DROSOPHILA INFORMATION SERVICE

60

June 1984

Material contributed by
DROSOPHILA WORKERS

and arranged by
P. W. HEDRICK

Prepared at the
DIVISION OF BIOLOGICAL SCIENCES
UNIVERSITY OF KANSAS
Lawrence, Kansas 66045 - USA

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Division of Biological Sciences
University of Kansas
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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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TECHNICAL NOTES

ERRATA

DIS 59 (1983), page 132: Valentin, J. "The maternal age effect on recombination is entirely reversed in mei-9b D.melanogaster". The fourth row, ct-f control recombination frequencies, should read: 36.2 34.4 36.5 36.1 36.0

DIS 59 (1983), page 161: Perez-Chiesa, Y. "Report of New Mutant". fs(1)se: female - sterile (1) small eggs: [should read] Topical applications of 0.03 µl [instead of 3] of a juvenile hormone analogue ....

25th ANNUAL DROSOPHILA RESEARCH CONFERENCE

Held April 26-29, 1984, at the Americana Congress Hotel in downtown Chicago, Illinois. Below is a list of invited speakers, topics and workshops:

Plenary Session - Genomic Plasticity — Thursday, April 26
(Gerald Rubin, Alan Templeton, David Ish-Horowicz)

Concurrent Sessions: — Friday, April 27
Developmentally Regulated Genes (Peter Wensink, Eric Fyrberg)
Sex Determination, X, Y (Tom Cline, Bruce Baker)
Pattern Formation and Early Development (Gerald Schubiger, Bob Holmgren)
Chromosome Structure and Gene Activation (Tom Grigliatti, John Lis)

Concurrent Sessions: — Saturday, April 28
ADH: From Molecular Genetics to Population Biology (Michael Ashburner)
Neurobiology (Jeff Hall)
Heat Shock (Susan Lindquist, Jose Bonner, Victor Corces, Carl Wu)
Population Genetics and Evolution (Rollin Richmond, Charles Aquadro, Tim Keith)
Gene-enzyme Systems (Ted Wright, Ross MacIntyre)

Plenary Session - Complex Developmental Loci — Sunday, April 29
(Organizer: Tom Kaufman; Speakers: Bill Gelbart, Michael Young, Michael Levine)

26th Annual Drosophila Conference will be held in Charleston, South Carolina, on Monday, April 1st thru Thursday, April 4, 1985, at the Sheraton-Charleston Hotel. It is a historic southern seaport and the Conference falls during a beautiful spring season.

Program and Registration Information will be mailed in October. Individuals who d not receive information or attend last year's conference and would like to receive a mailing, should contact: Jo Jack, NIEHS, PO Box 12233, Research Triangle Park, North Carolina 27709. Telephone: 919-541-3945.

27th Annual Drosophila Conference is planned for Asilomar, California, in 1986.

National Drosophila Species Resource Center has been transferred to Bowling Green State University. The Center is governed by a council appointed by American Society of Naturalists and is currently supported by a grant from the National Science Foundation. The purpose of the Center is to provide cultures of a wide variety of species to researchers, teachers and students.

The Center maintains approximately 350 species representing eight genera and 34 species groups in the family Drosophilidae. This is the largest collection of living eucaryotic organisms ever assembled whose evolutionary relationships and genetic biology have been extensively studied. Stocks of many species include strains having visable mutants, electromorphs and chromosomal rearrangements. A list of cultures and details on their maintenance are available from the Director. The Center intends to expand its holdings of species, especially those with mutants and chromosomal rearrangements and solicits information concerning them. New cultures will be added at the discretion of the Director.

Limited facilities are available for visiting scientists, including the large reference collection of pinned specimens assembled and provided by Prof. Emeritus Marshall B. Wheeler of the University of Texas, Austin, as well as a nearly complete reprint file on Drosophila systematics. Modern equipment for research on the genetics and biology of Drosophila is also available to qualified persons through the cooperation of the members of the genetics faculty in the Dept. of Biological Sciences, Bowling Green State University.

Cultures are provided at a cost of $20.00 for the first stock and $2.00 each for additional stocks. However, no reasonable request will be denied because of lack of funds.

CONTACT: Dr. Jong S. Yoon, National Drosophila Species Resource Center, Dept. of Biological Sciences, Bowling Green State University, Bowling Green, Ohio 43403. Telephone: 419-372-2742 or 372-2096.

REPRINT POLICY: The DIS printer does not have the capability to provide reprints for DIS contributions. Instead, each author should use photocopy services in her/his own institution. If you request the DIS office to send photocopies, we will do so and bill you for the photocopying charge plus postage.

NEW DIS TYPE STYLE appears for the first time in this issue. We have purchased a typing element called "Artisan" which you see here. It is a sans-serif style and we think provides a cleaner looking page, easier to read especially after the printer reduces the original copy. We will continue the policy of no italics and no underlining unless the situation requires it for clarification. Note that USNA has been used as the abbreviation for the United States of North America in all geographical listings, in order to distinguish it from other similar names in North & South America. It is also a helpful designator in our computer run of directory listings.

A new laboratory for research in Drosophila has been organized in the Departamento de Genetica of the Faculdade de Medicina de Ribeirão Preto of Saõ Paulo, Brasil. Dr. F. M. Sene is the head of this new laboratory, which will be devoted to research on Evolutionary Biology. Consult the Directory for a complete listing.

The 1984 revision of BIOCHEMICAL LOCI OF THE "FRUIT FLY" (Drosophila melanogaster), by L.G. Treat-Clemons and W.W. Doane, Dept. of Zoology, Arizona State University, Tempe, will appear in Genetic Maps, Volume 3, S.J. O'Brien, editor. This volume is scheduled to appear in late spring and will be published by the Cold Spring Harbor Laboratory, New York. It also appears in this issue as a Special Report, pages 17-42.

Drosophila Counter: Eric Bahn of the Institute of Genetics, University of Copenhagen, reports that the Drosophila counter developed by C. Barr of that Institute, and demonstrated at the Drosophila Conference at Asilomar and at the 8th European Drosophila Conference at Cambridge University, is now in production by, and can be ordered from, Brock & Michelsen A/S, Electronics Division, Blokken 76, DK-3460 Birkerød, Denmark. Tel (0)2 81 83 11; Telex: 39125; Order # BM8310. Price 9,250 Danish kr. (approx. $1,000 UA, plus taxes and shipping); includes instrument w/accessories, operator manual and service instructions, but does not include the pump. A KNF-Neuberger dry diaphragm pump (9 liters/min), obtainable in USA at $100-150, has proved satisfactory and is in use at the California Institute of Technology, Pasadena, to drive the vacuum system and was the pump used at the demonstration at Asilomar. Further information: Brock & Michelsen at above address. See also the description in this issue, Technical Notes: Barr & Sondergaard, page 214.

Space trip for Drosophila: In conjunction with the European Space Agency (ESA), Brunel University (Institute for Bioengineering), Uxbridge, England, is hoping to investigate the feasibility of having an unmanned space station with Biological (Life Science) experiments on board. At the present time [May 1984] we are investigating the parameters in which the material could survive, in the small automated craft. The material that may be going up includes a small green flowering plant (Arabidopsis thialina), uni-cellular organisms (Euglena gracilis or Tetrahymena rostrata) and an insect (Drosophila melanogaster). [Contributed by Debra Burbery; see Directory for complete address.]

Conference Announcement: A workshop on "Current Issues in Drosophila Development" will be held on the UCLA campus August 1-3, 1985. It is one of a series of pre- and post-conference "satellite" workshops held in connection with the 10th International Congress of the International Society of Development Biologists to be held in Los Angeles on August 4-9, 1985. For information, please contact: John Merriam or Judith Lengyel, Biology Department, University of California at Los Angeles, Los Angeles, CA 90024.

CALL FOR MATERIAL FOR DIS 61

Contributions are now being accepted for DIS 61, June 1985. Please double-space all contributions, and otherwise follow the format of DIS 59 & 60. Make sure complete information is included with every section of submissions--which will be separated from each other.

Please provide good quality black & white photographs or drawings; do not mount photographs (unless the situation requires it)--the layout person will do it according to printing specifications. Be sure your name is on all pages, tables, figures, etc., which accompany manuscripts. Note: italics and underlining are not used in the final copy formatting except in rare circumstances for clarity; scientific papers would look too cluttered with underlining otherwise.

