°C. There was no significant difference in the two sexes for enzyme activity per unit enzyme concentration, specific activity, Vmax and Km values. At pH 5.0 specific activity of the enzyme in crude extracts of S/S, F/F and F/S flies is 5.85, 10.78 and 16.39, respectively. Higher specific activity of the enzyme in crude extract of heterozygotes was also observed over a range of nine substrate concentrations (0.1 mM to 10.0 mM) and a range of 20 pH values (4.7 to 7.1). Thus with respect to specific activity, heterosis is being exhibited by heterozygotes. Vmax value of enzyme from S/S, F/F and F/S genotypes was found to be 1.51, 1.91 and 3.51 and Km value 0.50, 0.39 and 0.31, respectively. Thus enzyme from heterozygote has a better quality. For slow, fast and hybrid allozymes, partially purified from heterozygotes, specific activity was 4.50, 8.20 and 9.75, respectively. Thus was shown the higher efficiency and the better quality of the heterodimeric allozyme, which explains the heterosis observed in heterozygotes.

For homeostatic studies, specific activities in crude extracts from three genotypes were determined at pH values ranging from 4.7 to 7.1. The optimum pH of crude enzyme preparations from S/S, F/F and F/S genotypes was 5.9, 6.2 and 6.2, respectively. Partially purified slow, fast and hybrid allozymes showed peak activity at pH 5.9, 6.2 and 6.1, respectively. The enzyme from F/F extract showed a better homeostatic ability than that from S/S extract at pH value lower than the optimum pH, after which the relation is reversed. The extract from heterozygotes, however, exhibited higher stability than that from either homozygote on each side of the optimum pH. After comparing the homeostatic abilitty of the enzyme in a 1:1 mixture of crude extracts from the two homozygotes with those described above, it has been concluded that higher homeostatic ability of the heterozygotes is entirely due to the presence of heterodimeric allozyme. Subsequently, it was shown that homeostatic abiity of heterodimeric allozyme is higher than that of either homodimeric allozyme. Higher stability of ACPH in heterozygotes thus seems to arise from superior stability of heterodimeric allozyme.

For determining genotype x environment interaction, enzyme activities at different pH values and at different substrate concentrations were measured. It was observed that in most of the cases, for a similar change in pH or concentration of substrate, the enzyme in crude extract of S/S, F/F and F/S flies and partially purified allozymes from heterozygotes showed significantly different response.

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Weak points in nonsquashed salivary gland chromosome of **Drosophila melanogaster.**

Weak points (break, constrictions, attenuations, etc.) can be considered as best diagnostic characters for definition of intercalary heterochromatin in squashed polytene chromosomes. They are known since rediscovery of polytene chromosomes in early thirties (see Zhimulev et al. 1982, for ref.). However, it is not known whether they appear as a consequence of squashing the cells or exist in nonsquashed chromosomes. To clarify the situation, we analysed serial sections of nonsquashed salivary gland cells of late third instar larvae of giant (gt/gt^{13z}) stock.

Salivary glands were dissected in Ephrussi-Beadle solution, then fixed in 2.5% glutaraldehyde, postfixed in 1% osmium tetroxide, dehydrated in ethanol and acetone, and embedded into Epon.

Serial sections (ca 1 micron) of whole salivary glands made by Ultratome IV (LKB, Sweden) were put on slides, stained with methylene blue and analysed under light microscope.

On the section of nucleus chromosome twists can mimic weak points; therefore only those chromosome fragments were analysed which were clearly seen before and after weak point. Totally 12 nuclei were studied; weak points in each were mapped according to Bridges map (Table 1, Figure 1). If the exact location of weak point appeared to be difficult, such case was marked as "nonlocated" in the Table.

Weak points were found in 15 chromosome regions; the 35B was found to be broken in three and five regions (11A, 12E, 36D, 42B and 75C) in two different nuclei (Table 1). In all 15 regions, weak points in squashed preparations were found with high frequency (Zhimulev et al. 1982).

In one section thread of ectopic pairing was seen (Fig. 1f).

So, data obtained demonstrate that weak points are rather numerous in nonsquashed chromosomes.

Reference: Zhimulev, I.F. et al. 1982, Chromosoma 87:197-228.

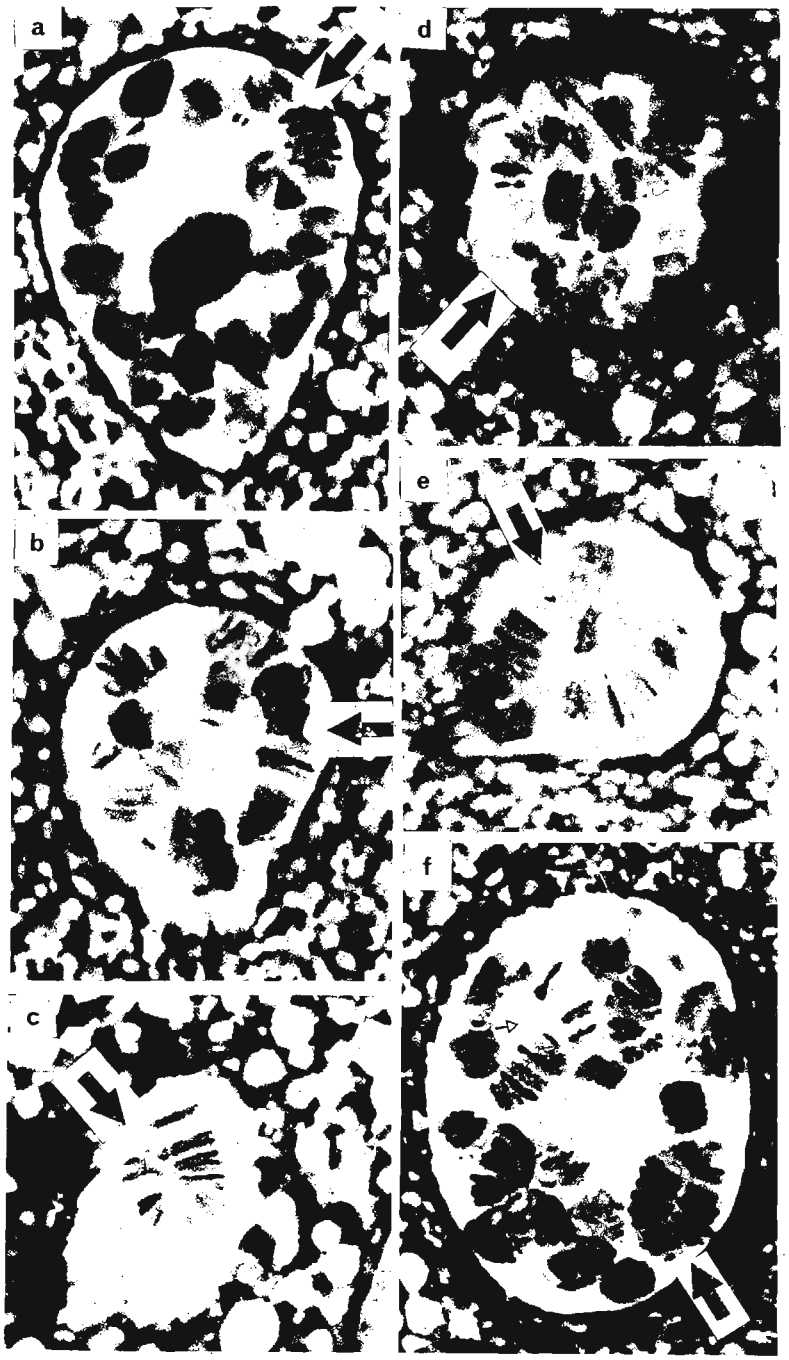


Figure 1. Polytene chromosome weak points in sections of salivary glands are shown with arrows: 12E(a), 35B(b), 98C(c), 11A(d), 42B(e) and 7B(f). Arrow with open head in (f) shows ectopic thread.

Table 1. Localization of weak points in sections of nonsquashed salivary gland nuclei.

No. of nuclei	Weak points in nucleus: Number	Location	No. of sections studied
1	1	non located	12
2	8	3C,7C,11A,12E,35B,36D, 42B,75C	13
3	2	35B, 75C	7
4	3	11A, 12E, 17A	5
5	3	22A, 42B, 67D	4
6	2	36D, 64C	5
7	2	35B, 70C	7
8	3	7B, 98C, non located	7
9	1	non located	2
10	0	---	5
11	0	---	2
12	0	---	1

Shyamala, B.V. and H.A. Ranganath. University of Mysore, India. Drosophila fauna of Mysore and Nallur (South India).

Mysore, located along 12°5' N latitude between 76° and 77° E longitude is a planar region, about 450 metres above the sea level with moderate rainfall. Nallur is located at the border of Western Ghats, 120 km from Mysore at an altitude of 900 m above sea level, with heavy rainfall and humid climate. Collections were made at 5 sites about 5 km apart in Mysore and in Nallur during the months of August and September 1985. Fermenting banana seeded with yeast was used as the bait. Collection records are given in Table 1.

Table 1. Distribution of different species of Drosophila in Mysore and Nallur.

Species	MYSORE Collection sites I	II	III	IV	Total	NALLUR
Subgenus: **Sophophora**						
D.malerkotliana	516	1017	78	116	1727	23
D.bipectinata	115	685	11	153	964	107
D.parabipectinata	2	4	--	2	8	--
D.pseudoananassae	5	3	--	--	8	--
D.takahashii	1	38	--	--	39	3
D.eugracilis	3	7	--	--	10	3
D.rajasekari	7	346	16	--	369	--
D.jambulina	1	44	--	4	40	--
D.punjabiensis	--	17	--	--	17	--
D.mysorensis	3	--	--	--	3	--
D.aqumbensis	--	2	--	--	2	--
D.nagarholensis	--	--	--	--	--	40
D.anomelani	2	2	--	1	5	4
Subgenus: **Drosophila**						
D.n.nasuta	543	673	3	116	1336	169
D.s.neonasuta	2	--	--	--	2	--
D.formosana	--	15	--	2	17	15
D.hypocausta	3	18	--	--	21	11
D.nigra	10	17	18	5	50	--
D.brindavani	--	53	10	--	63	--
D.repleta	8	44	--	--	52	--
Subgenus: **Dorsilopha**						
D.busckii	1	15	--	--	16	--
Subgenus: **Scaptodrosophila**						
D.coracina	5	12	5	--	22	--
Total no. of flies	1227	3023	141	399	4708	375
Total no. of species	17	19	7	8	21	9

A total of 22 species were recorded. Of these, 13 species belong to the subgenus **Sophophora**, 7 species to the subgenus **Drosophila**, one to the subgenus **Dorsilopha** and one to the subgenus **Scaptodrosophila**. **D.n.nasuta** of the **immigrans** species group is abundant in the population of both Mysore and Nallur indicating its ecological versatility. **D.malerkotliana** of the **melanogaster** species group is dominant in all populations of Mysore, but its frequency is remarkably low in the Nallur population. On the other hand, **D.bipectinata** and **D.nagarholensis** dominate Nallur population apart from **D.n.nasuta**.

The faunal constellations of Mysore and Nallur show qualitative and quantitative differences owing to macroenvironmental variations, but such differences found within Mysore populations can only be attributed to variations in microenvironmental factors.

Most of the species collected belong either to the **melanogaster** or to the **immigrans** species groups. This is in agreement with the earlier findings of Reddy (1973), Gowda (1979), Prakash (1979) and Muniyappa (1982) and supports the view of Bock & Wheeler (1972), that both these species groups have originated and diversified extensively in South East Asia.

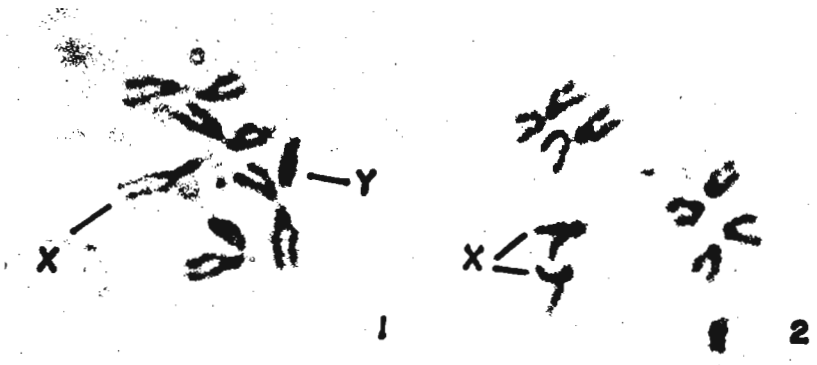
Acknowledgements: Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Post-Graduate Studies and Research in Zoology, University of Mysore, Manasagangotri, Mysore, for his help and encouragement, and to Prof. T. Okada for his advice in identifying the flies. One of the authors (BVS) wishes to thank UGC, New Delhi, for granting a research fellowship.

References: Bock, I.R. & M.R. Wheeler 1972, Univ. Tex. Publ. 7213:1-102; Gowda, L.S. 1979, Doctoral Diss., Univ. of Mysore, India; Muniyappa, N. 1982, Doctoral Diss., Univ. of Mysore, India; Prakash, H.S. 1979, Doctoral Diss., Univ. of Mysore, India; Reddy, G.S. 1973, Doctoral Diss., Univ. of Mysore, India.

Shyamala, B.V. and H.A. Ranganath. University of Mysore, India. Metaphase karyotype of **Drosophila nagarholensis.**

South East Asia is known to be a rich abode of species belonging to the **melanogaster** and the **immigrans** species groups of Drosophila. **D.nagarholensis** was described from South India by Prakash & Reddy (1980). It belongs to the **montium** subroup of the **melanogaster** species group of Drosophila. Its karyotype is described here for the first time.

Permanent air dry preparations of the neural ganglia were made as described by Ramachandra & Ranganath (1985). The male and female karyotypes are represented in Figures 1 and 2. The metaphase karyotype reveal the following: The diploid chromosome number (2n) is 8. It is made up of two pairs of metacentric chromosomes, a pair of dot chromosomes and a pair of acrocentric X chromosomes. In male, one of the X chromosomes is replaced by a submetacentric Y chromosome. The metaphase karyotype of



Figures 1 and 2. Male and female karyotypes of **D.nagarholensis.**

D.nagarholensis resembles the basic metaphase pattern of the **montium** species subgroup of the **melanogaster** species group of Drosophila as reported by Baimai (1980).

Acknowledgements: Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Zoology, for his help and encouragement. We are thankful to Mr. M.G. Vasudeva Rao, for preparing photographs. BVS is thankful to UGC, New Delhi, for granting a research fellowship.

References: Baimai, V. 1980, Jap. J. Genet. 55(3):165-175; Prakash, H.S. & G.S. Reddy 1980, Proc. Ind. Acad. Sci. 89(3):235-241; Ramachandra, N.B. & H.A. Ranganath 1985, Experientia 41:680-681.

Simmons, M.J., J.D. Raymond, E.A. Drier and G.J. Kocur. University of Minnesota, St. Paul, USNA.

Further studies with unstable X chromosomes.

Last year we reported that X chromosomes which had been unstable, as judged by high recessive lethal mutation rates, had lost their propensity to mutate to the lethal condition. These chromosomes were derived from an unstable X chromosome discovered by Lim (1979, 1981), which, in addition to high mutability, accumulated structural rearrangements and was able to confer these instabilities on previously stable X chromosomes (Lim et al. 1983). This last phenomenon, called homologue destablization, occurred independently of recombination between the stable and unstable X chromosomes and therefore suggested the action of a transposable element. The transposable element postulated to explain these instabilities has been called the L factor, where the letter "L" signifies its lethal-inducing properties. Evidently, this element has either been lost or inactivated in the stocks whose previously unstable X chromosomes have become stable.

Our recent efforts to study the instabilities attributed to the L factor have focused on lethal X chromosomes derived from Lim's unstable X, and on lethal X chromosomes which had been destabilized by it. Because these chromosomes were already lethal, we could not study their recessive lethal mutation rates; instead, we investigated their ability to destabilize nonlethal, stable X chromosomes in heterozygous females. As before, the primary criterion of instability was the propensity to mutate to the lethal condition. We therefore determined the recessive X-linked lethal mutation rates of the chromosomes which had been paired with the lethal and putatively unstable X's. A high mutation rate for one of these chromosomes would indicate that the lethal X had destabilized this chromosome, presumably through the action of the L factor.

To gather the data, we conducted several experiments following the same general plan. Males from a stock with a marked, stable X chromosome (symbolized "X") were mated at 25° to single FM6, y^{31d} sc^{8} dm B/ ℓ* females, where ℓ* denotes a marked, lethal X chromosome derived from Lim's unstable X or from an X chromosome destabilized by it. In the next generation, the ℓ*/"X" females were mated to their FM6 brothers to produce progeny (also at 25°), from which we selected only the males with the nonrecombinant "X" chromosome. The markers on the ℓ* and "X" chromosomes were chosen so that we could be sure that the males recovered from the ℓ*/"X" females had a nonrecombinant "X" chromosome. These males were then mated individually to FM7/sc^{7} ℓ females (see Simmons et al. 1980, for the full genotype of this balancer stock) to begin the X-linked lethal test. In the next generation, FM7/"X" females who had mated with their FM7 brothers were placed individually in culture tubes to produce progeny. If males with the "X" chromosome did not emerge, we tested their FM7/"X" sisters in mass cultures to determine whether or not the "X" chromosome had acquired a recessive lethal mutation. The methodological details for these X-linked lethal tests are given fully in Simmons et al. (1980). In addition to the "X" chromosomes that had been paired with ℓ* for one generation, we tested "X" chromosomes extracted directly from the stable stock. These latter tests documented the intrinsic stability of the "X" chromosomes used in the destabilization experiments.

Five different ℓ* chromosomes were tested, each in combination with one of three "X" chromosomes. The full genotypes of the potential destabilizing chromosomes, as well as those of the target "X" chromosomes, are given in Table 1. This table also gives the experimental results. Complementation

Table 1. Lethal mutation rates of stable and putatively destabilized X chromosomes.

Target "X" Chromosome	Destabilizing ℓ* Chromosome	No. Males tested	No. Chromosomes tested	No. Independent events	Number lethals	Mutation rate (%)
cho cv m	-----	373	2,739	2	2	0.07
cv v f	-----	397	3,795	5	5	0.16
H7 = y^{59b} z w^{i} ct^{6} f	-----	426[a]	3,628	0	0	---
cho cv m	I.14 = y^{59b} z w^{i} ct^{6} f	615	5,119	9	9	0.18
H7	II.10 = m	104[b]	975	7	14	1.43
cv v f	III.18 = y^{59b} z w^{i} ct^{6} m	551	5,530	25	32	0.58
cv v f	V.16.20 = y^{59b} z w^{i} ct^{6}	244	2,065	2	2	0.09
cv v f	V.16.38 = y^{59b} z w^{i} ct^{6}	178	1,591	3	3	0.19

a = Data from Simmons et al. 1985, DIS 61:157-159. b = For additional data, see text.

testing (Lim et al. 1983) had established that chromosomes I.14, II.10 and V.16.38 each had recessive lethal mutations uncovered by Df(1)ct^{78} (breakpoints 6F1-2 and 7C1-2); the lethal mutations on these chromosomes were also complemented by the y^{+} ct^{+} Y chromosome, suggesting that these were the only lethal mutations present. These results were confirmed immediately prior to the beginning of the destabilization experiments. Lim et al. (1983) showed that the lethal-containing region on these three chromosomes is a hotspot for L factor activity. The other two ℓ* chromosomes, III.18 and V.16.20, also had lethal mutations in this region, but by the time of these experiments, they had lost them. Their lethal mutations were outside the X chromosome segments carried by the y^{+} ct^{+} Y chromosome. It should also be noted that chromosomes V.16.20 and V.16.38 were derived from the ssame original lethal X chromosome; evidently, during the four years that elapsed between the identification of the original lethal chromosome and the time of these experiments, one lethal mutation reverted and another occurred, possibly as a result of L factor-induced instability.

As can be seen from the table, the "X" chromosomes are intrinsically quite stable. After having been paired with an ℓ* chromosome, only H7 showed any indication of an elevated mutation rate. Therefore, further tests were performed with this chromosome, using, as in the first set of experiments, the same ℓ* chromosome, II.10, in an attempt to destabilize it. In these additional tests, 1,463 H7 chromosomes were screened and only two proved to have acquired recessive lethal mutations. We also tested the destabilizing ability of the FM6 chromosome from the II.10 stock, just in case it had acquired L factor activity. H7 males were recovered from FM6/H7 females and used in a set of X-linked lethal tests. No lethals were detected in a sample of 1,166 H7 chromosomes screened. Therefore, we conclude that none of the ℓ* chromosomes has the ability to destabilize the specific target "X" chromosomes that were tested.

These results indicate that the five unstable or destabilized X chromosomes that we studied could not destabilize chromosomes paired with them for one generation. One explanation is that the target "X" chromosomes were immune to the destabilizing action of the L factor; however, in light of last year's report on the stabilization of several unstable X chromosomes, a more likely explanation is that the putatively unstable chromosomes that were tested also had become stable. These manifestly negative results may be turned to good use when the mutational hotspot for L factor activity has been characterized at the molecular level. It will then be possible to examine this locus in the stabilized X chromosomes and to infer the reasons for their stabilization.

Acknowledgements: This work was supported by the National Institute of Environmental Health Sciences (RO1 ES01960). M. Boedigheimer, C. McLarnon, R. Morrison and J. Zunt provided assistance.

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Singh, A.K. and B.N. Singh. Banaras Hindu University, Varanasi, India. Inversion karyotypes and crossing over in **Drosophila ananassae.**

Drosophila ananassae shows spontaneous crossing over in males (Kikkawa 1938; Moriwaki 1940; Mukherjee 1961; Ray-Chaudhuri & Kale 1966; Kale 1969; Hinton 1970). It has been suggested by various workers (Mukherjee 1961; Kale 1969; Hinton 1970; Moriwaki et al. 1970) that this extraordinary phenomenon is meiotic in origin. A large number of mutants are known in this species and linkage maps have been constructed (Moriwaki & Tobari 1975). **D.ananassae** presents a high degree of chromosomal polymorphism (for references, see Singh 1985). The three inversions, namely subterminal (alpha or In(2L)A) in 2L, terminal (delta or In(3L)A) in 3L and basal (eta or In(3R)A) in 3R, are coextensive with the species. The location of these inversions is shown in Figure 1. When the strains are maintained in the laboratory, these inversions have been found to persist for several years (Singh 1982, 1983). The suppressive effect of inversion heterozygosity on crossing over in **D.ananassae** has been indicated earlier (Moriwaki 1940; Mukherjee 1961; Hinton 1970). Singh (1973, 1974) reported the rate of crossing over between delta and eta inversions in the third chromosome in different strains of **D.ananassae.**

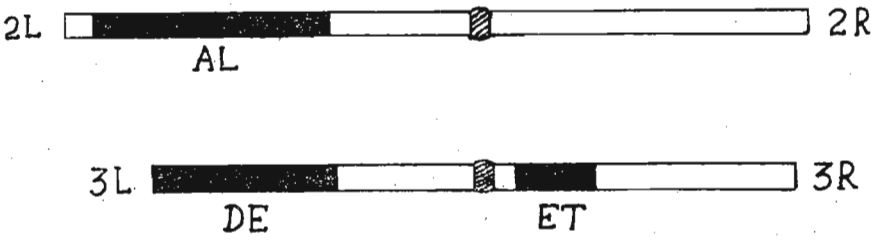


Fig. 1. Location of AL, DE & ET inversions in 2L, 3L & 3R, respectively.

In this note we report the results of our preliminary study on crossing over in females and males of **D.ananassae** under different karyotype combinations by using two wild type stocks and a triple recessive mutant stock.

cu b se mutant stock: The three markers, cu (curled wings), b (black body colour) and se (sepia eye colour) are located on the second chromosome. Kale used this trio of chromosome 2 markers in his study of crossing over. The cytological examination of this stock revealed that 2L is homozygous for AL inversion (AL/AL) whereas both the arms of third chromosome are homozygous for standard gene sequence (3L-ST/ST; 3R-ST/ST).

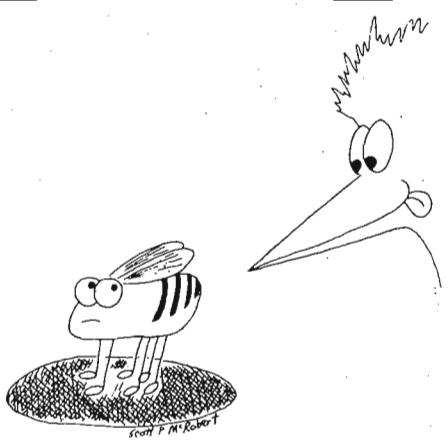
Wild stocks: Two wild stocks were used: (1) VN-ST; it is homozygous for standard gene order in 2L, 3L and 3R, and (2) VN-AL; it is homozygous for alpha gene arrangement in 2L (AL/AL) but homozygous for standard gene sequence in 3L and 3R (3L-ST/ST; 3R-ST/ST).

The results show that crossing over is completely absent in the first region (cu - b) in females and males when they are heterozygous for inversion (ST/AL). In the second region (b - se), the frequency of crossing over is above 40 percent in females and less than 1 percent in males. When F_1 flies are homozygous for alpha inversion (AL/AL), crossovers are found in both the regions in both the sexes. In the first region the rate of crossing over is nearly 30 percent and second region shows crossing over above 40 percent in females. In males crossing over is less than 1 percent in both the regions.

Further experiments to study intra- and interchromosomal effects of inversions on crossing over in **D.ananassae** are in progress.

Acknowledgements: The financial support from the Dept. of Science & Technology, New Delhi, is thankfully acknowledged. We also thank Miss Sujata Chatterjee for her help provided during this study.

References: Hinton, C.W. 1970, Genetics 66:663; Kale, P.G. 1969, Genetics 62:123; Kikkawa,H. 1938, Genetica 20:458; Moriwaki, D. 1940, Jap.J. Genet. 16:37; Moriwaki, D. & Y.N. Tobari 1975, in: Handbook of Genetics (R.C. King, ed.), Plenum Press; Moriwaki, D., Y.N. Tobari & Y. Oguma 1970, Jap. J. Genet. 45:411; Mukherjee, A.S. 1961, Am. Nat. 95:57; Ray-Chaudhuri, S.P. & P.G. Kale 1966, J. Cytol. Genet. 1:22; Singh, B.N. 1973, Genetica 44:602; ____ 1974, Caryologia 27:285; ____ 1982, Genetica 59:151; ____ 1983, Caryologia 36:333; ____ 1985, Nucleus 28:169.



Thompson, S.R. and T. Baum. Ithaca College, Ithaca, New York USNA. Locomotor activity, age and genotype effects on death rates in Drosophila population cages.

Thompson (1985) suggested that among the factors which might influence the rate at which flies emigrate to death vials in population cages were locomotor activity and genotype. This report is an examination of not only these two attributes, but also the effect of age on activity and death rate. The possible relationship of these factors to death vial emigration is also examined.

A rough estimate of locomotor activity was established by determining the distance, in centimeters, naive flies of known age, genotype and sex moved in an activity chamber during a five minute measurement period. The activity chamber was constructed of a 100 cm long segment of 1.0 cm i.d. glass tubing, capped at both ends witih plastic foam plugs, and mounted on a 0.625 x 7.5 x 150 cm lucite base. A metric tape was affixed to the lucite base, alongside the chamber, to facilitate measurement of the distance the flies moved. The chamber was illuminated along its entire length by two fluorescent bulbs, so that any phototropic effect would be minimized. For each measurement run, about twenty flies were inserted into the chamber by connecting an adaptor segment, containing the flies, to the activity chamber. The adaptor segment was a glass tube, one end of which was the same diameter as the activity chamber and which allowed quick connection to the chamber, the other end of the adaptor was expanded to the diameter of a standard shell vial (2.5 cm). This allowed flies to move from a shell vial into the adaptor, and then into the chamber, with little manipulation.

The effect of age and genotype on the overall death rate (% dead + moribund) was measured as in Thompson (1985). Flies of known age and genotype were inserted into population cages at a cage density of 2000 flies/cage, with equal numbers of females and males. The number of dead and moribund flies in the "death vial" was enumerated each day for a period of seven days, with a new death vial inserted at each count.

Table 1. Relationship of age, activity and death rate.

Age in days	sex	Oregon-R Activity	Oregon-R %D+M*	yellow Activity	yellow %D+M*
3	females	13.3±1.3	8.1	15.1±1.0	3.9
	males	18.3±1.2	7.3	19.4±1.7	4.7
7	females	13.5±1.3	15.0	8.0±1.8	21.3
	males	17.8±1.0	13.4	12.6±1.6	10.4
14	females	10.3±1.3	21.2	8.8±1.0	27.3
	males	10.3±1.0	19.4	8.8±0.8	19.7

* average of three separate experiments

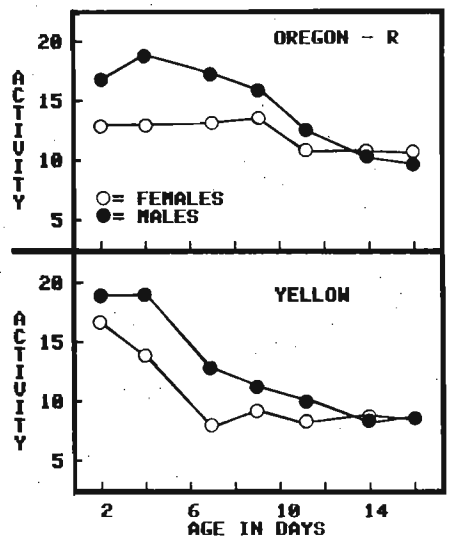


Figure 1. Relationship of locomotor activity with adult age in days.

Figure 1 shows the relationship between adult age and activity in Oregon-R and yellow flies; each point is based on no fewer than 200 flies. As can be seen, males have a higher activity, in both Oregon-R and yellow, until about eleven days at which time, and thereafter, there is no significant difference in activity between the sexes. Initially yellow flies have a higher activity rate than do Oregon-R flies, but with age the activity rate dropped at a younger chronological age and to a lower level with yellow than it did with Oregon-R.

Table 1, which is a comparison of locomotor activity and death rate in Oregon-R and yellow, shows that the influence of activity on death rate in population cages appears to be inversely related, i.e., flies with higher locomotor activity generally have a lower death rate and flies with lower locomotor activity have a higher death rate. The following specific relationships attest to this observation. (1) Young, three-day, yellow flies have a lower death rate than Oregon-R at the same age, and the activity of young yellow flies is higher. (2) Male flies usually have a lower death rate than females and their activity levels are generally higher. The exception to this is in young yellow flies, where the apparent death rate in males is somewhat higher than in females. (3) With increasing age, Oregon-R exhibits a lower death rate than yellow, and the locomotor activity is also higher.

Similar observations on average locomotor ability and death rate were seen in preliminary experiments with the mutants ebony and white, at seven days of age. In ebony, a mutant strain with poor optomotor behavior, the locomotor activity was the lowest observed (7.8) with a very high death rate (55.3%). The locomotor activity of white (11.1) was near that of yellow, but the white strain had the lowest death rate observed (2.8%). Comparison between strains is difficult as flies of the same chronological age may be of quite different biological ages. It may be that what is being examined, with both locomotor activity and death rate, is a reflection of different rates of aging.

References: Thompson, S.R. 1985, DIS 61:169-170.

Tobari, Y.N. and M. Matsuda. Tokyo Metropolitan University, Tokyo, Japan. Recombination in SM1/In(2L)t heterozygotes of **D.melanogaster.**

In previous studies of linkage disequilibrium in natural populations of **D.melanogaster**, we employed the balancer SM1 for chromosome 2 and Ubx^{130} for chromosome 3, as stated in Langley, Tobari & Kojima (1974). Over ten years, 615 2nd chromosomes were extracted from Katsunuma population by using the balancers. Of the 615, 90 chromosomes carried In(2L)t (23/123 in 1969, 36/269 in 1970, and 31/232 in 1979). Most In(2L)t's, 88 of 90, were linked with Adh^S; only 2 In(2L)t-Adh^F's were found, 1 in 1969 and 1 in 1979.

In view of the rare appearance of In(2L)t-Adh^F and the proximity of the breakpoints of In(2L)t and In(2L)Cy, we suspected that double crossing-over within 2L in SM1-Adh^F/In(2L)t-Adh^S heterozygous

Table 1. Recombination frequencies in SM1, Cy Bl/In(2L)t heterozygous females of **D.melanogaster.**

Chromosome	Nonrecombinant Cy Bl	Nonrecombinant +	Recombinant Cy	Recombinant Bl	No. of progeny	Recomb. freq.(%)
OK 1	314	322	7	6	649	2.00
OK 2	295	350	15	18	678	4.87
OK 3	219	271	11	14	515	4.85
OK 4	410	466	5	13	894	2.01
OK 5	401	459	5	8	873	1.49
OK 6	442	456	20	9	927	3.13
OK 7	281	210	5	1	497	1.21
OK 8	142	121	2	6	271	2.95
OK 9	159	159	2	6	326	2.45
OK 10	761	823	19	20	1623	2.40
OK 11	393	440	13	22	868	4.03
OK 12	563	584	6	5	1158	0.95
OK 13	314	276	4	3	597	1.17
OK 14	250	303	5	8	566	2.30
OK 15	154	217	2	6	379	2.11
OK 16	227	247	0	2	476	0.42
OK 17	243	249	8	1	501	1.80
OK 18	484	472	14	16	986	3.04
OK 19	197	344	14	7	562	3.74
OK 20	503	600	7	5	1115	1.08
OK 21	190	254	1	4	449	1.11
OK 22	283	310	5	3	601	1.33
OK 23	329	368	3	3	703	0.85
OK 24	449	438	6	5	898	1.22
OK 25	405	406	2	2	815	0.49
OK 26	479	485	9	6	979	1.53
OK 27	227	207	6	6	446	2.69
OK 28	360	403	5	1	769	0.78
OK 29	463	528	14	11	1016	2.46
OK 30	355	375	11	5	746	2.14
OK 31	440	440	13	20	913	3.61
OK 32	774	770	17	28	1589	2.83
OK 33	383	427	47	37	894	9.40
OK 34	126	126	3	3	258	2.33
OK 35	194	199	2	6	401	2.00
OK 36	229	284	23	31	567	9.52
OK 37	341	374	9	8	732	2.32
OK 38	206	265	6	4	481	2.08
OK 39	260	262	8	11	541	3.61
OK 40	338	337	8	11	694	2.74
OK 41	167	210	3	8	388	2.84
OK 42	280	338	11	18	647	4.48
OK 43	301	282	5	6	594	1.85
OK 44	131	126	7	4	268	4.10
OK 45	110	117	6	7	240	5.42
OK 46	163	175	4	2	344	1.74
OG 520	293	306	3	5	607	1.32
OG 3	104	155	2	1	263	1.52
OG 4	184	208	3	0	395	0.76
OG 10	251	285	3	0	539	0.56
OG 13	103	154	3	1	261	1.53
KT 1	461	483	10	5	957	1.57
KT 2	514	531	0	2	1047	0.19
KT 3	479	500	2	1	982	0.31
KT 4	440	447	1	0	888	0.11
KT 5	196	207	0	1	404	0.25
KT 6	437	483	5	3	928	0.86
KT 7	324	384	1	2	711	0.42
KT 8	430	490	5	6	931	1.18
AK 1	32	43	3	4	82	8.54
AK 2	40	61	2	2	105	3.81
AK 3	137	183	5	2	337	2.08
AK 4	119	169	2	5	295	2.37
AK 5	137	120	11	4	272	5.51
AK 6	113	141	5	2	261	2.68
AK 7	35	63	2	1	101	2.97
AK 8	42	65	1	0	108	0.93
AK 9	145	145	9	7	306	5.23
AK 10	41	52	4	2	99	6.06
Or-R	763	826	0	0	1589	0.00

Table 2. Recombination frequencies in SM1, Cy L/In(2L)t heterozygous females of **D.melanogaster.**

Chromosome	Nonrecombinant Cy L	Nonrecombinant +	Recombinant Cy	Recombinant L	No. of progeny	Recomb. freq.(%)
KT 2	663	670	5	1	1339	0.45
KT 3	175	249	1	1	426	0.47
KT 4	893	955	3	0	1851	0.16
KT 5	319	388	1	2	710	0.42
KT 6	181	189	0	0	370	0.00
KT 7	213	219	2	1	433	0.69
KT 9	145	132	0	2	279	0.72
OK 4	852	885	17	11	1765	1.59

Table 3. Linkage of recombinant chromosomes.

Population	Linkage of recombinant chromosomes				No. of recomb. tested
	Cy-Adh^F	Cy-Adh^S	Adh^F-Bl	Adh^S-Bl	
OK	73	29	20	45	167
OG	1	2	0	0	3
KT	0	6	1	1	8
Total	74	37	21	46	178

Table 4. Gene arrangement and linked alleles of recombinant chromosomes.

Recombinant chromosome	No. of chromosomes
In(2L)t, Cy Adh^F	37
In(2L)t, Cy Adh^S	21
In(2L)t, Adh^F Bl	0
In(2L)t, Adh^S Bl	0
SM1, Cy Adh^F	0
SM1, Cy Adh^S	0
SM1, Adh^F Bl	8
SM1, Adh^S Bl	26

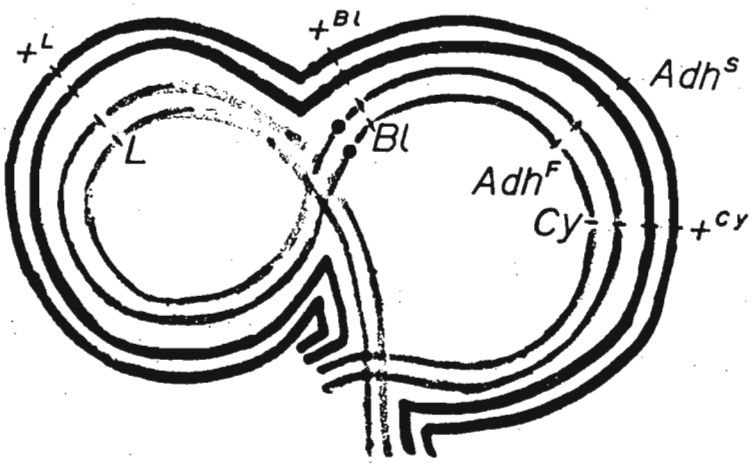


Figure 1. Schematic configuration of chromosome pairing in SM1/In(2L)t heterozygotes.

females (in the final step of making homozygotes of a single wild chromosome or to maintain them, Cy/+ ♀ x Cy/+ ♂) produced rare In(2L)t-Adh^F chromosomes.

In order to detect double recombination within chromosome 2, we synthesized SM1, Cy B1 and SM1, Cy L from SM1, Cy and S^2 Cy(2L, 2R) B1 L. In(2L)t chromosomes were sampled from Okinawa (OK), Ogasawara (OG), Katsunuma (KT) and Akayu (AK). Prior to the experiment Adh alleles of each In(2L)t were examined. In the test cross SM1, Cy B1/In(2L)t F_1 females or SM1, Cy L/In(2L)t F_1 females were individually mated to Or-R males. The F_2 progeny were scored for recombination of markers. Of the F_2 progeny recombinant alleles, Cy, B1, or L, were individually mated to Or-R females. After several days these F_2 males were analyzed for Adh alleles. Several F_3 larvae from each mating were individually examined for the inversions and Adh alleles simultaneously.

Sites of markers are approximately placed on chromosome loops which are to be formed when SM1 and In(2L)t pair (Fig. 1). Frequencies of viable recombinants in SM1/In(2L)t females are listed in Tables 1 and 2. Substantial recombinants were recovered in every female, in varioius proportions. This demonstrates that the SM1 did not balance chromosome 2 carrying In(2L)t. It is clear that there is a considerable chance that the Cy and/or Adh^F allele from the SM1 may have been introduced into In(2L)t. Inspection of Tables 3 and 4 indicates that Sm1/In(2L)t ♀ x SM1/In(2L)t ♂ crosses produced F_3 progeny composed of visibly indistinguishable blend of a variety of recombinants, including such as SM1, +^{Cy} and In(2L)t, Cy, and nonrecombinants.

Therefore, the existence of rare In(2L)t-Adh^F in natural populations of **D.melanogaster** seems very unlikely.

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Trejan, K.S. and K.S. Gill. Punjab Agricultural University, Ludhiana, India. Effect of inbreeding on wing morphology in **Drosophila malerkotliana.**

III effects of inbreeding on wings were studied while preparing isogenic lines for two acid phosphatase alleles (Acph$^{1.05}$ and Acph$^{0.95}$) in **Drosophila malerkotliana.** Homozygous stocks for the two alleles were established from wild-caught flies. From the running cultures of these two stocks, 20 single pair matings (one member homozygous for one allele and the other for the other allele) were set-up. From the progeny of a single mating pair, further 20 single pair matings F_1 x F_1) were set up. F_2 flies were taken to represent first inbred (I_1) generation. From the progeny of one of the 20 F_1 x F_1 matings, again 20 single pair matings (F_2 x F_2) were set up. After pupation the mating pairs were removed and subjected to electrophoresis, one of the vials, in which both the alleles were represented, was chosen for selecting the parents of next generation. This plan of mating

Table 1. Effect of inbreeding on wing morphology in **Drosophila malerkotliana.**

	No. of flies studied	Types of wing/abnormalities: one wing held out	both wings held out	shrivelled wings	% Total abnormal flies
I_4	1673	55	--	--	3.28
I_5	1444	132	--	--	9.14
I_6	1155	177	35	4	18.70
I_7	884	242	--	66	34.84
I_8	584	--	3	581	100%
I_9	No progeny				

was continued in each subsequent generation. Ill effects on wings, appearing for the first time in I_4 generation, were recorded on the progenies of five single pair matings in each generation.

No progeny were obtained in the I_9 generation; the above experiment was therefore repeated in each of the two subsequent years using freshly established stocks. In these experiments also I_9 generation was not obtained. Since no significant differences were observed in the number of flies showing abnormal wings appearing in the progeny of each of the five pair mates in each generation and for any specific generation in the three experiments, pooled data is being presented in Table 1.

In I_4 generation approximately 3% flies had one or the other wing permanently held out horizontally. The frequency of such flies increased in subsequent generations. Moreover, in I_6 generation approximately 16% of the abnormal flies had both the wings permanently held out and about 2% of the abnormal flies had shrivelled wing (resembling the vestigial wing mutant of **Drosophila melanogaster**). In I_7 generation abnormal flies with both the wings held out did not appear. However, the frequency of flies with shrivelled wings increased to approximately 21% of the abnormal flies. All the flies of I_8 generation were abnormal; more than 99% of the flies had shrivelled wings and only 3 flies had both the wings held out permanently. These flies were very weak, could not mate and died within a week.

A perusal of data in the table also indicates that the number of pooled flies was 1673 in I_4 and 584 in I_8 generation, and no progeny was obtained in I_9 generation. It then appears that inbreeding not only induces wing abnormalities but also drastically reduces the fertility.

Turelli, M., C. Burkhard, V. Fong, J. Moore, S. Van Horn and T. Prout. University of California, Davis, USNA. Does dusting distort Drosophila dispersal?

Since Crumpacker & Williams's (1973) original application of UV-fluorescent dusts to **Drosophila pseudoobscura**, these dusts have become a standard tool in the study of Drosophila field behavior. One of the most astounding results obtained is that Drosophila can disperse 10 km within 24 hr (Jones et al. 1981; Coyne et al. 1982). A central assumption in studies using dusts is that dusting does not significantly affect the behavior under investigation. Crumpacker (1974) performed control experiments which indicated that dusting does not decrease viability under laboratory conditions, but there are no comparable studies concerning the effects of dusts on dispersal. To test the hypothesis that dusts do not influence dispersal, we simultaneously released dusted and undusted **Drosophila simulans** homozygous for the recessive plum.

We reared approximately 100,000 flies in half-pint bottles. On August 31, 1982, we divided them at random into two groups of approximately equal size. The first group was heavily coated with red micronized UV-fluorescent dust (Radiant Color, Richmond, CA) by shaking the flies pooled from several culture bottles in a half-pint bottle containing several milligrams of dust. After two transfers to clean bottles to remove excess dust, the dusted flies were placed in a plexiglass cage, approximately 42 cm on a side. The second (control) group of approximately 50,000 was transferred from the culture bottles into bottles without dust then placed in a second plexiglass cage. No anesthesia was used and the flies ranged in age from 0-5 days.

At dusk, approximately three hours after we began collecting them from the culture bottles, we released the flies at an intersection of two rarely used roads crossing experimental fields on the Davis campus. The north-south road is asphalt and lined on both sides with mature olive trees. The east-west road is dirt with no vegetation along its borders. At the time of release, the red dust was only faintly visible on the marked flies. The next afternoon (day 1) we set out 41 five-liter buckets baited with orange/banana mash and Fleischmann's yeast. The buckets were placed along the roads at 30 m intervals, with ten in each direction and one at the intersection. During the hour and a half before nightfall, we collected the flies three times from each of the buckets. The baits were left out overnight. Because several flies had been captured in the outermost traps, we extended the north-south trapline during the afternoon of September 2 (day 2) by ten baits to the south and five to the north. During the hour and a half before nightfall, we again collected the flies three times from each bucket.

Table 1. $\bar{d}$, SD, N, and P for each direction and day.

		North	South	East	West	Overall
	Day 1					
dusted:	$\bar{d}$(N)	124.1(233)	96.0(740)	132.6(74)	66.5(32)	103.7
	SD	79.8	68.6	83.4	49.8	
undusted:	$\bar{d}$(N)	123.1(185)	106.8(584)	140.2(61)	96.1(44)	112.0
	SD	81.6	73.5	96.2	79.4	
P		>0.1	0.006	>0.1	>0.05	
	Day 2					
dusted:	$\bar{d}$(N)	147.4(141)	93.6(365)	163.6(31)	105.0(8)	111.7
	SD	95.5	82.3	60.4	69.9	
undusted:	$\bar{d}$(N)	149.9(304)	114.9(415)	189.4(32)	131.7(46)	132.2
	SD	89.9	94.7	87.7	72.0	
P		>0.1	<0.001	>0.1	>0.1	

Our results are summarized in Table 1. For each of the four directions on each day, we give the mean ($\bar{d}$) and standard deviation (SD) of the capture distance (in meters) from the release point (flies captured at the central bucket were ignored in these calculations), the number of released flies captured (N), and the significance level (P) of an approximate t-test (allowing for unequal variances, see Sokal & Rohlf 1981) for the hypothesis of equal mean dispersal distances for dusted and undusted flies. We have not statistically analyzed the data pooled across directions because of the heterogeneity of the environment.

Our data indicate that dusting tends to decrease dispersal distances slightly. From the two significantly different means, the estimated decrease is 10-20%. Thus, our major qualitative result is that estimates of dispersal distances based on dusting are fairly reliable but may be slightly conservative.

Other differences appear in our data. On day 1, we captured 1117 dusted and 893 undusted flies (including 38 dusted and 19 undusted from the central bucket). On day 2, we captured 554 dusted flies and 800 undusted flies (including 9 dusted and 3 undusted from the central bucket). Hence the dusted flies were significantly more likely to have been captured on day 1 and significantly less so on day 2) (X_1^2=68.2, P<0.001). Based on Nigro and Shorrocks's (1982) results concerning the persistence of dust on flies in the field, it is very unlikely that this difference is attributable to misclassifying dusted flies as undusted on day 2. The difference may have resulted, at least in part, from more of the more mobile undusted flies dispersing beyond the trapline on day 1, combined with lower viability of the dusted flies under field conditions. Alternatively, the dusted flies may have been more likely than the undusted flies to be captured on day 1, because they may have been more "stressed" (perhaps dessicated) and hence responded to the baits differently as observed by Hoffman & Turelli (1985). Chi-square tests also show that the proportions of dusted and undusted flies captured over the four directions were homogeneous on day 1 (X_3^2=5.6, P>0.1) but significantly heterogeneous on day 2 (X_3^2=43.9, P<0.001). Thus, even though dusts only slightly affect dispersal distances, they may alter field behavior. The significance of these effects for other types of experiments remains to be determined.

Acknowledgements: Supported by NIH Grant 22221 and Larry Harshman.

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MURDERS IN THE TIN-CAN MORGUE

by

Glenn Lucey, '85
UC-San Diego

I'll never forget the day I died!
A human friend to make I tried;
I waved and buzzed and did a dance
In hope that I might earn a glance;
And when his hand curled 'round my vial,
I thought perchance he'd seen my smile,
But then my world began to shake --
Inversion followed in its wake --
I coughed and struggled and fought for air
As ether fostered dark despair.
A bright light shone as I came 'round,
A poker pinned me to the ground.
As I slid off the porcelain plate
I realized what had sealed my fate.
The oil it clung and pulled me under
As I rued my fatal blunder;
My dance was not my human's gripe --
'Twas pure and simple my phenotype!

Ushakumari, A. and H.A. Ranganath. University of Mysore, India. Egg to adult viability and rate of development of five strains of Drosophila in four different media.

Viability and egg-to-adult developmental time are two important components of fitness in Drosophila and they can be related to phenomena such as larval facilitation, as well as concepts of microniches and microenvironments (Castro & Mensua 1985). Using these two parameters, experiments have been undertaken to study some aspects of nutritional requirements of Drosophila under laboratory conditions. The present note deals with four different types of wheat cream agar media. They are: (1) media without yeast and sugar (S^-/Y^-); (2) media with yeast and without sugar (S^-/Y^+); (3) media with sugar and without yeast (S^+/Y^-); and (4) media with yeast and sugar (S^+/Y^+).

The Drosophila strains used in this experiment are as follows: **D.sulfurigaster sulfurigaster**, P-11 Port Moresby, Papua, New Guinea; **D.s.albostrigata**, S-11 Sandakan, Sabah, Malaysia; **D.s.bilimbata** GUM-8 Guam; **D.s.neonasuta** 209.2 Mysore, India; **D.pulaua** S-18 Sandakan, Sabah, Malaysia.

These five strains belong to the orbital sheen complex of the **nasuta** subgroup of the **immigrans** species group of Drosophila. These members are morphologically indistinguishable from one another.

Eggs of approximately four hours of age of these five strains were collected following the modified procedure of Delcour (Ranganath & Krishnamurthy 1974). Ten vials were made for each strain in each medium. Each vial contains 50 eggs of any one strain in any one medium. After the onset of emergence the number of flies eclosed on each day was recorded and from this the mean developmental rate in days were calculated in four different media under study. The total number of flies emerged in 10 vials (10 vials used for the analysis of rate of development) in each medium were recorded and from this the egg to adult viability was estimated for the above said strains in four different media.

Table 1. Egg to adult viability of the 5 strains of Drosophila in four different media (500 eggs were sown in each group) with summarised chi-square test.

Strain	Media:	S^-/Y^-	S^-/Y^+	S^+/Y^-	S^+/Y^+	ΣX^2	df	P
D.s.sulfurigaster	(P-11)	62	76	107	174	71.17	3	<0.0001
D.s.albostrigata	(S-11)	2	10	84	185	308.11	3	<0.0001
D.s.bilimbata	(GUM-8)	15	28	52	83	60.25	3	<0.0001
D.s.neonasuta	(209.2)	167	284	290	303	45.86	3	<0.0001
D.pulaua	(S-18)	0	7	12	131	312.78	3	<0.0001
ΣX^2		403.63	673.57	422.46	153.45			
df		4	4	4	4			
P		<0.0001	<0.0001	<0.0001	<0.0001			

Table 2. Mean development time in days (mean ± SE) for the five strains of Drosophila in four different media.

Strain	Media:	S^-/Y^-	S^-/Y^+	S^+/Y^-	S^+/Y^+
D.s.sulfurigaster	(P-11)	13.00±0.10	12.76±0.14	13.36±0.15	13.43±0.09
D.s.albostrigata	(S-11)	17.00±0.00	17.00±0.00	12.48±0.14	11.79±0.12
D.s.bilimbata	(GUM-8)	21.00±0.00	18.54±0.19	14.50±0.14	13.46±0.14
D.s.neonasuta	(209.2)	16.59±0.13	13.52±0.11	15.42±0.11	13.14±0.09
D.pulaua	(S-18)		15.43±0.20	13.08±0.19	12.98±0.17

The viability of five strains in four different media recorded in Table 1 reveals the following: (1) in the media without yeast and without sugar, all the five strains have the least degree of viability while the highest was achieved in the media with yeast and with sugar; (2) of the five strains, **D.s.neonasuta** is the most versatile one as evidenced by the viability of its eggs in all the four types of media under study; (3) the performance of **D.pulaua** is inferior to all the other strains, except in the media with yeast and with sugar; (4) the egg to adult viability of the five strains in the media with yeast and with sugar is in the order of **D.s.neonasuta** > **D.s.albostrigata** > **D.s.sulfurigaster** > **D.pulaua** > **D.s.bilimbata.**

The results pertaining to the egg to adult rate of development are compiled in Table 2. The notable findings of this study include:

(1) In the four different types of media under study the rate of development of **D.s.sulfurigaster** ranges between 12.76 to 13.43 days. On the other hand, the spectrum of variation for **D.s.bilimbata**, is between 13.46 to 21.00 days.

(2) In the present experimental set up, the fastest rate of adult eclosion was noticed for the eggs of **D.s.albostrigata** in the media with sugar and with yeast, and the slowest rate of development was recorded in the media without sugar and without yeast for the eggs of **D.s.bilimbata.**

Thus, these experiments reveal (a) the importance of sugar and yeast on the manifestation of two parameters, namely the rate of development and viability, and (b) the differential response of closely related strains of Drosophila to different types of media.

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Ushakumari, A. and H.A. Ranganath. University of Mysore, India. Studies on the fecundity of closely related ten strains of Drosophila.

Fecundity is defined as the number of eggs laid by an individual. Experiments were conducted to assess the egg laying capacity of the ten strains of five species of the orbital sheen complex of the **nasuta** subgroup of the **immigrans** species group of Drosophila. Males and females from each strain were collected within four hours after eclosion, and after aging them for five days, pair matings were made. These pairs were transferred to fresh vials without etherisation once in every 24 hr. Then the number of eggs laid in each vial was recorded. Likewise egg counts for 60 pairs of flies were made for each strain for over a period of ten days. Chi-square test computed for these ten strains has revealed statistically significant differences in their fecundity.

Table 1. Fecundity of ten strains of the Orbital sheen complex of the **nasuta** subgroup of Drosophila.

Strains	No.of pairs	Total no. of eggs	Eggs/ individual	Eggs/individual/day
D.s.sulfurigaster (3019.8)	60	4337	72.28	7.23
D.s.sulfurigaster (P-11)	60	2550	42.50	4.25
D.s.albostrigata (W-3)	60	5480	91.33	9.13
D.s.albostrigata (S-11)	60	2580	43.00	4.30
D.s.bilimbata (GUM-8)	60	2802	46.70	4.67
D.s.bilimbata (HNL-III)	60	2350	39.17	3.92
D.s.neonasuta (polymorphic)	60	7401	123.35	12.34
D.s.neonasuta (monomorphic)	60	5267	87.78	8.78
D.pulaua (V-6)	60	4693	78.22	7.82
D.pulaua (S-18)	60	4716	78.60	7.86

$\Sigma X^2 = 5754.95$; df = 9; $P < 0.0001$.

The results summarized in Table 1 reveal the following: (1) the number of eggs per individual per day varies between 3 to 12; (2) except **D.pulaua,** the other four members show significant interstrain differences with regard to their egg laying capacity; (3) of the ten strains, the polymorphic strain of **D.s.neonasuta** has shown the highest fecundity, while HNL-III strain of **D.s.bilimbata** has the least.

The sequence as to the egg laying potentialities of the strains under study is **D.s.neonasuta** (polymorphic) > **D.s.albostrigata** (W-3) > **D.s.neonasuta** (monomorphic) > **D.pulaua** (S-18) > **D.pulaua** (V-6) > **D.s.sulfurigaster** (3019.8) > **D.s.bilimbata** (GUM-8) > **D.s.albostrigata** (S-11) > **D.s.sulfurigaster** (P-11) > **D.s.bilimbata** (HNL-111).

Even though all the ten strains are morphologically identical, phylogenetically and taxonomically closely related, they show significant differences in their egg laying capacity. The variability recorded in Table 1 reflects the genetic variability present in the ten strains under study for the character "fecundity".

Acknowledgements: Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Zoology, for help and encouragement; to the Indian National Science Academy, New Delhi, for awarding research grants to HAR; and to Prof. O. Kitagawa for sending flies.

Van Delden, W. and A. Kamping. University of Groningen, Haren, Netherlands. The relationship between ethanol tolerance and ethanol utilization in **Drosophila melanogaster.**

Many reports have demonstrated the relationship between the polymorphism at the alcohol dehydrogenase (Adh) locus in **D.melanogaster** and tolerance to alcohols (reviewed in Van Delden 1982). When the Adh genotypes, which differ in in vitro ADH activity, are compared for survival on toxic concentrations of ethanol or other alcohols, it is found that survival and ADH activity are positively correlated. It has further been shown that, in the absence of other food components, ethanol may serve as a food component when it is present at low concentrations (references in Van Delden 1982).

In the present study we tested strains of the Groningen population which had been selected for increased tolerance to ethanol, for their ability to utilize low concentrations of ethanol as the only food source. For this purpose we used one strain which was homozygous for the Adh^F allele (FFE) and one strain homozygous for the Adh^S allele (SSE). Both strains had been kept continuously on regular food

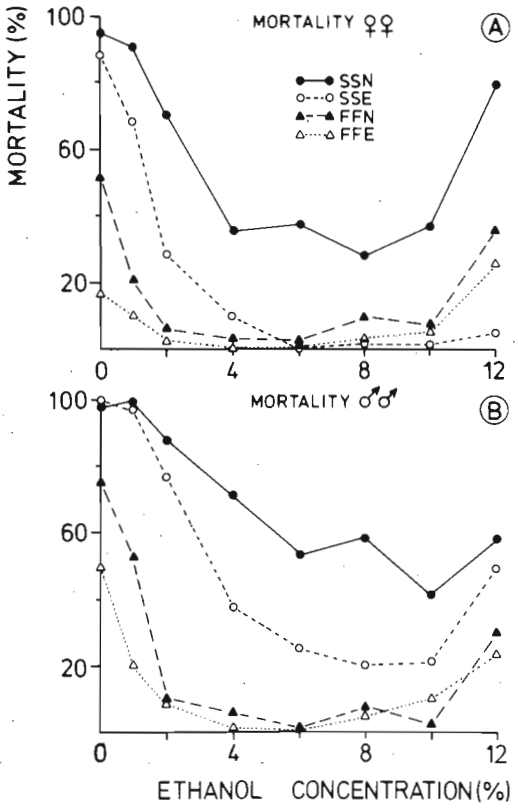


Figure 1. Adult mortalities after 100 h at various concentrations of ethanol supplemented to agar. SSE and FFE: strains selected for ethanol tolerance; SSN and FFN: control strains.

supplemented with ethanol for 112 generations (details in Kerver & Van Delden 1985). During that period the ethanol concentration was raised from an initial 12% by volume to 18% in three steps. These selected strains (FFE and SSE) were compared with their controls (FFN and SSN, respectively) which had been kept on regular food. The aim of our study was to establish whether the considerably increased tolerance to toxic concentrations of ethanol in the E lines is accompanied by an improved ability to utilize ethanol at low concentrations as the only food component.

Flies from each of the four strains were transferred to regular food and their offspring were used for the experiments. For the survival tests adults, 6-8 days old, were put into vials with 9 ml agar supplemented with 0, 1, 2, 4, 6, 8, 10 or 12 percent ethanol by volume. The number of flies (either males or females) per vial was 20; the number of replicates per sex and per strain was 5. The number of dead flies was recorded at regular intervals. The results are shown in Figure 1, where mortality after 100 h is given. Several conclusions can be drawn from the results. (1) The genotypes differ considerably in survival percentages, even when food is absent (0% ethanol): FF homozygotes perform better under starvation conditions than SS homozygotes. (2) Mortalities decrease with increasing ethanol concentrations until almost 8%, due to ethanol utilization. Mortalities at 12% ethanol are higher than at lower concentrations because at that concentration the toxic effects overcome the beneficial effects. (3) The increase in life span at the lower ethanol concentrations is higher in FF homozygotes than in SS homozygotes. This result confirms the findings of Daly & Clarke (1981), who measured survival at 2% ethanol. (4) The E strains survive better at the toxic ethanol concentrations than the N strains, which is in agreement with the findings of Kerver & Van Delden (1985) that tolerance is increased in the selected strains. (5) The E strains utilize ethanol at low concentrations better than their control strains. This is in agreement with the results of Dorado & Barbancho (1984) that strains selected for ethanol tolerance had a better ability to use ethanol (11%) as food than controls. Also Van Herrewege & David (1980) found this relationship, but not for all their strains. They therefore proposed different genetic mechanisms for tolerance and for utilization. Their selection procedure, however, was different from ours as selection for ethanol tolerance was only exercised during a limited number of days of the adult life phase.

The results show that selection for ethanol tolerance at high ethanol concentrations also improves the ability of the selected strains to utilize low concentrations of ethanol when present as the only food source. This relationship between ethanol tolerance and utilization has also been found, though in a different way, by Van Herrewege & David (1984) who selected for increased ethanol utilization and found an increase in ethanol tolerance at high ethanol concentrations. This result was, however, only found in one of the two strains tested.

The mechanism behind the observed relationship between tolerance and utilization could at first sight be sought in the difference in ADH activity of the strains in view of the different behavior of the SSN and FFN strains. Adults of the E strains used in our experiments, however, possess no higher ADH activities than their respective unselected controls (Kerver & Van Delden 1985); thus total ADH activity is apparently not the key factor involved.

References: Daly, K. & B. Clarke 1981, Heredity 46:219-226; Dorado, G. & M. Barbancho 1984, Heredity 53:309-320; Kerver, J.W.M. & W. van Delden 1985, Heredity 55:355-367; Van Delden, W. 1982, Evol. Biol. 15:187-222; Van Herrewege, J. & J.R. David 1980, Heredity 44:229-235; Van Herrewege, J. & J.R. David 1984, Genetica 63:61-70.

Villarroel, H. and P. Zamorano. Academia Superior de Ciencias Pedagogicas, Valparaiso, Chile. Distribution altitudinal of the Drosophila species which inhabit the National Park "La Campana", Valparaiso, Chile.

Genetic investigations carried out concerning the Drosophila genus are extensive and varied; however, very little is known about its ecology (Ford 1974), particularly in our zone. The studies carried out on these diptera at the National Park "La Campana" have shown the existence of nine Drosophila species (Villarroel & Zamorano 1984).

The purpose of the above mentioned work is to determine the composition and distribution of species of the Drosophila genus in one altitudinal transect at cerro "La Campana", a place forming part of the National Park under the same name, its maximum altitude being 1980 m above sea level (Serey, Ortiz, Meza and Solervicens 1976) and located at Olmue, at approximately 70 Km from Valparaiso.

The zone has a Mediterranean climate, where most of the more significant biotic communities of Central Chile are represented, such as: Nothofagus forest, hygrophilous forest, sclerophyll forest, matorral, bamboo thicket, succulent scrub, high altitude communities and palm forests (Oberdorffer 1960; Rundel & Weisser 1975).

Sample collection was made from October 1982 to March 1983. Three sampling stations were chosen at 560, 800 and 1160 m above sea level. Capture was performed by means of the usual trapping method (fermented banana bait).

Table 1. Altitudinal distribution of species Drosophila genus at "La Campana" (National Park), Valparaiso, Chile.

	Altitude (metres above sea level)			
	560 m	800 m	1100 m	TOTAL
SPECIES	no. flies(%)	no. flies(%)	no. flies(%)	no. flies(%)
D.amplipennis	94 (9.86)	21 (5.01)	77 (12.60)	192 (9.68)
D.araucana	174 (18.26)	51 (12.17)	344 (56.30)	569 (28.69)
D.busckii	---	---	1 (0.16)	1 (0.05)
D.immigrans	349 (36.62)	42 (10.02)	37 (6.06)	428 (21.58)
D.melanogaster más **D.simulans**	95 (9.97)	27 (6.44)	13 (2.13)	135 (6.81)
D.pavani	7 (0.74)	13 (3.10)	7 (1.15)	27 (1.36)
D.repleta	23 (2.41)	38 (9.07)	5 (0.82)	66 (3.33)
D.subobscura	211 (22.14)	227 (54.18)	127 (20.78)	565 (28.50)
TOTAL	953 (100.0)	419 (100.0)	611 (100.0)	1983 (100.0)

A total of 1983 flies was collected, corresponding to nine species of the 33 described for Chile by Brncic (1957a; 1962a) (see Table 1).

The distribution of each species at different altitudes indicates very particular behaviours, so we can distinguish three well defined groups.

I: D.amplipennis and **D.araucana** reach a maximum abundance at 1160 m above sea level. They are species belonging to the Southern zone of the country; therefore, the search of their native habitat will cause them to distribute more abundantly in natural places, away from man and where weather conditions are more or less resembling those of the south of Chile.

II: D.immigrans and **D.melanogaster + D.simulans** are more abundant at 560 m above sea level; they decrease while altitude increases. They are clearly a cosmopolitan species and therefore closely associated with man, hence they are preferably located at an altitude which is closer to human environment.

III: D.repleta, D.pavani and **D.subobscura** are more frequent at 800 m above sea level. At this altitude environmental conditions are a bit more xeric than in the remaining sampling stations, and they are more exposed to weather conditions such as wind and solar radiation; therefore, these species here would presumably find conditions resembling their natural environment. Moreover, a certain competition concerning food resource was apparently glimpsed between **D.subobscura** and other species.

Another interesting feature of the collection is a marked decrease in the number of individuals at 800 m above sea level. Afterwards it slightly increased at 1160 m above sea level. The intermediate altitude is characterized by a not very dense vegetation and by a floor of a scarce vegetable cover, while at maximum altitude a transition zone is placed where there exists a floristic convergence (Villasenor 1980), thus increasing the diversity of the area which would consequently cause an increase in the abundance of Drosophila at this altitude.

Under these considerations the 1160 m above sea level would constitute a favorable place for the establishment of the species.

Finally, we can indicate that environmental conditions play an interesting role in altitudinal distributions of the Drosophila species, not withstanding the importance of genetic factors in this particular situation.

References: Brncic,D. 1957a, Colecc. Monografias Biologicas Univ. de Chile, Santiago; _____ 1962a, Biologica 33:3-6; _____ 1970, "Essays in Evolution and Genetics" 14:401-436; Brncic, D. & M. Budnik 1980, DIS 55:20; Ford 1974, "Ecological Genetics"; Guenzalida,H. 1950, Clima 1:188; Biogeografia 1:371; Oberdorfer, E. 1960, Flora et Vegetatio Mundi, 2, Verlag J. Cramer, Pflanzensoziologische Studien in Chile, Ein Vergleich mit Europa; Rundel, P. & P. Weisser 1975, Biol. Conserv. 8:35-46; Villarroel, H. & P. Zamorano 1984, DIS 60:206; Villasenor, R. 1980, Ann. Mus. Hist. Nat. Valparaiso 13:65-70.

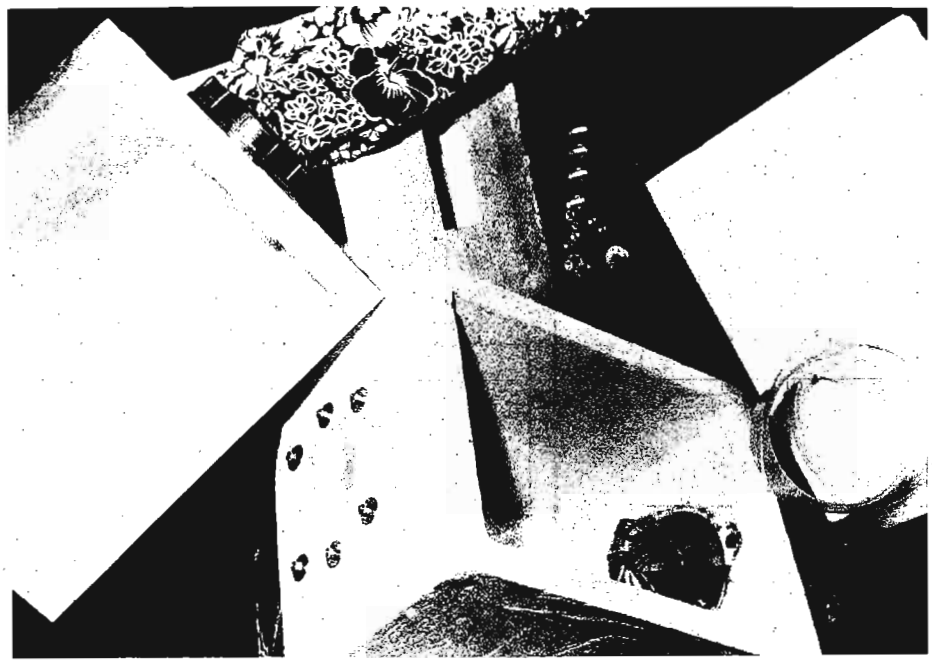
Arnason, E. University of Iceland, Reykjavik, Iceland. Yet another population cage.

Many different population cages have been used by drosophilists. The following is an extension of a design by Lewontin (1965); it is cheap, convenient in use and durable. The materials needed are: (1) a perishable shipper-foam unit only (available from Polyfoam Packers Corporation, 6415 N. Chicago Ave., Chicago, IL 60445 @ $4.35). Inside dimensions are $8\frac{1}{2}$" wide x 11-3/4" long x $11\frac{1}{2}$" deep with 1" thick walls made of high density polyfoam. (2) two 1000 ml polypropylene beakers, Tri-Pour design (@ $0.60) available from any biological supply house. (3) a piece of clear plexiglass $8\frac{1}{2}$" x 11-3/4" x 1/8" to fit on top. (4) strips of plexiglass $1\frac{1}{4}$" x 3/16" that can be cut and broken into desired lengths; plexiglass is available locally in most places. (5) a piece of 36 or 40 gauge nylon mesh 6" x 5" cut with a hot soldering iron to seal the edges. (6) A piece of broadcloth 15" x 20". (7) a tube of silicon rubber. (8) nine 1/16" metal bolts $1\frac{1}{4}$" long with 18 nuts and 18 washers to fit. (9) a role of duct tape or masking tape. The total cost of materials is about $6-7; labor is cheap.

Make the cage as follows: Cut the height of the box down to 8" using a sabre saw mounted with a knife edge blade. Cut a circular hole, $4\frac{1}{2}$" diameter, in the one end of the box, and a square hole, $3\frac{1}{2}$ x 2", in the other end. Cut the bottoms off the Tri-Pour beakers leaving a $1\frac{1}{2}$" rim with the triangular top of one and a 3/4" rim on the other. Saw a long sleeve from the broadcloth, tight enough to fit snugly on the beaker with the larger rim; tape the sleeve to the rim. Drill or punch holes large enough for the bolts in the corners of the beakers. Fit the beaker top with the sleeve through the circular hole, seal on the inside with silicon rubber and fit the other beakertop opposite on the outside. Push a sharp needle through the walls to make holes and push bolts through from the inside out. Use washers on both sides and fit and secure tightly two nuts on each bolt on the outside. Put some silicon rubber on top of the bolts and washers on the inside to prevent rust. Make two 6" long and two 2" long plexiglass strips and drill two holes in the longer strips and one in the shorter. Lay the nylon mesh in silicon rubber and fit over the square hole. Lay the plexiglass strips over and punch holes and bolt through the walls as before. Lay the clear plexiglass sheet in silicon rubber on top of the box and tape tightly. If desired, cut the lid of the polyfoam shipper to fit inside the box with holes cut out to hold food cups.

The cage is completely fly tight as constructed but care should be taken when introducing and removing food cups. I use disposable plastic cups made for yogurt as food cups. The cups measure 2" bottom diameter, 3" top diameter and 3" high and can hold 180 ml; the amount of medium is about 90-100 ml. A cage holds 12 such cups that can be rotated according to a schedule suitable for the species being studied.

The cage gets dirty after a while. To have a clear view into the cage, the inside of the plexiglass can be scraped clean with a razor blade. Alternatively, the food cups and the adults can be transferred to a clean cage. The adults are collected with a pooter using gentle suction provided by a diaphragm air pump (a suitable air pump is catalog number 1047-D10 from Thomas Scientific, Swedesboro, NJ). The top of the dirty cage can then be removed and the cage cleaned with a detergent and plenty of hot water. The top can be fitted in fresh silicon rubber and the cage reused.



Cages made after this design have been in continuous use in the laboratory for close to 5 years. They are still in good condition.

Acknowledgements: I thank Magnús Magnússon for the photography. During the designing of the cage I was supported by a Fulbright-Hays grant, a Fogarty International Research Fellowship NIH #F05 TWO3027-01, and a grant from the Icelandic Science Foundation (Visindasjodur Islands).

Reference: Lewontin, R.C. 1965, DIS 40:103.

Figure 1. Population cages.

Beukema, W.J., T.W. Nyboer and M.J. van den Berg. University of Groningen, Haren, Netherlands. An improved courtship song simulator for Drosophila.

It is well known that male song plays an important role in Drosophila courtship behavior (e.g., Bennet-Clark & Ewing 1967). This song which is produced during wing vibration consists of two elements: sine song and pulse song (Schilcher 1976). Several variables of this song are species specific (Ewing & Bennet-Clark 1968) and it is likely that such interspecific varation plays a role in sexual isolation. Furthermore, intraspecific variation in courtship song might play a role in mate choice. To study the role which these variables play in the courtship song, a simulator is necessary.

However, in trying to build the simulator developed by Johnson & Cowling (1980), we found that many of their time controlling elements were not locked in phase. This caused difficulties in tuning all the variables. For instance, the sine used to create one pulse would start at a random phase rather than phase zero, thus causing many uncontrolled harmonics. More seriously, however, even the smallest misalignment would change the original sequence: pulse; sine; silence to, for instance, pulse; sine; pulse; silence. Futhermore, Kyriacou & Hall (1980) showed that the interpulse interval is not constant, but modulated by a slow wave of approximately one minute. This modulation also proved to enhance mating speed (Kyriacou & Hall 1982); we therefore also wanted to incorporate such a modulation in our simulator. These two modifications (locking the time controlling elements in phase and the introduction of the modulation) necessitated the design of a new system. This new courtship song simulator will be discussed here.

The logic diagram indicates how the repeating song sequence, as shown by the output signal, is produced. (See Table 1 for the variable definitions.) Every t1 sec. a trigger pulse generator triggers the pulse song duration timer (IC7) to give a block of t2 sec. which enables the generation of ipi trigger pulses with an interval of t4 sec. during t2. These very short pulses are responsible for synchronising the pulse song generator (IC4) and for starting a pulse bit counter (IC6). The pulse bit counter's output signal gates the pulses from IC4 to the output mixer in such a way that, with the switch on N, only N pulses are passed

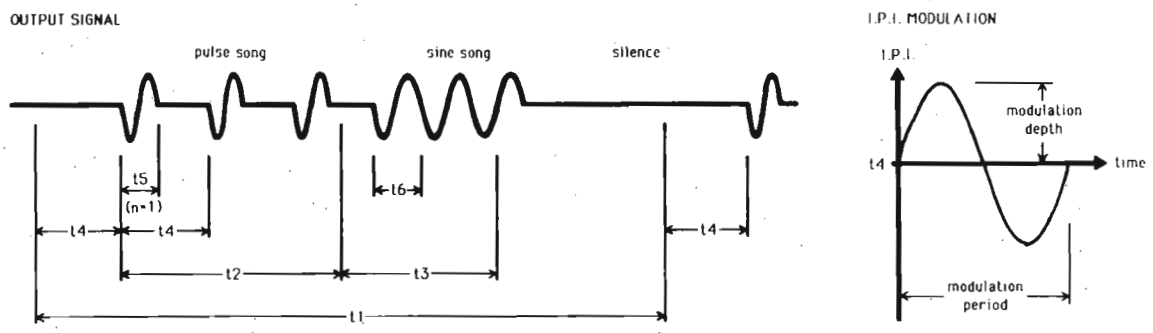


Figure 1. A schematic diagram of the output signal and the definitions of the variables.

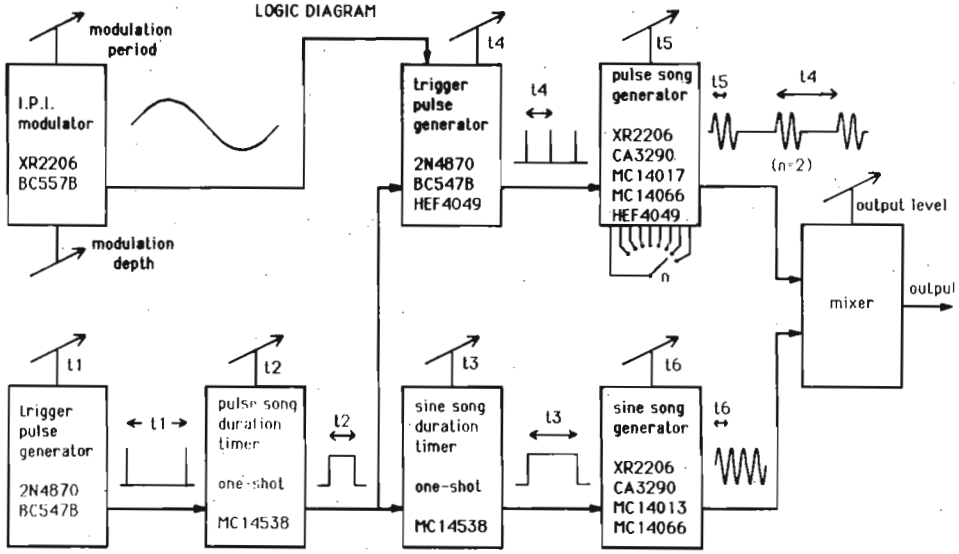


Figure 2. Logic diagram, showing how the different functions are interconnected.

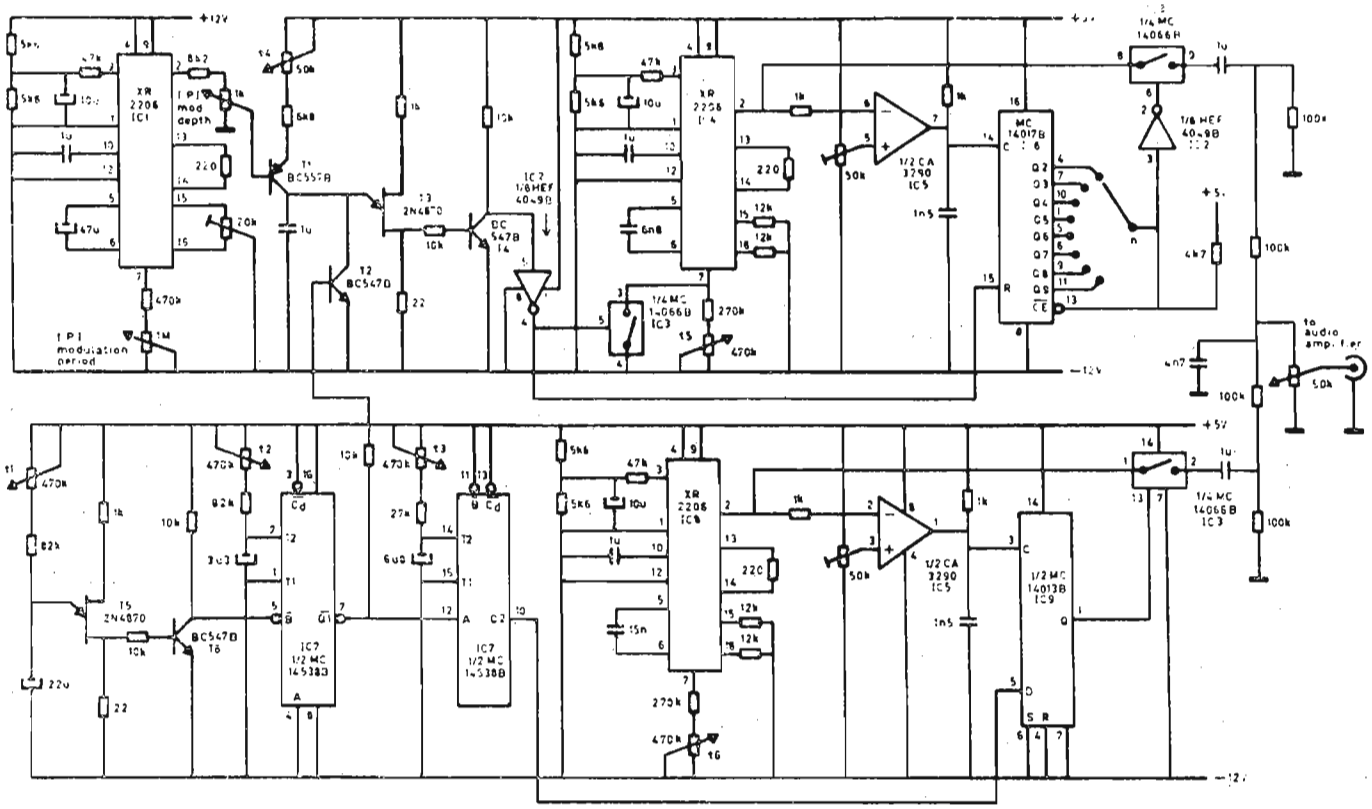


Figure 3. Circuit diagram of the sound simulator.

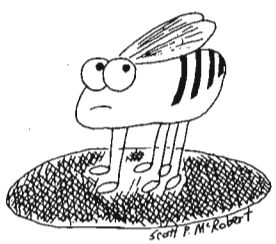
Table 1.

Variable	description	Control range
t1	length of one complete song sequence	1 to 10 sec
t2	length of pulse song	0.2 to 2 sec
t3	length of sine song	0.5 to 3 sec
t4	inter-ulse interval (I.P.I.)	15 to 100 msec
	- I.P.I. modulation period	30 to 90 sec
	- I.P.I. modulation depth	0 to 4 msec
t5	period of sine wave used for pulse song	2 to 5 msec
	- number of sine wave periods per pulse	1 to 8
t6	period of sine wave used for sine song	4 to 10 msec

to the mixer each t4. The trailing edge of t2 starts the sine song duration timer to give one block of t3 sec. During this time the sine song generator is gated to the output mixer and gives a pure tone with a wavelength of t6 sec. However, after the start of t3 the gate opens only when the sine signal crosses groundlevel for the first time. Similarly, after the end of t3 this generator will continue to finish its last wave.

The two timers symbolised by IC7 in the circuit diagram belong to the same integrated circuit 14578B. Similarly, MC14066B serves three functions, CA3290 two, and HEF4049B two functions. The controls are ordinary one turn potentiometers and specify all the times. The n switch, however, is a ten position switch and determines the exact number of cycles per pulse. The power supply, audio amplifier and speaker, being commercially available, are not given. The power consumption is 100 mA, the output (peak to peak) 1V, and the output impedance 50 KiloOhm.

References: Bennet-Clark, H.C. & A.W. Ewing 1967, Nature 215:669-671; Ewing, A.W. & H.C. Bennet-Clark 1968, Behaviour 31:228-301; Johnson, P. & D.E. Cowling 1980, DIS 55:152-154; Kyriacou, C.P. & J.C. Hall 1980, Genetics 11:6729-6733; Kyriacou, C.P. & J.C. Hall 1982, Anim. Behav. 30:794-801; Von Schilcher, F. 1976, Anim. Behav. 24:18-26.



Bhadra, U. and R.N. Chatterjee. University of Calcutta, India. A method of polytene chromosome preparation of salivary gland of Drosophila for in situ transcription and in situ hybridization technique.

Drosophila workers of tropical countries (like India, Bangladesh, etc.) repeatedly attempted to develop a cytological procedure that shows a better preparation of polytene chromosome of Drosophila for in situ transcription and in situ hybridization techniques. But, unfortunately, none of the procedure would give the optimum result after hybridization and transcription. This present procedure should exhibit well spread and evenly flattened polytene preparations, that are more essential for in situ transcription as well as in situ hybridization. Such a preparation shows a unique morhology of all the chromatins and gives the maximum transcription and hybridization signals.

The procedure is as follows:

[1] Salivary glands of well-nourished third instar larvae were dissected out by hand in buffered Ringer's solution (NaCl - 7.5 gms, KCl - 0.14 gms, $NaHCO_3$ - 0.2 gms, $CaCl_2$ - 0.12 gms, Na_2HPO_4 - 0.01 gm in 1 litre double distilled water at pH 7.2) (Berendes 1973).

[2] Two pairs of glands were shifted with a droplet of 50% acetic acid (the fixative) on a small siliconised coverslip (18 mm^2). Small coverslips provide such facilities that chromosomes will fix in a restricted zone. Ringer's solution should never be transferred along with the glands, since acetic acid dilution causes poor fixation of the glands.

[3] The glands were fixed for 2-3 minutes. A clean slide was slowly lowered on to the coverslip. The slide was turned over in upside down direction. The coverslip was tapped gently with a pointed object, such as the tip of the needle. With the help of this tapping, coverslip possibly moves slightly sideways. This step is intended to force the chromosomes out of the cells and to settle in a well spread manner. The prepartion was examined by a phase contrast microscope.

[4] The slides containing well spread chromosomes were placed on a filter paper with its coverslip down and wrapped with this paper. The coverslip was pressed very hard directly with the thumb. At this point, precaution should be taken that coverslip must not change its previous position. The thumb pressure will only flatten the chromosomal arms without improving its spreading, and thus make a good morphology.

[5] The prepared slides were placed on a hot plate (that was previously warmed to 40°C) for 10 seconds. The heat shock makes the chromosome to adhere to the surface of the slides and thus help the retention of maximum number of nuclei upon the slides.

[6] Immediately, after the heat shock, the slides were then placed into a horizontal coupline jar containing 50% acetic acid vapour and those were kept for one hour in coupline jar. In this step, maintaining appropriate time is very much vital; if the incubation time is reduced, it leads to poor morphology of the chromosome. Otherwise, if the incubation time is more than one hour, acetic acid vapour will reduce the amount of DNA, and will exhibit poor transcription and hybridization.

[7] The slides were immersed in a mixture of acetic acid and 50% ethanol (1:1) for 20 minutes, then the coverglass was flicked off by sliding a sharp razor blade under one corner of the slide within the mixture. Just after the removal of coverslips, the preparations were plunged into 70% ethanol at room temperature for 5 minutes.

[8] The slides were put through one wash in 90% ethanol and one more wash in 100% ethanol, 5 minutes each.

[9] The slides were allowed to air dry. In dry condition, slides could be stored for a longer period. The best preparation for in situ transcription and in situ hybridization is flat and grey chromosomes with no refractivity. The banding pattern should be easily distinguishable.

In this procedure, use of formaldehyde, acetomethanol as fixatives were avoided, because these interfere with the denaturation of the double stranded DNA. And fixative, like acetic acid:methanol (3:1) mixture may help to lose of large amount of DNA and reduced transcription and hybridization. Again, excessive acid treatment (such as aceto-orcein, aceto-carmine) may depurinate the DNA and exhibits the poor level of hybridization and transcription.

Acknowledgement: The authors are grateful to Council of Scientific and Industrial Research for financial assistance.

Reference: Berendes, H.D. 1973, Int. Rev. Cytol. 35:69-116.

Green. C.C., J.C. Sparrow and E. Ball.
University of York, Heslington, York, England.
Flight testing columns.

To test for flight ability, Benzer (1973) used a measuring cylinder whose walls were coated with paraffin oil. Flies were thrown down the cylinder and flighted flies flew to the sides and stuck to the oil. Flightless flies dropped to the bottom of the cylinder. Flies with reduced flight ability were found stuck to the lower levels of the cylinder having fallen further before reaching the sides of the column.

While using this test system we encountered the problem that the temperature-sensitive nature of the viscosity of the oil critically affected the efficiency of the cylinder to trap flies. To counter this problem, we have replaced the paraffin oil with a commercial compound for ecological sticky traps: "Oecotak" [Oecotak A5 from OECOS Ltd, 130, High Street, Kimpton, Hitchin, Herts., ENGLAND]. Its viscosity is unaffected by changes in temperature between 15-30°C. The "Oecotak" is applied with a hand roller to acetate sheets which are used to line the inside of a glass cylinder (G) (Figure 1). Flies are introduced directly from culture vials through a plastic funnel (F) and the flightless flies collected in a beaker (B). Measures of the flight ability of different strains can be made, either as a percentage of introduced flies recovered in the beaker, or from the positions of stuck "flighted" flies. In the latter case, the acetate sheets are removed from the cylinder, flattened, and placed on a grid to record the positions of stuck flies. This can also be done on a digitiser pad connected to a microcomputer. The sample mean and variance of the distance travelled from the point of introduction can then be computed.

Table 1.

No. of Ore in test vial	No. of FLI+ after 1st test	No. of FLI+ after re-test	No. of Ore in FLI+ vials	% Ore recovered
0*	9	4	--	--
6	7	5	5	83.3
12	14	11	10	83.3
18	20	16	14	77.8
24	21	23	21	87.5
30	31	29	28	93.3
36	39	39	36	100.0
42	39	41	40	95.2
48	47	49	47	97.9
54	55	52	51	94.4
60*	58	59	59	98.3

*thus the st p^p M320 strain is 93.3% flightless, whilst Oregon shows 1.7% flightlessness.

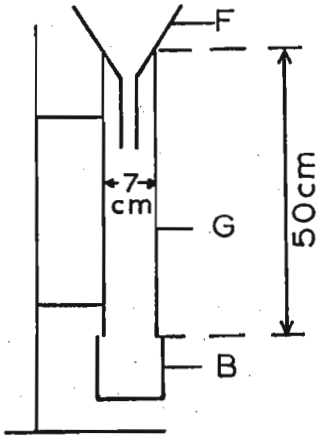


Figure 1.

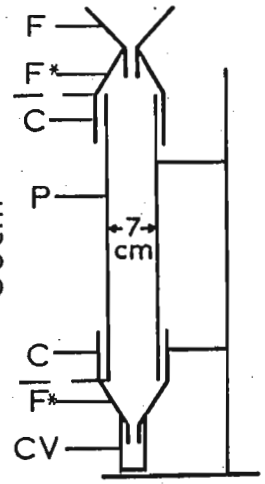


Figure 2.

Both Benzer's "greased" cylinder, and our "Oecotak" cylinder cause destruction of tested flies. This prevents repeated flight-testing of a sample. Such repetition could improve the separation of flighted from flightless flies, and facilitate the investigation of such phenomena as temperature sensitivity and age degeneration of flight ability. A non-destructive test could also be used to select flighted revertants of flightless mutations.

We have therefore devised a non-destructive flight-tester. In this system, the cylinder is formed from blotting paper (P) held in place by two PVC collars (C), onto which polypropylene funnels (F*) are taped (Figure 2). As before, flightless flies fall through the column and are collected. Flighted flies land and grip the paper. The first collecting vial (CV) is removed, and replaced by a new vial (whose base has been removed and replaced by a fine nylon mesh). The flighted flies are then simply blown out of the cylinder into the collecting vial.

The effectiveness of the paper column in separating flighted and flightless Drosophila is demonstrated in the following experiment: Wild-type Oregon flies were mixed in a vial in varying proportions with flies of a marked flightless strain, st p^p M320 (M320 is a mutation of actin 88F causing a reduction in IFM-specific actin). The flies were tested for flight ability in the paper column, and the flies in both the "flightless" and "flighted" vials were then re-tested (separately). The flies finally collected in the "flighted" vials were scored as to eye colour phenotype. The results are displayed in Table 1.

Using this non-destructive column, we plan to select for flighted revertants of flightless mutants. Such revertants may serve to isolate further genes involved in the flight system.

References: Benzer, S. 1973, Sci. Am. 229:24-37.

Handke-Kociok, M. and W. Liebrich. Institut fur Genetik, Universitat Dusseldorf, FR Germany.
A simple method for staining chromosomes in whole embryos of Drosophila.

A quick and easy method is described to stain the chromosomes of whole Drosophila embryos (prior to blastoderm formation) using the fluorescent dye, 4, 6-diamidino-2-phenylindole (DADI): eggs of **Drosophila hydei** in early developmental phases were collected and transferred into small baskets (baskets according to Widmer & Gehring 1974). During the whole procedure the eggs remained in them. Between each preparation step till fixation, they had been washed, pipetting Drosophila Ringer several times into the basket. After a careful wash in running water to remove medium, the eggs had been laid in, dechorionization was carried out placing the baskets in to a 3% sodium hypochloride solution (Hill 1945; Rickoll 1976) for 2-3 minutes. Proper dechorionization was checked by means of a stereo microscope. To permeabilize the vitelline membrane, the baskets were immersed into octane (Limbourg & Zalokar 1983) for 2 minutes. Fixation in a mixture of methanol and glacial acedic acid (3:1) followed during the ensuing 30 minutes. Thereafter eggs were carefully washed in Ringer's solution and placed into tris-buffered solution (tris-(hydroxy-methyl)-aminomethan; 0.18 M; pH 7.5) of DAPI (10^{-6} g/ml; Stohr et al. 1980). The eggs were then washed in tris-buffer and --after opening the baskets-- cautiously transferred into a troplet of tris-buffer on an object-slide. They were protected using a coverslip and observed by means of a fluorescence microscope (Zeiss No. II; filter combination: BP 365/10, FT 420, LP 418). In that way it is possible to visualize the chromosomes in situ. The technique is of interest for embryological as well as cytological studies (Fig. 1 and insert).

References: Hill, D.L. 1945, DIS 19:62; Limbourg, B. & M. Zalokar 1973, Dev. Biol. 35:382-387; Rickoll, W.L. 1976, Dev. Biol. 49:304-310; Stohr, M., K.-J. Hutter, M. Frank, G. Futterman & K. Goerttler 1980, Histochem. 67:179-190; Widmer, B. & W. Gehring 1974, DIS 51:149.