Sections you may contribute to are:

1. Announcements, requests, etc.
2. Research Notes
3. Technical Notes
4. Teaching Notes
5. Stock Lists, mel.
6. Stock Lists, spp.
7. Linkage Data
8. New Mutants
9. Directory
10. Special Reports

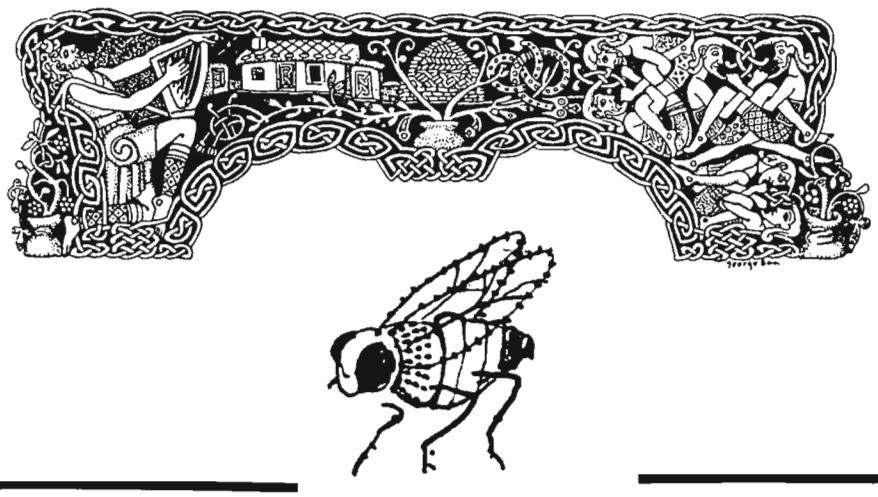
Note: A bibliography section is not being offered at this time since Dr. Herskowitz is no longer performing this task. The DIS office will continue to collect any bibliographic data you wish to send, holding it in reserve in the meantime.

DEADLINE FOR DIS 61 IS APRIL 1, 1985.

ORDER COPIES OF DIS IN ADVANCE if possible. *Subscribers paying in advance with the enclosed form or by written request will pay $5.00 per copy.* If the DIS office has to send out an invoice in December, the cost will be $6.00 per copy. Those who need additional copies of invoices are asked to photocopy the form to conserve on postage and paper work; if this is not suitable, please contact us and we will furnish a standard invoice at $6.00/copy. You may also request a standing order at this time. Orders may be air-mailed at your expense.

The Office still has on hand the following volumes:
38, 39, 40, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57(computerized stock list, 1982), 58, 59.

CHANGES OF ADDRESS FOR SUBSCRIPTIONS: please notify us before June 1st of each year; after this date we send all orders to the printer to be mailed automatically from their office and cannot cancel any mailings at that time. If you will be on leave, let us know your temporary address; many offices will not forward books. NOTE: when sending a change of address, please give your old address as well as the new one to avoid duplications.



SPECIAL REPORT

John Merriam
Biology Department
University of California
Los Angeles, CA 90024

Cloned DNA by chromosome location, May 22, 1984
Previous editions in: Genetic Maps, S.J.O'Brien (ed.)
Cold Spring Harbor Press 1984
See also: DIS 59:1-9, 1983

LOCATION	CLONE NUMBER	STOCK OF ORIGIN & OTHER INFORMATION	REF.
X chromosome			
1B1-2		Walk in Maniatis Library, Canton S, yellow achete loci	8
1B1,2-4,5		yellow, achaete, scute loci	57
1B5,8 and 2E	cos 4P	Oregon R, 70kb	57
1B11-13		su(s) locus	15
1B		adm 134E8	34
3B1,2		per locus	9
3B2 to 3C2		Oregon R, microdissection, 200Ks, white locus	62
3B	mDm112 C 10	Oregon R	1
3C1,2	λm1.2	White locus from Maniatis library	3
3C6,8	N2	Canton S, Notch locus	9
3C7		Notch locus	46
3C11,12		sgs 4 locus	45
3C7,3D1	pKdm 6B3	Intermolt I RNA	34
3, also 3R91	S24	Canton S	16
3,4	adm 136G5		34
4BC	mDm 109A7	Oregon R	1
4F5A	pkdm 35D12	late IV RNA	34
45 and 62	adm 106A10		34
4F5A	adm 139C12		34
5AB	adm 126D6		34
5C			12
5EF, and 6F64A	adm 140C11		34
5F (prox.)	B70	maternal restricted transcript	69
6F5		Sex lethal locus	66
7A5			67
7B3,4		Oregon R, cut locus 100kb	29
7D5,5 and short walk distal		Canton S, Oregon R	21
7E6,7F1,2		150 kb overlapping	44
78	adm 132H10		34
8A		100 kb overlapping	44
8D	PLZ-p	lozenge locus	2
8F9A	PYp1	Canton S, yolk protein 1 locus	2
8F9A	PYp2	Canton S, yolk protein 2 locus	2
8	56	Canton S	16
10A1,2		Vermillion locus	65
10C1,2		RNA polymerase II locus	51
10EF	adm 134A3	late V RNA	34
10EF, and 32AC	adm 130E12		34
10F	adm 10F.1	minor heatshock cDNA from Kc cells	39
11A		gastrulation defective locus, Maniatis lib.	38
12B,C	PYP3	Canton S, yolk protein 3 locus	2
12DE	pDt17R*	Ser 7 tRNA locus	27
12DE	pDt27*	Ser 4 tRNA locus	27
12DE	pDt73*	Ser 4-7 tRNA loci	27
12E	pDt16*	Ser 4-7 tRNA loci	27
12F	λ32-10	tRNA locus	58
12,13	adm 136F10		34
12	S21b	Canton S	16
14BC	adm 132B8		34

LOCATION	CLONE NUMBER	STOCK OF ORIGIN & OTHER INFORMATION	REF.
15A1		rudimentary locus	30
15A,B	548	Oregon R+ Head Specific RNA	31
16B3-5	PTE-1		2
16EF		Maniatis library: 50kb, Shaker locus	40
16F17	adm 135H4		34
17AB	λdmpt 61		58
18CD	A57	maternal restricted transcript	69
18D	λDmG21	G6PD locus, Oregon R+	28
19EF20AB	DCg2	collagen-like gene, from Maniatis library	25
19F	pDt67R	Lys 5 tRNA locus	27
2L			
21B	adm 142G5		34
21D	pD957		3
21F22A	adm 123D12, 123H3, 128B8		34
22B/C	adm 129E7		34
22F1,2		130 Kb, decapentaplegic complex	55
23A3-7		70 Kb, Maniatis library	59
23BC	B13	maternal restricted transcript	69
23E	pDt5*	Ser 7 tRNA locus	27
24C	mDm101A10	Oregon R	1
25BC	mDm109De	Oregon R	1
25C	DCg-1	collagen-like gene, from Maniatis library	25
25D	150-3(λ)	blastoderm-specific poly(A) RNA	47
25D1-4	MH5	from Gelbart library	6
26A7-9		beta galactosidase locus	6
26AB	A20	minor site at 88D	69
27C		GAR transformylase	7
27D	λ39-1	(Repetitive, also hybridizes to 91C and 43A, tRNA locus)	58
27F	adm 125G11		34
28A	551	Oregon R+, Head Specific RNA	31
28C	538	Oregon R+, Head Specific RNA	31
28C	λdmpt 49		58
28D9-12		CDNA, Kc cells	8
29A	pDt59R*	Lys 5 tRNA locus	27
29B1-4		CDNA, Kc cells	8
29C		SRC homologous	61
30B	λdmpt 75		58
30DE	adm 136De		34
30EF	λdmpt 104		58
31A	mDm 106A10	Oregon R	1
31C	adm 134G6		34
31C33 B	adm 142H3		34
31F, and 39F	adm 142F4		34
32AB	503	Oregon R+, Head Specific RNA	31
32CD	231	Maniatis library, myogenic cell RNA	63
33AB		extra sex combs locus; 250 kb	70
33B	adm 124D9		34
34AB	A34		69
34F	527	Oregon R+, Head Specific RNA	31
35B3-5	AC	alcohol dehydrogenase locus, Maniatis library	52
35B	mdm 103D5	Oregon R	1
35C36	adm 125E7		34
36B		myosin heavy chain locus, Maniatis library	13
36B		walked from myosin heavy chain locus	20
37B9C1,2		Df(2L)hk18	35
37B13-37C5	λDdc-1 thru-20	dopa decarboxylase locus, 100 Kb	35
38A6	2E2		35

LOCATION	CLONE NUMBER	STOCK OF ORIGIN & OTHER INFORMATION	REF.
39CD		cDNA, definitive ribosomal protein from Spradling Mahowald library	17
39DE		histone locus	54
39E, and 2L Base	adm 136D9		34
21 base (chromocenter)	adm 106H5, 123C3		
2 R			
2R base, and 3L base (Chromocenter)		adm 130B2	34
42A	mDm 106F8	Oregon R	1
42E	pDt 61	tRNA-Lys-2 locus	27
42EF	adm 126F7, 127A10		34
43AB	555	Oregon R+, Head Specific RNA	31
43BC	B17	maternal restricted transcript	69
43DE	B45	maternal restricted transcript	69
44CD	536	Oregon R+, Head Specific RNA	31
44D	adm 112C11, 126G12		34
44D	λDmLCP1-13	larval cuticle protein loci: 50kb	36,37
44F	129E7		3
44	L10	Canton S	16
45A	mDm103H10	Oregon R	1
45A	mDm108C7	Oregon R	1
45D, and chromocenter	mDm108A8		1
46B	B41	maternal restricted transcript	69
46DF, and chromocenter	236	Maniatis library, myogenic cell RNA	63
46E	549	Oregon R+, Head Specific RNA	31
47E	528	Oregon R+, Head Specific RNA	31
47F		50 Kb	14
47F48D	217	Maniatis library, myogenic cell RNA	63
48A		engrailed locus, Canto S 208 Kb	14
48B	pDt74	Met 2 tRNA locus	27
48C	adm 132A7		34
48E	adm 135E10		34
48F	543	Oregon R+, Head Specific RNA	31
49C	mDm101D3	Oregon R	1
49CD	mDm101D12	Oregon R	1
49DE	adm 140D1		34
49F	λDm1606	Troponin C locus from Maniatis library	22
50B	adm 142E9		34
50C	mDm3021	Oregon R	1
50CD	adm 133H7, 136F9, 138G8, 130H8		34
50	L6	Canton S	16
51A	S34	Canton S	16
51B	S14	Oregon R+, Head Specific RNA	31
51CD	A19	maternal restricted transcript	69
51D	adm 134E2		34
51DE	mDm102F11	Oregon R	1
51DE	mDm102B6	Oregon R	1
52B	mDm107A2	Oregon R	1
52DF	adm 139H3		34
53CD	λDm 32 (Class A)	Amy pseudogene, from Maniatis library	33
53F	λdmpt 116		58
53	L23	Canton S	16
54A1B1(54A)	λDm 65 (Class B)	Amy duplication locus, Canton S	33
54E	adm 54E.1	Minor heat shock cDNA	39
54F55A	adm 110A4, 132C9, 132E11, 132E12, 132G5, 134A4, 135D12		34
55BCD	adm 110G1, 110H1, 132D6		34

LOCATION	CLONE NUMBER	STOCK OF ORIGIN & OTHER INFORMATION	REF.
55F	B32	maternal restricted transcript	69
56C	DTB2	βtubulin locus, from Maniatis' library	25
56D412	KV 2-70a	βtubulin locus, from Maniatis' library	22
56EF	adm 135H8		34
56F	λDmt 56-6	$tRNA^{Gly}$ locus	58
57B			12
57C	525	Oregon R+, Head Specific RNA	31
58F	adm 132A3, 135D10, 135E6		34
60A (distal)	A8		69
60A (prox.)	B6	maternal restricted transcript	69
60A	adm 125C2		34
60A,and nucleolus	adm 106H6		34
60BC	B50	maternal restricted transcript	69
60C6-8	KV 1-11	β tubulin locus, from Maniatis' library	22
60C	DTB3	β tubulin locus, from Maniatis' library	25
3 L			
61A1-3	mDm105F3	Oregon R	1
62A	adm 112C10		34
62A	λ48-9	tRNA locus	58
62AB, 97C	adm 140F12		34
62CD	203	Maniatis library, myogenic cell RNA	63
62D	adm 142F6		34
63AC	227	Maniatis library, myogenic cell RNA	63
63B	bDm 4L	Oregon R, hsp 83 locus	39
63BC	λ6	Canton S, hsp 83 locus	10
63BC	pPW244, 301, 330	Oregon R, hsp 83 locus	10
63F	adm 63 F.1	minor hsp locus	39
63-66	S7	Canton S	16
64B	Drsrc	SRC homologous	60,61
64BC, and chromocenter	mDm104C1	Oregon R	1
64C	DHSV4	RAS homologous	60
64C	λdmpt 85		
64F	mDm106E3	Oregon R	1
64F	λdmpt 120		58
64F, 66C	adm 126B4		34
64F/65A	adm 135G4		34
65C	adm 111F10		34
66CD	adm 106E3		34
66D9-10	.8247, 30152, 3019		32
66D10-15		Oregon R, 85kb	57
66D11-15		100 kb overlapping	44
66D	507	Oregon R+, Head Specific RNA	31
66D	547	Oregon R+, Head Specific RNA	31
66F	λdmpt 121		58
67A5-7 to 67B1,2		Walk from Maniatis library	22
67B	λ88	Canton S, loci of hsp 22, 23, 26 and 28	10
67B	λDmp 67	hsp loci & flanking transcripts, from Canton S	43
67B	J1	includes hsp 28, 23, 26 loci, Oregon R	42
67C	DTA2	α tubulin locus, from Maniatis library	25
68C1-5	λcDm2021	In(3L)HR15	1
68C3-7; 68E	λbDm2054	Df(3L)vin	1
68C7-15	mDm148F7	Oregon R, sgs 3,7,8 loci	1
68C	pkdm 2G6	intermolt II RNA	34
68C	pkdm 2C1	intermolt III RNA	34
68C	pkdm 1H2	intermolt IV RNA	34
68C	adm 134C10		34
68EF	adm 133H1		34
69F	270	Maniatis library, myogenic cell RNA	63

LOCATION	CLONE NUMBER	STOCK OF ORIGIN & OTHER INFORMATION	REF.
69	L3g	Canton S	16
70A	adm 107A4		34
70AB	adm 128C11, 132B3		34
70BC	B20	maternal restricted transcript; minor homology to 2F-3A and chromocenter	69
70BC	pDt 55*	Val 4 tRNA locus	27
70C	adm 29D11		34
71A	2-5 (λ)	gastrula-differential poly(A) RNA	47
71AB	adm 123C4		34
71C3.4D1.2		EIP 28/29 locus	26
71 CE	λcDm 20,21,23, 23, 24	ecdysone induced late puff from Maniatis library	24
71DE	adm 134A9, 134A11, 134C11		34
71DE	pkdm 46B7	late I RNA	34
71DE	pkdm 38C9	late II, III RNA	34
71DE	pkdm 38C4	late II, III RNA	34
72BC	557	Oregon R+, Head Specific RNA	31
72DE	λdmpt 115		58
73B	Dash	Abelson SRC homologous	60,61
73D	adm 73D.1	minor heat shock locus	39
73DEF	521	Oregon R+, Head Specific RNA	31
74EF		early ecdysone responding puff, 300 Kb from Maniatis library	56
75C	adm 135F3		34
75	S39	Canton S	16
76A	adm 132D11		34
76DE	B48	maternal restricted transcript	69
76F	mDm 104G3	Oregon R	1
79B			12
79E1,2	13E5	Or,R PBR322	16
80C		Kc cells	8
3 L base (chromocenter)	adm 139A10		34
3 R			
3 R base (chromocenter)	adm 128F12		34
82A	S6-7	from Maniatis library	22
82F	506	Oregon R+, Head Specific RNA	31
83A	adm 136E4		34
83AB	adm 140E12		34
83A,B	pDt 66R2	Lys 5 tRNA locus	27
83B	adm 123G4		34
83C	mDm 105 B9	Oregon R	1
83CD	B21,B31	maternal restricted transcript	69
83F	adm 140C1		34
84A,B	pDt 12	Lys 5 tRNA locus	27
84A,B	pDt 39*	Lys 5 tRNA locus	27
84A4,5 to 84C1,2		Antennapedia complex, 440 Kb	49
84B1-3		Maniatis library: 240 Kb	49
84B3-6	λDm 2.55a	α tubulin locus, from Maniatis library	22
84B3-C1,2		75 Kb, Maniatis library	48
84B	DTA 1	α tubulin locus, from Maniatis library	25
84BC	adm 123D11		34
84C	Dm A 3a, 4a, 4b,	5a, 5b Maniatis library	4
84D3,4	1,2,3,10	30 kb from Maniatis library, overlaps Val 3b tRNA locus	19
84D4-8	λDm 5-1	α tubulin locus, from Maniatis library	25
84D	mDm 104H7	Oregon R	1
84D	pDt 78 RC*	Val3btRNA locus	27