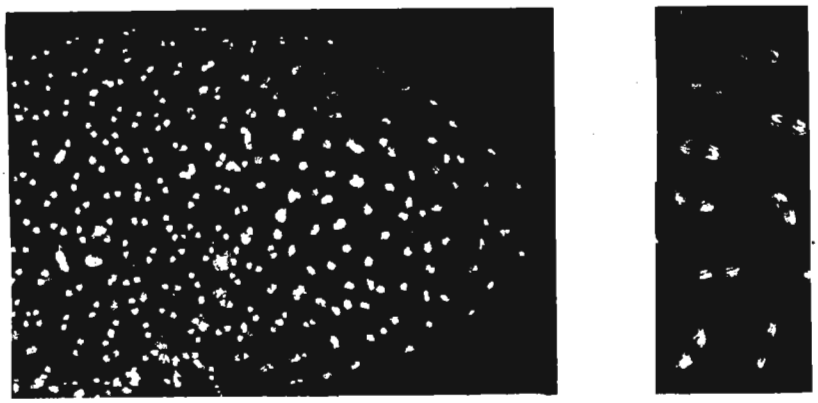


Figure 1. Whole Drosophila embryo with the chromosomes stained using DAPI. (x480, inset: x 1200).

Kalisch, W.-E. and T. Whitmore. Ruhr-Universität Bochum, FR Germany.
The SSP chromosome preparation technique as applied for **D.melanogaster.**

The intention of this technical note is to give a reply to the requests we have received asking for a more detailed description of the surface-spread polytene (SSP) chromosome preparation technique (Kalisch & Hägele 1981; Kalisch et al. 1984) and especially its adaption for the use with **D.melanogaster.** Quite recently, the technique was used successfully for high resolution mapping of in situ hybridized biotinylated DNA to salivary gland chromosomes of **D.melanogaster** (Kress et al. 1985). Since the technique used by Kress et al. (1985) differs in several methodological details from the optimized one we are now using, we feel it thus necessary to give the following up-to-date description of our technique with specific reference to the salivary gland chromosomes of **D.melanogaster.** The spreading effect and the chromosome cytology is given in Fig. 1 as an example and compares the reference map of Lefevre (1976) (from squash preparations) and a SSP chromosome preparation.

Preparation of salivary glands. After a brief washing of the larvae[1] in a pre-treatment solution[2], the salivary glands are excised with forceps[3] in ca. 16 µl of pre-treatment solution. It is not recommended to use Ringer-solution for this preparation step (for detailed reason, see Kalisch & Whitmore 1983). The glands are immediately transferred to a 10 µl vial, which is filled with 8 µl of pre-treatment solution (60 vials on one disposable test plate: No. 163118; NUNC GmbH, D-6200 Wiesbaden, FR Germany). After removal of any remaining parts of the fat bodies, the glands are mechanically lacerated with forceps. This procedure is controlled under the binocular, where the bursting of the cells is visible. The lacerating of the glands is, if necessary, continued until the glands disappear into a turbid 'chromosome suspension'. The pre-treatment procedure is terminated after 5 min (from excising the glands) by the spreading procedure.

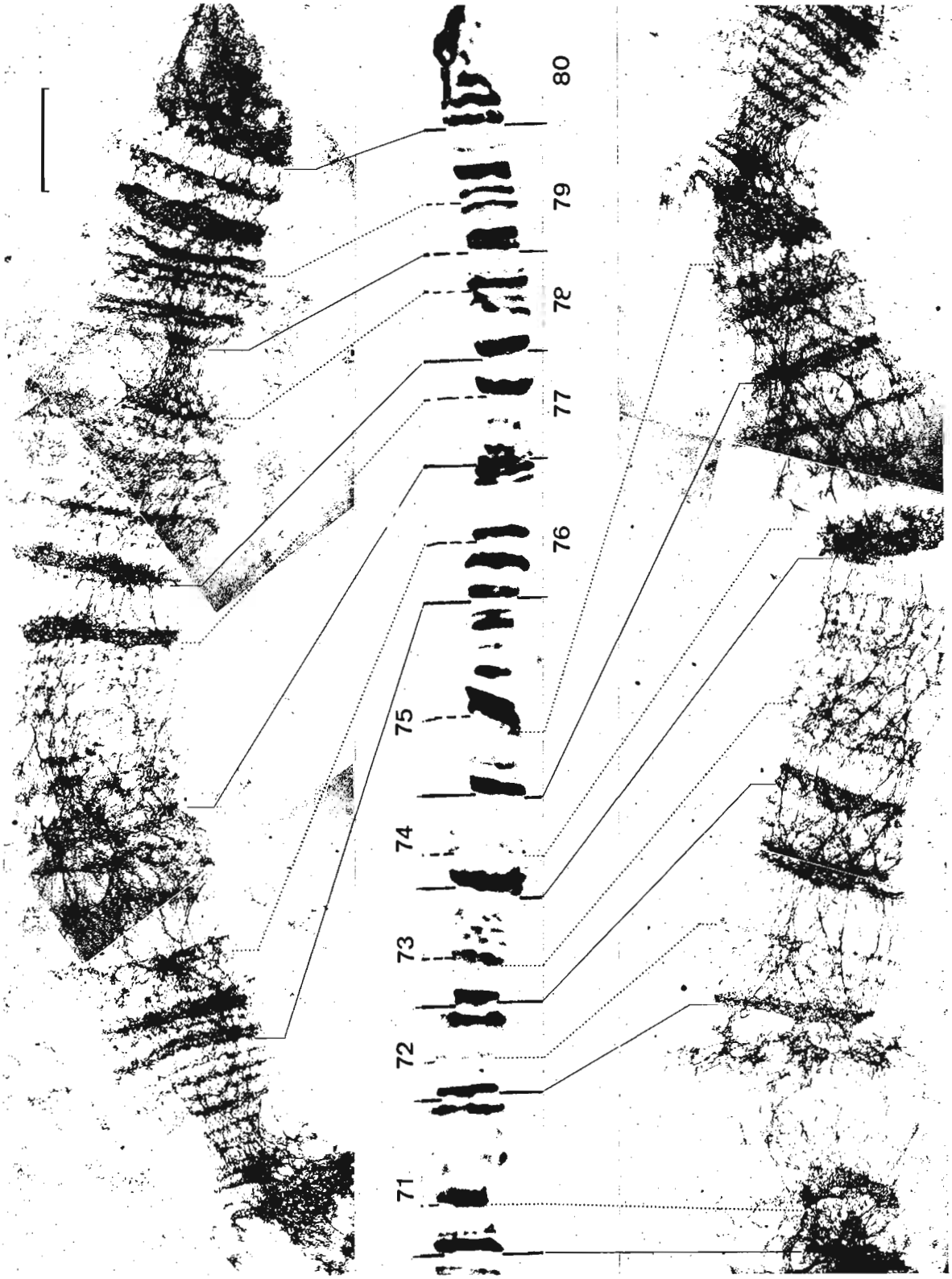


Figure 1. Salivary gland chromosome divisions 71-80 (3L) of **D.melanogaster:** Comparison between the electron micrograph of a single SSP chromosome preparation and the light micrograph of well-extended chromosome squash preparations (reference map of Lefevre 1976). Under the assumption that the squash preparation represents a chromosome diameter of 5-8 µm, then both chromosomes show the same magnification. It should be noted that the quality of the light micrograph is reduced by copying it from a reprint. Furthermore, it has to be considered, that Lefevre's reference map is a composition of several well-extended chromosome preparations. By this, the longitudinal (axial) degree of spreading of the SSP chromosome appears reduced from what is normally observed in comparison with routine squash preparations. Bar represents 10µm.

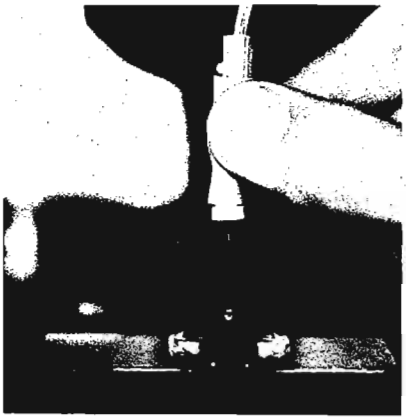


Figure 2. Illustration of the spreading procedure. The 'chromosome suspension' is hanging as a small drop at the lower end of the MICROCAP. This is immediately before the contact with the spreading medium (large drop). After the spreading process the microscope slide is moved using the object holder into the phase optic.

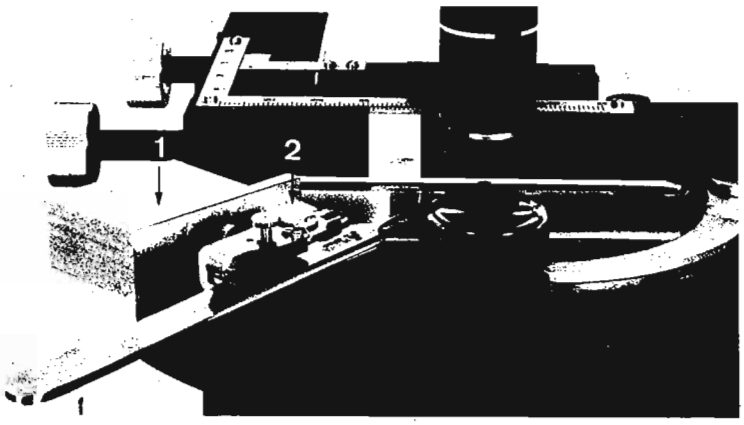


Figure 3. Picking up the chromosomes. The seesaw (see footnote **11**) holding the forceps with the grid can be moved freely on the microscope table to bring the mesh of the grid into alignment with the chromosomes (both of which can be seen with the phase optic). The actual distance is shown between the objects and objective being used.

Spreading procedure. (Fig. 2): Half a spherically shaped drop (Ø = ca. 1.5 cm) is formed by 1.5 ml of a spreading medium[4] on a double siliconized[5] microscope slide. A siliconized MICROCAP[6] is used to transfer the pre-treated chromosomes (the 'chromosome suspension') from the 10 µl vial to the surface of the spreading medium. For each spreading procedure, a ca. 4 µl drop of the 'chromosome suspension' is formed at the lower end of the MICROCAP and after waiting for about 30 sec the drop is brought into contact with the zenith of the spreading medium surface. At the moment when both drops touch each other the half spherically shaped drop of spreading medium is clearly flattened. In those cases where the spreading medium does not show this effect, the MICROCAP has either been pushed through the surface of the spreading medium or too small amount of 'chromosome suspension' has been used. In either case the spreading procedure has to be started again using a new drop of spreading medium.

SSP chromosomes: The spread chromosomes[7] remain on the surface of the spreading medium. They can be visualized using a phase optic (Achromat 10/0.22 Ph, No. 460401.9904.000, ZEISS). To enlarge the area of visibility with the phase optic in connection with the curved surface of the spreading medium, we mechanically flatten[8] the surface of the spreading medium. Spread chromosomes slipping to the rim of the surface of the spreading medium are brought back by blowing air[9] over the surface of the spreading medium from different directions.

Picking up the chromosomes (Fig. 3): SSP chromosomes are picked up on Carbon-coated Formvar grids[10]. In order to obtain the entire band-interband pattern of a chromosome or even an entire genome, we use grids with one 500 x 1000 µm mesh (Type G200, No. 01197; BALZERS GmbH, D-6200 Wiesbaden-Nordenstadt, FR Germany). For the process of picking up a chromosome, a pair of forceps (holding the grid) are secured in a seesaw[11] type construction. By this arrangement, it is possible to visualize both the grid and the SSP chromosome simultaneously allowing the proper adjustment of the mesh over the chromosome.

After picking up a SSP chromosome by touching the spreading medium with the grid, the EM grid is carefully washed[12] by floating the grid, filmed side down, for 10 min on the surface of aqua bidist., dipping several times in 50% and transferring to 96% isopropanol for 10 min. Air-drying of the grids (filmed side up) is done on a filter paper (since the grids have been bent slightly before being covered with the Formvar-film, a contact between the film and the filter paper can be avoided). There is no contrasting procedure used with this technique. The grids can be stored without a changing of the band-interband pattern, but time may weaken the strength of the Formvar film.

For LM analyses, SSP chromosomes can be picked up on subbed slides or cover slips (for this procedure, we hold the subbed slide at an angle of 90° to that of the slide where the spreading medium is on). If the SSP chromosomes are blown from the rim to the middle of the surface of the spreading medium after the pick up, it is possible to get even a second and third LM preparation from the same drop of spreading medium. The washing procedure is the same as for EM grids.

Cytological analysis: We start to analyze the degree of spreading and the quality of the SSP chromosomes on the EM grids by LM analysis (16/0.40 Ph, Zeiss). For the EM analysis of the SSP chromosomes we use a Siemens ELMISCOP 101 at 40 and 60 kV, respectively. Usually we take

micrographs with a x1,600 or a x2,400 magnification and use a final magnification of x3,200 and x4,800, respectively, unless overviews with a lower magnification have to be given as in Fig. 1.

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1. Raising of larvae: Third-instar larvae of a **D.melanogaster** wild-type strain (Berlin) were used in this case. Culture conditions were the same as commonly used by others for yielding proper chromosome squash preparations. Second- and third-instar larvae were raised at 18°C and fed with a daily supplement of baker's yeast. In order to ensure an optimal food supply third-instar larvae were transferred once to fresh medium.

2. Pre-treatment solution: For this 3.18 M citric acid 1-hydrate and 8.82 M proprionic acid solution, 10 g of a citric acid 1-hydrate (No. 244; MERCK, D-6100 Darmstadt, FR Germany) is dissolved in aqua bidist. by stirring and gentle heating and brought to 15 ml. Separately, 19.8 ml of propionic acid (No. 800605; MERCK) is mixed with 10.2 ml aqua bidist. Both acid solutions are carefully mixed together and can be stored in a tight vial for several days. Depending upon the conditions of the stock being studied, other ratios of the solutions than the 1:2 above (for example, 1 part propionic to 1 part citric acid) may be necessary to provide the required degree of chromosome spreading.

3. Forceps: For preparation of the salivary glands and for picking up the spread chromosomes from the surface of the spreading medium, we use the following forceps: EREM SWISS 5SA (BALZERS). For salivary glands preparations, the forceps are sharpened (we use a special instrument for sharpening: No. B8010 03 031; BALZER). For handling the EM grids during the washing procedures we use an extra forceps (No. B8010 03 097; BALZERS). Forceps are demagnetized (No. B8010 03 032; BALZERS) after sharpening and cleaned daily either in alcohol or ultrasonically.

4. Spreading medium: The 4 M urea and 0.1 M HCl solution is prepared as following: 24 g urea (No. 8487; MERCK) and 10 ml of 1M HCL are brought to 100 ml aqua bidist. A pH of 2.0 is achieved by additional 1M HCL.

5. Siliconizing: For siliconizing we use exclusively a commercially available silicone solution (No. 35130; SERVA, D-6900 Heidelberg, FR Germany). After siliconizing the microscope slides we dry them horizontally for 60 min at 100°. Otherwise, there will be a gradient of silicone from one side to the other, which results in a movement of the spreading medium to one side when the drop is formed or during the spreading process. The slides are siliconized twice. We use each slide for only one spreading preparation.

6. MICROCAP: The 10 µl MICROCAP (Drummond Scientific Co., USA) is connected (using the MICROCAP holder) with a parafin-oil filled plastic tube (see Fig. 2) which itself is connected with a parafin-oil filled 100 µl HAMILTON syringe (Typ 1710LL). The plunger of the syringe is connected directly with a micrometer (NSK, Japan Micrometer). An airspace of max. 3 cm should be left between the MICROCAP connection and the start of the parafin-oil. The siliconized MICROCAP is discarded after every few spreading procedures.

7. Spread chromosomes: Well-spread chromosomes are more or less free of those types of thick protein conglomerates which strongly reflect light as usually seen in squash preparations. Extremely well-spread chromosomes have a very low contrast by the optical system used making them difficult at first to locate. Contamination through too much accompanying cellular material can influence the quality and degree of spreading.

8. Flattening of the spreading medium: We use a disposable hypodermic syringe with a 20G1/2 (0.90 x 38 mm) needle which is connected with a rubber tube to a plastic tube connector which serves as a mouthpiece. The syringe can be injected at any area of the spreading medium. With the needle near the center of the drop (to avoid too much circulation) the spreading medium is sucked off very slowly to avoid a deformation of the drop at the rim. In those cases where inlets do appear, they can be removed by moving the hypodermic needle (together with the adherent spreading medium) to the old border of the rim.

9. Air blowing: We use a mouth controlled rubber tube, which is capped with a disposable PIPETMAN C20TJ tip. The extent to which this technique works depends upon the amount of cellular material accompanying the SSP chromosomes on the surface of the spreading medium. Furthermore, it should be emphasized, that a flattened surface of the spreading medium supports the efficiency.

10. Formvar grids: An extremely durable film is needed for the 500 x 1000 µm mesh of the grids used (No. B8010 01 197; BALZERS). We use a concentrated Formvar solution which yields a film ca. 3-4 times thicker than that normally used. The filming of the grids is carried out with certain modifications as described by Lickfield (1979), whereby the humidity used plays an important role in the preparation of optimal films: (1) Several microscope slides are cleaned with a linen cloth which has been boiled beforehand in distilled water. This is repeated with a new cloth. (2) A 0.25-0.35% solution of Formvar (Formvar 1595 E, No. 21740, SERVA) is prepared in chloroform (or in 1.2-dichlorethane) and stored in a bottle with an opening large enough for a microscope slide. (3) By 38% humidity one of the clean microscope slides is dipped into the Formvar solution, pulled out slowly and left to dry for ca. 24 hrs at the same humidity. (We use a plexiglass box for this step which has an opening for a hand. Use a vinyl (rubber) glove, otherwise the film may appear fogged. To keep the humidity at ca. 38% any of the typical dry gels should work. The opening in the plexiglass box should also be kept relatively closed with any type of

flexible (vinyl) plastic. (4) The dried, film-coated slide is scored along the edges with a razor blade. (5) The filmed microscope slide can be now slowly slid into a petri-dish filled with distilled water at an angle of ca. 5-10°. Thereby the film on the microscope slide floats onto the surface of the water. (6) The defilmed microscope slide is pulled out of the petri-dish. (7) Electron microscope grids are placed with the dull side down on the film. (8) A strip of either filter paper or parafilm is lowered onto the grids and as soon as it sticks to the film, the strip is pulled with the grids out of the petri-dish. To prevent a contact between the film and the filter paper the grids are bent slightly beforehand. (9) The filter paper (parafilm) strip is placed with the filmed EM grids on top in a petri-dish lined with filter paper to dry. (10) The petri-dish is closed with a glass lid. (11) In order to further stabilize the film we often carbon coat the grids.

11. Seesaw: A forceps frame consisting basically of two parts (1 and 2 in Fig. 3) which serve the function of a pivot point for the forceps. The forceps can be swivelled with the one end holding the grid and the other one being used for hand-directioning. Part 1 of the seesaw (a 25 x 20 x 15 mm aluminum block with a 4 x 10 x 15 mm lip on which the part 2 is attached with a screw) can be moved on the microscope table. Part 2 is an 'instrument holder' (part of the micromanipulator 'de Fonbrune', type PM1; BACHOFER GmbH, D-7410 Reutlingen, FR Germany). As a very primitive alternative, the forceps can even be secured by a clothespin.

12. Washing procedure: The Formvar film is not very strong when it is wet. For the wash in 50% isopropanol the grids should only be moved with the film in the vertical direction. We use exclusively plastic vials (35 x 10 mm, No. 150318; NUNC) for these processes, which are disposed of after half a day to avoid protein contamination.

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References: Kalisch, W.-E. & K. Hägele 1981, Europ. J. Cell Biol. 23:317-320; Kalisch, W.-E. & T. Whitmore 1983, Cytobios 37:37-43; Kalisch, W.-E., T. Whitmore & H. Reiling 1984, Cytobios 41:47-62; Kalisch, W.-E. & J. Böhm 1985, DIS 61:94-95; Kalisch, W.-E., T. Whitmore & A. Siegel 1985a, J. Microsc. 137:217-224; Kalisch, W.-E., T. Whitmore & G. Schwitalla 1985b, Chromosoma 92:265-272; Kalisch, W.-E., G. Schwitalla & T. Whitmore 1986a, Chromsoma, in press; _____ 1986b, Cytobios, in press; Kress, H., E.M. Meyerowitz & N. Davidson 1985, Chromosoma 93:113-122; Lefevre, G.J. 1976, in: The Genetics and Biology of Drosophila, Ashburner & Novitski (eds.), Academic Pr., Vol. 1a:31-66; Lickfield, K.G. 1979, Elektronenmikroskopie, UTB 965, Ulmer Verlag, Stuttgart.

Laverty, T.R. University of California, Berkeley, USNA. A device for dispensing instant Drosophila food.

When working with Drosophila sometimes one desires to rear them on media enriched with a specific chemical. Whether it is an antibiotic, ethanol, or some other chemical which is water soluble, it can be easily added to the water when preparing instant Drosophila media such as Formula 4-24 from Carolina Biological Supply Co. Described below is a simple, inexpensive device to dispense equal volumes of instant food quickly, reproducibly, and with little mess.

A three sided box is construced. The top has holes drilled in it to align with the vials to be filled and has a removable false bottom. The thickness of the top and the diameter of the holes are determined by the vial size. The hole size should be slightly less than the diameter of the vial. Then if the vials are slightly misaligned all the food will still fall into them.

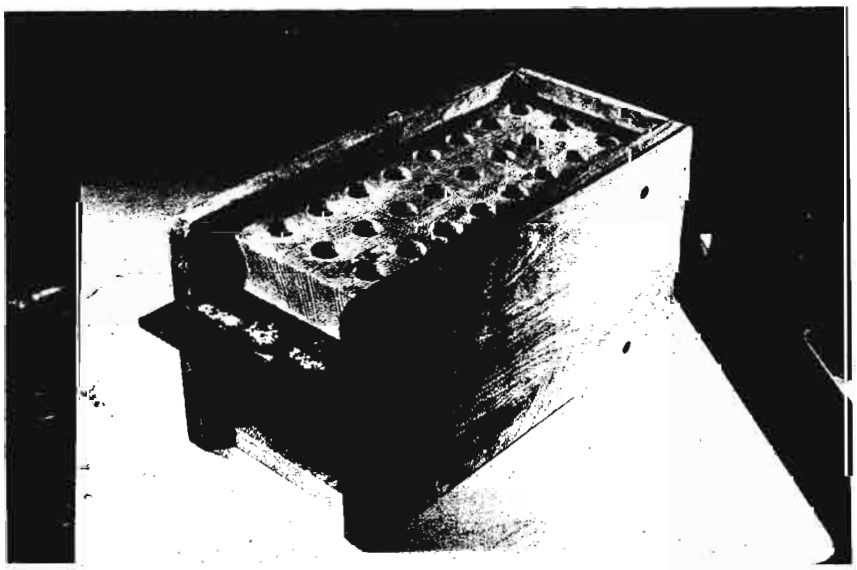

Figure 1. A device for dispensing instant Drosophila food.

The instant food is poured onto the top of the box. The food is spread over the top with a brush filling all the holes. The false bottom is then removed and the food falls into the vials. Water with the appropriate chemical can then be easily added to the food with a repeating syringe.

Using this device all the vials can be prepared identically and quickly. Therefore, any differences observed between crosses will not be due to inconsistencies in the media. Also this device is excellent to use when preparing instant food for normal uses.

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In situ hybridization of biotinylated DNA probes to polytene salivary chromosomes of Drosophila species.

There are several obvious advantages to using biotinylated probes for in situ hybridization in place of radioactive probes. Unfortunately, many laboratories have encountered serious difficulties in their attempts to make this technique work routinely. During the last few months, we have established a procedure which allows us to achieve consistently good results. Our protocol is derived from published accounts as well as personal communications of details that often escape journal reports. We are especially indebted to Dr. John Thomas of Stanford University for many valuable suggestions which were essential to the development of this procedure.

I. Preparing polytene chromosomes for in situ hybridization. Chromosome squashes were prepared essentially as described by Atherton & Gall (1972). Drosophila larvae were harvested from well-yeasted, uncrowded culture bottles. The best chromosome squashes were obtained from third instar larvae that had crawled up the side of the bottle and had stopped moving. Larvae were dissected in 45% acetic acid. The salivary glands were removed and most or all of the attached fat bodies were teased away. Dissection of some Drosophila species was particularly difficult. In such cases, larvae were dissected in Meyer's solution and the salivary glands were transferred immediately to 45% acetic acid.

After dissection, one pair of salivary glands was transferred to a 10-15 microliter drop of 45% acetic acid placed on a very clean (dust free) coverslip. [The coverslips were siliconized using the instructions contained with a bottle of Prosil-28 (SCM Specialty Chemicals), dried, and stored in the original container. Each day, the siliconized coverslips were placed in 95% ethanol until needed. Immediately before use, a single coverslip was dried with a paper lab wipe and blown clean with a stream of pressurized gas.] The drop was placed directly at the center of the coverslip so that it would be easy to locate the chromosomes later. The temptation to put more than one pair of glands under the coverslip should be avoided. In an ideal squash, the cellular debris flowed away from the chromosomes; too much tissue precluded this. An equally clean "subbed" slide (Gall & Pardue 1971) was lowered onto the coverslip so that three edges of the coverslip were closely parallel to and equidistant from the three edges of one end of the microscope slide. The coverslip was picked up with the subbed slide, with the coverslip "hanging" down. The coverslip was displaced laterally in all four directions in turn (to facilitate spreading of the chromosomes) by tapping the edges of the slide against a pencil (W.J. Dickinson, pers. comm.). The slide was turned over and examined with a phase contrast microscope to determine whether the nuclear envelope had been broken. If the chromosomes were sufficiently free of the nuclear envelope, the slide was placed on blotting paper, coverslip down, and the excess acetic acid was carefully removed by pressing lightly on the slide. As the acetic acid was blotted, the chromosomes spread. If too much pressure was exerted at this stage, the chromosomes were stretched and/or broken. The spreading was monitored frequently with the microscope. As the chromosomes were flattened onto the subbed slide, more pressure could be applied without stretching the chromosomes. The need for diligent care and patience at this stage cannot be over-emphasized. Sufficently flattened chromosomes had little or no refraction along their edges. The slide was placed coverslip up on a 45 C warming plate to increase flatness. After at least one minute, the warm slide was placed, coverslip face up, on a flat table top. With one sheet of blotting paper over the slide and the coverslip, the coverslip was pressed very firmly with the thumb. If the chromosomes were not squashed very flat, resolution was greatly impaired.

When the slide had cooled to room temperature (to prevent fracturing the glass), it was dipped into liquid nitrogen for a few seconds (until boiling subsided). The frozen coverslip was then removed immediately by inserting a razor blade under a corner of the coverslip and flipping it off. The slide was immediately plunged into freshly made absolute ethanol:acetic acid (3:1) which had been chilled to -20 C. After 2-3 minutes, the slide was transferred to 95% ethanol and left there for 30 minutes to 2 hours. It was then rinsed in 100% ethanol for about 2 minutes and air dried. Slides were examined without coverslips by phase microscopy to determine if they were suitable for hybridization. Suitable slides had undistorted chromosomes, very little refraction along the edges of the chromosomes, and clearly visible and distinct bands. If most of the chromosomes on a slide were not suitable, the slide was discarded.

The slide was incubated for 30 minutes in 2XSSC (SSC is standard saline citrate: 0.15 M NaCl, 0.015 M sodium citrate) at 70 C. The slide was then dehydrated in 70% and 95% ethanol for 5 minutes each and air dried. Chromosomal morphology seemed to be improved by the 2XSSC wash, air drying, and dehydration.

The chromosomes were denatured by soaking the slide in 0.07 N NaOH for 3-7 minutes. The time required to denature the chromosomes varied from species to species (**D.melanogaster** chromosomes denatured within 3-4 minutes; **D.silvestris** and **D.virilis** chromosomes seemed to require at least 7 minutes). The slide was transferred immediately to 70% ethanol, dehydrated through 95% ethanol, and air dried, as above. It was essential for good hybridization that the chromosomes were denatured sufficiently, even at the cost of some decrease in resolution of chromomeres. Chromosomes were denatured immediately before hybridization. Denatured chromosomes which had been stored before use did not hybridize well.

II. Nick translation. The probes which we used were recombinant plasmids which contained **D.melanogaster** DNA. The probes were purified using an alkaline lysis technique, RNaseA and proteinase K digestions, phenol extraction, and ethanol precipitations in the presence of ammonium sulfate and then sodium acetate. Where possible, the precipitated DNA was collected with a glass hook rather than by centrifugation. They were not purified by density gradient centrifugation or by gel exclusion chromatography. The $^{OD}260/^{OD}280$ ratio was generally in the range of 1.8. The preparations were free of bacterial DNA and all but a trace of digested RNA fragments as judged by ethidium bromide staining of agarose electrophoretic gels. DNA concentration was estimated assuming that a 50 mcg/ml solution has an $^{OD}260$ of 1.

Recombinant **D.melanogaster** DNA probes were labeled with biotinylated dUTP using a nick translation kit. (Although the details given below refer to the Bethesda Research Laboratories nick translation kit with biotin-11-dUTP, we have obtained equivalent results using the nick translation kit from Enzo Biochem with either biotin-11-dUTP or biotin-16-dUTP.) We followed the protocol on the BRL biotin11-dUTP data sheet or the protocol supplied with the Enzo Biochem kit. When using the BRL kit we stopped the reaction by adding stop buffer (omitting the SDS) and incubating at 65 C for 10 minutes. We no longer use radioisotopes to monitor the nick translation reaction, since we have never had one fail.

The labeled DNA was separated from unincorporated nucleotides and other molecules on a 0.9 cm diameter x 15 cm Sephacryl-200 (Pharmacia) column equilibrated with TE (0.01 M tris [tris(hydroxymethyl)aminomethane], 0.001 M EDTA, pH 7.5). Labeled DNA was mixed with 12.5 mcl of 0.5% blue dextran (BRL Focus 7(1):8). The blue dextran co-chromatographed with the DNA and did not interfere with hybridization. Fractions of 7 drops (approx. 39 mcl/drop) were collected. The DNA and blue dextran peak was contained in 2 or 3 tubes which were pooled. Polypropylene microcentrifuge tubes were used for the subsequent steps to minimize the loss of DNA. The DNA was precipitated with 0.1 volume of 1 M sodium acetate, 20 mM magnesium acetate, pH 7.7, and 2.5 volumes of 95% ethanol and placed at -20 C overnight or at -70 C for 10-15 minutes. The DNA was collected by centrifugation at 10,000 rpm for 10 minutes in a microcentrifuge at 4 C. The supernatant was carefully removed by aspiration and the blue pellet was air dried.

The pellet was dissolved in 100 mcl of distilled water and 17 mcl of 12 mg/ml sonicated salmon testes DNA and heated in a boiling water bath for 5 minutes. The tube was immediately plunged into an ice bath. The remaining ingredients of the hybridization solution were then added: 40 mcl 20XSSC, 4 mcl 50X Denhardt's solution (Denhardt 1966) and 40 mcl 10% dextran sulfate (MW 5,000). The final concentration of the labeled DNA was approximately 5 mcg/ml. This mixture can be frozen, thawed in an ice bath, and used for hybridization at least 5 times. Alternatively, the DNA solution can be divided into smaller portions so that it is thawed fewer times before use.

III. Hybridization. Although we have described the following procedure for a single slide, we have routinely hybridized and stained several slides at a time.

The probe concentration used in our standard procedure, described below, was higher than it needed to be for good hybridization. We have tried hybridizing two or four probes to the same slide, thus decreasing the amount of each probe accordingly. Two probes hybridized as well as a single probe. Four probes did not hybridize as well as a single probe. Therefore, we suspect that the probe concentration could be reduced to one-half or perhaps one-third of the concentration which we normally used.

About 12-14 mcl of the probe solution were placed over the chromosomes on a slide which had been prepared for hybridization as described in Section I above. A coverslip that had been washed briefly in 0.5 N HCl, rinsed in distilled water, and wiped and blown free of dust was set on the drop of probe solution. To prevent evaporation from the edge of the coverslip, it was sealed to the slide with rubber cement which had been thinned to an appropriate consistency. The rubber cement was allowed to flow from the brush as ropy strands around the edges of the coverslip. Aesthetic values were ignored.

In order to concentrate for possible poor seals, the slides were incubated in a humid chamber. A paper towel which had been cut to fit the bottom of a plastic sandwich box was placed in the box and saturated with 2XSSC. The slide was incubated in the sealed box at 65 C for about 12 hours. Some reports

have indicated that as little as 3 hours is sufficent for hybridization when using dextran sulfate and Denhardt's solution. Other reports have suggested that hybridization for more than 18 hours damages chromosomal DNA (Barbera et al. 1979).

The rubber cement and the coverslip were removed. The slide was washed four times in 2XSSC at 60 C for 10 minutes. It was then washed twice in 1XSSC at room temperature for 10 minutes. The slide was transferred from the final 1XSSC wash to the first wash in the next section without allowing it to dry.

IV. Detection of the probe. The probe was detected using the BRL DNA Detection Kit. The slide was washed for 60 minutes at room temperature in BRL Buffer #1 (0.1 M Tris-HCL, pH 7.5, 0.1 M NaCl, 2 mM $MgCl_2$, 0.05% (v/v) Triton X-100) supplemented with 1% BSA. (The BSA is used to remove label adhering to chromosomal proteins. Since BSA is an excellent bacterial nutrient, the solution should be sterilized or else BSA should be added to Buffer #1 immediately before use. All steps involving BSA or subsequent to its use should be done quickly, preferably the same day, to minimize bacterial growth. Bacterial proliferation may render the chromosomes unacceptable for microscopy.) After the 60 minute wash, the slide was allowed to drain but not to dry.

Several drops (about 50-200 mcl) of streptavidin solution (2 mcl of BRL streptavidin solution in 1 ml buffer #1) were dropped gently onto the chromosomes. Evaporation was minimized by placing a coverslip over the chromosomes, placing the slide in a shallow container, and covering the container with aluminum foil. The slides were incubated at room temperature for 2 hours (John Thomas, pers. comm.). The coverslip was removed and the slide was washed three times for 10 minutes each at room temperature in buffer #1. The slide was allowed to drain but not to dry.

Several drops (about 50-200 mcl) of poly-alkaline phosphatase solution (2 mcl of BRL poly-alkaline phosphatase solution in 2 ml Buffer #1) were layered over the chromosomes. Coverslips and an aluminum foil-covered container were used as before to minimize evaporation. The slides were incubated at room temperature for two hours. The coverslip was removed and the slide was washed twice with BRL Buffer #1 for 10 minutes at room temperature and twice with BRL Buffer #3 [0.1 M Tris-HCL (pH 9.5), 0.1 M NaCl, 50 mM $MgCl_2$] for 10 minutes at room temperature. The slide was allowed to drain but not to dry.

A few drops (about 50-200 mcl) of BRL dye solution (3.3 mcl NBT, 2.5 mcl BCIP, 0.75 ml Buffer #3) were placed on the slide under a coverslip and incubated for 1-4 hours at room temperature in the dark. Staining was occasionally monitored with the phase microscope. The stained band has sometimes become visible in as few as 15 minutes but more time may be required. When the staining was adequate as determined by observation with the microscope, the reaction was terminated by rinsing the slide extensively in distilled water. Extensive rinsing was needed to remove salt crystals which interfered with microscopy.

Slides were air-dried and stored dry without coverslips. For microscopic examination, re-hydration with distilled water under a coverslip provided excellent resolution for viewing and photographing with phase contrast.

The stained, hybrid bands did not fade over a period of 4-6 months when slides were stored in the dark. We routinely photographed chromosomes on color slide film through a phase microscope without filters. This procedure gave excellent resolution and provided a permanent record of the color contrast. Good results were also obtained with black-and-white film.

False-positive stained areas may occur at the broken ends of broken chromosomes and in folds where a chromosome is kinked or where two chromosomes cross each other. False positives in folds are usually much narrower and sharper looking than stained areas over hybridization sites.

V. Conclusion. While the above procedure works well in our laboratory, we recognize that it is almost certainly not optimal. There are several areas of uncertainty that should be mentioned: (1) In order to achieve hybridization on a chromosome which still has easily resolved chromomeres, there needs to be a balance between sufficient and excessive denaturation. Too little denaturation prevents DNA hybridization, but excessive denaturation causes the chromomeres to fade. This elusive "balance" is not the same for all species. (2) The details of the squashing technique, primarily timing and squashing pressure, are not the same for all species. (3) The percent homology between probe DNA and chromosomal DNA necessary to allow detectable hybridization is unclear. We have found that **D.melanogaster** coding sequences hybridize well to **D.virilis** and **D.silvestris** chromosomes, but non-coding sequences hybridize very poorly or not at all. However, we have not yet used a large number of probes. (4) We generally obtain hybridization on every slide, but we seldom see hybridization to all of the appropriate chromosomes on a single slide.

Notes added in proof: 1. We now add the carrier DNA to the labeled DNA immediatley after we stop the nick translation, before chromatography. 2. We have hybridized in the presence of dextran sulfate of two sizes (5,000 and 500,000 daltons) and both worked equally well. The smaller size is easier to work with because it is less viscous in solution. 3. We seem to get better hybridization if we lower the temperatures given above by about 2 to 5°C.

References: Atherton, D. & J. Gall 1972, DIS 49:131-133; Barbera, E., M.J. Caliani, M. Pages & C. Alonso 1979, Exptl. Cell Res. 119:151-162; Denhardt, D.T. 1966, Biochem. Biophys. Res. Com. 23:641-646; Gall, J.G. & M.L. Pardue 1971, in: L. Grossman & K. Moldave (eds.), Nucleic Acids, Part D, V.21:470-480.

Rabinow, L. and J. Birchler. Harvard University, Cambridge, Massachusetts USNA. Reciprocal translocations between balancers of the second and third chromosomes carrying a dominant temperature sensitive lethal and larval markers.

For a variety of reasons, we wished to construct a chromosome that would simultaneously balance both of the major autosomes as well as carry a dominant temperature sensitive (DTS) locus that would allow the investigator to eliminate individuals with this chromosome from the progeny of crosses involving it. It was also desired that this balancer would have at least two larval markers that could be readily scored without affecting the gross morphology. The simultaneous balancing of the major autosomes and subsequent elimination of the balancer in the second generation should be of general use in making homozygous recessive mutations or transformants.

The procedure used in the construction was as follows. The second chromosome balancer, CyO, carrying 1(2)513 DTS was crossed to a stock carrying the third chromosome balancer TM3, red e Ser. Males from the progeny of the above cross that had the Cy as well as the Ser markers were irradiated with 3000 rads and mated en masse to females of an Oregon-R stock. After three days the males were removed. Sons of this mating that had both the Ser and Cy markers were again mated to Oregon-R females. Of the 1012 pair matings, four gave progeny in which the two dominant markers invariably segregated together. These were the presumptive translocations between the two balancers.

One of these, designated #11, has been used to balance a variety of second and third chromosome lethals. Our experience with it to date suggests that it is an adequate balancer for mutants on either chromosome two or three. The 1(2) 513 DTS is reliably lethal at 29°C during larval and pupal stages, killing all progeny carrying the mutation. Adults are not affected, which permits crossing and laying at the restrictive temperature. Available upon request.

Redkar, V.D. Tata Institute of Fundamental Research, Bombay, India. A population cage for growing Drosophila.

We describe a new population cage designed to grow **Drosophila melanogaster** flies on a large scale. This cage can also be used to obtain embryos, larvae or pupae in large numbers. 20 to 30 gm of flies were obtained from this cage using about 500 ml of medium supplemented with glucose, sucrose and yeast tablet powder.

The cage consists of a rectangular box (27.5 cm x 23.0 cm x 28.0 cm), made of plexiglass glued with chloroform, with a plexiglass lid screwed on the flanged top, with a foam gasket (Fig. 1 to 4). The lid has windows fitted with fine nylon mesh for ventilation. Inside the cage there is an assembly of eight vertical plexiglass plates (pupation plates), held rigidly at its center by one end of a plexiglass screw with nuts N_1 and N_2 (Fig. 2 and 4). The other end of the screw passes through a smooth hole in the center of the lid and is held by a plexiglass nut N_3 from the top. By turning this nut N_3 the pupation plate assembly can be lowered or raised by about 2 cm guided by two plexiglass guide pins (Fig. 2 and 4). At the bottom of the cage a plexiglass tray (22.5 cm x 29.0 cm x 2.5 cm) slides smoothly inside the cage with a clearance of about 1 mm such that one such tray can be displaced and pushed out from one side of the cage by another similar tray into the cage from the other side.

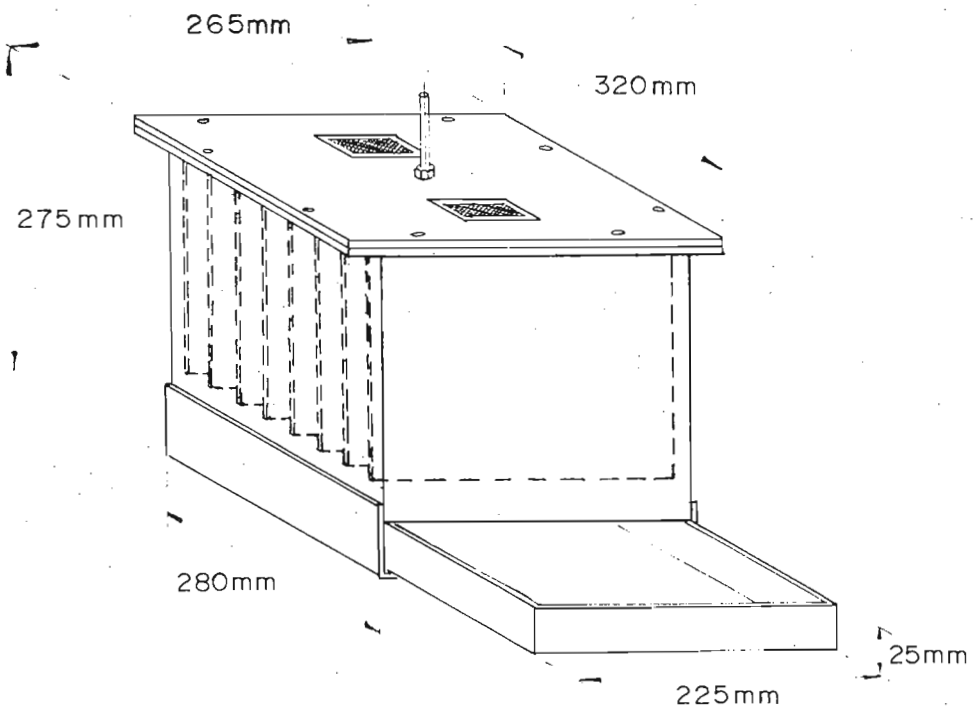

Figure 1. Perspective view of the population cage.

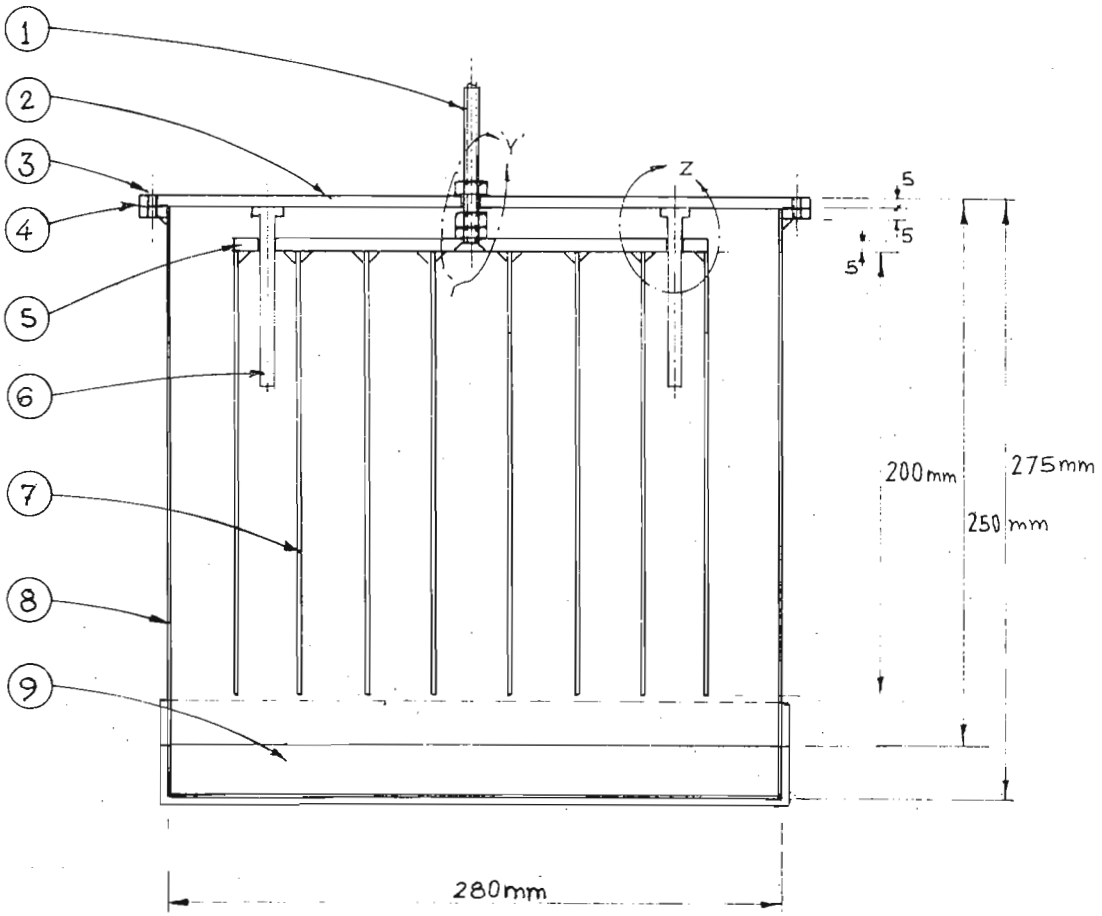

Figure 2. Sectional front view of the cage. (1) plexiglass screw 0.9 cm OD x 5 cm, that carries the pupation plate assembly. (2) Top lid of 0.5 cm plexiglass sheet. (3) Plexiglass screw, 0.5 cm OD x 2 cm, secures lid to the flanges. (4) Foam gasket 0.3 cm thick. (5) Plexiglass top plate of pupation plate assembly to which pupation plates are glued with chloroform and plexiglass strips. (6) Plexiglass guide pins, 0.5 cm x 5.0 cm. (7) A vertical plate in pupation plate assembly. (8) Outer wall of the cage of 0.3 cm plexiglass sheet. (9) Plexiglass tray.