LOCATION	CLONE NUMBER	STOCK OF ORIGIN & OTHER INFORMATION	REF.
84D	DTA 4	α tubulin locus, from Maniatis library	25
84E1,2		105 Kb, double sex locus and flanking, Maniatis library	48
84E11-12 to F4-5		Maniatis library: 240 Kb	50
84F2,3		In(3R)Hu	50
84F	B34	maternal restricted transcript	69
85A	λ50-8	tRNA locus	58
85C	λm 1:2	from Gelbart library	6
85D6-12	DTB 4	β tubulin locus, from Maniatis' library	25
85D	KV 1-22	β tubulin locus, from Maniatis' library	22
85D	542		16
85D	DHSV7	RAS homologous	60
85E6-10	λDm 5-22	α tubulin locus, from Maniatis' library	22
85E	DTA 3	α tubulin locus, from Maniatis' library	25
85E	mDm 3008	Oregon R	1
86B4C1		In(3R)Hu	50
86	adm 35E6		34
86	S35g	Canton S	16
87A7	pPW 223	Oregon R, hsp 70 locus	10
87A7		hsp 70 locus subclone	39
87A	56H8	hsp 70 locus and flanking	41
87A	GB	Hsp70, Sn cell DNA	42
87AB	540	Canton S	16
87C1	pPW232,pPW229	Oregon R, hsp 70 locus	10
87C1	132E3	hsp 70 locus and flanking	41
87C	G3	hsp 70, Sn cell DNA	42
87CF, 94D	adm 125G5		34
87D	mG31	Hsc 70; Oregon R	42
87E			12
88B	adm 88B.1	minor heat shock cDNA	39
88C	mDm 104D12	Oregon R	1
88E	mG34	Oregon R; hsc 70 locus	42
88F2-5	λDM 85	3 tropomyosin loci	22
88F		250 kb walk, actin locus	11
88F	λdmpt 73	tropomyosin locus	58
88F			12
88	S32	Canton S	16
89A	EU 27		23
89B	pDt 14*	Val 4, Phe 2 tRNA loci	27
89E1-4		300 kb walk, bithorax complex	18
90BC	pkdm 7E5	Intermolt V RNA, sgs locus	34,24
90BC	pDt 92RC*	Val 4 tRNA locus	27
90BC	pDt 120 RC*	Val 4 tRNA locus	27
90BC	pDt 41 RC4*	Val 3b, Pro tRNA loci	27
90BC	λbDm 1508	Oregon R, Hogness library	24
90BC	pDt 48*	Val 3b, Pro tRNA loci	27
90C	λ49-4	repetitive, also 85C and 84D, tRNA locus	58
91D	mDm 103G4	Oregon R	1
91	S24	Canton S, also X3	16
92A	mDm 101F8	Oregon R	1
92CD	512	Oregon R+, Head Specific RNA	28
92E	adm 124B10		34
92	S12g	Canton S	16
93D	adm 129F5		34
94A	adm 134C5, 135D2		34
94E	λdmpt 123		58
94F95A	156-1 (λ)	blastoderm-differential poly(A) RNA	47
95B	mDm 108E11	Oregon R	1
95D	pPW227	Oregon R, hsp 68 locus	10

95D	λ15	Canton S. hsp locus	10
96A	adm 137A2		34
96D	mDm 107D4	Oregon R	1
96F97A	adm 126D12		34
96F97C	adm 132C4, 132E7, 132H4		34
97A	λdmpt 50		58
97EF	DTB1	β-tubulin locus, from Maniatis library	25
97F	KV 3-12	β-tubulin locus, from Maniatis library	22
98,99	L2	Canton S	16
98E	B8	maternal restricted transcript	69
99C5-6	558	transient receptor potential locus, 45 kb	68
99C	559	Oregon R+, Head Specific RNA	31
99D	153-1 (λ)	blastoderm-specific poly(A) RNA	47
99D	rpro 49	ribosomal protein locus	53
99E1-3	36-1 (λ)	blastoderm-differential poly(A) RNA	47
99E	λDm 11-9	myosin light chain locus Maniatis library	22
99E	adm 132G9		34
99F	adm 142D9		34
100AB	5D7	OR.R PBR322	16
100B	mDm 103 F1	Oregon R	1
100B	λdmpt 31		58
100B	516	Oregon R+, Head Specific RNA	31
100C1-7	mDm 102A3	Oregon R	1
100D	mDm 105 H1	Oregon R	1
100	S2	Canton S	16
4th chromosome			
102C, also chromocenter	mDm 108 D1	Oregon R	1
102CD	116H2		3
102EF	λdmpt 101		58
Multiple			
5C, 42A, 57A, 79, 87F/88A, 88F	adm 105C6, 105G9, 108D11	Actin repeated locus	34
5CD, 24F, 30EF, 63F/64A	adm 136H5		34
21E, 82E, 95AC	pDm U1.4d RNA coding seq.		22
telomeres + β heterochromatin	T-A		22
	T-F		22
mitochondrial	710	Hind III C/Ch21A	22
	13	EcoRI C+B/Ch 4A	22
	23	EcoRI B /Ch 4A	22
	41	EcoRI C /Ch 4A	22
X base, 30F 48 D/E, 96	adm 135D5		34
25A/C, 44D, 64F65A, 66CD, 67B, 99C, 99F	adm 8G8, 26H2, "Jonah", 135A8, 135A10		34
48CD, 60A, 100C	adm 128A7		34
50BC, 50F, 58/59	adm 135D11		34
87C1, 42B, chromocenter	cDm 703	alpha beta repeated locus	39

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SPECIAL REPORT

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Chromosome rearrangements available for "jumping" with one breakpoint known within a cloned sequence.
May 31, 1984

TARGET BREAKPOINTS	CLONED LOCUS	STOCK DESCRIPTION	REF
X chromosome			
1B1-4C1	1B2,3 (achaete)	$In(1)ac^{3}$	5
1E2,3	1B2,3 (scute)	$In(1)sc^{260-22}$	5
1F	89E1-4 (BXC)	$T(1;3)Uab^{5}$	10
3A6-8	3C1-4 (from Notch)	$Df(1)64j4$	7
3B1,2	3C6 (Notch)	$Df(1)62d18$	7
3C1-2	3C11-12(Sgs-4)	$Df(1)w^{Nfm20}$	9
3C2-3	3C11-12(Sgs-4)	$Df(1)N^{fm21}$	9
3C2-3	3C11-12(Sgs-4)	$Df(1)N^{10}$	9
3C6	3C11-12(Sgs-4)	$Df(1)N^{541g}$	9
3C9,10	3C7,8	$In(1)N^{76b8}$	7
3D5,6	3C7,8	$Df(1)N^{62b1}$	7
3E4	3C11-12(Sgs-4)	$Df(1)dm^{75elg}$	9
4D	89E1-4 (BXC)	$T(1;3)bxd^{111}$	10
5B	89E1-4 (BXC)	$T(1;3)Ubx^{21560.8A}$complex	10
5D3-6	1B4-6 (scute)	$In(1)sc^{7}$	5
11D3-8	1B2,3 (scute)	$In(1)sc^{260-14}$	5
13A2-5	1B (scute)	$In(1)sc^{29}$	5
18A3,4	1A8B1 (yellow)	$In(1)y^{4}$	5
18B8,9	1B2,3 (scute)	$In(1)sc^{9}$	5
20C1	1B3,4 (scute)	$In(1)sc^{4}$	5
20C	1B3,4 (scute)	$In(1)sc^{L8}$	5
20D1	8B1,2 (achete)	$In(1)y^{3P}$	3
20D1	1B2,3 (scute)	$In(1)sc^{8}$	3,5
20D1	1B3,4 (scute)	$In(1)sc^{S1}$	5
20F	3B1,2 (per)	$Df(1)w^{-64d}$, In3B1,2-20F, Df3B1,2-3C2,3	7
20	2B1,2 (1(1)BA11)	$Dp(1;f)101$	8
20	89E	$T(1;3)Cbx^{rvR17.49A}$	10
20	89F	$T(1;3)P115$	10
20	89F	$Dp(3;1)P68$	10
Chromosome II			
2Ltip	89E	$T(3;2)bxd^{D36}$ complex	10
21L1,2	89E	$T(2;3)Ubx^{16160.18}$	10
22B1,2	89E	$T(2;3)Cbx^{rvR17.175}$	10
25A	1B4-7 (scute)	$T(1;2)sc^{19}$	5
29A-C	89E	$T(2;3)P10$	10
29	89E	T(2;3)Hm complex	10
31	89E	$T(2;3)Ubx^{18264.1}$	10
32	89E	T(2;3)Hm complex	10
34	89E	$T(2;3)Ubx^{4.30}$ (Madrid)	10
36D1-E1	37C1,3 (Ddc)	$Df(2L)VA18$	4
36E4-6	37B9-C1,2 (Ddc)	$Df(2L)hk18$	4
37F5-38A1	37C1,2 (Ddc)	$Df(2L)VA17$	4
38B1,2-C1,2	37C1-5 (Ddc)	$Df(2L)VA12$	4
38C1,2-D1,2	37C1-5 (Ddc)	$Df(2L)TE42-1$	4
38F4	37C1-5 (Ddc)	$Df(2L)TE42-1$	4
39	89E	$T(2;3)Ubx^{19286.8m}$	10
41A	89E	$T(2;3)Ubx^{17756.180}$	10
41A	89E	$T(2;3)Ubx^{18136.147}$	10
41A	89E	$T(2;3)Ubx^{19649.18}$	10

TARGET BREAKPOINTS	CLONED LOCUS	STOCK DESCRIPTION	REF
41A	89E	T(2;3)Ubx^{D1}	10
41A	89E	T(2;3)Cbx$^{rvR17.34}$	10
41A	89E	T(2;3)Cbx$^{rvR17.22x}$	10
41A	89E	T(2;3)bxd^{22044D}	10
41A	89E	T(2;3)bxd^{B231}	10
41F	89E	5(2;3)Ubx$^{16160.36}$	10
41	84B2 (Antp)	T(2;3)Antp^{NS+RC8}	2
42BC	89E	T(2;3)bxd$^{X22290.11x}$	10
44A	89E	T(Y;2;3)Mcp^{rvc10} complex	10
44C4	89E	T(2;3)P75Ubx$^{5T17.14-17}$	10
51E	89E	T(2;3)Cbx$^{rvR17.6F}$	10
52A-C	89E	T(2;3)Ubx$^{X6000.78A4}$	10
53C	89E	T(2;3)Ubx^{105}	10
54E	89E	In(3LR)89/75+T(2;3)54/75, Ubx 6-26 (Madrid)	10
59C	89E	T(2;3)bxd$^{29315.46}$ complex	10
59-60	89E	T(2;3)Mcp^{rvC1} complex	10
60B	89E	T(2;3)Mcp^{rv26105A}	10
60C1-3	1B4-7 (scute)	T(1;2)sc^{S2}	5
Chromosome III			
61F-62A	89E	In(3LR)Ubx^{300}	10
64D	68C1-5	In(3L)HR15	1
64E	87D(rosy-Ace)	In(3LR)3^{ry64}	10
66B	89E	Tp(3)P47	10
66C	89E	Tp(3)bxd^{100}	10
68A	89E	Tp(3)Ubx^{P20}	10
68E3,4	68C3-7	Df(3L)vin^{3}	1
68E	89E	Tp(3)Ubx7^{P20}	10
69B5-C2	68C10-12	Df(3L)vin^{7}	1
69C3-4	89E	In(3LR)bxd^{113}	10
70D	89E	In(3LR)Cbx$^{rvR17.42}$	10
71CD	1B4,5 (scute)	t(1;3)sc$^{260-15}$	5
71F	84B2 (Antp)	In(3LR)Antp^{PW}	2
72D11-72E1	89E	In(3LR)bxd^{106}	10
74F-75A	84B2 (Antp)	In(3LR)Antp^{NS+RC4}	2
74	89E	In(3)Ubx^{130}(TM2) complex	10
75C	89E	In(3LR)89/75+T(2;3)54/75, Ubx$^{6.26}$ (Madrid)	10
79C-E	89E	In(3LR)Cbx$^{rvR17.44v}$	10
80B	89E	In(3LR)Cbx$^{rvR17.16R}$	10
80F	89E	In(3LR)bxd$^{19409.2X}$	10
81	89E	In(3)Hab^{rvFC51}	10
81	89E	In(3)Ubx^{125}	10
81	89E	In(3)Mcp^{rvC1} complex	10
3R het	87D (rosy-Ace)	In(3R)ry^{54}	10
3R het	87D (1(512))	in(3R)ry^{PS11136}	10
84A1	84B2 (Antp)	Df(3R)Scr	2
84A1,2	84B2 (Antp)	Df(3R)JA99	2
84A4,5	83D4,5 (B31)	Tp(3;3)Dfd	6
84B1	84B2 (Antp)	Df(3R)ASCB	2
84D1,2	84B2 (Antp)	In(3R)Antp^{73b}	2
84D	84B2 (Antp)	Df(3R)Antp^{NS+RC7}	1
84D	89E	T(2;3)bxd^{DB6}	10
84F2,3	84B2-C1 (Hu)	In(3R)Hu	2
84F	89E	In(3)Mcp$^{rv29175.10}$	10
85E	84B2 (Antp)	In(3R)Antp^{B}	2
85F	84B2 (Antp)	In(3R)Hu	2
86B4-C1	84B2-C1 (Hu)	In(3R)Hu	2
87B	89E	In(3)Ubx^{882}	10

TARGET BREAKPOINTS	CLONED LOCUS	STOCK DESCRIPTION	REF
87C1-3	87E1,2 (rosy-Ace)	$Df(3R)ry^{81}$	10
87C7-8	87E1,2 (rosy-Ace)	$Df(3R)kar^{SZ11}$	10
87D1,2	87D14 (rosy-Ace)	$Df(3R)ry^{75}$	10
87D2-4	87D14 (rosy-Ace)	$Df(3R)ry^{614}$	10
87D2-4	87E1,2 (rosy-Ace)	$Df(3R)ry^{1402}$	10
87D2-4	87E1,2 (rosy-Ace)	$Df(3R)ry^{1301}$	10
87D3,4	87E1,2 (rosy-Ace)	$Df(3R)ry^{1607}$	10
87D5,6	87E1,2 (rosy-Ace)	$Df(3R)ry^{1608}$	10
87D12-14	89 (pic)	$In(3)Cbx^{rv21988B}$	10
87E1,2	89E	$In(3)Cbx^{+R1}$	10
87E11,F1	87E5,6 (rosy-Ace)	Df(3R)1C4a	10
87E12,F1	87D6--8 (rosy-Ace)	$Df(3R)ry^{619}$	10
87E-F	89	$In(3)Cbx^{rv21987A}$	10
87F11,12	87E1,2 (rose-Ace)	Df(3R)126c	10
87F	89E	$In(3)Cbx^{wt}$	10
87F	89E	$T(2;3)Cbx^{rvR17.6F}$	10
87F-88A	89E	In(3)Ubx80	10
88B	89E	$In(e)Ubx^{12.5}$ (Madrid)	10
88C4	89E	In(e)56A62(Szeged)	10
89A	89E	$In(3)Cbx^{3}$ (Cbx-like)	10
89A-B	89E	$In(3)bxd^{27830.C5A}$	10
89B21	2B3,4(Sta)	T(1;3)sta	8
89B	89E	In(3)Camel	10
89C1,2	89E	T(2;3)P10	10
89C	89E	$In(3)bxd^{183}$	10
90A	89E	$In(3)Ubx^{5.12}$ (Madrid)	10
90A	89E	$In(3)Sab\ Mcp^{rv29340.8}$	10
90B2	89E	$T(1;3)bxd^{111}$	10
90C	89E	$In(3)Ubx^{961.29}$	10
90E	89E	In(3)Tab	10
91B	89E	$In(3)Ubx^{3966.30}$	10
91C	89E	$In(3)cbx^{2}$	10
91D1,2	89E	$Tp(3)bxd^{110}$	10
91F-92A	84B2 (Antp)	$In(3R)Antp^{LC}$	2
92A1,2	89E	$Tp(3)bxd^{110}$	10
92A	89E	$In(3)Cbx^{rvR17.5E}$	10
92	89E	Tp(3)P47	10
93B	89E	$In(3)Ubx^{130}$(TM2) complex	10
94A	89E	Tp(e)Vno	10
94A	89E	$Tp(3)Mcp^{B277}$	10
96A	89E	$In(3)Ubx^{19286.76}$	10
96F	89E	$Tp(3)Mcp^{B277}$	10
96F-97A	89E	Tp(3)Vno	10
96F-97A	89E	$In(3)89E/97F-97A,\ Cbx^{rv21560.60}$	10
97CD	89E	$In(3)Ubx^{3798.68}$	10
97D	89E	Tp(3)abx	10
98B-C	89E	$In(3)Ubx^{X}$	10
98D-F	89E	$In(3)Mcp^{rvB315}$	10
98F1,2	83D4,5 (B31)	Tp(3;3)Dfd TRX1	6
98±	1B4,5 (scute)	$T(1;3)sc^{KA8}$	5
99E1-F1	87D8-10 (rosy-Ace)	$Df+In(3R)kar^{1g27}$	10
99	89E	$In(3)Ubx^{1928.16N}$	10
Chromosome IV			
101-102	1B4-C3 (scute)	$T(1;4)sc^{H}$	5
101F	89E	$T(3;4)bxd^{101}$	10
102	89E	$T(3;4)Ubx^{A}$ complex	10
102	3B1,2 (per)	T(1;4)JC43	7
4th	89E	$T(3;4)Cbx\ rvR17.40R$	10
4th het	87D12-13 (rosy-Ace)	T(3;4)ryP51149	10

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(All BXC rearrangements from E.Lewis at Cal Tech except when designated Madrid). Allele designations are tentative.

SPECIAL REPORT

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Transformed lines available for cloning or deleting DNA at specific chromosome sites.