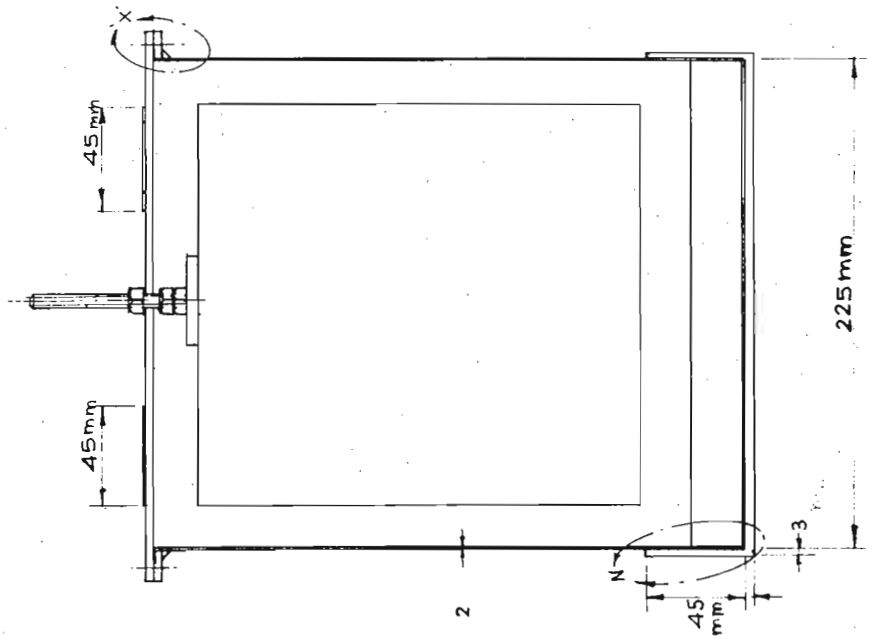

Figure 3. Sectional side view of the cage.

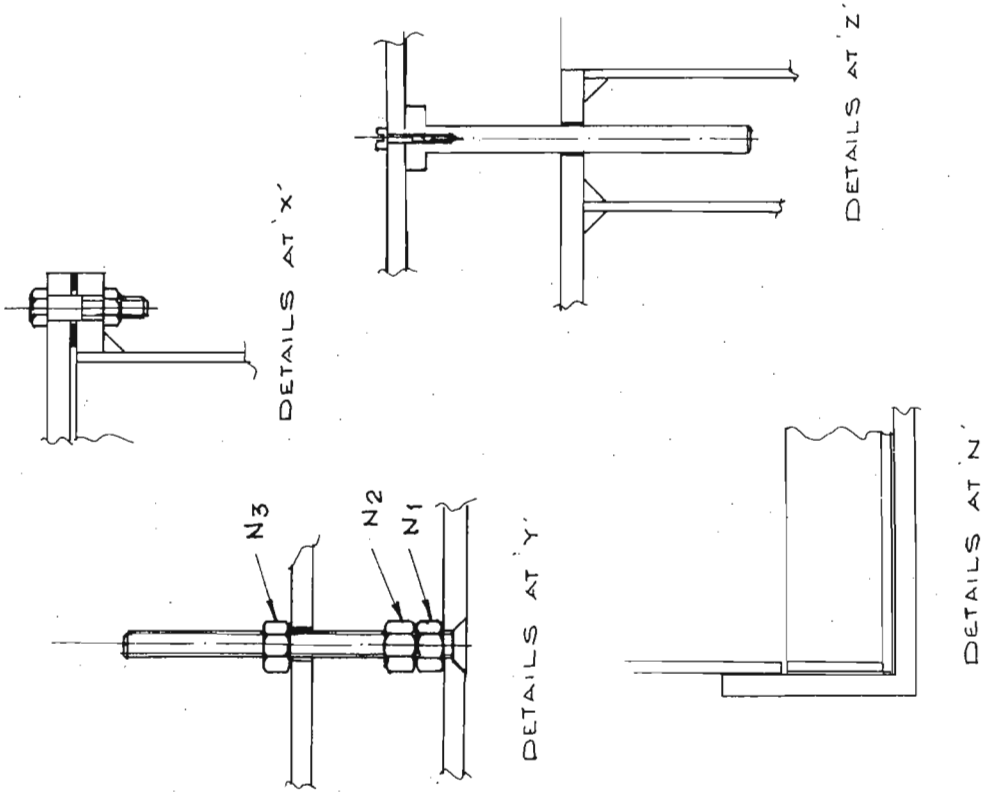

Figure 4. Shows details indicated in Figs. 2 and 3.

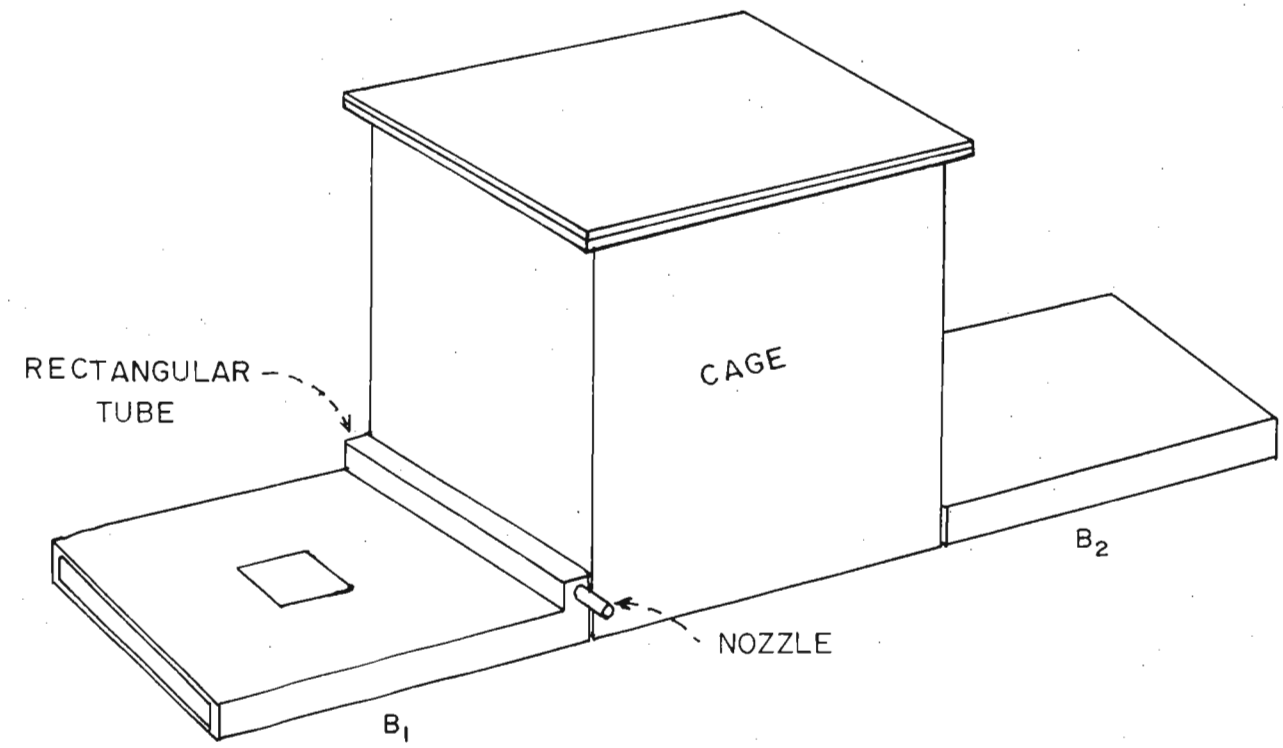

Figure 5. Transfer boxes, in perspective view, shown attached to the cage.

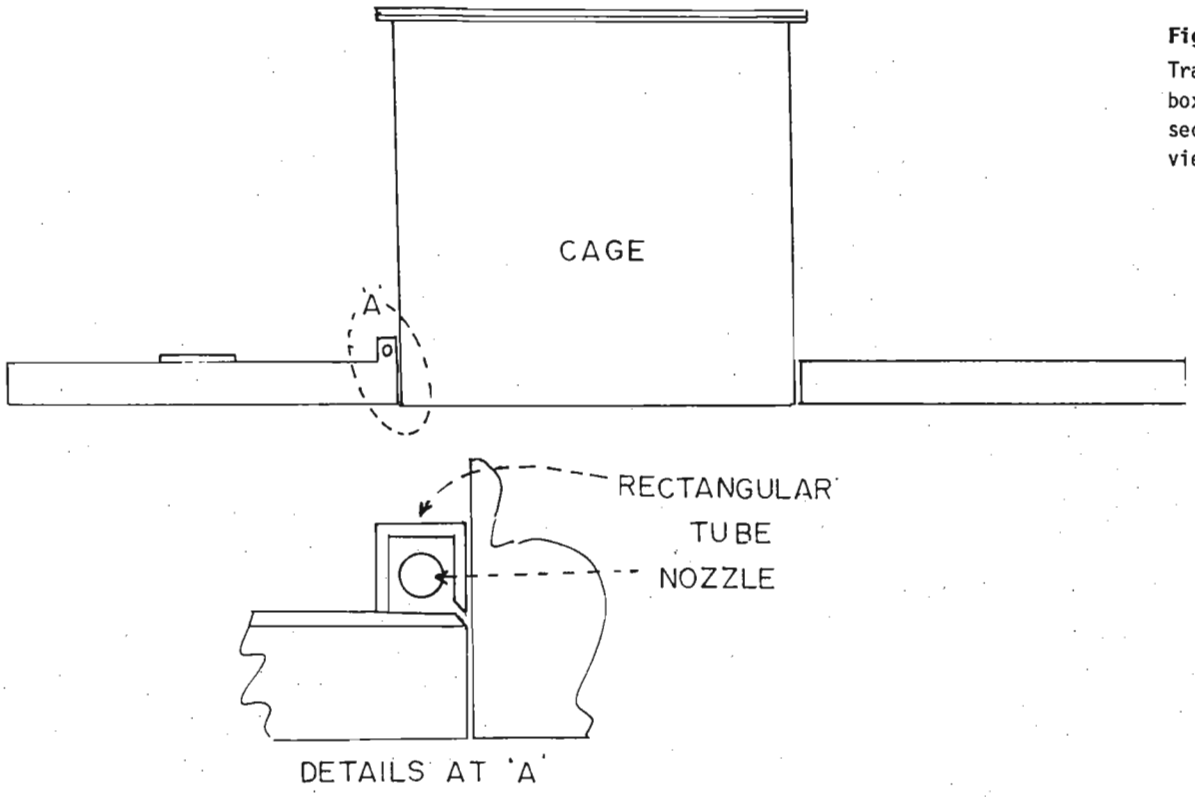

Figure 6. Transfer boxes, in sectional view.

Operation: The lid is fitted on the cage with the pupation plate assembly in raised position. 5 to 10 gm of ether-anaesthetised flies collected from bottle cultures are placed in a tray and the tray is introduced in the cage. The small gap between the tray and the cage is closed by cellophane-tape. Meanwhile another tray with normal agar-based Drosophila medium filled to about 5 to 7 mm below the top edge of the tray is prepared. After the medium cools and solidifies, 2 to 3 mm deep scratches, about 2 cm apart and parallel to the length and width of the tray are made on the surface. The surface of the medium is then made uniformly wet by 10 to 20 ml of an autoclaved solution containing 20% glucose and 10% sucrose. Finely ground yeast tablet powder is placed on the surface of the medium in small patches of about 0.5 cm in diameter with a clear space of about 2 cm in between. The yeast tablet powder patches should become wet; if not, some more sucrose-glucose solution is added. After this, half a gram of yeast granules (Baker's yeast) are sprinkled uniformly all over the surface. After the flies revive, the medium tray thus prepared is introduced in the cage by simultaneously pushing out the empty tray that was used to introduce the anaesthetised flies. To prevent flies from escaping while transferring the plates, two small flat boxes of plexiglass (see Fig. 5 and 6) are attached tightly to the two lower openings of the cage by cellophane-tape. Each of the two boxes is open from two sides and has inner dimensions same as a tray with a clearance of about 1 mm such that a tray slides smoothly through them. The left hand side box B1 has a small window in the centre at the top fitted with nylon mesh and a closed rectangular tube of plexiglass with a long slot of about 2 mm. In the righthand side box B2 the tray to be pushed inside the cage is inserted and then further pushed inside the cage by means of a wooden plate or another tray. While the tray inside the cage is being pushed out into the box B1, compressed air is forced in the rectangular plexiglass tube through the nozzle, connected by a tygon tube. This arrangement forces the flies sitting on the medium to fly back into the cage. After introducing the medium tray in the cage, the two boxes B1 and B2 are separated. The small gap between the tray and the cage is again sealed with cellophane tape. Any escaped flies in box B1 are ether-anaesthetised through the window and discarded. Eggs are collected on this medium for 24 hr with alternating 12 hr dark and light periods at 26°C. Using the transfer boxes B1 and B2 the egg-laden tray is removed and replaced by an empty tray. After introducing empty tray and sealing with cellophane tape, two tissue paper wads soaked in 1 to 2 ml of anaesthetic ether are placed on the two ventilation windows of the cage and covered with petridishes. After 15-20 minutes anaesthetised flies that drop on the tray are collected.

The medium on which eggs are laid is made moist by sprinkling some drops of the glucose-sucrose solution and this egg-laden tray is then introduced in cleaned, alcohol swabbed dry cage. This medium with

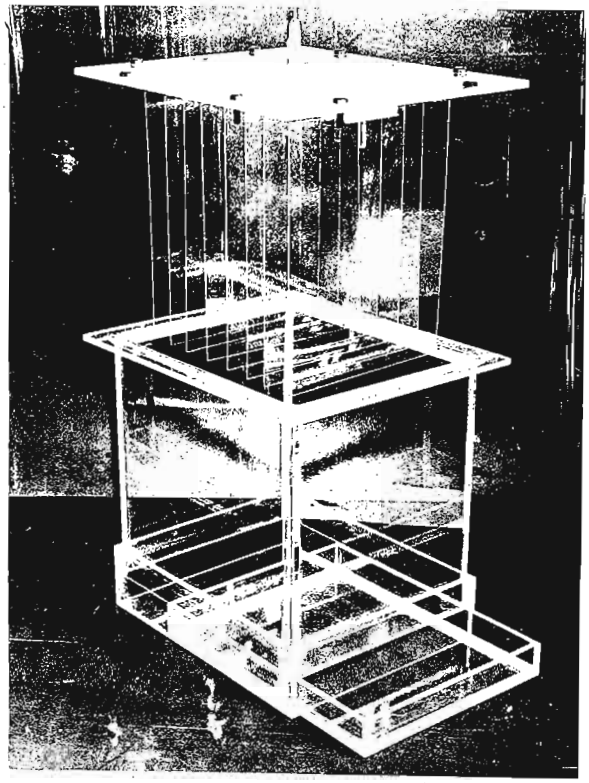

Fig. 7. Blow-up photograph of the mass culture unit.

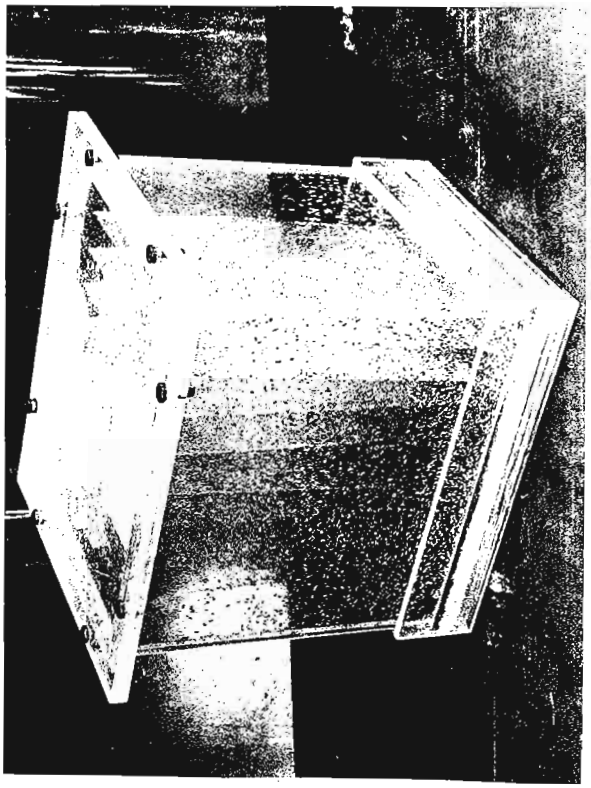

Figure 8. A photograph of the mass culture unit in operation showing flies and pupae inside the cage.

eggs is kept moist for two days by sprinkling 5-6 ml glucose-sucrose solution twice a day by sliding the medium tray out of the cage. On the third day when larvae appear to grow bigger, a little more glucose-sucrose solution is added to make the surface wet and fine powder of yeast tablets is sprinkled all over the surface in a thin uniform layer. At this stage the pupation plate assembly is lowered to touch the medium surface. This operation of adding sucrose-glucose solution and yeast tablet powder on the surface is continued for 5 to 7 days till all larvae pupate on the side walls of the cage and on the pupation plates. Every time glucose-sucrose solution and yeast powder have to be added, one has to raise the pupation plate assembly up and slide out the tray.

After the sixth or seventh day when no larvae are seen in the medium and before the flies start emerging from pupae, the bottom tray is removed , the lower part of the cage and pupation plate is washed and cleaned without disturbing the pupae, and a fresh medium tray is introduced every 24 hr. When all the flies have emerged and matured, the next batch of eggs is collected. Collection of flies is made in the manner described above by ether anaesthesia using the transfer boxes B1 and B2.

Comments: The cage is entirely made of plexiglass. All the morphogenic stages of the fly can be grown in one single unit of the cage. After initiation of the cage with 5 to 10 gm of flies from bottle cultures, the subsequent generations yield more flies. The humidity in the cage is maintained by the water-vapour evaporated from the medium. The flies lay eggs preferentially on the plane surface of the medium rather than on yeast powder. The hatched larvae grow well on the nutrient yeast powder with sugars, as seen by the crowding of larvae on yeast patches. Optimization of each of the operations and other factors such as size of the yeast tablet powder, moisture in the medium, humidity in the cage, feeding schedule of larvae, degree of crowding of flies, etc., can be achieved by experience.

Acknowledgements: The author is grateful to Prof. O. Siddiqi, Prof. U.W. Kenkare and Dr. K.S. Krishnan for useful suggestions and guidance.

Roberts, P.A. and D.G. Bedo.* Oregon State University, Corvallis, Oregon USNA. *C.S.I.R.O., Canberra, Australia. Improved Geimsa staining of in situ slides.

Geimsa staining of emulsion coated in situ slides often results in a precipitate from the stain solution appearing as dark spots similar to high background labelling. Although it can be distinguished from real silver grains, the precipitate impedes scoring of slides and prevents good photography. Filtering the Geimsa solution before use can help to prevent this problem, but we have found an easy and more convenient method to clean up slides after they are stained and dried. This is to immerse slides affected by deposits in 100% methanol for 1-2 minutes, then to air dry the slides. This treatment clears background deposits with no noticeable reduction in chromosome staining. If some deposits remain, the slide can be retreated with methanol but prolonged treatment can destain the chromosomes. Ethanol is not suitable as the deposits are not removed quickly and the chromosomes are subject to destaining.

Tsuno, K. Josai Dental University, Sakado, Saitama, Japan. A new apparatus for horizontal electrophoresis.

In order to facilitate the analysis of mutation experiments involving isoenzymes, I improved the apparatus used in a horizontal thin-layer agar gel electrophoresis method previously described (Sasaki 1974, Tsuno 1981). For the purpose of shortening the time for both the transference of homogenates onto a gel and penetration of sample juice into gels, the apparatus was modified by using sampling combs (spatula for sampling the juice of flies).

The apparatus consists of three parts: homogenate plates, sampling combs, and an insertion gauge. (a) Sampling comb (made from a 0.8 mm thick acrylic sheet): as seen in Figure 1, the comb has 10 teeth placed in register with one row of holes on the homogenate plate. The tips of the teeth are roughened with sandpaper to increase the amount of sample solution that can be taken. (b) Homogenate plate (made from an 8 mm thick acrylic plate): as shown in Fig. 1, the plate has 50 holes (10 holes x 5 rows), spaced at intervals of 13 mm, each of which is 5 mm in diameter, 3 mm in depth, and has a flat bottom. (c) Insertion gauge (made from 5 mm thick polyvinyl chloride plates): it consists of two plates (400 x 200 mm and 350 x 150 mm) connected by a firm hinge, one plate being bent at the edge where a scaled guide plate is fixed so that the sampling comb is correctly positioned, as seen in Figure 2. In the resting position, the gel is protected by a stopper pad from being damaged by the upper plate. Both attachment and detachment of the sampling comb and positioning of the stopper pad are done by hand for each application.

The procedure for electrophoresis of **D.virilis** esterases is as follows: (1) A drop of water or buffer solution and one or more flies is put into each hole of the homogenate plate. Then, the samples are homogenized by a motor-driven glass rod 4.8 mm in diameter. (2) The sampling comb is then dipped into the homogenates in order to load each of the ten tips. (3) Next, the comb is transferred and fitted to the scaled guide plate of the insertion gauge, and the sample-laden comb teeth are then inserted two or three times into a 0.8 mm thick thin-layer agar gel prepared previously on a glass plate (180 x 165 mm). [Agar

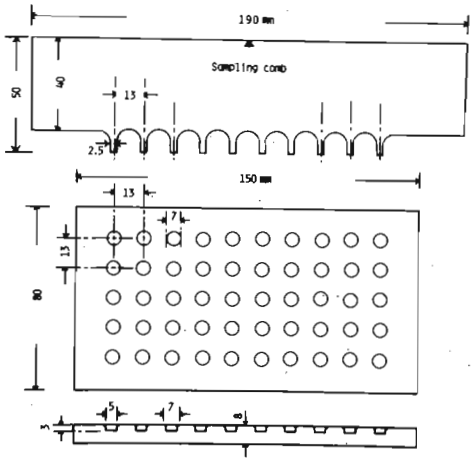

Figure 1. Sampling comb and homogenate plate.

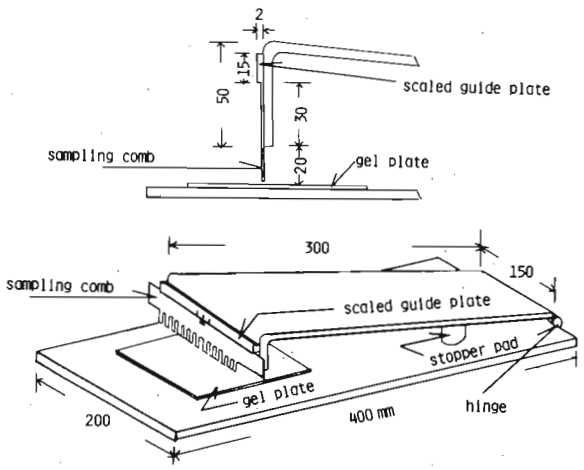

Figure 2. Insertion gauge prepared for delivery of sample into gel plate.

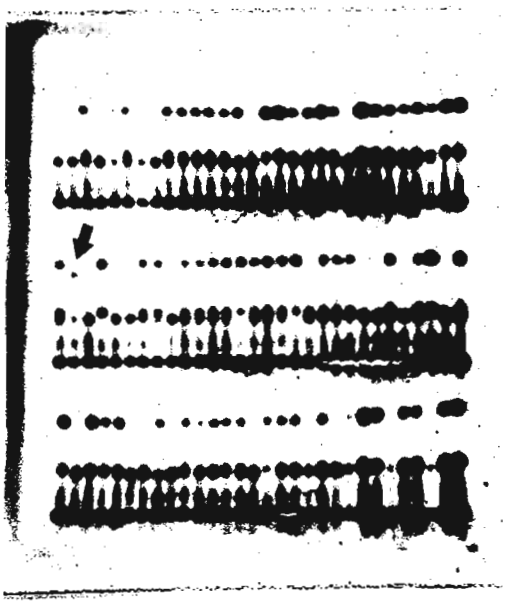

Figure 3. Examples of electrophoresis (α- and β-esterases of **D.virilis**).

gel: 0.7 gr agar purified + 2.0 gr polyvinylpyrolidone K-90 + 45 ml electrode buffer solution + 55 ml H_2O.] When the volume of sample solution on the teeth seems inadequate, steps (2) and (3) are repeated. Thus, ten different sample solutions can be applied at the same time to a gel plate. The next new application of samples, if necessary, may be fitted between the previous ones at a different scale position on the guide plate. By this procedure, one can insert a maximum of 30 kinds of sample solutions into a gel plate. Moreover, as a set of 30 samples or lanes, one can load three (or more) sets at different places on the same gel plate. So, 90 (or more) lanes can be electrophoresed at a time. (4) Then the gel plate is electrophoresed immediately to avoid diffusion of sample into the gel. The buffer system used in the electrode vessel is a discontinuous, potassium phosphate buffer [Electrode buffer: 100 ml 0.3M KH_2PO_4 + 100 ml 0.3M K_2HPO_4 + 2,200 ml H_2O]. A constant voltage of 16.5V/cm is applied for 90~120 min, and the procedure is carried out in a humid box kept at 3~5°C. After electrophoresis, the gel is treated with an appropriate substrate solution and staining solution as reported previously (Tsuno 1981).

Because of the possible necessity of further analysis, used homogenate plates having residual sample solution should be kept in a deep freezer to prevent drying until the electrophoretic results become clear. Sampling combs can be used repeatedly after a wash, but they should be dried vertically so as not to become bent.

Using this apparatus, I have carried out mutation experiments and examined about 1.5×10^6 alleles of **D.virilis** α-esterase (Tsuno 1985). An example of electrophoregrams are shown in Figure 3. In Fig. 3, in order to accelerate the analysis of the experiments, a drop of sample solution containing homogenate of two flies was applied to one lane, so 2 (flies) x 10 (teeth) x 3 (times of insertion) x 3 (sets) = 180 (samples) were examined on a single gel plate at a time. In this case, a mutant was found as indicated by the arrow on the gel.

So far, I have succeeded in electrophoresing isozymes of the following enzymes using this apparatus: α-esterase, β-esterase, acid phosphatase, alcohol dehydrogenase, α-glycerophosphate dehydrogenase in **D.virilis**, and some of the same ones in **D.simulans** and **D.melanogaster.** Moreover, a polyacrylamide gel instead of an agar one may be used in the test, and good results have been obtained with esterases of **D.virilis.** However, the agar gel method is better than the polyacrylamide gel one, because the preparation of agar gels is easier compared with that of polyacrylamide gels. Particularly, agar gels poured from agar boiled in an autoclave at 120°C and 1.2 kg/cm^2 for 25 min gave good results.

References: Sasaki, F. 1974, Jpn. J. Genet. 49:223-232; Tsuno,K. 1981, Jpn. J. Genet. 56:155-174; Tsuno, K. 1985, Jpn. J. Genet. 60:103-118.

Yamamoto, A.H. N.I.E.H.S., Research Triangle Park, North Carolina USNA. New protocol to prepare probes labelled by biotinylated dUTP for in situ hybridization of polytene chromosomes.

Biotinylated dUTPs have been used to label probe DNA because of its long life and the safety relative to radioisotopes. However, I have experienced difficulty in getting probes sufficiently labelled with biotinylated dUTP. According to the usual nick translation method for ^{3}H-dUTP or using nick translation kits supplied by several companies, incorporation rates of biotinylated-11 dUTP or biotinylated-16 dUTP measured by simultaneous incorporation of ^{3}H-dUTP was less than 5% and those probes did not work. Now I recommend to separate the reaction of DNase I from that of polymerase I, as follows. For two slides:

1st reaction:	10 x nick translation buffer (Maniatis et al. 1982)	1.0 µl
	DNA	0.5 µl
	DNase I (fresh 1:80 dil. of 0.1 mg/ml stock solution)	1.0 µl
	distilled H_2O	up to 10.0 µl

Incubate at room temperature for 15 to 60 min (depending on quality of DNA) and heat at 65°C for 15 min.

2nd reaction:	mixture of 1st reaction	10.0 µl
	10 x nick translation buffer	1.0 µl
	2 mM spermidine	1.0 µl
	dATP, dGTP, dCTP mix (0.3 mM each)	2.0 µl
	Bio-16-dUTP (0.3 mM, from Enzo Biochem)	5.0 µl
	^{3}H-dATP (as a tracer)	0.5 µl
	polymerase I	2.0 µl

Incubate at 14°C for 90 min. Add 2 µl of 0.5 M EDTA, and heat at 65°C for 15 min. Separate DNA from unincorporated dNTPs. Check incorporation rate (>10% is good enough).

The advantages of this protocol are not only being able to get probes of high quality but also being applicable to a range of DNA quality which may vary among batches, among methods for preparation or among persons who prepare them, by controlling the period of 1st reaction. Basically better quality DNA needs a shorter period for the 1st reaction.

References: Maniatis, T., E.F. Fritsch & J. Sambrook 1982, Molecular cloning, Cold Spring Harbor Laboratory, Cold Spring Harbor, New York.

SUBMITTED STOCK LISTS - all species

BERHAMPUR UNIVERSITY. Cellular Research Laboratory, Dept. of Zoology, Bhanja Bihar, Berhampur -7, Orissa, India.

Wild-type stock

1. **D.melanogaster** Oregon R$^+$
2. -do- 2
3. -do- local type
4. **D.ananassae**
5. **D.hydei**
6. **D.nasuta**
7. **D.virilis**

Mutant stocks

Chromosome 1

1. gt w^a
2. Ba sc (Muller - 5)
3. y w sn^3
4. y w f
5. Df(1)62 g^{18}
6. y w v g f l. e

Chromosome 2

1. vg
2. Cy

Chromosome 3

1. e

Multiple Chromosomes

1. $\frac{fs(1)\ K_{10}\ w}{C1B}$; $\frac{mwh\ se}{mwh\ se}$
2. $\frac{w^{co}}{w^{co}}$ $\frac{flr^3\ se}{TM_2Ubx\ se}$

HANYANG UNIVERSITY. School of Medicine, Dept. of Genetics, Seoul 133, Korea.

Wild stocks - **D.melanogaster**

Canton S
Oregon R
Seoul (Korea)

Chromosome 1

f
m
sc cv v f
v
w
w m f

Chromosome 2

al dp b pr ap blt bw/SM5
b
b vg
bw
Bl/In(2L + 2R)Cy, Cy bw^{45a} sp^2 or^{45a}
bw
cn bw
dp cn bw
vg
vg bw

Chromosome 3

cu
e^s
g^1
ru h ta st cu sr e^s ca/TM3 ru Sb Ser
Sb/In(3LR) Ubx130, Ubx130 e^s

Chromosome 4

ci sy^n
gvl

Chromosome 2-3

T(2: 3) Pm; Sb/Cy, Ins; Ubx, Ins

MAHARASHTRA ASSN. FOR CULTIVATION OF SCIENCE. Dept. of Zoology, Pune 411 004, India.

Mutant stocks of **D.melanogaster**

Df(1)w, y^2 sc z w spl
sc z $w^{+(TE)}$ sn (zeste unstable)
sc z $w^{+(TE)}$ sn (red unstable)
C(1)DX, y w f / Y / sc z $w^{+(TE)}$ f (zeste)
C(1)DX, y f / Y / sc z $w^{+(TE)}$ mei-9^a (zeste, excision-repair deficient)

UNIVERSITÄT MÜNSTER. Institut für Strahlenbiologie, Hittorfstr. 17, D-4400 Münster, FR Germany.

D.melanogaster:
1. + (Berlin wild)
2. B
3. y
4. w
5. e^{11}
6. y sc^{S1} In49 sc^8; bw; st p^p
7. y f:= & y sc^{S1} B In49 sc^8 ("Binscy")
8. y & y/y^+Y
9. y f;=/y^+Y & y/y^+Y
10. $y/y/y^+Y$ & y/y^+Y ("Trisom")
11. C(2L)RM, b; C(2R)RM, vg

UNIVERSITY OF TEACHER EDUCATION, Genetics Laboratory, Dept. of Biology, Tehran, 15614, Iran.

D.melanogaster:

Wild type
Amherst

Chromosome 1
Basc
C(1)RM, y w / $XY^L.Y^S$, y w f
dm sn / FM_3 / $In(1)w^{m4L}$ $N^{264-84R}$, y w^{m4} N^{264-74} sn
$In(1)w^{m4}$ w^h
w y v
w^a y w ct^6 m f

Chromosome 2
B1 L^2 / SM_5
bw
vg

Chromosome 3
e
se
se e
st

Multiple Chromosomes
bw ; st
In(2L)Cy, Cy / $In(2LR)bw^{V1}$, ds^{33k} bw^{V1}; In(3L)D, D / Sb

LINKAGE DATA

Report of J.S. Peterson and W.H. Petri.
Dept. of Biology, Boston College, Chestnut Hill, Massachusetts 02167 USNA.

Update of linkage information on the s70 chorion gene of **D.melanogaster.**

The s70 chorion protein gene was intially reported to be located within the A94 deletion (1E3-4;2B11-12) on the first chromosome. Using more detailed deletion mapping and the Staket strain which carries an electrophoretic variant of the protein, we have now shown the s70 gene to be within the S39 deletion (1E1-2;2B5-6) and outside the Sta deletion (1E1-2;2B3-4). This indicates that the S70 gene resides within the region 2B3-4 to 2B5-6.

References: Yannoni, C.Z. & W.H. Petri 1984, Dev. Biol. 102:504-508; Belyaeva, E.S. et al. 1982, DIS 58:184.

Report of I.D. Alexandrov and M.V. Alexandrova. Research Inst. of Medical Radiology, Obninsk, USSR.

Genetics and cytogenetics of the black mutations induced by gamma-rays, ^{252}Cf and fission neutrons.

The following list is a part of our general stocklist described in DIS 61 and contains information on the 117 out of 142 transmissible black mutations which were scored in various experiments designed for estimating the relative proportion of intra- versus inter-genic changes at the locus of interest after action of the low- (mainly gamma-rays of ^{60}Co) or high- (^{252}Cf, fission neutrons) LET radiation on the different post-meiotic germ cells of the wild type or c(3)G males (see the last column list) pre-treated with radiomodifiers used (the sixth column). All other conditions of the experiments have been described elsewhere (Alexandrov et al. 1985; Alexandrov, Research Note, this issue). The black mutations were named (first column) by the accepted alphanumeric code. The second, third, fourth and fifth columns are respectively giving information on the phenotype, pattern of complementation with both $su(b)^{31}$ and $su(b)^{18}$ as well as cytology for each mutation.

As seen, 61 transmissible and viable in homozygote black mutations as well as 24 non-viable ones, but with separable lethal phenotype, i.e., so-called "twin" black mutants (Alexandrov, ibid.), have a normal 34D region of 2L chromosome (the putative location of the gene in question) and can be accepted as true gene mutations, VVg, which found to be suppressed by both $su(b)^{31}$ and $su(b)^{18}$, whereas 31 out of 117 mutations preserved proved to be associated with either chromosome alterations among which deficiencies shown to be predominant chromosome changes (20 out of 31 rearrangements studied) after irradiation of any genotype by neutrons and of c(3)G genotype by gamma-rays. On the contrary, in the wild-type genome gamma-photons induce predominantly exchange-type rearrangements. Also, the "twin" black mutants found to arise more frequent (18 out of 24) after action of photons than after neutron irradiation. The data of genetic mapping of the chromosome rearrangement breakpoints listed are reported elsewhere (Alexandrova, this issue).

Acknowledgement: We are grateful to Dr. Al. Sherald, George Mason University, Virginia USNA, for supplying the $su(b)^{31}$ and $su(b)^{18}$ stocks.

References: Alexandrov, I.D., M.A. Ankina & M.V. Alexandova 1985, DIS 61:212-213; Lindsley, D.L. & E.H. Grell 1968, Carnegie Inst. Wash. Publ. 627.

1 Designation of mutation	2 Phenotype	3 Suppressed by su^{31}	3 Suppressed by su^{18}	4 Cytology	5 Modifier used, radiation dose	6 Genotype, germ cells irradiated
b^{661}	b*	+**	+	Normal	γ-rays, 40 Gy	D-32,MS***
b^{71k1}	lethal			Tp(2)34D2-4;34D8-E1,2;43C2-4	γ-rays, 40 Gy	D-18,MS
b^{71k2}	b	+	+	Normal	γ-rays, 40 Gy	D-18, L Sd
b^{74b2}	b	+	+	Normal	Caffeine + γ-rays, 40 Gy	D-32,M Sd
b^{74b4}	b	+	+	Normal	"	D-32,M Sd
b^{74b5}	b	+	+	Normal	"	D-32,M Sd
b^{74c2}	b	+	+	Normal	"	D-32,MS
b^{74c4}	b	+	+	Normal	"	D-32,MS
b^{74c5}	b	+	+	Normal	"	D-32,L Sd
b^{74c6}	Lethal			Df(2L)34D2-4;34D8-E1,2	"	D-32,MSd
b^{74d2}	b			Normal	"	D-32,L Sd
b^{74d4}	b	+		Normal	"	D-32,L Sd
b^{74d6}	b	+	+	Normal	"	D-32,M Sd
b^{75a}	b	+	+	Normal	γ-rays, 40 Gy	c(3)G,MS
b^{76b1}	b	+	+	Normal	Caffeine + γ-rays, 40 Gy	c(3)G,MS
b^{76b2}	b	+		Normal	"	c(3)G,MS
b^{76e1}	b	+	+	Normal	γ-rays, 40 Gy	c(3)G,MS
b^{76e2}	b	+	+	Normal	γ-rays, 40 Gy	c(3)G,MS
b^{76f3}	as b^{50d}*	+	+	Normal	γ-rays, 40 Gy	c(3)G,MS
b^{76j1}	b	+		Normal	Actinomycin-D + γ-rays, 40 Gy	D-32,MS
b^{76j2}	b	+	+	Normal	"	D-32,L Sd
b^{76j3}	b	+		Normal	"	D-32,M Sd
b^{76k1}	b	+	+	Normal	"	D-32,MS
b^{76k2}	b	+	+	Normal	"	D-32,L Sd
b^{77a1}	b	+	+	Normal	"	D-32,MS
b^{77a2}	b	+	+	Normal	"	D-32,MS
b^{77a3}	b	+	+	Normal	"	D-32,MS
b^{77a4}	b	+	+	Normal	"	D-32,MS
b^{77a5}	b	+	+	Normal	"	D-32,MS
b^{77c}	Lethal			Df(2L)34D2-4;34F4-35A1.2	"	c(3)G,MS

1	2	3		4	5	6
b^{77j}	b	+	+	Normal	"	c(3)G,MS
b^{78a}	b	+	+	Normal	γ-rays, 40 Gy	c(3)G,L Sd
b^{78f1}	b	+	+	Normal	NaF + -rays, 40 Gy	D-32,L Sd
b^{78f2}	b	+	+	Normal	"	D-32,M Sd
b^{78g}	b	+	+	Normal	"	D-32,E Sd
b^{78j}	Lethal			Df(2L)34D2-4;35A3-4	γ-rays, 40 Gy	c(3)G,E Sd
b^{78k1}	b	+	+	Normal	NaF + γ-rays, 40 Gy	D-32,MS
b^{78k2}	b	+	+	Normal	"	D-32,MS
b^{78k3}	b	+	+	Normal	"	D-32,M Sd
b^{78k5}	b	+	+	Normal	"	D-32,E Sd
b^{79a1}	b	+		Normal	0.85 MeV n,20 Gy	D-32,MS
b^{79a3}	Lethal			Df(2L)34D2-4;34D8-E1.2	"	D-32,MS
b^{79a4}	b	+	+	Normal	0.35 MeV n,20 Gy	D-32,MS
b^{79b1}	b	+	+	Normal	γ-rays, 40 Gy	D-32,MS
b^{79b3}	Lethal			Df(2L)34C7-D2;35A4$^{\pm}$	0.85 MeV n, 10 Gy	D-32,MS
b^{79b4}	Lethal			Df(2L)34D2-4;34E6-F1,2	"	D-32,MS
b^{79b7}	$b^{@}$	+	+	Normal	0.35 MeV n, 10 Gy	D-32,MS
b^{79b8}	Lethal			Df(2L)34D2-4;34F4-35A1,2	"	D-32,MS
b^{79d2}	$b^{@}$	+	+	Normal	γ-rays, 20 Gy	D-32,MS
b^{79d4}	b	+	+	Normal	0.85 MeV n, 10 Gy	D-32,MS
b^{79d5}	Lethal			In(2L)34D4$^{\pm}$;35B10$^{\pm}$	"	D-32,MS
b^{79d6}	Lethal			T(2;3)34A2-3;34D8-E1.2;79B;80C	0.85 MeV n + γ-rays, 10 + 10 Gy	D-32,MS
b^{79d8}	$b^{@}$ as b^{50d}	+	+	Normal	"	D-32,MS
b^{79d10}	b	+	+	Normal	"	D-32,MS
b^{79d11}	$b^{@}$	+	+	Normal	0.35 MeV n, 10 Gy	D-32,MS
b^{79d13}	b	+	+	Normal	γ-rays, 40 Gy	c(3)G,MS
b^{79df2}	b	+	+	Normal	"	c(3)G,MS
b^{79g2}	b	+	+	Normal	"	c(3)G,MS
b^{79h1}	Lethal			Tp(2L)34D2-4;34D8-E1.2;41	"	D-32,M Sd
b^{79h2}	b	+	+	Normal	"	c(3)G,M Sd
b^{79h3}	$b^{@}$		+	Normal	"	c(3)G,MS
b^{80k}	Lethal			Df(2L)34D2-4;35B10-C1	"	c(3)G,L Sd
b^{80l}	Lethal			Df(2L)34D2-4;34E1.2-E4.5	0.7 MeV n, 12 Gy	D-32,MS
b^{81a}	Lethal			Tp(2)34D2-4;34D8-#1.2;41D-E1	γ-rays, 60 Gy	D-32,MS
b^{81a2}	$b^{@}$	+	+	Normal	γ-rays, 60 Gy	D-32,MS
b^{81c}	b	+	+	Normal	0.7 MeV n, 20 Gy	D-32,MS
b^{81c2}	b	+	+	Normal	γ-rays, 40 Gy	D-32,M Sd
b^{81c17}	b	+	+	Normal	"	D-32,MS
b^{81f1}	Lethal			Df(2L)34D2-4;35A3-4	0.1 MeV n, 4 Gy	D-32,MS
b^{81f2}	Lethal			Df(2L)34D2-4;35B4-B5.6	0.1 MeV n, 8 Gy	D-32,MS
b^{81f3}	as b^{50d}	+	+	In(2L)34D2-4;35B10$^{\pm}$	0.1 MeV n, 15 Gy	D-32,MS
b^{81k}	$b^{@}$ as b^{50d}	+	+	Normal	γ-rays, 20 Gy	D-32,MS
b^{8117}	Lethal			In(2L)34D2-4;40F	"	D-32,MS
b^{81140}	b (sterile in homozygote)			Normal	γ-rays, 10 Gy	D-32,MS
b^{81142}	Lethal			Normal	"	D-32,MS
b^{82c3}	b	+	+	Normal	^{252}Cf, 14 Gy	D-32,MS
b^{82c7}	b	+	+	Normal	^{252}Cf, 7 Gy	D-32,L Sd
b^{82c16}	Lethal			Normal	^{252}Cf, 28 Gy	D-32,L Sd
b^{82c44}	$b^{@}$(sterile in homozygote)			In(2L)34D4$^{\pm}$;40F	^{252}Cf, 14 Gy	D-32,MS
b^{82c54}	$b^{@}$(sterile in homozygote)			Normal	^{252}Cf, 14 Gy	D-32,L Sd
b^{83b11}	$b^{@}$(sterile in homozygote)			Normal	1γ-rays, 40 Gy	D-32,MS
b^{83b2}	Lethal			In(2L)34D4$^{\pm}$;35B10$^{\pm}$	γ-rays, 40 Gy	D-32,MS
b^{83b40}	$b^{@}$	+	+	Normal	"	D-32,MS
b^{83c20}	b	+	+	Normal	"	D-32,L Sd
b^{83c25}	$b^{@}$	+	+	Normal	"	D-32,MS
b^{83c26}	$b^{@}$	+	+	Normal	"	D-32,MS
b^{83c35a}	b (sterile in homozygote)			Normal	"	D-32,MS
b^{83c35b}	$b^{@}$	+	+	Normal	"	D-32,M Sd
b^{83c36}	b	+	+	Normal	"	D-32,M Sd
b^{83c47}	b (sterile in homozygote)	+	+	Normal	"	D-32,M Sd

1	2	3	4	5	6
b^{83d29a}	Lethal		Df(2L)34D2-4;35E1.2-E5.6	^{252}Cf, 28 Gy	D-32,MS
b^{83d29b}	b	+ +	Normal	"	D-32,L Sd
b^{83d35}	b	+ +	Normal	^{252}Cf, 14 Gy	D-32,MS
b^{83d36}	$b^{@}$	+ +	Normal	^{252}Cf, 28 Gy	D-32,L Sd
b^{83f17}	$b^{@}$	+ +	Normal	γ-rays, 40 Gy	D-32,M Sd
b^{83f18}	b	+ +	Normal	"	D-32,L Sd
b^{83f51}	b	+ +	Normal	"	D-32,M Sd
b^{83f52}	$b^{@}$	+ +	Normal	"	D-32,M Sd
b^{82fXD}	b	+	Normal	X-rays, 40 Gy	c(3)G,L Sd
b^{831}	b	+ +	Normal	0.7 MeV n, 10 Gy	D-32,MS
b^{8311}	Lethal		Df(2L)34D2-4;34E1.2-E4.5	"	D-32,MS
b^{8312}	Lethal		Df(2L)34C2;34E4.5 + T(2;3) 34B12-C1;35A2-3;83A7-8;83C	0.7 MeV n, 10 Gy	D-32,MS
b^{84g}	b	+ +	Normal	X-rays, 40 Gy	c(3)G,L Sd
b^{84h14}	Lethal		Df(2L)34D2-4;34F4-35A1.2	X-rays, 20 Gy	c(3)G,MS
b^{84h34}	b	+ +	Normal	"	c(3)G,L Sd
b^{84h40}	$b^{@}$		Normal	"	c(3)G,MS
b^{84h50}	Lethal		Df(2L)34D2-4;35C1-C3	"	c(3)G,L Sd
b^{84h70}	$b^{@}$		Normal	γ-rays, 40 Gy	c(3)G,MS
b^{85b1}	Lethal		Df(2L)34D2-4;34E2-E4.5	γ-rays, 10 Gy	c(3)G,MS
b^{85b2}	Lethal		Df(2L)34D2-4;34D8-E1.2	γ-rays, 20 Gy	c(3)G,MS
b^{85b3}	b	+	Normal	γ-rays, 10 Gy	c(3)G,MS
b^{85b4}	b		Normal	"	c(3)G,MS
b^{85c1}	Lethal		Df(2L)34D2-4;34F4-35A1.2	0.85 MeV n, 10 Gy	c(3)G,MS
b^{85c2}	b (sterile in homozygote)		T(2;3)34C7-D1.2;34E1-2;95C4-D1	"	c(3)G,MS
b^{85c3}	b	+	Normal	0.85 MeV n, 5 Gy	c(3)G,MS
b^{85f1}	Lethal		Df(2L)34D2-4;34F3-4	γ-rays, 20 Gy	c(3)G,MS
b^{85f2}	Lethal		Df(2L)34D2-4;34E6-F1 + In(2L) 33A1-2;35E3-4	0.35 MeV n, 10 Gy	c(3)G,MS

* See Lindsley & Grell 1968; ** The existence of suppression, without the sign non-tested; *** MS - mature sperm, L, M, and E - late, middle and early spermatids (Sd), respectively; @ The "twin" black mutations with independent lethal separated by crossing over.

Report of F.C. Erk and A. Podraza.
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An extreme allele of vestigial in **D.melanogaster**: vg^{X}.