May 25, 1984

Cytological location of inserts	Transformant strain	Markers/genes present in construction	REF
X Chromosome			
1CD	BS2.7-12	chorion$^+$,ry$^+$	5
1F	R702.1	ry$^+$	5
1F	R704.2	ry$^+$	5
1F	DA24-14	Adh$^+$,Ddc$^+$	4
2B	Tf(1)GR304-1	ry$^+$	1
3B	Tf(1)GR420-3	ry$^+$	1
4D	R405.1	ry$^+$	5
6F	S6.9-2	chorion$^+$,lac$^+$, ry$^+$	5
7D	R403.1	ry$^+$	5
8BC	tAP-25,3.2	Adh$^+$	2
9A-D	R404.2	ry$^+$	5
9B		ry$^+$	7
9B	tAP-24B,3.2	Adh$^+$	2
9B	SB2.1-5	chorion$^+$, ry$^+$	5
9E	R701.1	ry$^+$	5
10BC	tAP-20,3.2	Adh$^+$	2
12A	tAP-17, 4.8	Adh$^+$	2
12BC	SRS3.9-1	chorion$^+$, ry$^+$	5
12D	R301.2	ry$^+$	5
13A-C,14F,69A,98C	BS2.7-5	chorion$^+$, ry$^+$	5
13CD	SB2.1-6	chorion$^+$, ry$^+$	5
15DE	BS2.7-10	chorion$^+$, ry$^+$	5
16D		ry$^+$	7
17DE	B1-2	w$^+$	3
18A	R704.3	ry$^+$	5
18D	BS2.7-3	chorion$^+$, ry$^+$	5
20A	Adh^{hs20A}	Adh$^+$, hsp 70	6

Cytological location of inserts	Transformant strain	Markers/genes present in construction	REF
Chromosome II			
21D	R602.1	ry^+	5
21DE	tAP1-10,4.8	Adh^+	2
22A	R604.1	ry^+	5
25C	R401.3	ry^+	5
28A	BS2.7-11	$chorion^+$, ry^+	5
29B	R308.1	ry^+	5
30C	S6.9-3	$chorion^+$, lac^+, ry^+	5
32BC	BS2.7-13	$chorion^+$, ry^+	5
35DE,45A,59E	S11.4-1	$chorion^+$	5
36C	tAP-8C,4.8	Adh^+	2
38BC	tAP19,4.8	Adh^+	2
38E,57F		ry^+	7
39BC	S6.9-8	$chorion^+$, lac^+, ry^+	5
39EF	$A^{R}4$-2	w^+, ry^+	3
39E,40F (2L)	$A^{R}4$-3	w^+, ry^+	3
42A	R301.1	ry^+	5
42A	tAP-13,4.8	Adh^+	2
42AB	R303.1	ry^+	5
42DE		ry^+	7
42E	R305.1	$chorion^+$, lac^+, ry^+	5
43	R704.1	ry^+	5
43C	R304.1	ry^+	5
43E,44CD	S6.9-7	$chorion^+$, lac^+, ry^+	5
44E	R3.9-4	$chorion^+$, ry^+	5
45E	DR-18	ry^+, Ddc^+	4
47A	tAP18, 4.8	Adh^+	2
50B	R306.1	ry^+	5
50B	S6.9-6	$chorion^+$, lac^+, ry^+	5
52A	DR-12	ry^+, Ddc^+	4
52B	tAP-21,3.2	Adh^+	2
52D	SRS3.9-1	$chorion^+$, ry^+	5
52F	R402.1	ry^+	5
53E	R3.9-1	$chorion^+$, ry^+	5
53EF	SB2.1-1	$chorion^+$, ry^+	5
54C	Adh^{hs54c}	Adh^+, hsp 70	6
56D	R3.9-6	$chorion^+$, ry^+	5
56F	DR-15	ry^+, Ddc^+	4
56F	DR-5	ry^+, Ddc^+	4
58EF	R3.9-5	$chorion^+$, ry^+	5
58F,59C	tAP-7A,4.8	Adh^+	2
59B	A3-1	w^+, ry^+	3
60A	R302.1	ry^+	5
60B	BS2.7-4	$chorion^+$, ry^+	5
60C		ry^+	7
60E	tAP-15A,4.8	Adh^+	2
60F	S3.8-4	$chorion^+$, ry^+	5
Chromosome III			
61A	Bg61	ry^+, $hsp70^+$, $lacZ^+$	8
61C	Adh^{hs61c}	Adh^+, hsp70	6
61E	SRS3.9-4	$chorion^+$, ry^+	5
62AB	tAP-27	Adh^+	2
64F-65A	SB2.1-3	$chorion^+$, ry^+	5
64C	R405.1	ry^+	5
64C	tAP-7B, 4.8	Adh^+	2
64D	Bf64 (lethal)	ry^+, $hsp70^+$, $lacZ^+$	8
66D-67A	DR-17	ry^+, Ddc^+	4
66E1,2	Tf(32)6A6.0-1	Adh^+	1

Cytological location of inserts	Transformant strain	Markers/genes present in construction	REF
68A	R7.7-1	chorion$^+$, ry$^+$	5
69CD	tAP-12,4.8	Adh$^+$	2
70AB	S3.8-3	chorion$^+$, ry$^+$	5
70C	S3.8-3	chorion$^+$	5
71F	tAP-11,4.8	Adh$^+$	2
75CD	R502.1	ry$^+$	5
75D	R706.1	ry$^+$	5
78BC	R603.1	ry$^+$	5
82B	Adh^{hs82B}	Adh$^+$, hsp 70	6
82BC	tAP-8B, 4.8	Adh$^+$	2
83A		ry$^+$	7
83BC	SB2.1-2	chorion$^+$, ry$^+$	5
83F	R3.9-3	ry$^+$	5
84BC, 96B	S3.8-1	chorion$^+$, ry$^+$	6
84C		ry$^+$	7
85A	R309.1	ry$^+$	5
85D	BS2.7-7	chorion$^+$, ry$^+$	5
85F	tAP-15B, 4.8	Adh$^+$	2
86D	R311.1	ry$^+$	5
87A	R307.1	ry$^+$	5
87CD	C1-1	w$^+$	3
87E		ry$^+$	7
87F	R308.2	ry$^+$	5
87F	R404.1	ry$^+$	5
87F	DRI-15	ry$^+$, Ddc$^+$	4
88C	S6.9-5	chorion$^+$, lac$^+$, ry$^+$	5
88E	BS2.7-9	chorion$^+$, ry$^+$	5
88E	R401.2	ry$^+$	5
88F	S6.9-1	chorion$^+$, lac$^+$, ry$^+$	5
89A	tAP-14,4.8	Adh$^+$	2
89A	B1-1	w$^+$	3
89B	BS2.7-6	chorion$^+$, ry$^+$	5
90CD	S3.8-2	chorion$^+$, ry$^+$	5
90EF	DR-1	ry$^+$, Ddc$^+$	4
91C	A2-1	w$^+$, ry$^+$	3
91F-92A	S3.8-5	chorion$^+$, ry$^+$	5
92A	R3.9-2	chorion$^+$, ry$^+$	5
92BC	B2-1	w$^+$	3
92F	BS2.7-2	chorion$^+$, ry$^+$	5
93AB	R310.1	ry$^+$	5
93D	SB2.1-4	chorion$^+$, ry$^+$	5
95A	R601.1	ry$^+$	5
95D	BS2.7-8	chorion$^+$, ry$^+$	5
95D		ry$^+$	7
96	DR-2	ry$^+$, Ddc$^+$	4
97A	tAP-16, 4.8	Adh$^+$	2
97F		ry$^+$	7
98A	BS2.7-1	chorion$^+$, ry$^+$	5
98C	R602.1	ry$^+$	5
98C	R705.1	ry$^+$	5
99AB	DA24-44	Adh$^+$, Ddc$^+$	4
99D	SRS3.9-3	chorion$^+$, ry$^+$	5
99D,1other	SRS3.9-5	chorion$^+$, ry$^+$	5
100D		ry$^+$	7
100F	A4-4	w$^+$, ry$^+$	3
Chromosome IV			
chromocenter	R401.1	ry$^+$	5

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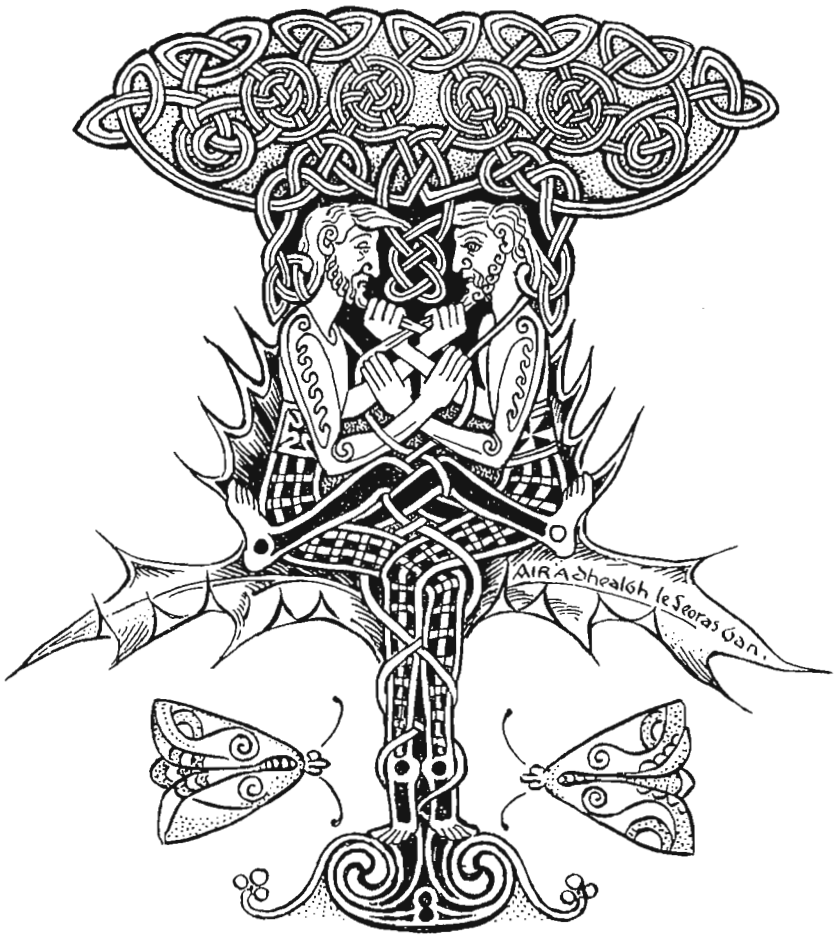

BIOCHEMICAL LOCI OF THE "FRUIT FLY" (*Drosophila melanogaster*)

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2N = 8

The genetic map of biochemical loci in *D. melanogaster* is an updated version of earlier revisions (see 547). Information from general references (92, 253, 268), and new material compiled by January 1, 1984 are included. An effort was made to screen all published articles, research notes, books and personal communications that have witheld the test of time. Some papers cited do not contain mapping data but were considered useful in the interpretation of mapping data. Abstracts were omitted if their data were published in subsequent journal articles. Certain loci have been so extensively studied that it is not feasible to give a full list of references for them; review articles are thus cited. Genetic symbols follow the rules (253) for naming mutant loci as closely as feasible and in accord with modifications that will appear in the revision of Genetic Variations of *Drosophila melanogaster* by Lindsley, Grell and Zimm (647). Genes located cytologically by segmental aneuploidy and/or by *in situ* hybridization studies, but not by recombination analysis, are listed with the chromosome, chromosome arm, or the inferred genetic locus in which each occurs shown parenthetically.

Loci for which there are physical mapping data from restriction enzyme and/or nucleotide sequencing studies are referenced by a superscript (P). Structural genes that code for the primary amino acid sequence of a given protein or polypeptide are indicated by a cross (+) where the data are convincing and by two crosses (++) where the data are suggestive but less critical. No information on mitochondrial genes or on dispersed repeated gene families such as copia, 412, 297 and other transposable elements is included. The tRNA genes, omitted in this revision, are listed in the accompanying map compiled by E. Kubli from tRNA and U-RNA *in situ* hybridization data; they have recently been reviewed in detail (624). Our revised map supercedes all earlier versions. It includes a table-figure of gene loci based on their chromosomal locations.

Gene Symbol	Genetic Map Locus	Cytological Location	Enzyme, Protein, or Nucleic Acid Affected	Reference
abo abnormal oocyte	2-44.0	31F-32E	rDNA redundancy	233 234 274 329 353 445 469P 647
+*Ace* (= *l(3)26*)	3-52.2	87E1,2	acetylcholinesterase (EC 3.1.1.7)	156 173 183 509P 529 579P 672 661 727P
+*Acph1* (= *Acph-1*)	3-101.1	99D-99E	acid phosphatase-1 (EC 3.1.3.2)	25 123 208 266 290-1
actin genes:				
+*Act5C* (= *act5C*)	(1-14)	5C3-4	actins II & III, cytoplasmic	130P 131P 396P 536P 562P 647 661 724P
+*Act42A* (= *act42A*)	(2-55.4)	42A	actins II & III (?), cytoplasmic	131P 396P 562P 661 647
+*Act57A* (= *act57A*)	(2-93)	57A	actin I, larval, pupal, adult muscle	130P 131P 396P 562P 661 647
+*Act79B* (= *act79B*)	(3-47.5)	79B	actin I (?), mostly in adult muscle	130P 131P 396P 455P 562P 647 661 679P 704P
+*Act87E* (= *act87E*)	(3-52.3)	87E	actin I (?), larval, pupal, adult muscle	130P 131P 396P 562P 661 647
+*Act88F* (= *act88F*)	(3-57)	88F	actin I, mostly adult muscle	130P 131P 396P 562P 647 661 679P 704P

Table-Figure 1. GENETIC MAP OF BIOCHEMICAL LOCI IN *Drosophila melanogaster*. Gene loci are listed according to chromosome or chromosome arm. The top entry in each column represents the leftmost locus and the bottom entry the rightmost locus. Cytological locations are consistent with the order given, where available.

X-CHROMOSOME		CHROMOSOME 2L		CHROMOSOME 2R	
0.0	*cin*	1.9	*Lsp1β*	55.2	*Dip-A*
0.0	*ewg*	3.0	*Got2*	(55.4)	*Act42A*
0.0	*su(b)*	5.0	*fs(2)B*	55.7	*bur*
0.0	*su(s)*	5.9	*Pgk*	55.8	*mle*
0.0	*l(1)npr1*	9.0	*msl2*	57.5	*cn*
0.63	*Pgd*	13.0	*dp*	58.6	*Pgi*
0.8	*pn*	13.9	*Sgs1*	62.0	*en*
1.0	*z*	(15)	*Cg25C*	62±	*Lcp1, Lcp2,*
1.5	*w*	20.5	*Gpdh, Gdt3*		*Lcp3, Lcp4*
3.0	*N*	20±	*βGal*	(62)	*HDL* family
3.6	*Sgs4*	?	*←Kf2*	?	*←Deb-A, Deb-B*
4.6	*dnc*	?	*←Gart*	?	*←Cmd*
(14)	*Act5C*	37±	*Mdh1*	?	*←Gapdh*
(18)	*Fum*	(37)	*Sucr*	72±	*l(2)me*
21±	*fs(1)1163, l(1)93p, vtw*	44.0	*abo*	73.5	*Hex-C*
23.1	*oc*	48.5	*b*	75±	*αGpo*
23.1	*s36, s38*	50.1	*Adh*	75.0	*Got1*
27.7	*su(r), lz*	51.3	*Bsh*	77.7	*Amy-p, Amy-d*
28	*gmp*	52±	*Ifm(2)1, Ifm(2)2, Ifm(2)3*	?	*←Hsp54E*
29.2	*Hex-A*	(52)	*Mhc36B*	80	*map*
29±	*Yp1, Yp2*	52.4	*tyr1*	80.6	*Phox*
31	*flp*	53.3	*msl1*	?	*←Eip40*
32.8	*ras*	53.9	*l(2)amd*	86±	*Aldox2*
?	*←pur1*	53.9	*Ddc*	89±	*sdh*
33.0	*v*	53.9	*l(2)37Bf*	?	*←βTub56C*
35.7	*RpII215*	(54)	*Tp1*	?	*←βTub56D*
?	*←Hsp10F*	54	*His1, His3, His4, His2a,*	(93)	*Act57A*
37.0	*Flu*		*His2b* (tandem repeats)	95±	5S RNA gene
38	*mfd*	54.5	*pr*	97±	*Pu*
39.5	*Lsp1α*	(55)	*Ifm(2)11*	100	*Adk-C*
42.0	*l(1)ts403*			107±	*Dat*
42.6	*Gpt*			107.0	*sp*
43.5	*int, up*				
44±	*Yp3, Yp3R*				
44.5	*fs(1)29*				
54.4	*Had*				
55.3	*r*				
55.5	*Su(b)*				
58	*gnd*				
59.5	*hdp*				
62.9	*Zw*				
64.8	*ma-l*				
(65)	*Cg19-20, sdby*				
65.9	*su(f)*				
66.0	*bb*				

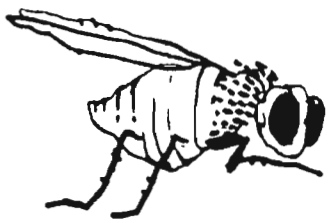


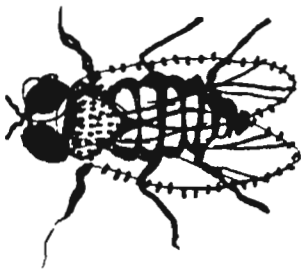
Table-Figure 1, continued

CHROMOSOME 3L

-1.41	*Lsp1γ*
3.03	*Aprt*
?	←*Hsp83*
?	←*Hsp63F*
19	*Ama1*
25.2	*Argt*
25.4	*Idh2*
26	*msl3*
26.5	*s15, s16, s18, s19*
?	←*Hsp22, Hsp23, Hsp26, Hsp28*
27.1	*Idh1*
?	←*αTub67C*
34.5	*lxd*
34.6	*Sod*
35.0	*rs*
35.5	*αFuc*
(36)	*Sgs3, Sgs7, Sgs8*
37±	*Lsp2*
36.8	*Est6*
?	←*Hsc1*
?	←*P1, P6*
?	←*Eip28, Eip29*
42.0	*Sgs6*
43.4	*Pgm*
(44)	*Dash*
?	←*Hsp73D*
?	←*Ars*
(44.3)	*Cat*
45.9	*DNase2*
47.3	*Aph1*
(47.5)	*Act79B*