The vg^{X} allele was extracted from a screen for X-linked lethals after gamma ray irradiation, using a Cs-137 source, of a highly inbred line of Oregon-R. The wings of homozygotes invariably appear as very small, veinless clubs; the halteres are reduced to tiny knobs or, in 80% of flies, are absent entirely on one or both sides; the postscutellar bristles are erect, and usually directed anteriad. These features, common to some other vestigial alleles, are accompanied by additional defects in the legs, abdominal tergites, and notum. Half or all of the notum is missing in 10% of flies, a consequence of failure of the dorsal mesothoracic disk to evert; one or more legs may be severely bent, or truncated terminally with loss of tarsi, or both; and abdominal tergites may be deleted on one side, or severely misaligned (8.5%). The metathoracic legs are most often affected, with the tibia being bent posteriorly 90° or more; walking is difficult, and the defective leg trails uselessly. Females are more than twice as likely to show severe leg bending (45%) as males (20%); occasionally mesothoracic legs are affected.

Both males and females show good viability, but females are completely sterile: ovaries appear to be structurally normal, but no eggs are laid. The allele can be maintained in heterozygous condition, and fertile homozygous males are regularly produced. Although somewhat smaller and virtually wingless, males have been capable of mating with all females with which they have been tested. Many heterozygous combinations of vg^{X} show mild to severe wing notching or scalloping.

Report of T. Kjaer. Inst. of Genetics, University of Copenhagen, DK-1353 Copenhagen K, Denmark.

su(dp^{ov})TK-84: suppressor of dumpy$^{oblique\text{-}vortex}$ 1-proximal half of chromosome T. Kjaer 1984e8.

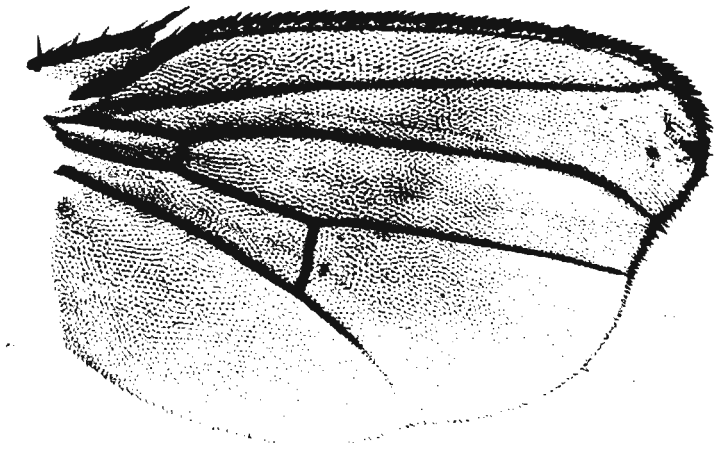

Figure 1. dp^{ov} wing phenotype: dp^{ov}/dp^{ov}.

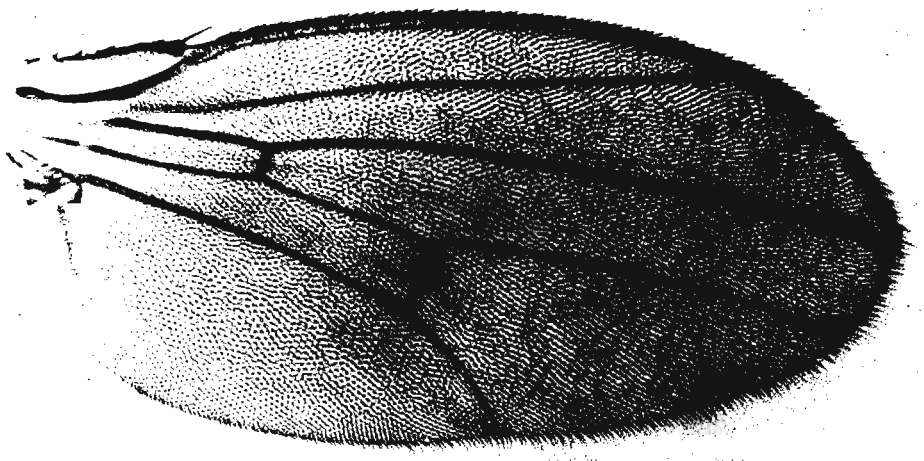

Figure 2. Suppressed wing phenotype: su(dp^{ov})$^{TK8\text{-}84}$;dp^{ov}.

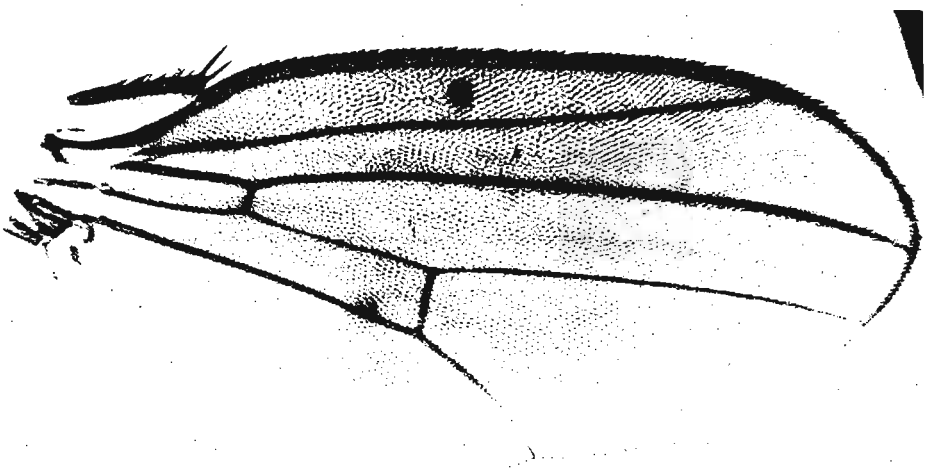

Figure 3. Wild type wing: +/+.

Three mutants, su(dp^{ov})TK8-84 su(dp^{ov})TK25-84 and su(dp)TK26-84 were induced by EMS. The suppressors were scored on dp^{ov} males (Fig. 1) with a normalized wing phenotype (Fig. 2 compared with Fig. 3). Additionally, it turned out that su(dp^{ov})TK8-84, but not su(dp^{ov})TK25-84 or su(dp^{ov})TK26-84, suppressed the vortex phenotype of dp^{ov}. However, none of the suppressors was able to suppress the vortex phenotype in a trans-configurated dp^{ov}/dp^{v2}. On the other hand, a trans-configurated dp^{ov}/dp^{o2} was totally suppressed at the wing phenotype. Of the three suppressors su(dp^{ov})TK26-84 has the lowest penetrance since only about 70% of the flies are totally suppressed, while the two other suppressors have approximately 100% penetrance. The three suppressors seem to be alleles, since none of them complement each other. Viability and fertility of both su(dp);dp^{ov} and su(dp);+ are good.

Report of S. Lenicek and D. Sesta.
C.U.O. "Braca Hanzek", Petrinja, Yugoslavia.

The new open wings mutant of **Drosophila melanogaster.**

In a sample of wild population of **D.melanogaster** from Petrinja locality (ca. 50 km southeast of Zagreb), cultured for several years in our laboratory, a phenotype with opened wings spontaneously appeared in 1983, indicating a new mutation. To determine the site of this mutation, we performed a genetic experiment with flies maintained nine generations by brother-sister mating. The crosses of our homozygous mutants with wild flies have shown that mutation is autosomal and recessive.

The crosses with brown-strain (bw 2-104.3) and scarlet-strain (st 3-440 showed that our mutant should be mapped on the third chromosome. In order to locate precisely the site of mutation, we applied the regular mapping procedure, by determining the frequency of crossing-over between the sepia and ebony markers (se 3-26; e 3-70.7). The obtained frequencies of recombinations (Table 1) suggested that the new mutation occurred at the locus 91.09 of the third chromosome. This is in the close neighborhood of well known locus crumpled (cmp 3-93), which has been supported by the appearance of cmp (up to 3%) on wings of the new phenotype.

Table 1. The frequencies of recombinations between jumper (ju) mutant vs sepia and ebony.

Parental types	+ + +	272	47.85%	
	se e ju	195		
single CO se-e	+ e ju	133	31.76%	recombinants
	se + +	177		
single CO e-ju	se e +	68	13.32%	
	+ + ju	62		
double CO	+ e +	43	7.07%	
	se + ju	26		
Total flies recorded		976		

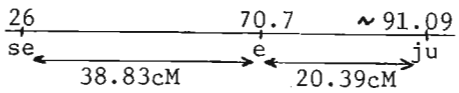

Figure 1. The phenotype of jumper mutant.

The main characteristics of the new phenotype is the presence of opened wings, oriented at the angle of 65° with respect to the longitudinal axis, and 10-30° to the horizontal axis (Figure 1). As of the other properties, the constant wing span and a decreased flying ability are so far the only recorded features, with a characteristic movement of individuals by jumping from one place to another. As far as we know (e.g., Zacharov 1979), this mutation is still not recorded in genetic maps of **D.melanogaster.**

We propose the following term to denotate this new mutation: jumper (ju 3-91.09).

Acknowledgements: The advices of Prof. D. Marinkovic and Asst. Z. Smit are very much appreciated.

References: Zacharov, J.A. 1979, in: Genetic maps of higher organisms, Nauka, Leningrad (in Russian).

Report of J.M. Mason,[1] R.A. Voelker,[1] D. Rosen,[2] A.R. Campos,[2] K. White[2] and J.K. Lim.[3]
1-NIEHS, Research Triangle Park, North Carolina USNA; 2-Brandeis University, Waltham, Massachusetts USNA; 3-University of Wisconsin, Eau Claire, Wisconsin USNA.

Localization of terminal deficiency breakpoints on the X chromosome.

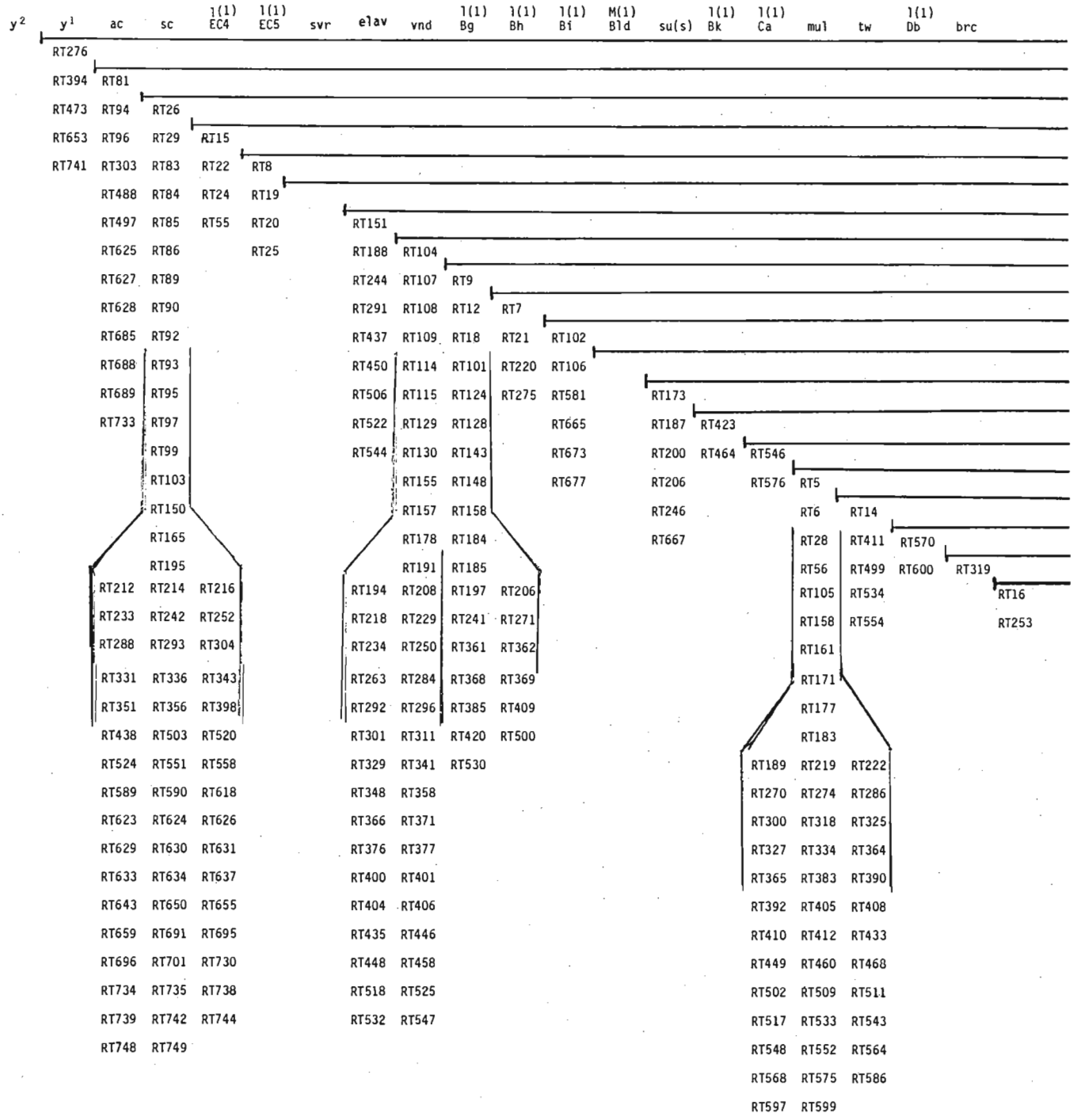

Figure 1. Positions of breakpoints for y deficiencies relative to nearby genetic markers. Names of genes are listed across the top. Most of these aberrations are true terminal deficiencies, although at least three (RT151, RT155, RT191 and possibly RT184) are half translocations.

Approximately 1500 putative terminal deficiencies with breaks between 1B1 and 1F were isolated by crossing irradiated +/+;mu-2/mu-2 females to y w/y^2 sc Y males (see Mason et al. 1984 for details). All y^- sc^+ deficiencies were examined for their effects on y and ac, approximately 500 sc^- deficiencies were examined for complementation with lethals in this region, and 129 random deficiencies were examined cytologically. The genetic complementation studies are summarized in the figure. The cytological positions of the breakpoints are not given because, with the number of lethal complementation groups available, and the absence of stretching in this region, the cytology does not have the resolving power of the genetic tests. Suffice it to say that the positions of the breakpoints as determined cytologically correlate well with the positions determined genetically. The cytological results also confirm the earlier conclusion that most of these deficiences extend to the tip of the X chromosome. About 8% are X-autosome translocations; the remainder are terminal deficiencies. Four translocations are included in the figure: [RT151=T(1;2R)1B7-10;57E10-F1. RT155=T(1;2L)1B9-12;21E4-F1. RT184 consistently ecotopically pairs with the tip of 3L, but no breakpoint is visible on chromosome 3. RT191=T(1;2L)1B4-10;21D1-2].

The apparent hot spots on the figure may not be real. The largest group of breakpoints lies between ac and sc, but the number of irradiated chromosomes screened to identify this sample is 2-3X larger than the number screened to identify breaks to the right of sc. The differences in the numbers of breakpoints in regions to the left of sc reflect differences in the physical distance between loci (Biessmann 1985, Campuzano et al. 1985). Apparent hot spots to the right of sc may be due to an incomplete genetic map as well as to differences in physical distance.

References: Biessmann, H. 1985, PNAS USA 82:7369-7373; Campuzano, S., L. Carramolino, C.V. Cabrera, M. Ruiz-Gomez, R. Villares, A. Boronat & J. Modollel 1985, Cell 40:327-338; Mason, J.M., E. Strobel & M.M. Green 1984, PNAS USA 81:6090-6094.

Report of D. Moriwaki and Y. N. Tobari.
Dept. of Biology, Tokyo Metropolitan University, Tokyo, Japan.

Description of new mutants of **Drosophila ananassae.**

X chromosome

f^{79b} : forked^{79b} Moriwaki 79i28. Spontaneous as 4 males in a wild strain collected at Bougainville Is., in 1979. Bristles twisted and forked. Allelic to f.

m^{84} : miniature^{84} Moriwaki 84e8. Spontaneous as two males of miniature wings accompanied with b ri-b, from a cross ♀ L bs/b ri-b x ♂ b ri-b. Wings only slightly longer than abdomen and somewhat round. Expression variable, sometimes indistinct. Allelic to m.

w^i : white-ivory Moriwaki 83 ℓ12. Spontaneous as a single male in a D ℓ^s stock. Eye color very light buff. Allelic to w. w^i > w.

w^{80} : white^{80} Moriwaki 80_g25. Spontaneous as a single male, white-eyed with D ℓ^s-wing, from an inbred line of D ℓ^s. Inseparable from w.

w^{84-2} : white$^{84-2}$ Moriwaki 84ℓ 27. Two males with white and Dℓ^{71} L phenotype spontaneously appeared in a D ℓ^{71} b L/ ℓ IM4 stock. Allelic to w.

y^{74} : yellow^{74} Moriwaki 74h17. Spontaneously a female of yellow phenotype among progeny of a cross ♀ ct y f w x ♂+ AM 1. Allelic to y.

ba-79 : balloon-79 Tobari 79b7. Spontaneously several flies arose in a M(2)b' stock. Wings inflated, blistered and a little extended. Penetrance incomplete.

bn-d : broken-d Moriwaki 83ℓ12. Spontaneously two males and one female appeared in a D ℓ^s stock. Posterior crossvein missing or broken. Sometimes weakly expressed in heterozygote.

D ℓ^s : Delta^s Moriwaki 80d7. Spontaneously a male and a female appeared in a cross, ♀ L b/D ℓ x ♂ mℓ(2)K159. More extreme than D ℓ. Reduced degree of expression depending on combined counterpart. Allelic to D ℓ.

M(2)83 : Minute(2)83 Moriwaki 83f1. Spontaneous as a single male in a cross, ♀ D ℓ^s x ♂ Pu^2/ve-TBU. Minute bristles; dominant; homozygous lethal.

M(2)b' : Minute(2)b' Moriwaki 83ℓ~84a. Arose spontaneously, in the M(3)b67 stock. M(3)b67 allele had been lost coincidently.

ml(2)K159 : minute-like(2)K159 Moriwaki 79k5. Spontaneously five males appeared in a wild strain, B30, collected in Thailand, in 1979. Bristles small as in Minutes.

pr : purple Moriwaki 77k12. Spontaneous as two females in a wild strain, PT-2, from Papua, New Guinea, in 1977. Eye color purplished ruby.

ri-b : radius incompletus-b Moriwaki 75d8. Spontaneously arose in a wild strain, A36, from Taiwan, in 1971. Vein L2 incomplete. Non allelic to ri. The name of ri, which was reported as radius interruptus in DIS 46 (1971) and in "**Drosophila ananassae**" (Handbook of Genetics, vol. 3, 1975), is revised, reading incompletus for interruptus.

se^{G103} : $sepia^{G103}$ Moriwaki 81h31. Spontaneously arose in a wild strain, G103, collected at Guam Is., in 1981. Eye color sepia. Allelic to se.

3rd chromosome

bri^{V} : $bright^{V}$ Moriwaki 81k11. Spontaneous as a male in a wild strain, VAV151, collected at Vavau, Tonga, in 1981. Eye color bright red; ocelli colorless. Allelic to bri.

c : curved Tobari 82b23. Spontaneoulsy arose in a wild strain, MYS178, collected at Mysore, India, in 1981. Wings curved downward throughout the length. Mixed with ones of curved and spread wings, which, by selection, were fixed as c^{sp} ($curved^{spread}$).

cn : cinnabar Moriwaki 84k5. Spontaneous as male and two females in a Dl^{s} stock. Eye color bright red, becoming dull with age.

Cy1 : Curlyoid Moriwaki 84k27. Spontaneously appeared in a cn stock. Wings curled up. Dominant; homozygous lethal. Dominancy incomplete, overlapping wild type occasionally.

dke : dark-eye Moriwaki 82a25. Spontaneous two males and one female in a wild strain, NAN80, collected in Fiji, in 1981. Eye color dark red.

dp : dumpy Moriwaki 79l3. Spontaneous as three males and two females in a wild strain, B13, collected in Thailand, in 1979. Wings obliquely truncated and reduced to 2/3 normal length; expression variable, often wings not shortened, narrow and somewhat pointed.

M(3)e : Minute(3)e Moriwaki 82j15. Spontaneously a male and two females arose in a cross, ♀ + $AABBg_1$ x ♂ ml ru. Minute bristles; dominant; homozygous lethal. Expression variable.

ml : minute-like Tobari 78f1. Spontaneously arose in a M(3)d ru stock. The locus seems likely near M(3)d or an allele of the M(3)d, but undetermined.

ml(3)B164 : minute-like(3)B164 Moriwaki 80l17. Spontaneous as four females in a wild strain, B164, collected in Thailand, in 1979. Bristles small as in Minutes.

ml(3)B30 : minute-like(3)B30 Moriwaki 79114. Spontaneously five males appeared in a wild strain, B30, collected in Thailand, in 1979. Bristles small as in Minutes.

pm : plum Moriwaki 81k18. Spontaneously two males and one female appeared in a wild strain, TBU130, collected in Tongatapu, in 1981. Eye color brownish wine, darkening to garnet with age. Combination of pm bri gives colorless eye and pm cn showing diluted orange color.

px^{d} : $plexus^{d}$ Moriwaki 77k12. Spontaneous as a female arose in a wild strain, 2S, collected in Papua, New Guinea, in 1977. Extra venation slightly near the tip of marginal cell; only dot-like at times. Allelic to px.

px^{3} : $plexus^{3}$ Moriwaki 7913. Spontaneously appeared in a wild strain, B8, collected in Thailand, in 1979. Venation plexus, an extra vein usually in submarginal cell. Allelic to px.

px^{4} : $plexus^{4}$ Moriwaki 83h8. In a M(3)d dke stock, spontaneously appeared as many flies. Venation plexus, extra vein mostly in submarginal cell and marginal cell. Allelic to px.

rf : roof Tobari 74j4. Spontaneously appeared in a cd Dl^{74} stock. Wings slanted at roof-like angle similar to Rf-phenotype. Probably allelic to Rf, as recessive.

rfl : rooflike Moriwaki 81c2. Spontaneous as a single male appeared in a wild strain, B13, collected in Thailand, in 1979. Wings drooped at sides like a roof, often elevated upward.

wp-89 : warped-79 Tobari 79a9. Spontaneously appeared in a cross derived from M(3)74. Wings warped and slightly divergent.

4th chromosome

bb^{HYD} : bobbed^{HYD} Moriwaki 84e21. Spontaneously several females with bobbed bristles appeared in a spa^{82} stock. spa^{82} bb^{HYD} stock was fixed. Allelic to bb.

spa : sparkling Moriwaki 79k5. Spontaneous as a female and ten males in a wild strain, D6, collected in India, in 1979. Eye surface rough and mottled in varying degrees. A linkage relation in spa-bb (4th chromosome) was confirmed. A similar mutant mo (mottled), found before the War(36k24), though discarded, also located in 4th chromosome (Moriwaki 1938).

spa^{82} : sparkling^{82} Tobari 82a20. Arose spontaneously in a wild strain, HYD3, collected in Hyderabad, India, in 1981. Somewhat extreme spa allele.

Report of C. Najera. Dept. of Genetics, Faculty of Biology, University of Valencia, Spain.

List of the different eye colour mutants of **Drosophila melanogaster** obtained in two different captures carried out in a pine-wood from La Canada, Valencia (Spain).

Localized mutants

se^{80i} (sepia-80)
cd^{80i} (cardinal-80) - two alleles at same capture
cd^{80d} (cardinal-80) - two alleles at same capture
sf^{80i} (safranin-80) - four alleles at same capture
sf^{80d} (safranin-80) - four alleles at same capture
sed^{80d} (sepiaoid-80)
v^{80d} (vermilion-80)
pr^{80d} (purple-80)

Non-localized mutants

65,80. eye colour chestnut
66. eye colour light chocolate
67,83. eye colour dark red
68.82. eye colour like wild darkening with age
69. eye colour dull red darkening with age

Non-localized (cont.)

70,81. eye colour bright brown
71. eye colour bright brown darkening with age
72. eye colour caramel
73. eye colour bright garnet darkening with age
74,91. eye colour bright red
75. eye colour vermilion
76,84. eye colour dark wine
77. eye colour reddish brown
78. eye colour coppery red
79. eye colour reddish brown darkening with age
(two alleles)
85. eye colour dark chocolate
86. eye colour ruby
87. eye colour purple
88. eye colour orange red darkening with age
89. eye colour orange red
90. eye colour shining red

Report of Yong K. Paik. Dept. of Genetics, Hanyang University, School of Medicine, Seoul 133, Korea.

New inversions of Korean **D.melanogaster** recovered from natural populations.
All inversions are maintained as balanced stocks.

Inversion	Breakpoints	Inversion	Breakpoints
In(2L)KA	22A;26B	In(3R)KA	82E/F;99E/F
In(2L)KB	22D;(2R)57A	In(3R)KB	83D/E;86D
In(2L)KC	24C/D;36B/C	In(3R)KC	83C/D;93A
In(2L)KD	26B;29B/C	In(3R)KD	84E;94D/E
In(2L)KE	26C;29B/C	In(3R)KE	85C;88F
In(2L)KF	26C/D;32B/C	In(3R)KF	86B;87B/C
In(2L)KG	28A/B;32D	In(3R)KG	86D/E;97C
In(2R)KA	42A;57F	In(3R)KH	88C;96D
In(2R)KB	42A;58C/D	In(3R)KI	88D/94A
In(3L)KA	66C;71B/C	In(3R)KJ	90D;93B
In(3L)KB	68C;(3R)91D	In(3R)KK	92E;97C

Report of C. Pla,*° J.B. Toral* and A. Fontdevila.*
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Description, localization and recombination values of several mutants of **Drosophila buzzatii.**

Table 1. Recombination values between some of the mutants of **Drosophila buzzatii.**

chromosome	markers*	cross-overs	sample size	recombination value
X	Fum - N	68	289	0.2352 ± 0.0059
	Fum - w	94	243	0.3868 ± 0.0121
	Fum - y	22	187	0.1176 ± 0.0028
	Fum - v	68	204	0.3333 ± 0.0110
	N - w	68	2420	0.0280 ± 0.0009
	N - y	152	971	0.1565 ± 0.0018
	N - v	145	993	0.1460 ± 0.0016
	w - y	204	1007	0.2026 ± 0.0026
	w - v	93	901	0.1032 ± 0.0101
	y - v	152	518	0.2934 ± 0.0059
2	-Est - mah	9	128	0.0703 ± 0.0016
	Pept-2 - se	7	62	0.1129 ± 0.0045
3	Adh - fo	103	307	0.3355 ± 0.0090
4	Pgm - 4s	10	64	0.1562 ± 0.0071
	Pgm - st	17	182	0.0934 ± 0.0020
5	bw - vs	275	529	0.5198

* Fum, -Est, Pgm and Adh stands for Fumarase, -Esterase, Phosphoglucomutase and Alcohol dehydrogenase. 4s refers to inversion breakpoints of chromosome 4.

The biological interest of **D.buzzatii** as a species to perform evolutionary studies has been emphasized several times (Fontdevila 1982). However, design of genetic experiments with this species has been hampered in many cases by the lack of appropriate morphological and biochemical markers. We report here the first list of morphological mutants of **D.buzzatii** obtained from natural populations and laboratory strains. Initially, chromosomal rearrangements, known to be located in specific chromosomes (Pla et al. 1984; Ruiz et al. 1985). Mutant naming and morphological description analogous to those of **D.melanogaster** have been adopted only when homologies are well established (Stone 1955; Zouros 1976). In other cases, appropriate names have been adopted. Genetic naming follows that one by Lindsley & Grell (1968).

Description of mutants

bw : **brown.** Eye color brown. Recessive. Located in chromosome 5. Probably homologous to brown of **D.melanogaster** (chromosome 2R). Originated from a natural population from Sitges (Barcelona, Spain), (Pla et al. 1984a).

ey : **eyeless.** Eye more reduced to normal area. Recessive. Could be homologous to eyeless of **D.m.** (chromosome 4). Originated from a natural population from Mazarron (Murcia, Spain).

fo : **folded.** Wings remain unexpanded. Recessive. Located in chromosome 3. Induced by X-ray in a laboratory stock.

mah : **mahogany.** Eye color brown (or maroon) and darkening with age. Recessive. Located in chromosome 2. Probably homologous to mahogany of **D.m.** (chromosome 3R). Originated from a natural population from Sitges.

N : **Notch.** Wings incised at tips and often along edges. Terminal part of vein L5 often opened. Variable expression. Dominant. Located in chromosome X. Male and homozygous female lethal. Probably homologous to Notch of **D.m.** but we have not detected any deficiency. Induced by X-ray in a laboratory stock.

st : **scarlet.** Eye color bright red-scarlet. Recessive. Located in chromosome 4. Probably homologous to scarlet of **D.m.** (chromosome 3L). Originated from a natural population from Sitges (Barcelona, Spain).

se : **sepia.** Eye color dark brown and darkening to sepia with age. Recessive. Located in chromosome 2. Originated spontaneously from a laboratory stock. Produces orange eyes in combination with vermilion.

v : **vermilion.** Eye color bright red. Recessive. Located in chromosome X. Probably homologous to vermilion of **D.m.** (chromsome X). Originated from a natural population from Sitges (Barcelona, Spain). With sepia produces orange eyes.

vs : **vesiculated.** Wings warped, wrinkled, blistered and rough texture. Variable expression. Penetration of 90%. Recessive. Located in chromosome 5. Induced by X-ray in a laboratory stock.

w : **white.** Eye color white. Recessive. Located in chromosome X. Probably homologous to white of **D.m.** (chromosome X). Originated spontaneously from a laboratory stock.

y : **yellow.** Body color yellow. Recessive. Located in chromosome X. Probably homologous to yellow of **D.m.** (chromosome X). Originated spontaneously from a laboratory stock.

References: Fontdevila, A. 1982, in: Ecological Genet. and Evol., ch 6; Lindsley, D.L. & E.H. Grell 1968, Carn. Inst. Wash. Publ. 627; Pla, C., J.B. Toral, H. Naveira & A. Fontdevila 1984, Experientia 41:507-508; Pla, C., J.B. Toral & A. Fontdevila 1984a, DIS 60:163; Ruiz, A., H. Naveira & A. Fontdevila 1985, Genetica Iberica, in press; Stone, W.S. 1955, Cold Spring Harbor Symp. Quant. Biol. 20:256-270; Zouros, E. 1976, Genetics 83:169-179.

Report of T. Prout and M.M. Green.
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We here report the identification of a new locus in **D.melanogaster.** The recessive phenotype is identical with that of "eagle", e.g., 3-47.3, but is linked to chromosome 2 mapping 26.5 ± 0.9 centimorgans to the left of B1, and so is located at position 28.3 ± 0.9. We propose the name "nesher" (Hebrew for "eagle"), symbol "nr". Nesher and eagle combined exhibit the eagle phenotype. Nesher was isolated from 8 independent isofemale lines derived from two natural populations; one Apple Hill in northern California and the other Furnace Creek in Death Valley, California. Nesher occurred among the much more numerous independent appearances of eagles found by J. Bundgaard in isofemale lines from the above localities as well as several others in California.

Report of J. Sved.
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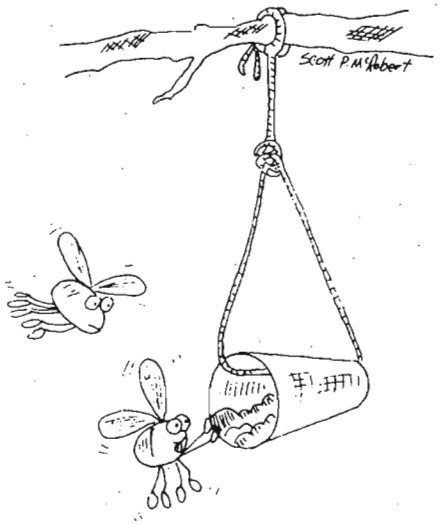
Eyes Absent (eya)

This mutation was uncovered several years ago during screening of a winery population using chromosome homozygosis for chromosomes II via the Cy/Pm technique. Attempts to map the mutation were unsuccessful, and it was found to be associated with an inversion with breakpoints at 22D and 34B.

The phenotype is characterised by complete absence of eye facets. Other aspects of head development appear normal, although the head size is considerably reduced, and the pattern of bristles surrounding the residual eye socket is variable. The mutation is fully recessive. The viability in pure culture is high, although reduced when measured in competitive viability tests.



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Langelan, R Postdoctoral scholar -6997
Woods, D Postdoctoral Scholar -6997
Jursnich, V grad student -6997
Sponaugle, D grad student -6997
Jo Wen Wu grad student -6997
Hsei, B research assoc -6997
Wilson, L research assoc -6997

USNA: Raleigh, North Carolina 27650: North Carolina State Univ, Genetics Dept Tel 919-737- [UPDATE]
delete: Sederoff, R R

USNA: St. Paul, Minnesota 55112: Bethel College, Biology Dept, 3900 Bethel Drive Tel 612-638-6400
Jones, C W PhD structure & function of ecdysone-responsive genes in Drosophila [NEW LISTING]

USNA: Seattle, Washington 98195: Univ of Washington, Zoology Dept (NJ-15) [UPDATE]
add: Sederoff, R R PhD molecular genetics

USNA: Stony Brook, New York 11794: State Univ of New York, Biochemistry Dept Tel 516-246-5043 [UPDATE]
add: Bingham, P M PhD physiological genetics, transposons
Carlson, E A PhD mutagenesis, mosaicism
Erk, F C PhD developmental genetics

NOTE: The Alphabetical Directory is being omitted in this issue; it will appear in volume 65 (1987).

THE MOLECULAR GENOME OF DROSOPHILA MELANOGASTER

CATALOGS OF CLONED DNA, BREAKPOINTS AND TRANSFORMED INSERTS BY CHROMOSOME LOCATION

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The force of Drosophila cytogenetics has been strong in identifying specific genes as mutant alleles. With the advent of molecular cloning and in situ hybridization, this cytogenetic approach can be extended, so far uniquely to Drosophila, to cloning genes, or identifying cloned genes, starting from chromosome locations. To do so via chromosome walks, chromosome jumps or transposon tagging requires preexisting clones or breakpoints or other genes inserted by transformation in a location. Sources of knowledge of these preexisting starting points are critical for experimental design. The community of Drosophila workers is cooperative about sharing this information. Beginning in 1983 with the National Annual meeting and with extensions to the international community we have sought to collect and summarize what is known about the molecular genome of Drosophila. As these reports in the D.I.S. have grown, the format of presenting the information has also changed. The present report consists of four tables: MASTER LIST, TABLE 1, TABLE 2, and REFERENCES. A suggested report form is appended to copy in order to send in more recent additions.

MASTER LIST contains data by chromosome location with the following information listed:

LABEL	DESCRIPTION OF INFORMATION
CLONE NAME	Name or number of cloned sequence
GENES	Genes contained in cloned sequence
BREAKPOINT	Name of rearrangement with breakpoint(s) in cloned region
CROSS LIST	Cross listing of other breakpoints of rearrangement
INSERTION	Transformants inserted at this location
CROSS LIST	Cross listing of chromosome location of genes contained in the insert
REF	Literature citation for information on clones, breakpoints, and inserts.

TABLE 1 contains other information on clones listed by chromosome location. Information on stock of origin, KB size, DNA library, reference to mapping sequence, and miscellaneous informatioin is provided when available under the heading OTHER INFORMATION.

TABLE 2 contains information on inserts listed in MASTER LIST. Inserts are listed by chromosome location with gene markers contained in the inserts listed under the heading, MARKERS.

REFERENCES are listed in the last table by reference number used in the MASTER LIST.

This work was supported by NIH Grant LM04344 from the National Library of Medicine.

MASTER LIST OF CLONES,BREAKPOINTS, AND INSERTS
JUNE 4, 1986

1A8B1
CLONE NAME: unn REF: 105
GENES: yellow
BREAKPOINT: In(1)y4 REF: 105
CROSS LIST: 18A3,4

1A
CLONE NAME: lambda T-A REF: 156
GENES: telomeres
CROSS LIST: 21A, 60F, 61A, 100F, 102F

1B
INSERTION: 27X-F REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: unn REF: 105
GENES: scute
CLONE NAME: lambda y1 REF: 77
GENES: yellow
CLONE NAME: y+p13-2,yp3A2 REF: 74
GENES: yellow
CLONE NAME: adm134E8 REF: 34
GENES:
BREAKPOINT: In(1)sc29 REF: 105
CROSS LIST: 13A2-5
INSERTION: g71:1 REF: 114
CROSS LIST: 68C3-5, 87D12

1B1,2
CLONE NAME: unn REF: 8
GENES: yellow, achete
BREAKPOINT: In(1)y3p REF: 103
CROSS LIST: 20D1

1B1,2-B4,5
CLONE NAME: sc64-sc133 REF: 152
GENES: y, ac, sc-alpha and sc-beta

1B2,3
CLONE NAME: unn REF: 105
GENES: achete, scute
BREAKPOINT: In(1)ac3, In(1)sc260-22, In(1)sc8, In(1)sc9 In(1)sc260-14 REF: 105
CROSS LIST: 1B14C1, 1E2,3, 20D1, 18B8,9, 11D3,8

1B3,4
CLONE NAME: unn REF: 105
GENES: scute
BREAKPOINT: In(1)sc4, In(1)scL8, In(1)scS1 REF: 105
CROSS LIST: 20C1, 20C, 20D1

1B4,5
CLONE NAME: unn REF: 105
GENES: scute
BREAKPOINT: T(1;3)scKA8, T(1;3)sc260-15 REF: 105
CROSS LIST: 98 (+ OR -), 71CD

1B4,6
CLONE NAME: unn REF: 105
GENES: scute
BREAKPOINT: In(1)sc7 REF: 105
CROSS LIST: 5D3,6

1B4,7
CLONE NAME: unn REF: 105
GENES: scute
BREAKPOINT: T(1;2)sc19, T(1;2)scS2 REF: 105
CROSS LIST: 25A, 60C1,3

1B4-C6
CLONE NAME: unn REF: 105
GENES: scute
BREAKPOINT: T(1;4)scH REF: 105
CROSS LIST: 101-102

1B4,5-1B8,9
CLONE NAME: REF: 206
GENES: elav
BREAKPOINT: In(1)63 REF: 206

1B5,8
CLONE NAME: cos 4P REF: 57
GENES:
CROSS LIST: 2E

1B11-13
CLONE NAME: unn REF: 15
GENES: suppressor of sable

1B1,2-4,5
CLONE NAME: unn REF: 57
GENES: yellow, achete, scute

1B14C1
BREAKPOINT: In(1)ac3 REF: 105
CROSS LIST: 1B2,3

1CD
INSERTION: Bs2.71-2 REF: 115
CROSS LIST: 66D11-15, 87D12

1E 2-3
INSERTION: Strain 23-2 REF:
CROSS LIST: 87D12, 5S ribosomal genes

1E2,3
BREAKPOINT: In(1)sc260-22 REF: 105
CROSS LIST: 1B2,3

1F
BREAKPOINT: T(1;3)Uab5 REF: 110
CROSS LIST: 89E1-4
INSERTION: R704.2, R702.1 REF: 115
CROSS LIST: 87D12
INSERTION: DA24-14 REF: 114
CROSS LIST: 35B2-3,37C1,2

2A
INSERTION: P[(w,ry)E]5 REF: 176
CROSS LIST: 3C1,2; 87D
INSERTION: 27P X/X-F REF: 200
CROSS LIST: 67B, 87D12

2B
INSERTION: P(ry,hsp0-1)8 REF: 137
CROSS LIST: 87D12
INSERTION: Tf(1)Gr304-1 REF: 111
CROSS LIST: 87D12

2B1,2
CLONE NAME: unn REF: 108
GENES: l(1)BA11
BREAKPOINT: Dp(1;f)101 REF: 108
CROSS LIST: 20

2B1,2-5,6
CLONE NAME: unn REF: 98
GENES: occ

2B1,2
CLONE NAME: Dm156, Dm159, Dm160 REF: 108
GENES:

2B3,4
CLONE NAME: unn REF: 108
GENES: sta
BREAKPOINT: T(1;3)sta REF: 108
CROSS LIST: 89B21
CLONE NAME: unn REF: 108
GENES: sta
BREAKPOINT: T(1;3)sta REF: 108
CROSS LIST: 89B21
CLONE NAME: Dm174, Dm 340 REF: 108
GENES:

2B13-18
INSERTION: g711:2 REF: 114
CROSS LIST: 68C3-5,87D12

2C
INSERTION: Pc[ry(delta0-1)]48 REF: 137
CROSS LIST: 87D12

2C1-2E3
CLONE NAME: REF: 161
GENES: vsp, csw, ph, Pgd, kz, 11 lethals

2D2-3
BREAKPOINT: Df(1)pu3; Df(1)Pgd-kz;Df(1)JA52;Dp(1)dorY18T REF: 161

2EF
CLONE NAME: unn REF: 80
GENES: fs(1)pecanex,pcx1

2E
CLONE NAME: cos 4P REF: 57
GENES:
CROSS LIST: 1B5,8

2E2-F3
CLONE NAME: unn REF: 98
GENES: fs(1)K10,crooked neck,pcx,kurz

2F5-6
CLONE NAME: draf1 REF: 129
GENES: raf oncog. homo.; pos. ser/thr kinase
CROSS LIST: minor homology to 43A2-5

3
CLONE NAME: S24 REF: 16
GENES:

3A
INSERTION: 7-1;7-2 REF: 140
CROSS LIST: 3C11,12;87D; 3C11,12;ry+

3A1-4
CLONE NAME: REF: 166
GENES: zeste
BREAKPOINT: In(1)e(bx); Df(1)64C4; Df(1)w258-11; Df(1)wrj REF: 166
1; Df(1)62g18
CROSS LIST: 4F

3A6-8
BREAKPOINT: Df(1)64j4 REF: 107
CROSS LIST: 3C1-4

3B
CLONE NAME: lambda DT2,5 REF: 217
GENES:
CLONE NAME: mDm112 C 10 REF: 1
GENES:
INSERTION: Tf(1)GR420-3 REF: 111
CROSS LIST: 87D12

3B1,2
CLONE NAME: REF: 107
GENES: per, L(1)BA11
BREAKPOINT: Df(1)w-64D, In(1)3B1, 2-20F, Df(1)3B1,2-3C2,3 REF: 107
Dp(1;f)101, Df(1)62d18, T(1;4)JC43
CROSS LIST: 20F, 20, 3C6, 102
CLONE NAME: unn REF: 9
GENES: per

3B2-3C2
CLONE NAME: unn REF: 62
GENES: white

3BC
CLONE NAME: M187 REF: 84
GENES:

3C
CLONE NAME: unn REF: 95
GENES: distal to white

3C1-4
BREAKPOINT: Df(1)64j4 REF: 107
CROSS LIST: 3A6-8

3C1,2
CLONE NAME: lambda m1.2 REF: 3
GENES: white
BREAKPOINT: Df(1)wNfm20 REF: 109
CROSS LIST: 3C11-12

3C2-3
BREAKPOINT: Df(1)Nfm21, Df(1)N10 REF: 109
CROSS LIST: 3C11-12

3C6-8
CLONE NAME: unn REF: 107
GENES: Notch
BREAKPOINT: Df(1)62d18 REF: 107
CROSS LIST: 3B1,2

3C6
BREAKPOINT: Df(1)N541g REF: 109
CROSS LIST: 3C11-12

3C7,8
CLONE NAME: unn REF: 9
GENES: Notch
BREAKPOINT: In(1)N76b8, Df(1)N62b1 REF: 107
CROSS LIST: 3C9,10, 3D5,6

3C7
CLONE NAME: unn REF: 46
GENES: Notch
CLONE NAME: pKdm 6B3 REF: 34
GENES: intermolt I RNA
CROSS LIST: 3D1
CLONE NAME: REF: 177
GENES: notch-epidermal growth factors

3C9,10
BREAKPOINT: In(1)N76b8 REF: 107
CROSS LIST: 3C7,8

3C11,12
CLONE NAME: unn REF: 45
GENES: Sgs 4
CLONE NAME: REF: 109
GENES: Sgs-4
BREAKPOINT: Df(1)WNfm20, Df(1)Nfm21, Df(1)N10, Df(1)N541g REF: 109
Df(1)dm75elg
CROSS LIST: 3C1-2, 3C2-3, 3C6, 3E4

3C11
CLONE NAME: pOM3, pOW3 REF: 134
GENES: Sgs-4
CROSS LIST: pSME3, pSWE3
CLONE NAME: pOM3,pOW3 REF: 134
GENES: Sgs-4
CROSS LIST: pSME3, pSWE3

CROSS LIST:

3D1
CLONE NAME: pKdm 6B3 REF: 34
GENES: intermolt I RNA
CROSS LIST: 3C7

3D5,6
BREAKPOINT: Df(1)N62b1 REF: 107
CROSS LIST: 3C7,8

3E4
BREAKPOINT: Df(1)dm75elg REF: 109
CROSS LIST: 3C11,12

3F
INSERTION: g71dx:2 REF: 114
CROSS LIST: 68C3-5,87D12

3,4
CLONE NAME: adm 136G5 REF: 34
GENES:

4C5-6
INSERTION: Strain 23-2 REF:
CROSS LIST: 87D12, 5S ribosomal genes

4B
INSERTION: cHB delta -59 REF: 118
CROSS LIST: 87,lacZ,87D12

4BC
CLONE NAME: mDm 109A7 REF: 1
GENES:
CLONE NAME: unn REF: 90
GENES: no receptor potential

4C
CLONE NAME: M97 REF: 84
GENES:

4D
BREAKPOINT: T(1;3)bxd111 REF: 110
CROSS LIST: 89E1,4
INSERTION: R405.1 REF: 115
CROSS LIST: 87D12

4F5A
CLONE NAME: pKdm 35D12 REF: 34
GENES: late IV RNA
CLONE NAME: adm 139C12 REF: 34
GENES:

5AB
CLONE NAME: adm 126D6 REF: 34
GENES:

5B
BREAKPOINT: T(1;3)Ubx21560.8A complex REF: 110
CROSS LIST: 89E1,4

5C
CLONE NAME: pDmAz REF: 179
GENES: actin
CLONE NAME: unn REF: 12
GENES:

5D
CLONE NAME: unn REF: 85
GENES: ribosomal protein 7/8

5D3-6
BREAKPOINT: In(1)sc7 REF: 105
CROSS LIST: 1B4,6

5EF
CLONE NAME: adm 140C11 REF: 34
GENES:
CROSS LIST: 63F64A

5F (prox)
CLONE NAME: B70 REF: 69
GENES: maternal restricted transcript

6AB
INSERTION: P[(w,ry)H]4 REF: 176
CROSS LIST: 3C1,2; 87D

6F
INSERTION: S6.9-2 REF: 115
CROSS LIST: 66D11-15,lacZ,87D12

6F5
CLONE NAME: unn REF: 66
GENES: sex lethal
CLONE NAME: S1, S2A, S2B REF: 223
GENES: Sex-lethal (sxl)
BREAKPOINT: In(1)sxl-af; Df(1)sxl-ra REF: 223

7A
CLONE NAME: unn REF: 67
GENES:

7B3,4
CLONE NAME: unn REF: 29
GENES: cut

7D
CLONE NAME: m58 REF: 84
GENES:
INSERTION: R403.1 REF: 115
CROSS LIST: 87D12

7D5
CLONE NAME: unn REF: 21
GENES: fs(1)homeotic, l(1)myospheroid

7D5-6
CLONE NAME: REF: 202
GENES: fs(1) homeotic;l(1) B104 leth. myospher.
BREAKPOINT: T(1:3) N72 REF: 202
CROSS LIST: 7D1,2

7E6
CLONE NAME: unn REF: 44
GENES:
CROSS LIST: 7F1,2

7E11-7F1,2
CLONE NAME: unn REF: 115
GENES: s36, s38
CROSS LIST: see Table 2

7F1,2
CROSS LIST: 7E6

7F1
CLONE NAME: REF: 204
GENES: ovarian tumor (otu)

7,8
CLONE NAME: adm 132H10 REF: 34
GENES:

8
CLONE NAME: 56 REF: 16
GENES:

8A
CLONE NAME: unn REF: 44
GENES:

8BC
INSERTION: tAP-25,3.2 REF: 112
CROSS LIST: 35B2-3

8D
CLONE NAME: PLZ-p REF: 2
GENES: lozenge

8E
INSERTION: cHB delta-59 REF: 118
CROSS LIST: 87,lacZ,87D12

8F9A
CLONE NAME: PYp2 REF: 2
GENES: yolk protein 2
CLONE NAME: PYp1 REF: 2
GENES: Yolk protein 1

9E3-4
CLONE NAME: lambda EMBL4-LF1 REF: 138
GENES: raspberry, P-element

9A-D
INSERTION: R404.2 REF: 115
CROSS LIST: 87D12

9B
INSERTION: unn REF: 117
CROSS LIST: 87D12
INSERTION: tAP-24B,3.2 REF: 112
CROSS LIST: 35B2-3
INSERTION: SB2.1-5 REF: 115
CROSS LIST: 66D11-15,87D12

9C
INSERTION: S6.9-9 REF: 115
CROSS LIST: 66D11-15,lacZ,87D12

9D
INSERTION: P(ry,hsp0-1)22 REF: 137
CROSS LIST: 87D12

9E
INSERTION: R701.1 REF: 115
CROSS LIST: 87D12
INSERTION: cHB lambda -23 REF: 118
CROSS LIST: 87-lacZ,87D12

10A
CLONE NAME: unn REF: 79
GENES: vermilion: tryptophan oxygenase

10A1
CLONE NAME: REF: 186
GENES: vermilion

10A1,2
CLONE NAME: lambda vDT1 REF: 217
GENES: vermilion (tryp oxygenase)

10A2
CLONE NAME: sevenless REF: 173
GENES: photoreceptor cell development

10B
CLONE NAME: unn REF: 91
GENES:

10BC
INSERTION: tAP-20,3.2 REF: 112
CROSS LIST: 35B2-3

10C1,2
CLONE NAME: lamda DmRpll-1 REF: 51
GENES: RNA polymerase II largest subunit
BREAKPOINT: Df(1) GA112; Df(1) HA85; Df(1) M259-4 REF: 147
CROSS LIST: 10B1,2; 11A1,2; 10E1,2

10D
INSERTION: AR4-038 REF: 126
CROSS LIST: 3C1,2

10EF
CLONE NAME: adm134A3, adm130E12 REF: 34
GENES: late V RNA

10F
CLONE NAME: adm 10F.1 REF: 39
GENES: minor heat shock cDNA from Kc cells

11A
CLONE NAME: unn REF: 38
GENES: gastrulation defective
CLONE NAME: lambda Dm (Can S) LSP1 alpha: 1 REF: 130
GENES: LSP1 alpha

11A2-4
CLONE NAME: REF: 174
GENES: gastrulation defective

11D3-8
BREAKPOINT: In(1)sc260-14 REF: 105
CROSS LIST: 1B2,3

12
CLONE NAME: S21b REF: 16
GENES:

12A
INSERTION: tAP-17,4.8 REF: 112
CROSS LIST: 35B2-3

12B
INSERTION: P[(w,ry)E]2 REF: 176
CROSS LIST: 3C1,2; 87D

12B,C
CLONE NAME: PYP3 REF: 2
GENES: yolk protein 3
INSERTION: SRS3.9-1 REF: 115
CROSS LIST: 66D11-15, 87D12

12BC
INSERTION: AR4-032(X) REF: 126
CROSS LIST: 3C1,2

12D
INSERTION: R301.2 REF: 115
CROSS LIST: 87D12

12DE
CLONE NAME: pDt17R, pDt27,pDt73 REF: 27
GENES: ser 7,4,4-7 tRNA, respectively

12E
INSERTION: E 7-10 REF: 134
CROSS LIST: 3C1-2
CLONE NAME: pDt16 REF: 27
GENES: ser 4-7 tRNA
INSERTION: cHB delta-73 REF: 118
CROSS LIST: 87-lacZ, 87D12

12F
CLONE NAME: lambda 32-10 REF: 58
GENES: tRNA

12F1,2
CLONE NAME: Dm2L1 REF: 75
GENES: tandem repeated 2L1 sequence

12
CLONE NAME: adm 136F10 REF: 34
GENES:

13A2-5
BREAKPOINT: In(1)sc29 REF: 105
CROSS LIST: 1B

13A-C
INSERTION: BS.27-5 REF: 115
CROSS LIST: 66D11-15,87D12

13CD
INSERTION: SB2.1-6 REF: 115
CROSS LIST: 66D11-15,87D12

13EF
INSERTION: P[(w,ry)E]3g;P[(w,ry)G]4 REF: 176
CROSS LIST: 3C1,2; 87D; 3C1,2; 87D
CLONE NAME: M3,6,8,10 REF: 178
GENES: c-myb

13F
CLONE NAME: G2 REF: 135
GENES: Glyceraldehyde-3-phosphate dehydrogenase

14A
INSERTION: 21-1 REF: 140
CROSS LIST: 3C11,12;87D

14BC
CLONE NAME: adm 132B8 REF: 34
GENES:

14D
CLONE NAME: M75 REF: 84
GENES:

15A1
CLONE NAME: unn REF: 30
GENES: rudimentary

15A,B
CLONE NAME: 548 REF: 31
GENES: head specific RNA

15B
CLONE NAME: unn REF: 85
GENES: ribosomal protein S 18

15DE
INSERTION: BS2.7-10 REF: 115
CROSS LIST: 66D11-15,87D12

15E
INSERTION: P15-1 REF:

15F
CLONE NAME: Dmf3 REF: 158
GENES: forked

CLONE NAME: lambda f1 REF: 77
GENES: forked

16B3-5
CLONE NAME: PTE-1 REF: 2
GENES:

16BC
INSERTION: S6.9-11 REF: 145
CROSS LIST: 66D11-15, lacZ, 87D12

16C
INSERTION: H1 REF: 221
CROSS LIST: 87D12

16D
INSERTION: unn REF: 117
CROSS LIST: 87D12

16E
INSERTION: 27 N/P-A REF: 200
CROSS LIST: 67B, 87D12

16EF
CLONE NAME: unn REF: 40
GENES: shaker

16F
INSERTION: 27C X/X-A REF: 200
CROSS LIST: 67B, 87D12

16F17
CLONE NAME: adm 135H4 REF: 34
GENES:

17AB
CLONE NAME: lambda dmpt 61 REF: 58
GENES:

17C
INSERTION: tAP-5 REF: 181
CROSS LIST: 35B3-5

17DE
INSERTION: B1-2 REF: 113
CROSS LIST: 3C1-2

18A
INSERTION: R704.3 REF: 115
CROSS LIST: 87D12

18A3,4
BREAKPOINT: In(1)y4 REF: 105
CROSS LIST: 1A8B1

18B8,9
BREAKPOINT: In(1)sc9 REF: 105
CROSS LIST: 1B2,3

18CD
CLONE NAME: A57 REF: 69
GENES: maternal restricted transcript

18D
INSERTION: 16-3 REF: 140
CROSS LIST: 3C11,12;87D
CLONE NAME: lamda DmG21 REF: 28
GENES: G6PD

INSERTION: BS2.7-3 REF: 125
CROSS LIST: 66D11-15, 87D12
CLONE NAME: trc 18 REF: 201
GENES:

18E
CLONE NAME: Dm14 REF: 165
GENES: G6PD
CLONE NAME: lambda Dm G6PD 14 REF: 128
GENES: G6PD

19A
INSERTION: cHB delta-89 REF: 118

19E
INSERTION: +65 REF: 118
INSERTION: tAP-1 REF: 181
CROSS LIST: 35B3-5

19E8
CLONE NAME: PP95 REF: 160
GENES: unc

19EF
CLONE NAME: DCg2 REF: 25
GENES: collagen-like gene
CROSS LIST: 20AB

19F
INSERTION: P[(w,ry)E]1 REF: 176
CROSS LIST: 3C1,2; 87D
CLONE NAME: pDt67R REF: 27
GENES: Arg rTNA locus

20
BREAKPOINT: Dp(1;f)101 REF: 108
CROSS LIST: 2B1,2
BREAKPOINT: T(1;3)CbxrvR17.49A, T(1;3)P115, Dp(3;1)P68 REF: 110
CROSS LIST: 89E,89F

20A
INSERTION: 2 REF: 123
CROSS LIST: 88F
INSERTION: Adh hs20A REF: 116
CROSS LIST: 35B2-3,87

20AB
CLONE NAME: DCg2 REF: 25
GENES: collagen-like gene
CROSS LIST: 19EF

20C
BREAKPOINT: In(1)scL8 REF: 105
CROSS LIST: 1B3,4

20C1
BREAKPOINT: In(1)sc4 REF: 105
CROSS LIST: 1B3,4

20CD
INSERTION: P(ry,HsAFP) REF: 141
CROSS LIST: 87D12

20D
INSERTION: AR4-024 REF: 126
CROSS LIST: 3C1,2

20D1
BREAKPOINT: In(1)scS1 REF: 105
CROSS LIST: 1B3,4
BREAKPOINT: In(1)sc8, In(1)y3P REF: 103
CROSS LIST: 1B2,3, 1B1,2

20F
BREAKPOINT: Df(1)w-64D= In(1)3B1,2-20F+, REF: 107
Df(1)3B1,2-3C2,3
CROSS LIST: 3B1,2

21 tip
BREAKPOINT: T(3;2)bxd-D36 complex REF: 110
CROSS LIST: 89E

21A
CLONE NAME: lambda alpha 8; lambda 8001 to 8014 REF: 131
GENES: lethal(2) giant larvae
CLONE NAME: alpha 8 REF: 154
GENES: l(2)gl
CLONE NAME: lambda T-A REF: 156
GENES: telomeres
CROSS LIST: 1A, 60F, 61A, 100F, 102F

21A1,2
BREAKPOINT: T(2;3)Ubx16160.18 REF: 110
CROSS LIST: 89E

21B
INSERTION: P[(w,ry)G]1 REF: 176
CROSS LIST: 3C1,2; 87D
CLONE NAME: adm 142G5 REF: 34
GENES:

21C
CLONE NAME: unn REF: 88
GENES: double sex cognate

21D
CLONE NAME: pDm6A65 REF: 151
GENES: U1 snRNA
CROSS LIST: 82E, 95C
INSERTION: P[(w,ry)F]4-2 REF: 176
CROSS LIST: 3C1,2; 87D
CLONE NAME: lambda Dm9 REF: 219
GENES: LSP-1 beta
CLONE NAME: pD957 REF: 3
GENES:
INSERTION: R602.1 REF: 115
CROSS LIST: 87D12
CLONE NAME: lambda Dm (Can S) LSP1 beta: 1 REF: 130
GENES: LSP1 beta

21DE
INSERTION: tAP-10,4.8 REF: 112
CROSS LIST: 35B2-3

21F
CLONE NAME: adm 123D12,123H3,128B8 REF: 34
GENES:
CROSS LIST: 22A

22A
CLONE NAME: adm 123D12, 123H3, 128B8 REF: 34
GENES:
CROSS LIST: 21F
INSERTION: R604.1 REF: 115
CROSS LIST: 87D12

22AC
CLONE NAME: REF: 193
GENES: 4S RNA

22B
CLONE NAME: mDm103H10, mDm108C7 REF: 1
GENES:
CROSS LIST: 45A
INSERTION: w20.2,w20.10 REF: 120
CROSS LIST: 3C1-2

22B1,2
BREAKPOINT: T(2;3)Cbx rvR17.175 REF: 110
CROSS LIST: 89E

22B,C
CLONE NAME: adm 129E7 REF: 34
GENES:

22F1,2
CLONE NAME: unn REF: 55
GENES: decapentaplegic complex
CLONE NAME: pDm22F Ya,Yb REF: 189
GENES: tyr tRNA

22F-23A
INSERTION: P13-1 REF:
CROSS LIST: 87D12

23A
INSERTION: +65 REF: 118
CROSS LIST: 87,87D12

23A3-7
CLONE NAME: unn REF: 59
GENES:

23BC
CLONE NAME: B13 REF: 69
GENES: maternal restricted transcript
INSERTION: g5:2 REF: 124
CROSS LIST: 68C3-5, 87D12

23E
CLONE NAME: pDt5 REF: 27
GENES: ser 7 tRNA

24AB
INSERTION: P[(w,ry)D]4 REF: 176
CROSS LIST: 3C1,2; 87D
CLONE NAME: M98 REF: 84
GENES: 0-1 hrs.
CROSS LIST: 67C4,5

24C
CLONE NAME: mDm101A10 REF: 1
GENES:

24CD
INSERTION: AR4-24 REF: 126
CROSS LIST: 3C1,2

25A
BREAKPOINT: T(1;2)sc19 REF: 105
CROSS LIST: 1B4-7
INSERTION: 6-1 REF: 140
CROSS LIST: 3C11,12;87D

25B
INSERTION: alpha T3.1 REF: 82
CROSS LIST: 11A
INSERTION: alpha T3.21 REF: 82
CROSS LIST: 11A

25BC
CLONE NAME: mDm109D3 REF: 1
GENES:

25C
INSERTION: P[(w,ry)D]1 REF: 176
CROSS LIST: 3C1,2; 87D
CLONE NAME: DCg-1 REF: 25
GENES: collagen-like gene
INSERTION: R401.3 REF: 115
CROSS LIST: 87D12

25D
CLONE NAME: 150-3(lambda) REF: 47
GENES: blastoderm-specific poly(A) RNA

25D1-4
CLONE NAME: MH5 REF: 6
GENES:

25F
INSERTION: 27P X/X-B REF: 200
CROSS LIST: 67B, 87D12

25F5
CLONE NAME: Gpdh 411 REF: 139
GENES: glycerol-3-phosphate dehydrogenase

26A
CLONE NAME: DmcMM115, LS1 REF: 72
GENES: vitelline

26A3-5
CLONE NAME: Gpdh 411 REF:
GENES: glycerol-3-phosphate dehydrogenase

26A7-9
CLONE NAME: unn REF: 6
GENES: beta galactosidase

26AB
CLONE NAME: A20 REF: 69
GENES:

26B
INSERTION: D4 REF: 119
CROSS LIST: 99D,87D12
INSERTION: D1 REF: 119
CROSS LIST: 99D, 87D12

27A-C
INSERTION: icarus-neo REF: 185
CROSS LIST: heatshock, neo

27C
INSERTION: 33-4 REF: 140
CROSS LIST: 3C11,12;87D
CLONE NAME: Pupal cuticle protein REF: 86
GENES: within GAR transformylase intron
CLONE NAME: unn REF: 7
GENES: Gar transformylase,synthetase,AIR synth.

27D
CLONE NAME: lambda 39-1 REF: 58
GENES:
CROSS LIST: 43A, 91C

27F
CLONE NAME: adm 125G11 REF: 34
GENES:

28A
CLONE NAME: 551 REF: 31
GENES: head specific RNA
INSERTION: BS2.7-11 REF: 115
CROSS LIST: 66D11-15, lacZ, 87D12

28C
CLONE NAME: 538 REF: 31
GENES: head specific RNA
CLONE NAME: lambda dmpt 49 REF: 58
GENES:

28D5
INSERTION: E 7-1 REF: 134
CROSS LIST: 3C1-2

28D9-12
CLONE NAME: unn REF: 8
GENES: CDNA, Kc cells

29
BREAKPOINT: T(2;3)Hm complex REF: 110
CROSS LIST: 89E

29A
CLONE NAME: pDt59R REF: 27
GENES: lys 5 tRNA locus

29A-C
BREAKPOINT: T(2;3)P10 REF: 110
CROSS LIST: 89E

29B
INSERTION: R308.1 REF: 115
CROSS LIST: 87D12

29B1-4
CLONE NAME: unn REF: 8
GENES: CDNA, Kc cells

29C
CLONE NAME: unn REF: 61
GENES: SRC homologous

30A
INSERTION: 28C-B REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28C-B REF: 121
CROSS LIST: 87D12, 67B
INSERTION: cHB delta-89 REF: 118
CROSS LIST: 87-lacZ,87D12

30A9-30B1,2
CLONE NAME: lambda Dm65 REF: 219
GENES: P6

30B
CLONE NAME: lambda dmpt 75 REF: 58
GENES:

30C
INSERTION: S6.9-3 REF: 115
CROSS LIST: 66D11-15,lacZ,87D12
INSERTION: A4-N22 REF: 126
CROSS LIST: 3C1,2

30DE
CLONE NAME: adm 136D3 REF: 34
GENES:

30EF
CLONE NAME: lambda dmpt 104 REF: 58
GENES:

31
BREAKPOINT: T(2;3) Ubx18264.1 REF: 110
CROSS LIST: 89E

31A
CLONE NAME: mDm 106A10 REF: 1
GENES:
CLONE NAME: M35 REF: 84
GENES:

31B
INSERTION: cHB delta-89 REF: 118
CROSS LIST: 87,lacZ,87D12

31C
CLONE NAME: adm 134G6 REF: 34
GENES:
CLONE NAME: adm 142H REF: 34
GENES:
CROSS LIST: 33B

31F
CLONE NAME: adm 142F4 REF: 34
GENES:
CROSS LIST: 39F

32
BREAKPOINT: T(2;3)Hm complex REF: 110
CROSS LIST: 89E

32AB
CLONE NAME: 503 REF: 31
GENES: head specific RNA

32BC
INSERTION: BS2.7 REF: 115
CROSS LIST: 66D11-15,87D12

32C-F
CLONE NAME: DmcMM99 REF: 210
GENES: oocyte-specific cDNA
CLONE NAME: E2 REF: 210
GENES: pupal cDNAs

32CD
INSERTION: cp70 delta B REF: 118
CROSS LIST: 87,lacZ,87D12
CLONE NAME: 231 REF: 63
GENES: myogenic cell RNA

32EF
CLONE NAME: Dmc MM99 REF: 72
GENES: vitelline

32F
INSERTION: g6:5 REF: 124
CROSS LIST: 68C3-5,87D12

32F-33E
CLONE NAME: REF: 218
GENES: paired

33AB
CLONE NAME: unn REF: 70
GENES: extra sex combs

33B
CLONE NAME: adm 142H REF: 34
GENES:
CROSS LIST: 31C

CLONE NAME: adm 124D9 REF: 34
GENES:
CLONE NAME: M146 REF: 84
GENES: 0-1,2-5,3-5

33B1,2
CLONE NAME: REF: 218
GENES: esc

34
BREAKPOINT: T(2;3) Ubx4.3 (madrid) REF: 110
CROSS LIST: 89E

34AB
CLONE NAME: A34 REF: 69
GENES:

34C
CLONE NAME: DmcMM99 REF: 72
GENES: vitelline, Oregon R ovaries, cDNA

34D
INSERTION: g7:4 REF: 124
CROSS LIST: 68C3-5,87D12

34E
INSERTION: P(w)11P REF: 136
CROSS LIST: 3C1,2

34EF
INSERTION: g711:1 REF: 124
CROSS LIST: 68C3-5,87D12

34F
CLONE NAME: 527 REF: 31
GENES: head specific RNA

35A
INSERTION: 15-1;15-2 REF: 140
CROSS LIST: 3C11,12;87D; 3C11,12;87D

35A4-35B1
CLONE NAME: REF: 157
GENES: Adh, outspread, no ocelli

35AB
CLONE NAME: lambda CHD1 REF: 133
GENES:

35B1-3
CLONE NAME: lambda ob 5 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: Tp(2;2)ScoR+12;In(2LR)ScoR+1;In(2L)noc2; REF: 132
Df(2L)A267;Df(2L)A379
CROSS LIST: 34A8-B1; 44C3-5; 36D3; 35B9-10; 57A8-10-60
CLONE NAME: lambda w4.04 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: Df(2L)osp29 REF: 132
CROSS LIST: 35E6

CLONE NAME: lambda w3.13 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: T(2;3)osp90 REF: 132
CROSS LIST: 89B9-11
CLONE NAME: lambda ob 9.04 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: Df(2L)A446;In(2LR)noc4;In(2L)fr27;T(2;3)Mpe REF: 132
CROSS LIST: 49B1-3; 41; 35D1-2; 86C1-2
CLONE NAME: lambda ob 3.04 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: In(2L)ScoR+17;Df(2L)osp18;Df(2L)b84a4 REF: 132
CROSS LIST: 35D1-2; 35C4-5; 35B3-4
CLONE NAME: lambda ob 2.01 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: In(2L)ScoR+11;Df(2L)A220;Df(2L)A264 REF: 132
CROSS LIST: 35D1-2; 35B9; 35B8-9

35B1,2

CLONE NAME: ob 9.04 REF: 167
GENES: no-ocelli

35B1-3

CLONE NAME: lambda ob 1.12 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: Df(2L)TE146(Z)GW8 REF: 132
CROSS LIST: 35A2-4 to B1-2
CLONE NAME: lambda gAC2 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: Df(2L)A48;Df(2L)A72 REF: 132
CROSS LIST: 35D5-7; 35B7-8
CLONE NAME: lambda ob 4.01 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: Df(2L)b84a8 REF: 132
CROSS LIST: 35B3-4
CLONE NAME: lambda ob 7.06 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: In(2L)ScoR+8;Df(2L)b81a1;Df(2L)TE146(Z)GW4; REF: 132
Df(2L)TE146(Z)GW10
CROSS LIST: 35D1-2; 34D3; 34F1-2; 34F1
CLONE NAME: lambda ob 8.10 REF: 132
GENES: Adh, noc, osp, l(2)br22
CLONE NAME: lambda ob 6 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: In(2LR)ScoR+9;In(2LR)noc7;Df(2L)nNxF2 REF: 132
CROSS LIST: 41; 46B1-2; 35B10
CLONE NAME: lambda gAC3 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: In(2L)osp59 REF: 132
CROSS LIST: 38B3-6
CLONE NAME: lambda w2.0 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: Df(2L)noc20 REF: 132
CROSS LIST: 35B2
CLONE NAME: lambda ob 10.02 REF: 132
GENES: Adh, noc, osp, l(2)br22
BREAKPOINT: Df(2L)n7813 REF: 132
CROSS LIST: 35D5-7

35B3-5

CLONE NAME: AC REF: 52
GENES: alcohol dehydrogenase

35B9-C1
CLONE NAME: REF: 149
GENES: crinkled locus
INSERTION: TE 36 REF: 149
CROSS LIST: 3C1,2-3,4; FB elements

35C36
CLONE NAME: adm 125E7 REF: 34
GENES:

35DE
INSERTION: S11.4-1 REF: 115
CROSS LIST: 66D11-15

36A
INSERTION: tAP-3 REF: 181
CROSS LIST: 35B3-5
INSERTION: unn REF: 118
CROSS LIST: 87,lacZ,87D12

36B
CLONE NAME: unn REF: 13
GENES: myosin heavy chain
CLONE NAME: unn REF: 20
GENES: walked from myosin heavy chain

36C
CLONE NAME: iH-6 REF: 172
GENES: dorsal
BREAKPOINT: In(2L)dlT; In(2L)dlH REF: 172
CROSS LIST: 21E,F; 37C1,2
INSERTION: tAP-8C,4.8 REF: 112
CROSS LIST: 35B2-3

36D1-E1
BREAKPOINT: Df(2L)VA18 REF: 35
CROSS LIST: 37C1,3

36E4-6
BREAKPOINT: Df(2L)hk18 REF: 35
CROSS LIST: 37B9-C1,2

36F
CLONE NAME: mdm 103D5 REF: 1
GENES:

37A
INSERTION: P[(w,ry)G]2 REF: 176
CROSS LIST: 3C1,2; 87D
INSERTION: unn REF: 118
CROSS LIST: 67B,lacZ,87D12

37B9-C1,2
CLONE NAME: unn REF: 35
GENES: dopa decarboxylase
BREAKPOINT: Df(2L)hk18 REF: 35
CROSS LIST: 36E4,6

37B13-C5
CLONE NAME: lambda Ddc-1 thru-20 REF: 35
GENES: dopa decarboxylase

37BC
CLONE NAME: DmcMM109 REF: 72
GENES: Oregon R ovaries
INSERTION: unn REF: 122
CROSS LIST: 35B2-3,63BC1

37C1,5
BREAKPOINT: Df(2L)TE42-1 REF: 35
CROSS LIST: 38F4
CLONE NAME: unn REF: 35
GENES: Ddc
CROSS LIST: see Table 2
BREAKPOINT: Df(2L)VA18, Df(2L)VA17, Df(2L)TE42-1 REF: 35
Df(2L)VA12
CROSS LIST: 36D1-E1, 37F5-38A1, 38C1, 2D1,2, 38B1,2-C1,2

37F5-38A1
BREAKPOINT: Df(2L)VA17 REF: 35
CROSS LIST: 37C1,5

38A6
CLONE NAME: 2E2 REF: 35
GENES:

38B
INSERTION: P(ry, HsAFP)2 REF: 141
CROSS LIST: 87D12
INSERTION: cHB delta-59 REF: 118
CROSS LIST: 87,lacZ,87D12

38B1,2-C1,2
BREAKPOINT: Df(2L)VA12 REF: 35
CROSS LIST: 37C1-5

38BC
INSERTION: tAP-19,4.8 REF: 112
CROSS LIST: 35B2-3

38C1,2-D1,2
BREAKPOINT: Df(2L)TE42-1 REF: 35
CROSS LIST: 37C1-5

38D
INSERTION: 1 REF: 123
CROSS LIST: 88F

38E
CLONE NAME: F33 REF: 143
GENES: caudal
INSERTION: unn REF: 117
CROSS LIST: 87D12

38F4
BREAKPOINT: Df(2L)TE42-1 REF: 35
CROSS LIST: 37C1-5

39
BREAKPOINT: T(2;3)Ubx19286.8m REF: 110
CROSS LIST: 89E

39B
INSERTION: 28P-C REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28P-C REF: 121
CROSS LIST: 87D12, 67B

39BC
INSERTION: E 5-5 REF: 134
CROSS LIST: 3C1-2
INSERTION: S6.9-8 REF: 115
CROSS LIST: 66D11-15,lacZ,87D12

39DE
INSERTION: H5 REF: 221
CROSS LIST: 87D12
CLONE NAME: unn REF: 54
GENES: histone

39E
INSERTION: AR4-3 REF: 113
CROSS LIST: 3C1-2, 87D12
CLONE NAME: adm 136D9 REF: 34
GENES: chromocenter
INSERTION: B4 REF: 119
CROSS LIST: 99D 87D12

39EF
INSERTION: AR4-2 REF: 113
CROSS LIST: 3C1-2, 87D12

39F
CLONE NAME: adm 142F4 REF: 34
GENES:
CROSS LIST: 31F

40
CLONE NAME: adm 106H5, 123C3, adm136D9 REF: 34
GENES: chromocenter

40F
INSERTION: AR4-3 REF: 113
CROSS LIST: 3C1-2, 87D12

41
CLONE NAME: adm 130B2 REF: 34
GENES: chromocenter
CROSS LIST: 80
BREAKPOINT: T(2;3)AntpNS+RC8 REF: 102
CROSS LIST: 84B2

41A
BREAKPOINT: T(2;3)bxd22044D REF: 110
CROSS LIST: 89E
BREAKPOINT: T(2;3)Ubx17756.180, T(2;3)Ubx18136.147 REF: 110
T(2;3)Ubx19649.18, T(2;3)UbxD1, T(2;3)bxdB231
T(2;3)CbxrvR17.22x, T(2;3)rvR17.34
CROSS LIST: 89E

41F
BREAKPOINT: T(2;3)Ubx16160.36 REF: 110
CROSS LIST: 89E

42A
CLONE NAME: 4 tRNA genes REF: 190
GENES: cluster of asn, arg, lys, ile tRNAs
CLONE NAME: mDm 106F8 REF: 1
GENES:
INSERTION: R301.1 REF: 115
CROSS LIST: 87D12
INSERTION: +411 REF: 118
CROSS LIST: 87,87D12
INSERTION: tAP-13,4.8 REF: 112
CROSS LIST: 35B2-3

42AB
INSERTION: R303.1 REF: 115
CROSS LIST: 87D12

42BC
BREAKPOINT: T(2;3)bxd x22290.11x REF: 110
CROSS LIST: 89E
CLONE NAME: lambda st11-205.16 REF: 83
GENES:

42DE
INSERTION: unn REF: 117
CROSS LIST: 87D12

42E
CLONE NAME: pDt 61 REF: 27
GENES: tRNA-lys-2
INSERTION: R305.1 REF: 115
CROSS LIST: 66D11-15, lacZ, 87D12

42EF
CLONE NAME: adm 126F7, 127A10 REF: 34
GENES:

42F
INSERTION: 28X-C REF: 180
CROSS LIST: 67B, 87D
INSERTION: S6.9-4 REF: 115
CROSS LIST: 66D11-15,lacZ,87D12

43
INSERTION: R704.1 REF: 115
CROSS LIST: 87D12

43A
CLONE NAME: lambda 39-1 REF: 58
GENES:
CROSS LIST: 27D, 91C

43A2-5
CLONE NAME: draf2 REF: 129
GENES: raf oncogene homologous
CROSS LIST: minor homology to 2F5-6

43AB
CLONE NAME: 555 REF: 31
GENES: head specific RNA

43BC
CLONE NAME: B17 REF: 69
GENES: maternal restricted transcript

43C
INSERTION: R304.1 REF: 115
CROSS LIST: 87D12

43CD
INSERTION: +411 REF: 118
CROSS LIST: 87,87D12

43DE
CLONE NAME: B45 REF: 69
GENES: maternal restricted transcript

43E
CLONE NAME: G3 REF: 135
GENES: Glyceraldehyde-3-phosphate dehydrogenase
INSERTION: S6.9-7 REF: 145
CROSS LIST: 66D11-15, lacZ, 87D12
INSERTION: g7:7 REF: 124
CROSS LIST: 68C3-5,87D12

44A
BREAKPOINT: T(Y;2;3)Mcp rvc10 complex REF: 110
CROSS LIST: 89E

44
CLONE NAME: L10 REF: 16
GENES:

44C
CLONE NAME: lambda e8 e9 REF: 71
GENES:
INSERTION: S6.9-7 REF: 145
CROSS LIST: 66D11-15, lacZ, 87D12

44C4
BREAKPOINT: T(2;3)P75Ubx 5T17.14-17 REF: 110
CROSS LIST: 89E

44CD
CLONE NAME: 536 REF: 31
GENES: head specific RNA
INSERTION: S6.9-7 REF: 115
CROSS LIST: 66D11-15,lacZ,87D12

44D
CLONE NAME: lambda DmLCP1-13 REF: 36
GENES: larval cuticle protein

44E
INSERTION: R3.9-4 REF: 115
CROSS LIST: 66D11-15,87D12

44EF
CLONE NAME: M51 REF: 84
GENES:
CROSS LIST: 64B

44F
CLONE NAME: 129E7 REF: 3
GENES:
INSERTION: cp70ZT REF: 118
CROSS LIST: 87-lacZ,87D12

45A
CLONE NAME: mDm103H10, mDm108C7 REF: 1
GENES:
CROSS LIST: 22B
INSERTION: S11.4-1 REF: 115
CROSS LIST: 66D11-15

45AB
INSERTION: F4 REF: 119
CROSS LIST: 99D,87D12

45B
CLONE NAME: M199 REF: 84
GENES: 0-3.5 hrs

45D
CLONE NAME: mDm108A8 REF: 1
GENES:
INSERTION: AR4-020(11) REF: 126
CROSS LIST: 3C1,2

45E
INSERTION: DR-18 REF: 114
CROSS LIST: 87D12,37C1,2
INSERTION: cHB delta-73 REF: 118
CROSS LIST: 87,lacZ,87D12

46B
CLONE NAME: B41 REF: 69
GENES: maternal restricted transcript

46C
INSERTION: 27 X/A-2-A;27 X/A-2-B;27 X/A-2-C;27 X/A-2-D REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: S72 REF: 187
GENES: eve
INSERTION: A4-N21 REF: 126
CROSS LIST: 3C1,2

46DF
CLONE NAME: 236 REF: 63
GENES: myogenic cell RNA

46E
CLONE NAME: 549 REF: 31
GENES: head specific RNA

47A
INSERTION: tAP-18,4.8 REF: 112
CROSS LIST: 35B2-3
INSERTION: A1-1 REF: 113
CROSS LIST: 3C1-2,87D12

47C
INSERTION: cHB delta-89 REF: 118
CROSS LIST: 87,lacZ,87D12

47D
INSERTION: P[(w,ry)E]8 REF: 176
CROSS LIST: 3C1,2; 87D
INSERTION: 27 N/P-B REF: 200
CROSS LIST: 67B, 87D12

47E
CLONE NAME: 528 REF: 31
GENES: head specific RNA

47F
CLONE NAME: unn REF: 14
GENES:

47F48D
CLONE NAME: 217 REF: 63
GENES: myogenic cell RNA

48A
CLONE NAME: unn REF: 14
GENES: engrailed

48AB
INSERTION: g7:3 REF: 124
CROSS LIST: 68C3-5,87D12

48B
INSERTION: hsp26-lacZ REF: 118
INSERTION: tAP-6 REF: 181
CROSS LIST: 35B3-5
CLONE NAME: pDt74 REF: 27
GENES: Met 2 tRNA

48C
CLONE NAME: adm 132A7 REF: 34
GENES:

48D
CLONE NAME: Dm-15 REF: 188
GENES: F1, female enriched RNA (all stages)
CROSS LIST: homology to F2 at 100E
INSERTION: DR-9 REF: 114
CROSS LIST: 87D12,37C1,2

48E
CLONE NAME: adm 135E10 REF: 34
GENES:

48E-F
INSERTION: tAP-4 REF: 181
CROSS LIST: 35B3-5

48EF
CLONE NAME: aDmS7 REF: 198
GENES: Deb-A, Deb-B

48F
CLONE NAME: 38B10 REF: 196
GENES: his tRNA genes
CLONE NAME: 543 REF: 31
GENES: head specific RNA

49A
CLONE NAME: p500, p2.2 REF: 12
GENES: calmodulin

49A12B3
CLONE NAME: unn REF: 73
GENES: possible site aristapedioid

49B
INSERTION: unn REF: 118
CROSS LIST: 67B,lacZ,87D12

49C
INSERTION: P(w)24S REF: 136
CROSS LIST: 3C1,2
CLONE NAME: mDm101D3 REF: 1
GENES:

49CD
CLONE NAME: mDm101D12 REF: 1
GENES:

49D
INSERTION: -51 REF: 118
CROSS LIST: 87,87D12
INSERTION: A1 REF: 119
CROSS LIST: 99D,87D12
INSERTION: S38M-5 REF: 115
CROSS LIST: 7E11
INSERTION: AR4-042 REF: 126
CROSS LIST: 3C1,2

49DE
CLONE NAME: adm 140D1 REF: 34
GENES:

49DEF
CLONE NAME: lambda 120 REF: 214
GENES: vestigial

49E5F1
CLONE NAME: unn REF: 73
GENES: possible site aristapedioid

49EF
INSERTION: S3.8-6 REF: 115
CROSS LIST: 66D11-15,87D12

49F
CLONE NAME: Dm1606 REF: 22
GENES: muscle specific Troponin C(ca++)
INSERTION: tAP-9,4.8 REF: 112
CROSS LIST: 35B2-3

50A
INSERTION: P[(w,ry)F]1 REF: 176
CROSS LIST: 3C1,2; 87D

50
CLONE NAME: L6 REF: 16
GENES:

50AB
CLONE NAME: 7 tRNA genes REF: 191
GENES: cluster of leu and ile tRNAs

50B
CLONE NAME: adm 142E9 REF: 34
GENES:
INSERTION: R306.1, S6.9-6 REF: 115
CROSS LIST: 87D12, lacZ, 66D11-15

50BC
CLONE NAME: 7 tRNA genes REF: 192
GENES: cluster of lys tRNAs

50C
CLONE NAME: mDm3021 REF: 1
GENES:

50CD
CLONE NAME: adm 133H7,136F9,138G8,130H8 REF: 34
GENES:

50F
CLONE NAME: unn REF: 88
GENES: double sex cognate

51A
CLONE NAME: S34 REF: 16
GENES:

51B
CLONE NAME: S14 REF: 31
GENES: head specific RNA

51CD
CLONE NAME: A19 REF: 69
GENES: maternal restricted transcript

51D
CLONE NAME: adm 134E2 REF: 34
GENES:

51DE
CLONE NAME: mDm102F11, mDm102B6 REF: 1
GENES:

51E
BREAKPOINT: T(2;3)Cbx rvR17.6F REF: 110
CROSS LIST: 89E

52A1,2
CLONE NAME: M144 REF: 84
GENES:

52A
INSERTION: DR-12 REF: 114
CROSS LIST: 87D12,37C1,2

52A3,6
CLONE NAME: M137 REF: 84
GENES:

52A-C
BREAKPOINT: T(2;3)Ubx X6000.78A4 REF: 110
CROSS LIST: 89E

52B
CLONE NAME: mDm107A2 REF: 1
GENES:
INSERTION: cHB delta-89 REF: 118
CROSS LIST: 87,lacZ,87D12
INSERTION: tAP-21,3.2 REF: 112
CROSS LIST: 35B2-3

52BC
INSERTION: P(w)5P REF: 136
CROSS LIST: 3C1,2

52CD
INSERTION: H3 REF: 221
CROSS LIST: 87D12

52D
CLONE NAME: unn REF: 88
GENES: not double sex cognate
INSERTION: SRS3.9-1 REF: 115
CROSS LIST: 66D11-15,87D12

52D6,15
CLONE NAME: M222 REF: 84
GENES: 07, 0-6

52DF
CLONE NAME: adm 139H3 REF: 34
GENES:

52F
INSERTION: S6.9-10 REF: 115
CROSS LIST: 66D11-15,lacZ, 87D12

53
CLONE NAME: L23 REF: 16
GENES:

53A
INSERTION: S38M-6 REF: 115
CROSS LIST: 7E11, M13+, 87D12

53BC
INSERTION: E 7-3 REF: 134
CROSS LIST: 3C1-2

53C
BREAKPOINT: T(2;3)Ubx 105 REF: 110
CROSS LIST: 89E

53CD
CLONE NAME: rpA1 REF: 199
GENES: rpA1
CLONE NAME: rp A1 REF: 212
GENES: ribosomal protein

39CD
CLONE NAME: unn REF: 17
GENES: ribosomal protein A1

52CD
CLONE NAME: lambda Dm32 (class A) REF: 33
GENES: amy pseudogene

53E
INSERTION: R3,9-1 REF: 115
CROSS LIST: 66D11-15,87D12

53EF
INSERTION: SB2.1-1 REF: 115
CROSS LIST: 66D11-15,87D12

53F
CLONE NAME: lambda dmpt116 REF: 58
GENES:
INSERTION: g4:1 REF: 124
CROSS LIST: 68C3-5,87D12

54A
INSERTION: 2 REF: 123
CROSS LIST: 88F
INSERTION: 2 REF: 123
CROSS LIST: 88F

54A1B1
CLONE NAME: lambda Dm 65 (class B) REF: 33
GENES: amylase duplication

54C
INSERTION: Adh,hs54c REF: 116
CROSS LIST: 35B2-3,87

54E
CLONE NAME: adm 54E.1 REF: 39
GENES: minor heat shock cDNA
BREAKPOINT: In(3LR)89/75+T(2;3)54/75,Ubx 6-26 Madrid REF: 110
CROSS LIST: 89E

54F55A
CLONE NAME: adm 110A4,132C9,132E11, 132E12 REF: 34
GENES:
CLONE NAME: adm 132G5, 134A4, 135D12 REF: 34
GENES:

55BCD
CLONE NAME: adm 110G1,110H1,132D6 REF: 34
GENES:

55F
CLONE NAME: B32 REF: 69
GENES: maternal restricted transcript

56AB
INSERTION: H2 REF: 221
CROSS LIST: 87D12

56C
CLONE NAME: DTB2 REF: 25
GENES: B tubulin

56D
CLONE NAME: pTu56 REF: 71
GENES: B 1 tubulin
INSERTION: R3.9-6 REF: 115
CROSS LIST: 66D11-15,87D12

56D412
CLONE NAME: KV 2-70A REF: 22
GENES: B tubulin

56EF
CLONE NAME: adm 135H8 REF: 34
GENES:

56F
CLONE NAME: REF: 194
GENES: 5S RNA
CLONE NAME: lambda Dmt 56-6 REF: 58
GENES: tRNA Gly
INSERTION: DR-15, DR-5 REF: 114
CROSS LIST: 87D12,37C1,2

57
INSERTION: HB4/Sc1 REF: 82
CROSS LIST: 21D

57A
INSERTION: alpha T3.21 REF: 82
CROSS LIST: 11A

57AB
INSERTION: 27P X/X-A;27P X/X-E REF: 200
CROSS LIST: 67B, 87D12

57B
INSERTION: P[(w,ry)E]6;P[(w,ry)F]4-1 REF: 176
CROSS LIST: 3C1,2; 87D; 3C1,2; 87D
INSERTION: 27S-B REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: unn REF: 12
GENES:

57C
CLONE NAME: unn REF: 92
GENES: punch
CLONE NAME: 525 REF: 31
GENES: head specific RNA
CLONE NAME: unn REF: 93
GENES: tudor

57F
INSERTION: unn REF: 117
CROSS LIST: 87D12

CLONE NAME: unn REF: 60
GENES: c-erbB, EGF receptor protein

58C
CLONE NAME: REF: 208
GENES: en-like homeobox
CROSS LIST: 95A-B

58D
INSERTION: 27 X/A-1-B REF: 200
CROSS LIST: 67B, 87D12
INSERTION: AR4-043 REF: 126
CROSS LIST: 3C1,2

58EF
INSERTION: R3.9-5 REF: 115
CROSS LIST: 87D12,37C1,2

58F
CLONE NAME: adm 132A3,135D10,135E6 REF: 34
GENES:
INSERTION: cp70 delta B REF: 118
CROSS LIST: 87,lacZ,87D12
INSERTION: tAP-7A,4.8 REF: 112
CROSS LIST: 35B2-3

59-60
BREAKPOINT: T(2;3)Mcp rvC1 complex REF: 110
CROSS LIST: 89E

59B
INSERTION: A3-1 REF: 115
CROSS LIST: 3C1-2,87D12

59C
INSERTION: tAP-7A,4.8 REF: 112
CROSS LIST: 35B2-3
BREAKPOINT: T(2;3)bxd 29315.46 REF: 110
CROSS LIST: 89E

59D
INSERTION: 27C X/X-B REF: 200
CROSS LIST: 67B, 87D12

59E
INSERTION: S11.4-1 REF: 115
CROSS LIST: 66D11-15
CLONE NAME: trc 59 REF: 201
GENES:

60A
INSERTION: R302.1 REF: 115
CROSS LIST: 87D12
CLONE NAME: adm 125C2 REF: 34
GENES:
CLONE NAME: A8 REF: 69
GENES:
CLONE NAME: adm 106H6 REF: 34
GENES:
CLONE NAME: B6 REF: 69
GENES: maternal restricted transcript

60AB
INSERTION: g1 REF: 124
CROSS LIST: 68C3-5,87D12

60B
CLONE NAME: pTu60 REF: 71
GENES: B 3 tubulin locus
BREAKPOINT: T(2;3) Mcp rv26105A REF: 110
CROSS LIST: 89E
INSERTION: BS2.7-4 REF: 115
CROSS LIST: 66D11-15,87D12

60BC
INSERTION: 27P X/X-B REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: B50 REF: 69
GENES: maternal restricted transcript

60C
INSERTION: C2 REF: 119
CROSS LIST: 99D,87D12
CLONE NAME: DTB3 REF: 25
GENES: B tubulin locus

60C1-3
BREAKPOINT: T(1;2)scS2 REF: 105
CROSS LIST: 1B4-7

60C
INSERTION: unn REF: 117
CROSS LIST: 87D12

60C6-8
CLONE NAME: KV 1-11 REF: 22
GENES: B tubulin locus

60E
INSERTION: S38Z-1 REF: 115
CROSS LIST: 7E11-7F1,2,lacZ,87D12
INSERTION: tAP-15A,4.8 REF: 112
CROSS LIST: 35B2-3

60F
CLONE NAME: lambda T-A REF: 156
GENES: telomeres
CROSS LIST: 1A, 21A, 61A, 100F, 102F
INSERTION: S3.8-4 REF: 115
CROSS LIST: 66D11-15,87D12

60F3
CLONE NAME: REF: 97
GENES: Kruppel

61A
CLONE NAME: lambda T-A REF: 156
GENES: telomeres
CROSS LIST: 1A, 21A, 60F, 100F, 102F
INSERTION: 27C+G-B REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: lambda Dm60 REF: 219
GENES: LSP-1 gamma

INSERTION: Bg61 REF: 118
CROSS LIST: 87D12, 87, lacZ
INSERTION: S38Z-5 REF: 115
CROSS LIST: 7E11-7F1,2,lacZ,87D12
CLONE NAME: lambda Dm (Can S) LSP1 gamma: 1 REF: 130
GENES: LSP1 gamma

61A1-3
CLONE NAME: mDm105F3 REF: 1
GENES:

61A-B
INSERTION: tAP-2 REF: 181
CROSS LIST: 35B3-5

61C
INSERTION: Adh hs61C REF: 116
CROSS LIST: 35B2-3, 87

61D
INSERTION: 28P-A REF: 180
CROSS LIST: 67B, 87D
INSERTION: cp70 delta B REF: 118
CROSS LIST: 87-lacZ, 87D12
INSERTION: 28P-A REF: 121
CROSS LIST: 87D12, 67B

61E
INSERTION: SRS3.9-4 REF: 115
CROSS LIST: 66D11-15,87D12

61F
INSERTION: P(ry,HsAFP)3 REF: 141
CROSS LIST: 87D12
CLONE NAME: REF: 88
GENES: double sex cognate

61F-62A
BREAKPOINT: In(3LR)Ubx 300 REF: 110
CROSS LIST: 89E

62A
INSERTION: P[(w,ry)D]2 REF: 176
CROSS LIST: 3C1,2; 87D
CLONE NAME: 5 tRNA genes REF: 195
GENES: cluster of glu tRNAs
CLONE NAME: adm 112C10 REF: 34
GENES:
CLONE NAME: lambda 48-9 REF: 58
GENES: tRNA locus

62AB
INSERTION: tAP-27 REF: 112
CROSS LIST: 35B2-3
CLONE NAME: adm 140F12 REF: 34
GENES:
CROSS LIST: 97C

62B7-12
CLONE NAME: REF: 211
GENES: apr+
CROSS LIST: 62B9

62B9
CLONE NAME: REF: 211
GENES: apr+
CROSS LIST: 62B7-12

62CD
CLONE NAME: 203 REF: 63
GENES: myogenic cell RNA

62D
CLONE NAME: adm 142F6 REF: 34
GENES:

62E
CLONE NAME: REF: 85
GENES: ribosomal protein L12 locus

63-66
CLONE NAME: S7 REF: 16
GENES:

63AB
INSERTION: HB4/Sc2 REF: 82
CROSS LIST: 21D

63AC
CLONE NAME: 227 REF: 63
GENES: myogenic cell RNA

63B
CLONE NAME: bDm 4L REF: 39
GENES: hsp 83

63BC
INSERTION: 28C-A REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28C-A REF: 121
CROSS LIST: 87D12,67B
CLONE NAME: lambda 6, pPW244,301,330 REF: 10
GENES: hsp 83

63C
INSERTION: S38Z-6 REF: 115
CROSS LIST: 7E11-7F1,2,lacZ,87D12

63E
INSERTION: 23-3 REF: 140
CROSS LIST: 3C11,12;87D
CLONE NAME: IMP-E2 REF: 164
GENES: 20-hydroxyecdysone inducible

63F
CLONE NAME: adm 63 F.1 REF: 39
GENES: minor hsp

63F64A

CLONE NAME: adm 140C11 REF: 34
GENES:
CROSS LIST: 5EF

64B

CLONE NAME: IMP-L2 REF: 164
GENES: 20-hydroxyecdysone inducible
INSERTION: P[(w,ry)F]3 REF: 176
CROSS LIST: 3C1,2; 87D
CLONE NAME: M51 REF: 84
GENES:
CROSS LIST: 44EF
CLONE NAME: Drsrc REF: 60
GENES: SRC homologous
CLONE NAME: pDMRS64B REF: 76
GENES: ras oncogene

64BC

CLONE NAME: mDm104C1 REF: 1
GENES:

64C

INSERTION: R405 REF: 115
CROSS LIST: 87D12
INSERTION: tAP-7B,4.8 REF: 112
CROSS LIST: 35B2-3
CLONE NAME: DHSV4 REF: 60
GENES: RAS homologous

64D

BREAKPOINT: In(3L)HR15 REF: 110
CROSS LIST: 68C1,5

64E

BREAKPOINT: In(3LR)3 ry64 REF: 110
CROSS LIST: 87D

64F

CLONE NAME: lambda dmpt 120 REF: 58
GENES:
CLONE NAME: mDm 106E3 REF: 1
GENES:
CLONE NAME: adm 126B4 REF: 34
GENES:
CROSS LIST: 66C
CLONE NAME: adm 135G4 REF: 34
GENES:
CROSS LIST: 65A
INSERTION: SB2.1-3 REF: 115
CROSS LIST: 66D11-15,87D12
INSERTION: cHB delta -73 REF: 118
CROSS LIST: 87,lacZ,87D12

65A

INSERTION: SB2.1-3 REF: 115
CROSS LIST: 66D11-15, 87D12
CLONE NAME: adm 135G4 REF: 34
GENES:
CROSS LIST: 64F

65AB
INSERTION: w47.1 N8 REF: 120
CROSS LIST: 3C1-2

65B
CLONE NAME: IMP-L3 REF: 164
GENES: 20-hydroxyecdysone inducible

65C
CLONE NAME: adm 111F10 REF: 34
GENES:

65D
INSERTION: 28A-B REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28A-B REF: 120
CROSS LIST: 87D12,67B

65D-66B
INSERTION: S38Z-7 REF: 145
CROSS LIST: 7E11, lacZ, 87D12

65F
INSERTION: g711:3 REF: 124
CROSS LIST: 68C3-5,87D12

66A
INSERTION: 28X-D REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28X-D REF: 121
CROSS LIST: 87D12,67B

66B
CLONE NAME: REF: 216
GENES: leu tRNA
BREAKPOINT: Tp(3)P47 REF: 110
CROSS LIST: 89E

66C
CLONE NAME: IMP-E1 REF: 164
GENES: 20-hydroxyecdysone inducible
CLONE NAME: adm 126B4 REF: 34
GENES:
CROSS LIST: 64F
BREAKPOINT: Tp(3)bxd 100 REF: 110
CROSS LIST: 89E

66CD
CLONE NAME: adm 106E3 REF: 34
GENES:

66D
CLONE NAME: 507, 547 REF: 31
GENES: head specific RNA
INSERTION: DR-17 REF: 114
CROSS LIST: 87D12,37C1,2

66D9-10
CLONE NAME: .8247,30152,3019 REF: 32
GENES:

66D10-15
CLONE NAME: unn REF: 57
GENES:

66D11-15
CLONE NAME: unn REF: 44
GENES: chorion protein genes
CROSS LIST: see Table 2

66D12-15
CLONE NAME: AB REF: 148
GENES: gene s18-1, s15-1, s19-1

66E1,2
INSERTION: Tf(3L)Ga6.0-1 REF: 111
CROSS LIST: 35B2-3

66E
INSERTION: AR4-012 REF: 126
CROSS LIST: 3C1,2

66F
CLONE NAME: lambda dmpt 121 REF: 58
GENES:

67A5-7-67B1,2
CLONE NAME: unn REF: 22
GENES:

67B
CLONE NAME: overlapping REF: 171
GENES: hsp22, hsp23, hsp26, hsp27, 1, 2, 3
CLONE NAME: lambda 88 REF: 10
GENES: loci of hsp 22,23,26, and 28
CLONE NAME: lambda Dmp 67 REF: 43
GENES: hsp and flanking transcripts
CLONE NAME: J1 REF: 42
GENES: includes hsp 28,23,26

67BC
INSERTION: 27P X/X-D REF: 200
CROSS LIST: 67B, 87D12

67C
CLONE NAME: DTA2 REF: 25
GENES: delta tubulin

67C4,5
CLONE NAME: M98 REF: 84
GENES: 0-1 hrs.
CROSS LIST: 24AB

67DE
CLONE NAME: gt11.205.23 REF: 83
GENES:

67E
INSERTION: 27 C X/X-A REF: 200
CROSS LIST: 67B, 87D12

67F
CLONE NAME: l(3) 1902 REF: 209
GENES:

68A
INSERTION: R7.7-1 REF: 115
CROSS LIST: 66D11-15,87D12
BREAKPOINT: Tp(3)Ubx P20 REF: 110
CROSS LIST: 89E

68B5-C2
BREAKPOINT: Df(3L)vin 7 REF: 1
CROSS LIST: 68C10-12

68C
INSERTION: 28X-A REF: 180
CROSS LIST: 67B, 87D
CLONE NAME: pkdm1H2, adm 134C10,pkdm2C1,2G6 REF: 34
GENES: intermolt IV, III, II RNA
INSERTION: 28X-A REF: 121
CROSS LIST: 87D12,67B
INSERTION: g7:5, g71:2 REF: 124
CROSS LIST: 68C3-5,87D12

68C1-5
CLONE NAME: lambda cDm2021 REF: 1
GENES:
BREAKPOINT: In(3L)HR15 REF: 1
CROSS LIST: 64D

68C3-7
CLONE NAME: REF: 153
GENES: Sgs proteins
CLONE NAME: unn REF:
GENES: Sgs protein genes
CROSS LIST: see Table 2
BREAKPOINT: Df(3L)vin 3 REF: 1
CROSS LIST: 68E3,4

68C4-6
CLONE NAME: aDm 1501-10 REF: 1
GENES: sgs 3,7,8

68C7-15
CLONE NAME: mDm148F7 REF: 1
GENES:

68C10-12
CLONE NAME: unn REF: 1
GENES:
BREAKPOINT: Df(3L)vin 7 REF: 1
CROSS LIST: 68B5-C2

68D
INSERTION: S38Z-3 REF: 115
CROSS LIST: 7E11-7F1,lacZ,87D12

68E
CLONE NAME: lambda Dm104 REF: 219
GENES: LSP-2

BREAKPOINT: Tp(3)Ubx7 P20 REF: 110
CROSS LIST: 89E
INSERTION: g6:4 REF: 124
CROSS LIST: 68C3-5,87D12

68E3,4
BREAKPOINT: Df(3L)vin 3 REF: 1
CROSS LIST: 68C3-7

68EF
CLONE NAME: adm 133H1 REF: 34
GENES:

69
CLONE NAME: L3g REF: 16
GENES:

69C3-4
BREAKPOINT: In(3LR)bxd 113 REF: 110
CROSS LIST: 89E

69CD
INSERTION: tAP-12,4.8 REF: 112
CROSS LIST: 35B2-3

69D
CLONE NAME: M37 REF: 84
GENES:

69F
CLONE NAME: 270 REF: 63
GENES: myogenic cell RNA

70A
CLONE NAME: IMP-L1 REF: 164
GENES: 20-hydroxyecdysone inducible
CLONE NAME: 63H5 REF: 196
GENES: asp tRNA genes
CLONE NAME: adm 107A4 REF: 34
GENES:

70AB
INSERTION: S3.8-3 REF: 115
CROSS LIST: 66D11-15,87D12
CLONE NAME: adm 128C11, 132B3 REF: 34
GENES:

70BC
CLONE NAME: B20 REF: 69
GENES: maternal restricted transcription
CLONE NAME: pDt 55 REF: 27
GENES: Val 4 tRNA locus

70C
CLONE NAME: adm 29D11 REF: 34
GENES:
INSERTION: S11.4-2 REF: 115
CROSS LIST: 66D11-15

70D
BREAKPOINT: In(3LR)Cbx rvR17.42 REF: 110
CROSS LIST: 89E

70D1-3
CLONE NAME: lambda Dm117 REF: 219
GENES: P1

70D4,5
CLONE NAME: Q111,Q131,Q112 REF: 81
GENES: frizzled locus

71A
CLONE NAME: 2-5 (lambda) REF: 47
GENES: gastrula differential poly(A) RNA

71AB
INSERTION: -51 REF: 118
CROSS LIST: 87,87D12
CLONE NAME: adm 123C4 REF: 34
GENES:

71C
INSERTION: S38M-1 REF: 115
CROSS LIST: 7E11-7F1,2, M13+, 87D12

71C3,4D1,2
CLONE NAME: REF: 26
GENES: EIP 28/29 locus

71CD
BREAKPOINT: T(1;3)sc260-15 REF: 105
CROSS LIST: 1B4,5

71CE
CLONE NAME: lambda cDm 20-24 REF: 24
GENES: ecdysone induced late puff

71DE
CLONE NAME: adm 134A9,134A11,134C11,pkdm 46B7 REF: 34
GENES: late I RNA
CLONE NAME: pkdm 38C9, 38C4 REF: 34
GENES: late II,III RNA

71F
INSERTION: tAP-11,4.8 REF: 112
CROSS LIST: 35B2-3
BREAKPOINT: In(3LR)AntpPW REF: 102
CROSS LIST: 84B2
INSERTION: g7:2 REF: 124
CROSS LIST: 68C3-5,87D12

72BC
CLONE NAME: 557 REF: 31
GENES: head specific RNA

72D11-72E1
BREAKPOINT: In(3LR)bxd 106 REF: 110
CROSS LIST: 89E

72DE
CLONE NAME: lambda dmpt 115 REF: 58
GENES:

73A
CLONE NAME: REF: 88
GENES: double sex cognate, not transformer
CLONE NAME: REF: 87
GENES: transformer

73A3-4
CLONE NAME: lambda stR4 REF: 197
GENES: scarlet

73A3-B1,2
CLONE NAME: stdb-st tra DIS-5 cABL std5 REF: 201
GENES:

73A3,4-73B
CLONE NAME: REF: 215
GENES: tra

73B
INSERTION: Pc[ry(delta0-1)]2 REF: 137
CROSS LIST: 87D12
INSERTION: 27S-D REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: Dash REF: 60
GENES: Abelson SRC homologous

73D
CLONE NAME: adm 73D.1 REF: 39
GENES: minor heat shock locus

73DEF
CLONE NAME: 521 REF: 31
GENES: head specific RNA

73E
INSERTION: P(ry,HsAFP)6 REF: 141
CROSS LIST: 87D12

74
BREAKPOINT: In(3)Ubx 130 (TM2) complex REF: 110
CROSS LIST: 89E

74EF
CLONE NAME: REF: 56
GENES: early ecdysone responding puff

74F-75A
BREAKPOINT: In(3LR)Antp NS+RC4 REF: 102
CROSS LIST: 84B2

75
CLONE NAME: S39 REF: 16
GENES:

75C
INSERTION: P[(w,ry)H]2-2 REF: 176
CROSS LIST: 3C1,2; 87D

BREAKPOINT: In(3LR)89/75+T(2;3)54/75,Ubx6.26 Madrid REF: 110
CROSS LIST: 89E
CLONE NAME: adm 135F3 REF: 34
GENES:

75CD
INSERTION: R502.1 REF: 115
CROSS LIST: 87D12

75D
CLONE NAME: trc 75 REF: 201
GENES:
INSERTION: R706.1 REF: 115
CROSS LIST: 87D12

76A
CLONE NAME: adm 132D11 REF: 34
GENES:
INSERTION: A38M-4 REF: 115
CROSS LIST: 7E11-7F1,2, M13+, 87D12

76DE
CLONE NAME: B48 REF: 69
GENES: maternal restricted transcript

76F
CLONE NAME: mDm 104G3 REF: 1
GENES:

77A
INSERTION: cHB delta-73 REF: 118
CROSS LIST: 87, lacZ, 87D12

77DE
INSERTION: P(ry, HsAFP)1 REF: 141
CROSS LIST: 87D12

78BC
INSERTION: R603.1 REF: 115
CROSS LIST: 87D12

78CD
INSERTION: P[(w,ry)H]1 REF: 176
CROSS LIST: 3C1,2; 87D

78D
INSERTION: cHB delta-89 REF: 118
CROSS LIST: 87-lacZ, 87D12
INSERTION: cHB delta-89 REF: 118
CROSS LIST: 87-lacZ, 87D12

78D7,8
CLONE NAME: lambda Dm-208B REF: 182
GENES: polycomb

79-80
CLONE NAME: REF: 222
GENES: ribosomal protein-21

79B
CLONE NAME: unn REF: 12
GENES:

79C-E
BREAKPOINT: In(3LR)Cbx rvR17.44V REF: 110
CROSS LIST: 89E

79E
INSERTION: +411 REF: 118
CROSS LIST: 87,87D12

79E1,2
CLONE NAME: 13E5 REF: 16
GENES:

79F
INSERTION: 8-1 REF: 140
CROSS LIST: 3C11,12;87D

80
CLONE NAME: adm 130B2 REF: 34
GENES: chromocenter
CROSS LIST: 41

80A
INSERTION: B1,F2 REF: 119
CROSS LIST: 99D,87D12

80B
BREAKPOINT: In(3LR)Cbx rvR17.16R REF: 110
CROSS LIST: 89E

80C
CLONE NAME: REF: 8
GENES: Kc cells

80F
CLONE NAME: adm 139A10 REF: 34
GENES: chromocenter
BREAKPOINT: In(3LR)bxd 19409.2X REF: 110
CROSS LIST: 89E

81
CLONE NAME: adm 128F12 REF: 34
GENES: chromocenter
BREAKPOINT: In(3)Hab rvFC51, In(3)Ubx 125 REF: 110
In(3)Mcp rvC1 complex
CROSS LIST: 89E
BREAKPOINT: In(3R)ry 54, In(3R)ry PS11136 REF: 110
CROSS LIST: 87D

82A
CLONE NAME: S6-7 REF: 22
GENES:
INSERTION: w47.4L REF: 120
CROSS LIST: 3C1-2

82B
INSERTION: Adh hs82b REF: 116
CROSS LIST: 35B2-3,87

82BC
INSERTION: tAP-8B,4.8 REF: 112
CROSS LIST: 35B2-3
INSERTION: g4:3 REF: 124
CROSS LIST: 68C3-5,87D12

82C
INSERTION: 28X-E REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28X-E REF: 121
CROSS LIST: 87D12,67B

82E
CLONE NAME: pDm6A65, Dm 525 REF: 151
GENES: U1 snRNA
CROSS LIST: 21D, 95C
CLONE NAME: lambda Dm525 REF: 220
GENES: 2 SnRNA U1 pseudogenes (inverted)

82F
INSERTION: 5-1 REF: 140
CROSS LIST: 3C11,12;87D
CLONE NAME: 506 REF: 31
GENES: head specific RNA
INSERTION: AR4-025 REF: 126
CROSS LIST: 3C1,2

83A
CLONE NAME: adm 136E4 REF: 34
GENES:
INSERTION: unn REF: 117
CROSS LIST: 87D12

83A,B
CLONE NAME: pDt 66R2 REF: 27
GENES: Lys 5 tRNA locus

83AB
CLONE NAME: adm 140E12 REF: 34
GENES:

83B
INSERTION: 27S-E REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: adm 123G4 REF: 34
GENES:

83BC
INSERTION: SB2.1-2 REF: 115
CROSS LIST: 66D11-15,87D12

83C
CLONE NAME: mDm 105 B9 REF: 1
GENES:

83CD
CLONE NAME: B21, B31 REF: 69
GENES: maternal restricted transcript

83D4,5
CLONE NAME: B31 REF: 69
GENES:
BREAKPOINT: Tp(3;3)Dfd, Tp(3;3)DfdTRX1 REF: 69
CROSS LIST: 84A4,5, 98F1,2

83F
CLONE NAME: adm 140C1 REF: 34
GENES:
INSERTION: R3.9-3,9/ S38M-2 REF: 115
CROSS LIST: 87D12/ 7E11-7F1,2, M13+, 87D12

84A1
BREAKPOINT: Df(3R)Scr REF: 102
CROSS LIST: 84B2

84A1,2
BREAKPOINT: Df(3R)JA99 REF: 102
CROSS LIST: 84B2

84A4,5
BREAKPOINT: Tp(3;3)Dfd REF: 69
CROSS LIST: 83D4,5

84A1
CLONE NAME: REF: 213
GENES: zen

84A4,5-84C1,2
CLONE NAME: REF: 49
GENES: antennapedia complex

84A,B
CLONE NAME: pDt 12, pDt 39 REF: 27
GENES: Lys 5 tRNA Ylocus

84B
INSERTION: 27P X/X-E REF: 200
CROSS LIST: 67B, 87D12

84B1
BREAKPOINT: Df(3R)ASCB REF: 102
CROSS LIST: 84B2

84B
CLONE NAME: DTA 1 REF: 25
GENES: delta tubulin

84B2
CLONE NAME: unn REF: 102
GENES: Antp
BREAKPOINT: T(2;3)AntpNS+RC8, Df(3R)Scr REF: 102
CROSS LIST: 41, 84A1
BREAKPOINT: In(3R)Antp LC, IN(3LR)AntpPW REF: 102
In(3LR)Antp NS+RC4, Df(3R)JA99, In(3R)Antp73B
CROSS LIST: 91F-92A, 71F, 74F-75A, 84A1,2, 84D1,2
BREAKPOINT: Df(3R)ASCB, In(3R)Hu, Df(3R)Antp NS+RC7 REF: 102
In(3R)AntpB
CROSS LIST: 84B1, 84F2,3, 85F, 84D, 85E

84B2-C1
CLONE NAME: unn REF: 102
GENES: hu
BREAKPOINT: In(3R)Hu REF: 102
CROSS LIST: 84F2,3, 86B4-C1

84B3-6
CLONE NAME: lambda Dm 2.55a REF: 22
GENES: delta tubulin

84B3-C1,2
CLONE NAME: REF: 48
GENES: double sex

84BC
CLONE NAME: adm 123D11 REF: 34
GENES:
INSERTION: S3.8-1 REF: 116
CROSS LIST: 66D11-15,87D12

84C
INSERTION: unn REF: 117
CROSS LIST: 87D12

84C8
CLONE NAME: DmA 3a,4a,4b,5a,5b REF: 4
GENES: glucose dehydrogen., eclosion genes A-D

84D
INSERTION: 28X-B REF: 180
CROSS LIST: 67B, 87D
CLONE NAME: lambda 49-4 REF: 58
GENES: tRNA
CROSS LIST: 85C, 90C
INSERTION: unn REF: 118
CROSS LIST: 67B-lacZ,87D12
CLONE NAME: mDm 104H7 REF: 1
GENES:
CLONE NAME: pDt 78 RC REF: 27
GENES: Val 3b tRNA locus
CLONE NAME: DTA 4 REF: 25
GENES: delta tubulin locus
BREAKPOINT: T(2;3)bxd DB6 REF: 110
CROSS LIST: 89E
INSERTION: 28X-B REF: 121
CROSS LIST: 87D12,67B
BREAKPOINT: Df(3R)Antp NS+RC7 REF: 101
CROSS LIST: 84B2

84D1,2
BREAKPOINT: In(3R)Antp 73b REF: 102
CROSS LIST: 84B2

84D3,4
CLONE NAME: 1,3,6,10 REF: 19
GENES: overlaps Val 3b tRNA

84D4-8
CLONE NAME: lambda Dm 5-1 REF: 22
GENES: delta tubulin

84E
CLONE NAME: IMP-E3 REF: 164
GENES: 20-hydroxyecdysone inducible
INSERTION: cHB delta-194, g6:2 REF: 118
CROSS LIST: 87,lacZ,87D12
INSERTION: g6:2 REF: 124
CROSS LIST: 68C3-5,87D12

84E1,2
CLONE NAME: REF: 48
GENES: double sex locus and flanking

84E11-12 - F4-5
CLONE NAME: unn REF: 50
GENES:

84F
CLONE NAME: 14C4 REF: 196
GENES: cluster of arg and asn tRNAs
CLONE NAME: B34 REF: 69
GENES: maternal restricted transcript
BREAKPOINT: In(3)Mcp rv29175.10 REF: 110
CROSS LIST: 89E
INSERTION: 1 REF: 123
CROSS LIST: 88F
INSERTION: g71:3 REF: 124
CROSS LIST: 68C3-5,87D12
INSERTION: A3 REF: 119
CROSS LIST: 99D,87D12

84F2,3
CLONE NAME: unn REF: 50
GENES:
BREAKPOINT: In(3R)Hu REF: 102
CROSS LIST: 84B2-C1

85
CLONE NAME: adm 123B10 REF: 34
GENES:

85A
INSERTION: 27 X/A-1-A REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: lambda 50-8 REF: 58
GENES: tRNA locus
INSERTION: R309.1 REF: 115
CROSS LIST: 87D12

85AB
INSERTION: 814,CH8 REF: 119
CROSS LIST: 99D,87D12

85BC
INSERTION: 24-1 REF: 140
CROSS LIST: 3C11,12;87D

85C
CLONE NAME: lambda 49-4 REF: 58
GENES: tRNA
CROSS LIST: 84D, 90C
CLONE NAME: p85C REF: 27

GENES: Arg tRNA
CLONE NAME: lambda m 1:2 REF: 6
GENES:

85D
INSERTION: P[(w,ry)H]3 REF: 176
CROSS LIST: 3C1,2; 87D
INSERTION: P(ry,HsAFP)5 REF: 141
CROSS LIST: 87D12
INSERTION: BS2.7-7 REF: 115
CROSS LIST: 66D11-15,87D12
CLONE NAME: KV 1-22 REF: 22
GENES: delta tubulin locus
CLONE NAME: 542 REF: 16
GENES:
CLONE NAME: DHSV 7 REF: 60
GENES: RAS homologous
CLONE NAME: REF: 71
GENES: delta 2 tubulin locus
INSERTION: g6:1 REF: 124
CROSS LIST: 68C3-5,87D12

85D,E
CLONE NAME: REF: 88
GENES: double sex cognate

85D6-12
CLONE NAME: DTB 4 REF: 25
GENES: delta tubulin locus

85E
CLONE NAME: M253 REF: 84
GENES: 0-1 hr.
CLONE NAME: DTA 3 REF: 25
GENES: delta tubulin
CLONE NAME: mDm 3008 REF: 1
GENES:
BREAKPOINT: In(3R)AntpB REF: 50
CROSS LIST: 84B2

85E6-10
CLONE NAME: lambda Dm 5-22 REF: 22
GENES: delta tubulin locus

85E10-15
CLONE NAME: cDm51 REF: 162
GENES: metallothionein
CLONE NAME: lambda Dm13 REF: 163
GENES: metallothionein
CLONE NAME: lambda Dm13 REF: 183
GENES: metallothionein (Mtn)

85F
INSERTION: P[(w,ry)H]2-1 REF: 176
CROSS LIST: 3C1,2; 87D
INSERTION: tAP-15B,4.8 REF: 112
CROSS LIST: 35B2-3
BREAKPOINT: In(3R)Hu REF: 50
CROSS LIST: 84B2

86

CLONE NAME: adm 35E6 REF: 34
GENES:
CLONE NAME: S35g REF: 16
GENES:

86A

INSERTION: 20-1 REF: 140
CROSS LIST: 3C11,12;87D

86B4-C1

BREAKPOINT: In(3R)Hu REF: 50
CROSS LIST: 84B2-C1

86C

INSERTION: P[(w,ry)F]2 REF: 176
CROSS LIST: 3C1,2; 87D

86D

INSERTION: R311.1 REF: 115
CROSS LIST: 87D12
INSERTION: g7:6 REF: 124
CROSS LIST: 68C3-5,87D12

86E

INSERTION: S38Z-2 REF: 115
CROSS LIST: 7E11-7F1,2,lacZ,87D12

87A

CLONE NAME: GB REF: 42
GENES: Hsp 70, Sn cell DNA
CLONE NAME: 56H8 REF: 41
GENES: hsp 70 and flanking
INSERTION: R307.1 REF: 115
CROSS LIST: 87D12

87A7

CLONE NAME: pPw 223 REF: 10
GENES: hsp 70
CLONE NAME: REF: 39
GENES: hsp 70 subclone

87AB

INSERTION: 28A-C REF: 180
CROSS LIST: 67B, 87D
CLONE NAME: 540 REF: 16
GENES:
INSERTION: 28A-C REF: 121
CROSS LIST: 87D12,67B

87B

BREAKPOINT: In(3)Ubx 882 REF: 109
CROSS LIST: 89E

87C

CLONE NAME: G3 REF: 42
GENES: hsp 70, Sn cell DNA

87C1

CLONE NAME: 132E3 REF: 41
GENES: hsp 70 and flanking

87C1-3
BREAKPOINT: Df(3R)ry 81 REF: 110
CROSS LIST: 87E1,2

87C7-8
BREAKPOINT: Df(3R)kar SZ11 REF: 110
CROSS LIST: 87E1,2

87C10
CLONE NAME: pPW232, pPW229 REF: 10
GENES: hsp 70

87CD
INSERTION: C1-1 REF: 113
CROSS LIST: 3C1-2

87CF
CLONE NAME: adm 125G5 REF: 34
GENES:
CROSS LIST: 94D

87D
CLONE NAME: mG31 REF: 42
GENES: Hsc 70
CLONE NAME: unn REF: 144
GENES: rosy ace, 1512
BREAKPOINT: In(3LR)3 ry64, In(3R)ry54, In(3R)ryPS11136 REF: 144
CROSS LIST: 64E, 81

87D1,2
BREAKPOINT: Df(3R)ry75 REF: 144
CROSS LIST: 87D14

87D2-4
BREAKPOINT: Df(3R)ry614, Df(3R)ry1301, Df(3R)ry1402 REF: 144
CROSS LIST: 87D14, 87E1,2

87D3,4
BREAKPOINT: Df(3R)ry 1607 REF: 144
CROSS LIST: 87E1,2

87D5-87E5
CLONE NAME: REF: 5
GENES: rosy and Ace

87D5,6
BREAKPOINT: Df(3R)ry 1608 REF: 144
CROSS LIST: 87E1,2

87D6-8
CLONE NAME: unn REF: 144
GENES: rosy ace
BREAKPOINT: Df(3R)ry 619 REF: 144
CROSS LIST: 87E12,F1

87D8-10
CLONE NAME: unn REF: 144
GENES: rosy ace
BREAKPOINT: Df+In(3R)kar 1g27 REF: 144
CROSS LIST: 99E1-F1

87D12-13
CLONE NAME: unn REF: 144
GENES: rosy ace
CROSS LIST: see Table 2
BREAKPOINT: T(3;4)ryP51149 REF: 144
CROSS LIST: 4th het

87D12-14
BREAKPOINT: In(3)Cbx rv21988B REF: 144
CROSS LIST: 89

87D14
CLONE NAME: unn REF: 144
GENES: rosy ace
BREAKPOINT: Df(3R)ry75 REF: 144
CROSS LIST: 87D12, 87D2,4

87D17
CLONE NAME: unn REF: 144
GENES: rosy ace

87E
INSERTION: unn REF: 117
CROSS LIST: 87D12
CLONE NAME: unn REF: 12
GENES:
INSERTION: unn REF: 117
CROSS LIST: 87D12

87E1,2
CLONE NAME: unn REF: 110
GENES: rosy ace
BREAKPOINT: Df(3R)ry 81, Df(3R)karSZ11,In(3)Cbx+R1 REF: 110
Df(3R)ry1402, Df(3R)ry1301
Df(3R)ry1607, Df(3R)ry1608
CROSS LIST: 87C1,3, 87C7,8, 89E, 87D2,4, 87D3,4, 87D5,6
BREAKPOINT: Df(3R)126c REF: 110
CROSS LIST: 87F11,12

87E5,6
BREAKPOINT: Df(3R)1C4a REF: 110
CROSS LIST: 87E11,F1

87E11,F1
BREAKPOINT: Df(3R)1C4a REF: 110
CROSS LIST: 87E5,6

87E12,F1
BREAKPOINT: Df(3R)ry 619 REF: 110
CROSS LIST: 87D6,8

87E-F
BREAKPOINT: In(3)Cbx rv21987A REF: 110
CROSS LIST: 89

87F
BREAKPOINT: In(3)Cbx wt REF: 110
CROSS LIST: 89E
BREAKPOINT: T(2;3)Cbx rvR17.6F REF: 110
CROSS LIST: 89E
INSERTION: R308.2, R404.1 REF: 115
CROSS LIST: 87D12
INSERTION: R404.1 REF: 115
CROSS LIST: 87D12
INSERTION: DRI-15 REF: 114
CROSS LIST: 87D12,37C1,2

87F11,12
BREAKPOINT: Df(3R)126c REF: 110
CROSS LIST: 87E12

87F-88A
BREAKPOINT: In(3)Ubx80 REF: 110
CROSS LIST: 89E

88
CLONE NAME: S32 REF: 16
GENES:

88A
INSERTION: cHB delta-23 REF: 118
CROSS LIST: 87-lacZ,87D12

88B
CLONE NAME: adm 88B.1 REF: 39
GENES: minor heat shock cDNA
BREAKPOINT: In(e)Ubx 12.5 (Madrid) REF: 110
CROSS LIST: 89E

88C
CLONE NAME: mDm 104D12 REF: 1
GENES:
INSERTION: S6.9-5 REF: 115
CROSS LIST: 66D11-15,lacZ,87D12

88C4
BREAKPOINT: In(e)56A62Y REF: 110
CROSS LIST: 89E

88D
CLONE NAME: REF: 88
GENES: double sex cognate

88E
CLONE NAME: mG34 REF: 42
GENES: hsc 70
INSERTION: BS2.7-9, R401.2 REF: 115
CROSS LIST: 66D11-15,87D12/ 87D12

88F
CLONE NAME: REF: 169
GENES: muscle specific tropomysin
CLONE NAME: REF: 170
GENES: actin
CLONE NAME: unn REF: 11

GENES: actin
CLONE NAME: lambda dmpt 73 REF: 58
GENES: tropomyosin
INSERTION: cp70 delta B REF: 118
CROSS LIST: 87-lacZ,87D12
INSERTION: S6.9-1 REF: 115
CROSS LIST: 66D11-15,lacZ,87D12

88F2-5
CLONE NAME: lambda DM 85 REF: 22
GENES: 3 tropomyosin loci

89
CLONE NAME: unn REF: 110
GENES: pic
BREAKPOINT: In(3)Cbx rv21987A, In(3)Cbx rv21988B REF: 110
CROSS LIST: 87E-F,87D12-14

89A-B
BREAKPOINT: In(3)bxd27830.C5A REF: 110
CROSS LIST: 89E

89A
BREAKPOINT: In(3)Cbx 3 (Cbx-like) REF: 110
CROSS LIST: 89E
INSERTION: +204 REF: 118
CROSS LIST: 87,87D12
INSERTION: B1-1 REF: 113
CROSS LIST: 3C1-2

89B
INSERTION: 28-2 REF: 140
CROSS LIST: 3C11,12;87D
INSERTION: 27P X/X-C REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: pDt 14 REF: 27
GENES: Val 4 Phe 2 tRNA
BREAKPOINT: In(3)Camel REF: 110
CROSS LIST: 89E
INSERTION: BS2.7-6 REF: 115
CROSS LIST: 66D11-15,87D12
INSERTION: g7:8 REF: 124
CROSS LIST: 68C3-5,87D12

89B4
CLONE NAME: REF: 207
GENES: sb

89B21
BREAKPOINT: T(1;3)sta REF: 108
CROSS LIST: 2B3,4

89BC
CLONE NAME: EV27 REF: 23
GENES:

89C
BREAKPOINT: In(3)bxd 183 REF: 110
CROSS LIST: 89E

89C1,2

BREAKPOINT: T(2;3)P10 REF: 110
CROSS LIST: 89E

89E

BREAKPOINT: T(2;3)bxdB231,T(2;3)P75Ubx5T17.14-17 REF: 110
T(2;3)CbxrvR17.6F,T(2;3)UbxX6000.78A4
T(2;3)Ubx105
CROSS LIST: 41A,44C4,51E,52A-C,53C
BREAKPOINT: In(3)89E/97F-97A, Cbx rv21560.60 REF: 110
Tp(3)Vno, Tp(3)abx, In(3)Ubxx,
In(3)Mcp rvB315
CROSS LIST: 90A
BREAKPOINT: T(2;3)McprvC1complex, T(2;3)bxd29315.46, REF: 110
Tp(3)bxd100, Tp(3)UbxP20, Tp(3)Ubx7P20,
Tp(3)P47, In(3LR)bxd113, In(3LR)CbxrvR17.42
CROSS LIST: 59-60, 59C, 66C, 66C, 68A, 68E,66B,69C3,4,70D
BREAKPOINT: In(3)Ubx 882 REF: 109
CROSS LIST: 87B
BREAKPOINT: In(3)Ubx 130 (TM2) complex REF: 110
CROSS LIST: 74
BREAKPOINT: In(3)Cbx rvR17.5E, Tp(3)P47, REF: 110
In(3)Ubx130(TM2)complex, Tp(e)Vno,
Tp(3)McpB277, In(3)Ubx19286.76
CROSS LIST: 96F-97A, 96F-97A, 97CD, 97D, 98D-F
BREAKPOINT: In(3LR)Ubx130TM2 complex REF: 110
In(3LR)bxd106, In(3LR)bxd19409.2x
CROSS LIST: 74, 72D11-72E1, 80F
BREAKPOINT: In(3)Hab rvFC51, In(3)Ubx125, REF: 110
In(3)Mcp rvC1 complex, In(3LR)Cbx rvR17.44V
In(3LR)Cbx rvR17.16R
CROSS LIST: 81, 79C-E, 80B, 84D
BREAKPOINT: In(3)bxd27830.C5A, In(3)Ubx5.12 Madrid, REF: 110
T(1;3)bxd111, In(3)Camel, In(3)bxd183
In(e)56A62 (szedged)
CROSS LIST: 89A,B, 90A, 90B2, 89B, 89C, 88C4
BREAKPOINT: In(3LR)89/75+T(2;3)54/75,Ubx 6-26 Madrid REF: 110
T(1;3)Ubx21560.8A Complx,T(1;3)Uab5
T(1;3)bxd111,Dp(3;1)P68,T(1;3)P115
CROSS LIST: 54E,5B,1F,4D,20
BREAKPOINT: In(3)Ubx 1928.16N, T(3;4)bxd101, REF: 110
T(3;4)UbxA complex, T(3;4)Cbx rv R17.40R
CROSS LIST: 99, 101F, 102, 4
BREAKPOINT: T(2;3)CbxrvR17.34,T(2;3)rvR17.22x REF: 110
T(2;3)bxd22044D,5(2;3)Ubx16160.36
T(y;2;3)Mcprvc10 complx,T(2;3)bxd x22290.11x
CROSS LIST: 41A,41F,44A,42BC
BREAKPOINT: In(3)Cbx +R1 REF: 101
CROSS LIST: 87E1,2
BREAKPOINT: T(2;3)P10, In(3)Ubx961.29, In(3)Tab, REF: 110
In(3)Ubx3966.30, In(3)cbx2, Tp(3)bxd110,
Tp(3)bxd110
CROSS LIST: 89C1,2, 90C, 90E, 91B, 91C, 91D1,2, 92A1,2
BREAKPOINT: T(3;2)bxdD36 complx,T(2;3)Ubx18136.147 REF: 110
T(2;3)Ubx19286.8m,T(2;3)Ubx17756.180
T(2;3)Ubx19649.18,T(2;3)UbxD1
CROSS LIST: 2Ltip,41A,39
BREAKPOINT: T(2;3)Cbx rvR17.6F REF: 110
CROSS LIST: 87F
BREAKPOINT: In(3)Sab Mcp rv29340.8 REF: 110
CROSS LIST: 90A

BREAKPOINT: T(1;3)CbxrvR17.49A,T(2;3)Ubx16160.18 REF: 110
T(2;3)Ubx18264.1,T(2;3)Ubx4.3,T(2;3)Ubx4.3 Md
T(2;3)CbxrvR17.175,T(2;3)Hm complx,T(2;3)P10
CROSS LIST: 20,31,21L12,34,32,29,29A-C,22B1,2,2L tip
BREAKPOINT: In(3)Mcp rv29175.10, In(3)Cbx wt REF: 110
In(3)Ubx80, In(e)Ubx12.5 Madrid,
In(3)Cbx3 (Cbx-like)
CROSS LIST: 84F, 87F, 87F-88A, 88B, 89A
CLONE NAME: unn REF: 110
GENES:
BREAKPOINT: T(2;3) Mcp rv26105A REF: 110
CROSS LIST: 60B

89E1-4
CLONE NAME: unn REF: 18
GENES: bithorax complex
BREAKPOINT: T(1;3)Uab5, T(1;3)bxd111, REF: 110
T(1;3)Ubx21560.8A complex
CROSS LIST: 1F, 4D, 5B

89F
BREAKPOINT: T(1;3) P115, Dp(3;1) P68 REF:
CROSS LIST: 20

90A
BREAKPOINT: In(3)Ubx 5.12 (madrid) REF: 110
In(3)Sab Mcp rv29340.8
CROSS LIST: 89E, 89E

90B2
BREAKPOINT: T(1;3)bxd 111 REF: 110
CROSS LIST: 89E

90BC
CLONE NAME: pkdm 7E5 REF: 34
GENES: Intermolt V RNA, sgs
CLONE NAME: pDt 48 REF: 27
GENES: val 3b, pro tRNA
CLONE NAME: lambda bDm 1508 REF: 24
GENES:
CLONE NAME: pDt 92RC, pDt 120RC, pDt 41 RC4 REF: 27
GENES: Val tRNA, val 4 tRNA, Cal 3b, pro tRNA

90C
CLONE NAME: lambda 49-4 REF: 58
GENES: tRNA
CROSS LIST: 84D, 85C
BREAKPOINT: In(3)Ubx 961.29 REF: 110
CROSS LIST: 89E

90CD
INSERTION: S3.8-2 REF: 115
CROSS LIST: 66D11-15,87D12

90E
INSERTION: P[(w,ry)D]3 REF: 176
CROSS LIST: 3C1,2; 87D
BREAKPOINT: In(3)Tab REF: 110
CROSS LIST: 89E

90EF
INSERTION: DR-1 REF: 114
CROSS LIST: 87D12,37C1,2

91
CLONE NAME: S24 REF: 16
GENES:

91AB
INSERTION: AR4-01 REF: 126
CROSS LIST: 3C1,2

91B
BREAKPOINT: In(3)Ubx 3966.30 REF: 110
CROSS LIST: 89E
INSERTION: cHB delta-194 REF: 118
CROSS LIST: 87-lacZ,87D12

91C
CLONE NAME: lambda 39-1 REF: 58
GENES:
CROSS LIST: 27D, 43A
BREAKPOINT: In(3)cbx 2 REF: 110
CROSS LIST: 89E
INSERTION: A2-1 REF: 113
CROSS LIST: 3C1-2,87D12

91D
CLONE NAME: mDm 103G4 REF: 1
GENES:
INSERTION: g4:2 REF: 124
CROSS LIST: 68C3-5,87D12

91D1,2
BREAKPOINT: Tp(3)bxd 110 REF: 110
CROSS LIST: 89E

91F-92A
BREAKPOINT: In(3R)Antp LC REF: 102
CROSS LIST: 84B2
INSERTION: S3.8-5 REF: 115
CROSS LIST: 66D11-15,87D12

92
BREAKPOINT: Tp(3)P47 REF: 110
CROSS LIST: 89E
CLONE NAME: S12g REF: 16
GENES:

92A
BREAKPOINT: In(3)Cbx rvR17.5E REF: 110
CROSS LIST: 89E
CLONE NAME: mDm 101F8 REF: 1
GENES:
INSERTION: R3.9-2 REF: 115
CROSS LIST: 66D11-15,87D12

92A1,2
BREAKPOINT: Tp(3)bxd 110 REF: 110
CROSS LIST: 89E

92A2-3
CLONE NAME: HP-clones REF:
GENES: delta
BREAKPOINT: T(3.3) niII13 REF:

92B
INSERTION: AR4-032(111);AR4-01(111) REF: 126
CROSS LIST: 3C1,2

92B8-11
CLONE NAME: DmRh1 REF: 159
GENES: opsin
CLONE NAME: REF: 99
GENES: rhodopsin (nina E)

92BC
INSERTION: H4 REF: 221
CROSS LIST: 87D12
INSERTION: B2-1 REF: 113
CROSS LIST: 3C1-2

92CD
CLONE NAME: 512 REF: 28
GENES: head specific RNA

92E
CLONE NAME: adm 124B10 REF: 34
GENES:

92F
INSERTION: BS2.7-2 REF: 115
CROSS LIST: 66D11-15,87D12

93AB
INSERTION: R310.1 REF: 115
CROSS LIST: 87D12

93B
BREAKPOINT: In(3)Ubx 130 (TM2) complex REF: 110
CROSS LIST: 89E
CLONE NAME: unn REF: 78
GENES:

93CD
CLONE NAME: unn REF: 127
GENES: ebony, 93D heat shock locus

93D
CLONE NAME: cos7, p5A, cDm E1 REF: 146
GENES: heat-shock
INSERTION: SB2.1-4 REF: 115
CROSS LIST: 66D11-15,87D12
CLONE NAME: adm 129F5 REF: 205
GENES: heat shock

93D6,7
INSERTION: g5:1 REF: 124
CROSS LIST: 68C3-5,87D12

94A
CLONE NAME: adm 134C5,135D2 REF: 34
GENES:
BREAKPOINT: Tp(3)Vno, Tp(3)McpB277 REF: 110
CROSS LIST: 89E

94B
INSERTION: 28P-D REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28P-D REF: 121
CROSS LIST: 87D12,37C1,2

94D
INSERTION: P[(w,ry)E]7 REF: 176
CROSS LIST: 3C1,2; 87D
CLONE NAME: adm 125G5 REF: 34
GENES:
CROSS LIST: 87CF

94E
CLONE NAME: lambda dmpt 123 REF: 58
GENES:
INSERTION: AR4-020 REF: 126
CROSS LIST: 3C1,2

94F
CLONE NAME: 0018 REF: 41
GENES: oocyte RNA

95A
INSERTION: R601.1 REF: 115
CROSS LIST: 87D12

95A-B
CLONE NAME: REF: 208
GENES: en-like homeobox
CROSS LIST: 58C

95AB
INSERTION: 27S-C REF: 200
CROSS LIST: 67B, 87D12

95B
CLONE NAME: mDm 108E11 REF: 1
GENES:

95C
CLONE NAME: pDm6A65 REF: 151
GENES: U1 snRNA
CROSS LIST: 82E, 21D
CLONE NAME: 156-1(lambda) REF: 47
GENES: blastoderm-differential poly(A) RNA
INSERTION: D1 REF: 119
CROSS LIST: 99D,87D12

95D
INSERTION: 27S-A REF: 200
CROSS LIST: 67B, 87D12
CLONE NAME: pPW227, lambda 15 REF: 10
GENES: hsp 68

INSERTION: BS2.7-8 REF: 115
CROSS LIST: 66D11-15, 87D12
INSERTION: unn REF: 117
CROSS LIST: 87D12

95F

INSERTION: 28A-A REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28A-A REF: 121
CROSS LIST: 87D12, 67B

96

INSERTION: DR-2 REF: 114
CROSS LIST: 87D12, 37C1,2

96A

BREAKPOINT: In(3)Ubx 19286.76 REF: 110
CROSS LIST: 89E
CLONE NAME: adm 137A2 REF: 34
GENES:

96AB

INSERTION: P[(w,ry)G]3 REF: 176
CROSS LIST: 3C1,2; 87D

96B

INSERTION: S3.8-1 REF: 116
CROSS LIST: 66D11-15, 87D12
INSERTION: S38M-3 REF: 115
CROSS LIST: 7E11-7F1,2,87D12

96D

CLONE NAME: mDm 107D4 REF: 1
GENES:

96F

BREAKPOINT: Tp(3)Mcp B277 REF: 110
CROSS LIST: 89E

96F97A

BREAKPOINT: Tp(3)Vno REF: 110
In(3)89E/97F-97A, Cbx rv21560.60
CROSS LIST: 89E
CLONE NAME: adm 126D12 REF: 34
GENES:

96F97C

CLONE NAME: adm 132C4, 132E7, 132H4 REF: 34
GENES:

97A

INSERTION: 28N REF: 200
CROSS LIST: 67B, 87D12
INSERTION: tAP-16,4.8 REF: 112
CROSS LIST: 35B2-3
CLONE NAME: lambda 50 REF: 58
GENES:

97AB
INSERTION: +65 REF: 118
CROSS LIST: 87, 87D12
INSERTION: 28-term REF: 121
CROSS LIST: 87D12, 67B

97B
INSERTION: P[(w,ry)F]4-3 REF: 176
CROSS LIST: 3C1,2; 87D
INSERTION: 28P-B REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28P-B REF: 121
CROSS LIST: 87D12, 67B

97C
CLONE NAME: adm 140F12 REF: 34
GENES:
CROSS LIST: 62AB

97CD
BREAKPOINT: In(3)Ubx 3798.68 REF: 110
CROSS LIST: 89E

97D
BREAKPOINT: Tp(3)abx REF: 110
CROSS LIST: 89E

97D1,2
CLONE NAME: REF: 203
GENES: toll

97EF
CLONE NAME: DTB1 REF: 25
GENES: delta tubulin

97F
INSERTION: unn REF: 117
CROSS LIST: 87D12
CLONE NAME: KV 3-12 REF: 22
GENES: delta tubulin

98
CLONE NAME: L2 REF: 16
GENES:
CROSS LIST: 99

98 (+ or -)
BREAKPOINT: T(1;3)scKA8 REF: 105
CROSS LIST: 1B4,5

98A
INSERTION: BS.27-1 REF: 115
CROSS LIST: 66D11-15,87D12

98B-C
BREAKPOINT: In(3)Ubx-x REF: 110
CROSS LIST: 89E

98B
INSERTION: cHB delta-89 REF: 118
CROSS LIST: 87-lacZ, 87D12

98C
INSERTION: R602.1, R705.1 REF: 115
CROSS LIST: 87D12

98D-F
BREAKPOINT: In(3)Mcp rvB315 REF: 110
CROSS LIST: 89E

98E
CLONE NAME: B8 REF: 69
GENES: Maternal restricted transcript

98F
CLONE NAME: b11 REF: 175
GENES: maternal RNA
CLONE NAME: REF: 168
GENES: myosin alkali light chain
CLONE NAME: M55 REF: 84
GENES: 0-2.5 hrs.

98F1,2
BREAKPOINT: Tp(3;3)DfdTRX1 REF: 69
CROSS LIST: 83D4,5

99
CLONE NAME: L2 REF: 16
GENES:
CROSS LIST: 98
BREAKPOINT: In(3)Ubx 1928.16N REF: 110
CROSS LIST: 89E

99A
INSERTION: g6:3 REF: 124
CROSS LIST: 68C3-5, 87D12

99AB
INSERTION: DA24-44 REF: 114
CROSS LIST: 35B2-3, 37C1,2

99B
CLONE NAME: REF: 50
GENES: Homeo box

99C
CLONE NAME: 559 REF: 31
GENES: Head specific RNA

99C5-6
CLONE NAME: 559 REF: 68
GENES: transient receptor potential

99CF
CLONE NAME: adm 129B8 REF: 34
GENES:

99D
CLONE NAME: EH8 REF: 142
GENES: serendipity alpha, beta, delta
INSERTION: SRS3.9-3 REF: 115
CROSS LIST: 66D11-15, 87D12
CLONE NAME: rpro 49 REF: 53
GENES: ribosomal protein, Minute

CLONE NAME: 153-1 (lambda) REF: 47
GENES: blastoderm-specific poly(A) RNA

99E
CLONE NAME: REF: 47
GENES: myosin light chain 2
CLONE NAME: dmpT57 REF:
GENES: myosin light chain-2
INSERTION: B 25 REF: 134
CROSS LIST: 3C1-2
CLONE NAME: lambda Dm 11-9 REF: 22
GENES: myosin light chain
CLONE NAME: adm 132G9 REF: 34
GENES:

99E1-3
CLONE NAME: 36-1 (lambda) REF: 47
GENES: blastoderm differential poly(A)

99E1-F1
BREAKPOINT: Df+In(3R)kar 1g27 REF: 110
CROSS LIST: 87D8-10

99F
CLONE NAME: adm 142D9 REF: 34
GENES:

100
CLONE NAME: S2 REF: 16
GENES:

100AB
CLONE NAME: 5D7 REF: 16
GENES:

100B
CLONE NAME: DM 24B10 REF: 150
GENES: Ag24B10
CLONE NAME: mDm 103 F1 REF: 1
GENES:
CLONE NAME: lambda dmpt 31 REF: 58
GENES:
CLONE NAME: 516 REF: 31
GENES: head specific RNA

100C
CLONE NAME: REF: 88
GENES: double sex cognate

100C1-7
CLONE NAME: mDm 102A3 REF: 1
GENES:

100CD
CLONE NAME: 11B2 REF: 94
GENES: cell death-5 locus

100D
INSERTION: unn REF: 117
CROSS LIST: 87D12

CLONE NAME: mDm 105 H1 REF: 1
GENES:

100E
CLONE NAME: Dm-17 REF: 188
GENES: F2, pupal mRNA in both males and females
CROSS LIST: homology to F1 at 48D

100F
CLONE NAME: lambda T-A REF: 156
GENES: telomeres
CROSS LIST: 1A, 21A, 60F, 61A, 102F
CLONE NAME: lambda 5c,lm.R15,lm.gt11.20516 REF: 83
GENES: microtubule associated protein locus
INSERTION: A4-4 REF: 113
CROSS LIST: 3C1-2, 87D12

100G
BREAKPOINT: T(3;4)ryP51149 REF: 110
CROSS LIST: 87D12-13
BREAKPOINT: T(3;4)Cbx rvR17.40R REF: 110
CROSS LIST: 89E

101-102
BREAKPOINT: T(1;4)scH REF: 105
CROSS LIST: 1B4-C6

101F
BREAKPOINT: T(3;4)bxd101 REF: 110
CROSS LIST: 89E

102
BREAKPOINT: T(3;4)Ubx A complex REF: 110
CROSS LIST: 89E
BREAKPOINT: T(1;4)JC43 REF: 107
CROSS LIST: 3B1,2

102C
CLONE NAME: mDm 108D1 REF: 1
GENES:

102CD
CLONE NAME: 116H2 REF: 3
GENES:

102EF
CLONE NAME: lambda dmpt101 REF: 58
GENES:

102F
CLONE NAME: lambda T-A REF: 156
GENES: telomeres
CROSS LIST: 1A, 21A, 60F, 61A, 100F

TABLE 1: OTHER INFORMATION ON CLONES

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
1A8B1	unn	0		
1A	lambda T-A	0		
1B	unn	0		
1B	lambda y1	0		
1B	y+p13-2,yp3A2	0		
1B	adm134E8	0		
1B1,2	unn	70	Maniatis library, Canton-S	
1B1,2-B4,5	sc64-sc133	113		
1B2,3	unn	0		
1B3,4	unn	0		
1B4,5	unn	0		
1B4,6	unn	0		
1B4,7	unn	0		
1B4-C6	unn	0		
1B4,5-1B8,9		70		
1B5,8	cos 4P	0		
1B11-13	unn	0		
1B1,2-4,5	unn	0		
2B1,2	unn	0		
2B1,2-5,6	unn	230	Maniatis library	
2B1,2	Dm156, Dm159, Dm160	0		
2B3,4	unn	0		
2B3,4	unn	0		
2B3,4	Dm174, Dm 340	0		
2C1-2E3		0	Oregon-R	210 kb walk
2EF	unn	0		
2E	cos 4P	0		
2E2-F3	unn	200		
2F5-6	draf1	8	Oregon R	late embryo
3	S24	0	Canton S	
3A1-4		200		microdissection
3B	lambda DT2,5	0		
3B	mDm112 C 10	0	Oregon R	
3B1,2		0		
3B1,2	unn	40		nucleotide sequence det.
3B2-3C2	unn	200	Oregon R	Microdissection
3BC	M187	0		0-6 hrs.
3C	unn	25		
3C1,2	lambda m1.2	0	Maniatis library	
3C6-8	unn	0		
3C7,8	unn	0	Canton S	
3C7	unn	0		
3C7	pKdm 6B3	0		
3C7		40		nucleotide sequence det.
3C11,12	unn	0		
3C11,12		0		
3C11	pOM3, pOW3	3	Oregon-R	
3C11	pOM3,pOW3	3	Oregon-R	
3D1	pKdm 6B3	0		
3,4	adm 136G5	0		

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
4BC	mDm 109A7	0	Oregon R	
4BC	unn	40		
4C	M97	0		8-24 hrs.
4F5A	pKdm 35D12	0		
4F5A	adm 139C12	0		
5AB	adm 126D6	0		
5C	pDmAz	0		
5C	unn	0		
5D	unn	0	Maniatis library	
5EF	adm 140C11	0		
5F (prox)	B70	0		
6F5	unn	0		
6F5	S1, S2A, S2B	0	Canton-S	
7A	unn	99	Maniatis library	
7B3,4	unn	100	Oregon R	
7D	m58	0		0-6 hrs.
7D5	unn	90	Canton S, Oregon R	
7D5-6		0		
7E6	unn	150		overlapping
7E11-7F1,2	unn	0		
7F1		85		
7,8	adm 132H10	0		
8	56	0	Canton S	
8A	unn	100		100 KB overlapping
8D	PLZ-p	0		
8F9A	PYp2	0	Canton S	
8F9A	PYp1	0	Canton S	
9E3-4	lambda EMBL4-LF1	20	PM dysgenic ras-L strain NJS	
10A	unn	0		
10A1		0		
10A1,2	lambda vDT1	18	Maniatis library	homology to rat cDNA
10A2	sevenless	0		
10B	unn	0		
10C1,2	lamda DmRpl1-1	0		
10EF	adm134A3, adm130E12	0		
10F	adm 10F.1	0		
11A	unn	0	Maniatis library	
11A	lambda Dm (Can S) LSP1 alpha:	0		alpha subunit of Larval Serum Protein 1
11A2-4		0		100 kb walk
12	S21b	0	Canton S	
12B,C	PYP3	0	Canton S	
12DE	pDt17R, pDt27,pDt73	0		
12E	pDt16	0		
12F	lambda 32-10	0		
12F1,2	Dm2L1	0		
12	adm 136F10	0		
13EF	M3,6,8,10	30		nucleotide sequence det.
13F	G2	2	Maniatis library	XhoI-XhoI fragment in Charon-insert

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
14BC	adm 132B8	0		
14D	M75	0		0-1 hrs.
15A1	unn	90	Maniatis library	
15A,B	548	0	Oregon R	
15B	unn	0		
15F	Dmf3	40		
15F	lambda f1	0		
16B3-5	PTE-1	0		
16EF	unn	50	Maniatis library	
16F17	adm 135H4	0		
17AB	lambda dmpt 61	0		
18CD	A57	0		
18D	lamda DmG21	0	Oregon R	
18D	trc 18	0		
18E	Dm14	0		17-base oligonucleotide
18E	lambda Dm G6PD 14	13	High-G6PD mutant strain	2 EcoRI sites within insert
19E8	PP95	0		
19EF	DCg2	0	Maniatis library	
19F	pDt67R	0		
20AB	DCg2	0	Maniatis library	
21A	lambda alpha 8 lambda 8001 to 8014	0	Charon-4 lambda recombinant library	40 kb walk
21A	alpha 8	40		
21A	lambda T-A	0		
21B	adm 142G5	0		
21C	unn	0		
21D	pDm6A65	0		nucleotide sequence det.
21D	lambda Dm9	0	Maniatis library	overlaps clone lambda Dm34
21D	pD957	0		
21D	lambda Dm (Can S) LSP1 beta: 1	0		
21F	adm 123D12,123H3,128B8	0		
22A	adm 123D12, 123H3, 128B8	0		
22AC		7		
22B	mDm103H10, mDm108C7	0		
22B,C	adm 129E7	0		
22F1,2	unn	130		
22F1,2	pDm22F Ya,Yb	0		
23A3-7	unn	70	Maniatis library	
23BC	B13	0		
23E	pDt5	0		
24AB	M98	0		
24C	mDm101A10	0	Oregon R	
25BC	mDm109D3	0	Oregon R	

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
25C	DCg-1	0	Maniatis library	
25D	150-3(lambda)	0		
25D1-4	MH5	0	Gelbart library	
25F5	Gpdh 411	1	Adult lambda gtll library	1300 bp cDNA clone
26A	DmcMM115, LS1	0	Oregon R	cDNA, embryos genomic
26A3-5	Gpdh 411	0		
26A7-9	unn	0		
26AB	A20	0		minor site at 88D
27C	Pupal cuticle protein	0		
27C	unn	0		
27D	lambda 39-1	0		
27F	adm 125G11	0		
28A	551	0	Oregon R	
28C	538	0	Oregon R	
28C	lambda dmpt 49	0		
28D9-12	unn	0		
29A	pDt59R	0		
29B1-4	unn	0		
29C	unn	0		
30A9-30B1, 2	lambda Dm65	55	Canton-S	expressed in fat body
30B	lambda dmpt 75	0		
30DE	adm 136D3	0		
30EF	lambda dmpt 104	0		
31A	mDm 106A10	0	Oregon R	
31A	M35	0		0-6 hrs.
31C	adm 134G6	0		
31C	adm 142H	0		
31F	adm 142F4	0		
32AB	503	0	Oregon R	
32C-F	DmcMM99	0		
32C-F	E2	200		
32CD	231	0	Maniatis library	
32EF	Dmc MM99	0		cross hybridizes with MM99 & MM115
32F-33E		999		walk from esc
33AB	unn	250		
33B	adm 142H	0		
33B	adm 124D9	0		
33B	M146	0		
33B1,2		380		microdissection
34AB	A34	0		
34C	DmcMM99	0	Oregon R	
34F	527	0	Oregon R	
35A4-35B1		165		nucleotide sequence Adh
35AB	lambda CHD1	0	Maniatis library	
35B1-3	lambda ob 5	0	Maniatis library	
35B1-3	lambda w4.04	0	Maniatis library	
35B1-3	lambda w3.13	0	Maniatis library	
35B1-3	lambda ob 9.04	0	Maniatis library	

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
35B1-3	lambda ob 3.04	0	Maniatis library	
35B1-3	lambda ob 2.01	0	Maniatis library	
35B1,2	ob 9.04	0		TE146 and fold back sequences inserted
35B1-3	lambda ob 1.12	0	Maniatis library	
35B1-3	lambda gAC2	0	Maniatis library	
35B1-3	lambda ob 4.01	0	Maniatis library	
35B1-3	lambda ob 7.06	0	Maniatis library	
35B1-3	lambda ob 8.10	0	Maniatis library	
35B1-3	lambda ob 6	0	Maniatis library	
35B1-3	lambda gAC3	0	Maniatis library	
35B1-3	lambda w2.0	0	Maniatis library	
35B1-3	lambda ob 10.02	0	Maniatis library	cross hybridizes in situ to 3A1
35B3-5	AC	0	Maniatis library	
35B9-C1		0		
35C36	adm 125E7	0		
36B	unn	0	Maniatis library	
36B	unn	0		
36C	iH-6	75		
36F	mdm 103D5	0	Oregon R	
37B9-C1,2	unn	0		
37B13-C5	lambda Ddc-1 thru-20	100		
37BC	DmcMM109	0	Oregon R	cDNA
37C1,5	unn	0		
38A6	2E2	0		
38E	F33	0	Maniatis library	
39DE	unn	0		
39E	adm 136D9	0		
39F	adm 142F4	0		
40	adm 106H5, 123C3, adm136D9	0		
41	adm 130B2	0		
42A	4 tRNA genes	94		
42A	mDm 106F8	0	Oregon R	
42BC	lambda st11-205.16	0		
42E	pDt 61	0		
42EF	adm 126F7, 127A10	0		
43A	lambda 39-1	0		
43A2-5	draf2	7	Oregon R	
43AB	555	0	Oregon R	
43BC	B17	0		
43DE	B45	0		

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
43E	G3	3	Maniatis library	EcoRI-Hind III frag in Charon-4 insert
44	L10	0	Canton S	
44C	lambda e8 e9	0	Oregon, EMBL 4 library	
44CD	536	0	Oregon R	
44D	lambda DmLCP1-13	50		
44EF	M51	0		0-24 hrs.
44F	129E7	0		
45A	mDm103H10, mDm108C7	0		
45B	M199	0		
45D	mDm108A8	0		
46B	B41	0		
46C	S72	0		
46DF	236	0	Maniatis library	
46E	549	0	Oregon R	
47E	528	0	Oregon R	
47F	unn	50		
47F48D	217	0	Maniatis library	
48A	unn	208	Canton S	
48B	pDt74	0		
48C	adm 132A7	0		
48D	Dm-15	0		nucleotide sequence det.
48E	adm 135E10	0		
48EF	aDmS7	0	Canton-S	
48F	38B10	0		
48F	543	0	Oregon R	
49A	p500, p2.2	0		
49A12B3	unn	0		
49C	mDm101D3	0	Oregon R	
49CD	mDm101D12	0	Oregon R	
49DE	adm 140D1	0		
49DEF	lambda 120	0		
49E5F1	unn	0		
49F	Dm1606	35	Maniatis library	
50	L6	0	Canton S	
50AB	7 tRNA genes	2		
50B	adm 142E9	0		
50BC	7 tRNA genes	0		
50C	mDm3021	0	Oregon R	
50CD	adm 133H7,136F9,138 G8,130H8	0		
50F	unn	0		
51A	S34	0	Canton S	
51B	S14	0	Oregon R	
51CD	A19	0		
51D	adm 134E2	0		
51DE	mDm102F11, mDm102B6	0	Oregon R	
52A1,2	M144	0		0-1 hrs.
52A3,6	M137	0		0-1 hrs.
52B	mDm107A2	0	Oregon R	
52D	unn	0		

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
52D6,15	M222	0		
52DF	adm 139H3	0		
53	L23	0	Canton S	
53CD	rpA1	2	Oregon-R	
53CD	rp A1	0		
39CD	unn	0	Spradling and Mahowald library	
52CD	lambda Dm32 (class A)	0	Maniatis library	
53F	lambda dmpt116	0		
54A1B1	lambda Dm 65 (class B)	0	Canton S	
54E	adm 54E.1	0		
54F55A	adm 110A4,132C9,132 E11, 132E12	0		
54F55A	adm 132G5, 134A4, 135D12	0		
55BCD	adm 110G1,110H1,132 D6	0		
55F	B32	0		
56C	DTB2	0	Maniatis library	
56D	pTu56	0	Oregon R, EMBL 4 library	
56D412	KV 2-70A	0	Maniatis library	
56EF	adm 135H8	0		
56F		0		
56F	lambda Dmt 56-6	0		
57B	unn	0		
57C	unn	60		
57C	525	0	Oregon R	
57C	unn	200		
57F	unn	0		
58C		0		
58F	adm 132A3,135D10,13 5E6	0		
59E	trc 59	0		
60A	adm 125C2	0		
60A	A8	0		
60A	adm 106H6	0		
60A	B6	0		
60B	pTu60	0	Oregon R, EMBL 4 library	
60BC	B50	0		
60C	DTB3	0	Maniatis library	
60C6-8	KV 1-11	0	Maniatis library	
60F	lambda T-A	0		
60F3		50	Oregon R	microdissected from Oregon
61A	lambda T-A	0		
61A	lambda Dm60	0	Maniatis library	overlaps clone lambda Dm35

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
61A	lambda Dm (Can S) LSP1 gamma: 1	0		
61A1-3	mDm105F3	0	Oregon R	
61F		0	Maniatis library	
62A	5 tRNA genes	0		
62A	adm 112C10	0		
62A	lambda 48-9	0		
62AB	adm 140F12	0		
62B7-12		0		cluster of metabolic housekeeping genes
62B9		0		microdissection
62CD	203	0	Maniatis library	
62D	adm 142F6	0		
62E		0		
63-66	S7	0	Canton S	
63AC	227	0	Maniatis library	
63B	bDm 4L	0	Oregon R	
63BC	lambda 6, pPW244,301,330	0	Canton S	
63E	IMP-E2	15	Maniatis library	insert of Canton-S embryonic DNA
63F	adm 63 F.1	0		
63F64A	adm 140C11	0		
64B	IMP-L2	15	Maniatis library	insert of Canton-S embryonic DNA
64B	M51	0		0-24 hrs.
64B	Drsrc	0		
64B	pDMRS64B	0	Maniatis library	nucleotide sequence det.
64BC	mDm104C1	0	Oregon R	
64C	DHSV4	0		
64F	lambda dmpt 120	0		
64F	mDm 106E3	0	Oregon R	
64F	adm 126B4	0		
64F	adm 135G4	0		
65A	adm 135G4	0		
65B	IMP-L3	15	Maniatis library	insert of Canton-S embryonic DNA
65C	adm 111F10	0		
66B		0		517 bp in M13, originally in lambda Ch4A
66C	IMP-E1	15	Maniatis library	insert of Canton-S embryonic DNA
66C	adm 126B4	0		
66CD	adm 106E3	0		
66D	507, 547	0	Oregon R	
66D9-10	.8247,30152,3019	0		
66D10-15	unn	85	Oregon R	
66D11-15	unn	100		overlapping
66D12-15	AB	0		nucleotide sequence det.
66F	lambda dmpt 121	0		
67A5-7-67B 1,2	unn	0	Maniatis library	walk

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
67B	overlapping	0		nucleotide sequence det.
67B	lambda 88	0	Canton S	
67B	lambda Dmp 67	0	Canton S	
67B	J1	0	Oregon R	
67C	DTA2	0	Maniatis library	
67C4,5	M98	0		
67DE	gt11.205.23	0		
67F	l(3) 1902	0		
68C	pkdm1H2, adm 134C10,pkdm2C1, 2G6	0		
68C1-5	lambda cDm2021	0		
68C3-7		0		
68C3-7	unn	0		
68C4-6	aDm 1501-10	0		
68C7-15	mDm148F7	0	Oregon R	
68C10-12	unn	1		
68E	lambda Dm104	0	Maniatis library	overlaps clone lambda Dm95
68EF	adm 133H1	0		
69	L3g	0	Canton S	
69D	M37	0	0-24 hrs.	
69F	270	0	Maniatis library	
70A	IMP-L1	15	Maniatis library	insert of Canton-S embryonic DNA
70A	63H5	0		
70A	adm 107A4	0		
70AB	adm 128C11, 132B3	0		
70BC	B20	0		minor homology to 2F
70BC	pDt 55	0		
70C	adm 29D11	0		
70D1-3	lambda Dm117	85	Canton-S	ecdysone-inducible expressed in fat body
70D4,5	Q111,Q131,Q112	60	Maniatis library	
71A	2-5 (lambda)	0		
71AB	adm 123C4	0		
71C3,4D1,2		0		
71CE	lambda cDm 20-24	0	Maniatis library	
71DE	adm 134A9,134A11,13 4C11,pkdm 46B7	0		
71DE	pkdm 38C9, 38C4	0		
72BC	557	0	Oregon R	
72DE	lambda dmpt 115	0		
73A		0		
73A		0		
73A3-4	lambda stR4	0		
73A3-B1,2	stdb-st tra DIS-5 cABL std5	0		
73A3,4-73B		0		transformation
73B	Dash	0		
73D	adm 73D.1	0		
73DEF	521	0	Oregon R	

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
74EF		300	Maniatis library	
75	S39	0	Canton S	
75C	adm 135F3	0		
75D	trc 75	0		
76A	adm 132D11	0		
76DE	B48	0		
76F	mDm 104G3	0	Oregon R	
78D7,8	lambda Dm-208B	200	Maniatis library	
79-80		0		
79B	unn	0		
79E1,2	13E5	0		Or, R PBR 322
80	adm 130B2	0		
80C		0		
80F	adm 139A10	0		
81	adm 128F12	0		
82A	S6-7	0	Maniatis library	
82E	pDm6A65, Dm 525	0		nucleotide sequence det.
82E	lambda Dm525	0	Maniatis library	
82F	506	0	Oregon R	
83A	adm 136E4	0		
83A,B	pDt 66R2	0		
83AB	adm 140E12	0		
83B	adm 123G4	0		
83C	mDm 105 B9	0	Oregon R	
83CD	B21, B31	0		
83D4,5	B31	0		
83F	adm 140C1	0		
84A1		0		
84A4,5-84C 1,2		440		
84A,B	pDt 12, pDt 39	0		
84B	DTA 1	0	Maniatis library	
84B2	unn	0		
84B2-C1	unn	0		
84B3-6	lambda Dm 2.55a	0	Maniatis library	
84B3-C1,2		75	Maniatis library	
84BC	adm 123D11	0		
84C8	DmA 3a,4a,4b,5a,5b	152	Maniatis library	
84D	lambda 49-4	0		
84D	mDm 104H7	0	Oregon R	
84D	pDt 78 RC	0		
84D	DTA 4	0		
84D3,4	1,3,6,10	30	Maniatis library	
84D4-8	lambda Dm 5-1	0	Maniatis library	
84E	IMP-E3	15	Maniatis library	insert of Canton-S embryonic DNA
84E1,2		105	Maniatis library	
84E11-12 - F4-5	unn	240	Maniatis library	

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
84F	14C4	18		
84F	B34	0		
84F2,3	unn	0		
85	adm 123B10	0		
85A	lambda 50-8	0		
85C	lambda 49-4	0		
85C	p85C	0		
85C	lambda m 1:2	0	Gelbart library	
85D	KV 1-22	0	Maniatis library	
85D	542	0		
85D	DHSV 7	0		
85D		0	Oregon R, EMBL 4 library	
85D,E		0		
85D6-12	DTB 4	0	Maniatis library	
85E	M253	0		
85E	DTA 3	0	Maniatis library	
85E	mDm 3008	0	Oregon R	
85E6-10	lambda Dm 5-22	0	Maniatis library	
85E10-15	cDm51	0		400 bp
85E10-15	lambda Dm13	13		
85E10-15	lambda Dm13	14		cloned in lambda EMBL4 EcoRI site
86	adm 35E6	0		
86	S35g	0	Canton S	
87A	GB	0		
87A	56H8	0		
87A7	pPw 223	0	Oregon R	
87A7		0		
87AB	540	0	Canton S	
87C	G3	0		
87C1	132E3	0		
87C10	pPW232, pPW229	0	Oregon R	
87CF	adm 125G5	0		
87D	mG31	0	Oregon R	
87D	unn	0		
87D5-87E5		315		
87D6-8	unn	0		
87D8-10	unn	0		
87D12-13	unn	0		
87D14	unn	0		
87D17	unn	0		
87E	unn	0		
87E1,2	unn	0		
88	S32	0	Canton S	
88B	adm 88B.1	0		
88C	mDm 104D12	0	Oregon R	
88D		0		
88E	mG34	0	Oregon R	
88F		0		
88F		0		
88F	unn	0		
88F	lambda dmpt 73	0		

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
88F2-5	lambda DM 85	0		
89	unn	0		
89B	pDt 14	0		
89B4		0		
89BC	EV27	0		
89E	unn	0		
89E1-4	unn	400		
90BC	pkdm 7E5	0		
90BC	pDt 48	0		
90BC	lambda bDm 1508	0	Oregon R, Hogness library	
90BC	pDt 92RC, pDt 120RC, pDt 41 RC4	0		
90C	lambda 49-4	0		
91	S24	0	Canton-S	
91C	lambda 39-1	0		
91D	mDm 103G4	0	Oregon R	
92	S12g	0	Canton S	
92A	mDm 101F8	0	Oregon R	
92A2-3	HP-clones	140		
92B8-11	DmRh1	0		nucleotide sequence det.
92B8-11		0		nucleotide sequence det.
92CD	512	0	Oregon R	
92E	adm 124B10	0		
93B	unn	90		
93CD	unn	300		
93D	cos7, p5A, cDm E1	0		
93D	adm 129F5	0		
94A	adm 134C5,135D2	0		
94D	adm 125G5	0		
94E	lambda dmpt 123	0		
94F	0018	16		
95A-B		0		
95B	mDm 108E11	0	Oregon R	
95C	pDm6A65	0		nucleotide sequence det.
95C	156-1(lambda)	0		
95D	pPW227, lambda 15	0	Oregon R	
96A	adm 137A2	0		
96D	mDm 107D4	0	Oregon R	
96F97A	adm 126D12	0		
96F97C	adm 132C4, 132E7, 132H4	0		
97A	lambda 50	0		
97C	adm 140F12	0		
97D1,2		0		
97EF	DTB1	0	Maniatis library	
97F	KV 3-12	0	Maniatis library	
98	L2	0	Canton S	
98E	B8	0		
98F	b11	14	Maniatis library	preblastoderm RNA
98F		0		

LOCATION	CLONE NAME	KB	STRAIN	OTHER INFORMATION
98F	M55	0		
99	L2	0	Canton-S	
99B		0		
99C	559	0	Oregon R	
99C5-6	559	45		
99CF	adm 129B8	0		
99D	EH8	0		
99D	rpro 49	0		
99D	153-1 (lambda)	0		
99E		0		
99E	dmpT57	0		nucleotide sequence det.
99E	lambda Dm 11-9	0	Maniatis library	
99E	adm 132G9	0		
99E1-3	36-1 (lambda)	0		
99F	adm 142D9	0		
100	S2	0	Canton S	
100AB	5D7	0		OR .R PBR 322
100B	DM 24B10	0		nucleotide sequence det.
100B	mDm 103 F1	0	Oregon R	
100B	lambda dmpt 31	0		
100B	516	0	Oregon R	
100C		0	Maniatis library	
100C1-7	mDm 102A3	0	Oregon R	
100CD	11B2	0		
100D	mDm 105 H1	0	Oregon R	
100E	Dm-17	0		
100F	lambda T-A	0		
100F	lambda 5c,lm.R15,lm.gt 11.20516	0		
102C	mDm 108D1	0		
102CD	116H2	0		
102EF	lambda dmpt101	0		
102F	lambda T-A	0		

TABLE 2: MARKERS IN INSERTS

LOCATION	TRANSFORMANT STRAIN	MARKERS IN INSERT
1B	27X-F	hsp 28, ry
1B	g71:1	sgs3+, ry+
1CD	Bs2.71-2	chorion+, ry+
1E 2-3	Strain 23-2	ry+, 21 copies D. reissieri 5S genes
1F	R704.2, R702.1	ry+
1F	DA24-14	adh+,Ddc+
2A	P[(w,ry)E]5	w, ry
2A	27P X/X-F	hsp 28, ry
2B	P(ry,hsp0-1)8	ry, hsp
2B	Tf(1)Gr304-1	ry+
2B13-18	g711:2	Sgs3+, ry+
2C	Pc[ry(delta0-1)]48	ry
3A	7-1	sgs4+, ry+
	7-2	sgs4+, ry+
3B	Tf(1)GR420-3	ry+
3F	g71dx:2	Sgs3+, ry+
4C5-6	Strain 23-2	ry+, 21 copies D. reissieri 5S genes
4B	cHB delta -59	Hsp 70-lacZ, ry+
4D	R405.1	ry+
6AB	P[(w,ry)H]4	w, ry
6F	S6.9-2	chorion+,lac+,ry+
7D	R403.1	ry+
8BC	tAP-25,3.2	Adh+
8E	cHB delta-59	Hsp70-lacz,ry+
9A-D	R404.2	ry+
9B	unn	ry+
9B	tAP-24B,3.2 .	Adh+
9B	SB2.1-5	chorion+, ry+
9C	S6.9-9	chorion+,lacZ+, ry+
9D	P(ry,hsp0-1)22	ry, hsp
9E	R701.1	ry+
9E	cHB lambda -23	Hsp70-lacZ, ry+
10BC	tAP-20,3.2	Adh+
10D	AR4-038	w
12A	tAP-17,4.8	Adh+
12B	P[(w,ry)E]2	w, ry
12B,C	SRS3.9-1	chorion+ & ry+
12BC	AR4-032(X)	w
12D	R301.2	ry+
12E	E 7-10	Sgs-4
12E	cHB delta-73	Hsp70-lacZ & ry+
13A-C	BS.27-5	Chorion+, ry+
13CD	SB2.1-6	chorion+, ry+
13EF	P[(w,ry)E]3g	w, ry
	P[(w,ry)G]4	w, ry
14A	21-1	Sgs4+, ry+
15DE	BS2.7-10	chorion+, ry+
15E	P15-1	gl+, neo+
16BC	S6.9-11	ch, lacZ, ry
16C	H1	ry
16D	unn	ry+
16E	27 N/P-A	hsp 28, ry

LOCATION	TRANSFORMANT STRAIN	MARKERS IN INSERT
16F	27C X/X-A	hsp 28, ry
17C	tAP-5	Adh+
17DE	B1-2	w+
18A	R704.3	ry+
18D	16-3	sgs4+, ry+
18D	BS2.7-3	chorion+,ry+
19A	cHB delta-89	
19E	+65	
19E	tAP-1	Adh+
19F	P[(w,ry)E]1	w, ry
20A	2	Act88F+
20A	Adh hs20A	adh+, hsp 70
20CD	P(ry,HsAFP)	ry, HsAFP
20D	AR4-024	w
21B	P[(w,ry)G]1	w, ry
21D	P[(w,ry)F]4-2	w, ry
21D	R602.1	ry+
21DE	tAP-10,4.8	Adh+
22A	R604.1	ry+
22B	w20.2,w20.10	w+
22F-23A	P13-1	gl+, ry+
23A	+65	Hsp70,ry+
23BC	g5:2	Sgs3+ & ry+
24AB	P[(w,ry)D]4	w, ry
24CD	AR4-24	w
25A	6-1	sgs4+, ry+
25B	alpha T3.1	LSP1 alpha
25B	alpha T3.21	LSP1 alpha
25C	P[(w,ry)D]1	w, ry
25C	R401.3	ry+
25F	27P X/X-B	hsp 28, ry
26B	D4	rp49+,ry+
26B	D1	rp49+, ry+
27A-C	icarus-neo	
27C	33-4	sgs4+, ry+
28A	BS2.7-11	chorion+&lac+&ry+
28D5	E 7-1	Sgs-4
29B	R308.1	ry+
30A	28C-B	
30A	28C-B	ry+,hsp28
30A	cHB delta-89	Hsp70-lacZ,ry+
30C	S6.9-3	chorion+,lac+,ry+
30C	A4-N22	w
31B	cHB delta-89	Hsp70-lacZ,ry+
32BC	BS2.7	chorion+,ry+
32CD	cp70 delta B	Hsp70-lacZ,ry+
32F	g6:5	Sgs3+,ry+
34D	g7:4	Sgs3+,ry+
34E	P(w)11P	w
34EF	g711:1	Sgs3+,ry+
35A	15-1	sgs4+, ry+
	15-2	sgs4+, ry+
35B9-C1	TE 36	w+, rst+
35DE	S11.4-1	chorion+
36A	tAP-3	Adh+
36A	unn	Hsp26-lacZ,ry+
36C	tAP-8C,4.8	Adh+
37A	P[(w,ry)G]2	w, ry
37A	unn	Hsp26-lacZ,ry+

LOCATION	TRANSFORMANT STRAIN	MARKERS IN INSERT
37BC	unn	Adh+ Hsp82
38B	P(ry, HsAFP)2	ry, HsAFP
38B	cHB delta-59	Hsp70-lacZ,ry+
38BC	tAP-19,4.8	Adh+
38D	1	Act88F
38E	unn	ry+
39B	28P-C	
39B	28P-C	ry+ hsp28
39BC	E 5-5	Sgs-4
39BC	S6.9-8	chorion+,lac+,ry+
39DE	H5	ry
39E	AR4-3	w+,ry+
39E	B4	rp49+,ry+
39EF	AR4-2	w+,ry+
40F	AR4-3	w+, ry+
42A	R301.1	ry+
42A	+411	Hsp70,ry+
42A	tAP-13,4.8	Adh+
42AB	R303.1	ry+
42DE	unn	ry+
42E	R305.1	chorion+&lac+&ry+
42F	28X-C	
42F	S6.9-4	chorion+,lac+,ry+
43	R704.1	ry+
43C	R304.1	ry+
43CD	+411	Hsp70,ry+
43E	S6.9-7	ch, lacZ, ry
43E	g7:7	Sgs3+,ry+
44C	S6.9-7	ch, lacZ, ry
44CD	S6.9-7	chorion+,lac+,ry+
44E	R3.9-4	chorion+,ry+
44F	cp70ZT	Hsp70-lacZ,ry+
45A	S11.4-1	chorion+
45AB	F4	rp49+,ry+
45D	AR4-020(11)	w
45E	DR-18	ry+,Ddc+
45E	cHB delta-73	Hsp70-lacZ,ry+
46C	27 X/A-2-A	hsp 28, ry
	27 X/A-2-B	hsp 28, ry
	27 X/A-2-C	hsp 28, ry
	27 X/A-2-D	hsp 28, ry
46C	A4-N21	w
47A	tAP-18,4.8	Adh+
47A	A1-1	w+,ry+
47C	cHB delta-89	Hsp70-lacZ,ry+
47D	P[(w,ry)E]8	w, ry
47D	27 N/P-B	hsp 28, ry
48AB	g7:3	Sgs3+,ry+
48B	hsp26-lacZ	
48B	tAP-6	Adh+
48D	DR-9	ry+,Ddc+
48E-F	tAP-4	Adh+
49B	unn	Hsp26-lacZ,ry+
49C	P(w)24S	w
49D	-51	Hsp70,ry+
49D	A1	rp49+,ry+
49D	S38M-5	s38+,M13+,ry+
49D	AR4-042	w
49EF	S3.8-6	chorion+,ry+

LOCATION	TRANSFORMANT STRAIN	MARKERS IN INSERT
49F	tAP-9,4.8	Adh+
50A	P[(w,ry)F]1	w, ry
50B	R306.1, S6.9-6	ry+,lac+,chorion+
52A	DR-12	ry+,Ddc+
52B	cHB delta-89	Hsp70-lacZ,ry+
52B	tAP-21,3.2	Adh+
52BC	P(w)5P	w
52CD	H3	ry
52D	SRS3.9-1	chorion+,ry+
52F	S6.9-10	chorion+,lac+,ry+
53A	S38M-6	s38+,M13+,ry+
53BC	E 7-3	Sgs-4
53E	R3,9-1	chorion+,ry+
53EF	SB2.1-1	chorion+,ry+
53F	g4:1	Sgs3+,ry+
54A	2	Act88F+
54A	2	Act88F+
54C	Adh,hs54c	Adh+,hsp70
56AB	H2	ry
56D	R3.9-6	chorion+,ry+
56F	DR-15, DR-5	ry+,Ddc+
57	HB4/Sc1	LSP1 beta
57A	alpha T3.21	LSP1 alpha
57AB	27P X/X-A	hsp 28, ry
	27P X/X-E	hsp 28, ry
57B	P[(w,ry)E]6	w, ry
	P[(w,ry)F]4-1	w, ry
57B	27S-B	hsp 28, ry
57F	unn	ry+
58D	27 X/A-1-B	hsp 28, ry
58D	AR4-043	w
58EF	R3.9-5	chorion+,ry+
58F	cp70 delta B	Hsp70-lacZ,ry+
58F	tAP-7A,4.8	Adh+
59B	A3-1	w+,ry+
59C	tAP-7A,4.8	Adh+
59D	27C X/X-B	hsp 28, ry
59E	S11.4-1	chorion+
60A	R302.1	ry+
60AB	g1	Sgs3+,ry+
60B	BS2.7-4	chorion+,ry+
60BC	27P X/X-B	hsp 28, ry
60C	C2	rp49+,ry+
60C	unn	ry+
60E	S38Z-1	s38,lacZ,ry
60E	tAP-15A,4.8	Adh+
60F	S3.8-4	chorion+,ry+
61A	27C+G-B	hsp 28, ry
61A	Bg61	ry+,hsp70+,lacZ+
61A	S38Z-5	s38+,lacZ+,ry+
61A-B	tAP-2	Adh+
61C	Adh hs61C	Adh+,hsp70
61D	28P-A	
61D	cp70 delta B	Hsp70-lacZ,ry+
61D	28P-A	ry+hsp28
61E	SRS3.9-4	chorion+,ry+
61F	P(ry,HsAFP)3	ry, HsAFP
62A	P[(w,ry)D]2	w, ry
62AB	tAP-27	Adh+

LOCATION	TRANSFORMANT STRAIN	MARKERS IN INSERT
63AB	HB4/Sc2	LSP1 beta
63BC	28C-A	
63BC	28C-A	ry+ hsp28
63C	S38Z-6	s38+,lacZ+,ry+
63E	23-3	sgs4+, ry+
64B	P[(w,ry)F]3	w, ry
64C	R405	ry+
64C	tAP-7B,4.8	Adh+
64F	SB2.1-3	chorion+, ry+
64F	cHB delta -73	Hsp70-lacZ,ry+
65A	SB2.1-3	chorion+, ry+
65AB	w47.1 N8	w+
65D	28A-B	
65D	28A-B	ry+,hsp28
65D-66B	S38Z-7	s38, lacZ, ry
65F	g711:3	sgs3+,ry+
66A	28X-D	
66A	28X-D	ry+ hsp28
66D	DR-17	ry+,Ddc+
66E1,2	Tf(3L)Ga6.0-1	Adh+
66E	AR4-012	w
67BC	27P X/X-D	hsp 28, ry
67E	27 C X/X-A	hsp 28, ry
68A	R7.7-1	chorion+,ry+
68C	28X-A	
68C	28X-A	ry+ hsp28
68C	g7:5, g71:2	Sgs3+,ry+
68D	S38Z-3	s38+,lacZ+,ry+
68E	g6:4	Sgs3+,ry+
69CD	tAP-12,4.8	Adh+
70AB	S3.8-3	chorion+,ry+
70C	S11.4-2	chorion+
71AB	-51	Hsp70,ry+
71C	S38M-1	s38+,M13+,ry+
71F	tAP-11,4.8	Adh+
71F	g7:2	Sgs3+,ry+
73B	Pc[ry(delta0-1)]2	ry
73B	27S-D	hsp 28, ry
73E	P(ry,HsAFP)6	ry, HsAFP
75C	P[(w,ry)H]2-2	w, ry
75CD	R502.1	ry+
75D	R706.1	ry+
76A	A38M-4	s38+,M13+,ry+
77A	cHB delta-73	hsp 70-lacZ, ry+
77DE	P(ry, HsAFP)1	ry, HsAFP
78BC	R603.1	ry+
78CD	P[(w,ry)H]1	w, ry
78D	cHB delta-89	hsp 70-lacZ, ry+
78D	cHB delta-89	Hsp70-lacZ, ry+
79E	+411	Hsp70,ry+
79F	8-1	sgs4+, ry+
80A	B1,F2	rp49+,ry+
82A	w47.4L	w+
82B	Adh hs82b	Adh+,hsp70
82BC	tAP-8B,4.8	Adh+
82BC	g4:3	Sgs3+,ry+
82C	28X-E	
82C	28X-E	ry+ hsp28
82F	5-1	sgs4+, ry+

LOCATION	TRANSFORMANT STRAIN	MARKERS IN INSERT
82F	AR4-025	w
83A	unn	ry+
83B	27S-E	hsp 28, ry
83BC	SB2.1-2	chorion+,ry+
83F	R3.9-3,9/ S38M-2	ry+/ s38+, M13+, ry+
84B	27P X/X-E	hsp 28, ry
84BC	S3.8-1	chorion+,ry+
84C	unn	ry+
84D	28X-B	
84D	unn	Hsp26-lacZ,ry+
84D	28X-B	ry+, hsp28
84E	cHB delta-194, g6:2	Hsp70-lacZ,ry+
84E	g6:2	Sgs3+,ry+
84F	1	Act88F+
84F	g71:3	Sgs3+,ry+
84F	A3	rp49+,ry+
85A	27 X/A-1-A	hsp 28, ry
85A	R309.1	ry+
85AB	814,CH8	sry+,ry+
85BC	24-1	sgs4+, ry+
85D	P[(w,ry)H]3	w, ry
85D	P(ry,HsAFP)5	ry, HsAFP
85D	BS2.7-7	chorion+,ry+
85D	g6:1	Sgs3+,ry+
85F	P[(w,ry)H]2-1	w, ry
85F	tAP-15B,4.8	Adh+
86A	20-1	sgs4+, ry+
86C	P[(w,ry)F]2	w, ry
86D	R311.1	ry+
86D	g7:6	Sgs3+,ry+
86E	S38Z-2	s38+,lacZ+,ry+
87A	R307.1	ry+
87AB	28A-C	
87AB	28A-C	ry+hsp28
87CD	C1-1	w+
87E	unn	ry+
87E	unn	ry+
87F	R308.2, R404.1	ry+
87F	R404.1	ry+
87F	DRI-15	ry+,Ddc+
88A	cHB delta-23	Hsp70-lacZ,ry+
88C	S6.9-5	chorion+,lac+,ry+
88E	BS2.7-9, R401.2	chorion+,ry+/ ry+
88F	cp70 delta B	Hsp70-lacZ,ry+
88F	S6.9-1	chorion+,lac+,ry+
89A	+204	Hsp70,ry+
89A	B1-1	w+
89B	28-2	sgs4+, ry+
89B	27P X/X-C	hsp 28, ry
89B	BS2.7-6	chorion+,ry+
89B	g7:8	sgs3+,ry+
90CD	S3.8-2	chorion+,ry+
90E	P[(w,ry)D]3	w, ry
90EF	DR-1	ry+,Ddc+
91AB	AR4-01	w
91B	cHB delta-194	Hsp70-lacZ,ry+
91C	A2-1	w+,ry+
91D	g4:2	Sgs3+,ry+
91F-92A	S3.8-5	chorion+,ry+
92A	R3.9-2	chorion+,ry+

LOCATION	TRANSFORMANT STRAIN	MARKERS IN INSERT
92B	AR4-032(111)	w
	AR4-01(111)	w
92BC	H4	ry
92BC	B2-1	w+
92F	BS2.7-2	chorion+,ry+
93AB	R310.1	ry+
93D	SB2.1-4	chorion+,ry+
93D6,7	g5:1	Sgs3+,ry+
94B	28P-D	
94B	28P-D	ry+hsp28
94D	P[(w,ry)E]7	w, ry
94E	AR4-020	w
95A	R601.1	ry+
95AB	27S-C	hsp 28, ry
95C	D1	rp49+,ry+
95D	27S-A	hsp 28, ry
95D	BS2.7-8	chorion+,ry+
95D	unn	ry+
95F	28A-A	
95F	28A-A	ry+ hsp28
96	DR-2	ry+,Ddc+
96AB	P[(w,ry)G]3	w, ry
96B	S3.8-1	chorion+, ry+
96B	S38M-3	s38+,M13+,ry+
97A	28N	hsp 28, ry
97A	tAP-16,4.8	Adh+
97AB	+65	Hsp70,ry+
97AB	28-term	ry+,hsp28
97B	P[(w,ry)F]4-3	w, ry
97B	28P-B	
97B	28P-B	ry+ hsp28
97F	unn	ry+
98A	BS.27-1	chorion+,ry+
98B	cHB delta-89	Hsp70-lacZ,ry+
98C	R602.1, R705.1	ry+
99A	g6:3	Sgs+,ry+
99AB	DA24-44	Adh+,Ddc+
99D	SRS3.9-3	chorion+,ry+
99E	B 25	Sgs-4
100D	unn	ry+
100F	A4-4	w+,ry+

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Use this form to report new information on clones, breakpoints known within a cloned sequence, and transformed inserts.

I. Report on Cloned DNA

A. Chromosome Location:

B. Clone Name:

C. Genes in Clone:

D. Other information (strain, size, DNA sequence,restriction map references, misc.):

E. Literature Reference to Clone:

II. Report on Locating Rearrangement Breakpoints

A. Chromosome Location (of previously reported clone):

B. Rearrangement name:

C. Additional Rearrangement Breakpoints (not in clone):

D. New Order (if known):

E. Literature reference to mapping rearrangement within clone:

III. Report on Location of Tranformation Inserts

A. List Transformant Name, Insertion sites, and Markers present: Denote gene name and allele symbol (whether + or altered): <continue on back if necessary.>

B. Literature reference to Transformation:

IV. Lab Information: (Names, Lab Address, and Phone #)

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