CHROMOSOME 3R

47.8	*Antp*
(47.8)	*αTub84B*
48	*Gld*
(48)	*αTub84D*
48	*Est-C*
48	*dsx*
48±	*Dhod*
48.3	*Ali*
48.5	*B2t*
(49)	*Mtn, αTub85E*
49.2	*Odh*
(51)	*Dip-C*
(51)	*Hsp70*
51.7	*Men*
?	←*Hsc2*
52.0	*ry*
52.2	*Ace*
(52.3)	*Act87E, Mhc87E*
53.6	*Dip-B*
53.6	*red*
?	←*Hsp88B*
(55)	*Ifm(3)1, Ifm(3)2, Ifm(3)3, Ifm(3)4, Ifm(3)5, Ifm(3)6, Ifm(3)7*
55.4	*R3-55.4*
56.7	*m-Est*
(57)	*Act88F, Mhc88F*
57.2	*lpo*
57.2	*Aldox1*
58.8	*bx*
58.8	*Cbx*
58.8	*Ubx*
58.8	*bxd*
58.8	*pbx*
61.8	*DNase1*
62	*sr*
62.6	*Mdh2*
64.5	*Sodh*
64.6	*Cha*
66	*ninaE*
?	←*Kf1*
70±	*r-l*
?	←*Hsr93D*
?	←*Hsp68*
81.7	*Gdh*
91.5	*Ald*
95.4	*rsd*
98.3	*Lap-A, Lap-D*
100	*Ama2*
101.1	*Acph1*
101.3	*Tpi*



βTub97F?

Rbp49? *Mlc99E?* *βGlu?*

+*Adh*	2-50.1	35B2-3	alcohol dehydrogenase (EC 1.1.1.1), with cis control	31[P] 32[P] 142 149[P] 162 275 279 303 354 407 410 434-5 482 499[P] 502 512-4[P] 543 571[P] 584[P] 623[P] 625[P] 630[P] 658 661
----	2-not near *Adh*		ADH trans control loci	184 391
++*Adk-C* (= *Ak-C*)	2-100, approximate		adenylate kinase C (EC 2.7.4.3)	414 647
+*Ald*	3-91.5	97AB	aldolase (EC 4.1.2.13)	308 414 416
+*Aldox1* (= *Aldox*= *Ao* = *aldox*)	3-57.2	89A	aldehyde oxidase (EC 1.2.1.3), with cis control	73 77 84 90 91 116 282 368 414 431 493 544
Aldox2	2-86		aldehyde oxidase-2	29
+*Ali* (= *ali*= *ali-est*)	3-48.3		ali-esterase	306 307
Ama1	3-19		α-amanitin resistance 1	691
Ama2	3-100		α-amanitin resistance 2	691
amd: see *l(2)amd*				
+*Amy-p* +*Amy-d*	2-77.7	54A1-B1	α-amylase (EC 3.2.1.1), proximal & distal (gene duplicated)	12-14 93 94 97 228 253 548 568[P] 641
Antennapedia Complex (ANT-C):				
Antp Antennapedia	3-47.8	84B1-2	3.5 and 5.0 kb transcripts, tissue localizations	253 528[P] 542 551 563[P] 578[P] 583 607 634[P] 645-6 711 733
+*Aph1*	3-47.3		alkaline phosphatase 1 (EC 3.1.3.1)	22 421 647
Aph2	2-		alkaline phosphatase in adult hindgut	647 708
+*Aprt* (= *aprt*)	3-3.03	62B7-12	adenine phosphoribosyl transferase (EC 2.4.2.7)	210 253 601 647
+*Argt* (= *Ak*)	3-25.2	66B-D11	arginine kinase (EC 2.7.3.3)	128 560-1
++*Ars*	(3L)	74A-79D	arylsulfatase (EC 3.1.6.1)	267 647
b black	2-48.5	34E5-35D1	β-ureidopropionase (?), β-alanine deficient	16 253 471 647
+*B2t* (= *ms(3)KK*^{D})	3-48.5	85D7-11	β_2-tubulin, testis specific	220 221 609 647 661
bb bobbed	1-66.0, Y-proximal to *ks1* and *ks2*	right of 20F	rRNA: 2S 5.8S 18S 28S	61 87[P] 140 146 147[P] 148[P] 195[P] 212[P] 213[P] 224[P] 225[P] 230[P] 259-61[P] 273[P] 319[P] 331[P] 340 341 346-8[P] 429[P] 443[P] 452[P] 534[P] 545[P] 610 616-7[P] 627[P] 663[P] 706 715[P] 718[P]
bur burgundy (= *qua2*?)	2-55.7		inosinate dehydrogenase activity in *burqua2-1*	253 676

Bithorax Complex (BX-C): units in complex listed left to right				
bx bithorax			cis control unit	
Cbx Contrabithorax			cis control due to transposition of *pbx*+ DNA into middle of *Ubx* unit	
+*Ubx* Ultrabithorax	3-58.8	89E1,2 (in or close to)	structural unit producing 4.3, 3.2, 1.6 & 1.4 kb poly(A)+ RNA transcripts and 4.7 kb poly(A)- RNA transcript	253 497 510P 632 644 647 661 687P
+*bxd* bithoraxoid			structural unit producing 1.28 & 1.15 kb transcripts	
pbx postbithorax			cis control unit	
++*Cat*	(3-44.3)	75D-76A	catalase (EC 1.11.1.6)	265 647
+*Cha* (= *Cat*)	3-64.6	91B-D	choline acetyltransferase (EC 2.3.1.6)	154 172 174 647
++*Cg19-29*	(1-65)	19E-20B	collagen	647 661 669P 678P
++*Cg25C*	(2-15)	25C	collagen	
chorion protein: see shell protein (*s*) genes				
cin cinnamon	1-0 (.0017cM left of *y*)		modifier of Mo hydroxylases (AO, PO, XDH) & sulfite oxidase	17 30 49 116 314 423 459 493
+*Cmd*	(2R)	49	calmodulin	749P
++*cn* cinnabar	2-57.5	43E3-14	kynurenine hydroxylase (EC 1.99.1.5)	141 253 318 385 453 557
collagen-like proteins: see *Cg*				
cuticle proteins: see *Lcp*				
cr compensatory response	(1)	20C1-2	rDNA redundancy	329 556
+*Dat*	2-107	60B1-10	dopamine acetyl transferase (EC 2.3.1.5)	194 277
+*Ddc*	2-53.9 (.025cM right of *hk*)	37C1,2	dopa decarboxylase (EC 4.1.1.26)	186 276 437-41 589P 661 709P 747
+*Dhod*	3-48	85A-C	dihydroorotate dehydrogenase (EC 1.3.3.1), mitochondrial	333 337
+*Dip-A*	2-55.2	41A	dipeptidase-A	309 412 414 631
+*Dip-B*	3-53.6	87F12-88C3	dipeptidase-B	309 631
+*Dip-C*	(3-51)	87B5-10	dipeptidase-C	309 631
+*DNase1*	3-61.8	90C2-E	deoxyribonuclease-1 (EC 3.1.4.5)	89 160 730
++*DNase2*	3-45.9		deoxyribonuclease-2	160
+*dnc* dunce	1-4.6	3D4	cAMP-phosphodiesterase, form II (EC 3.1.4.17) with cis control, .09 cM right of *dnc*	50 85 101 226 227 608 613 702-3 717 725
dp dumpy	2-13.0	25A1-2	orotate phosphoribosyl transferase (EC 2.4.2.10)	43 151 253 409

Drosophila oncogene sequences:				
Dash (=*S16*)(*v-abl* & *v-src* homologous)	(3-44)	73B	Protein kinases, cAMP-dependent (?)	661 719^{p} 790-1^{p}
Dsrc (=*S24*)(*v-src* homologous)	(2L)&(3L)	29A,C & 64B		
dsx double-sex	3-48	84E1-2	YP regulation; 3.7 & 1.65 kb larval transcripts; adult transcript	253 521 645 647 661 746^{p}
ecdysteroid-inducible genes for polypeptide (EIPs), proteins & RNAs:				
(see *Ace*, *Act*, *Ddc*, *l(2)37Bf*, *Lcp*, *Lsp*, *Sgs*, *Yp*)				
Eip28	(3L)	71C3-D2	EIP 28 (I, II & III),	58 461 530^{p} 661
Eip29			EIP 29 (I & II)	
Eip40	(2R)	55B-D	EIP 40 (I & II)	
----	(3L)	63F2-4	ecdysone-inducible mRNA	597^{p}
----	(3L)	74EF	ecdysone-inducible RNA	523^{p} 661
en engrailed	2-62.0	48A1-4	3.5 kb & 7.5 kb RNA transcripts	253 553 564 619-20 626^{p} 633 671
+*Est-C*	3-48	84D3,4-11,12	esterase-C	23 308 310 401 524
+*Est6* esterase-6	3-36.8	69A1-5	carboxylesterase (EC 3.1.1.1)	2 23 70 123 344 345 436 655 694
Est9	2-		esterase-9	264
fs(1)29 (= *fs29*)	1-44.5	12E1-F1	sequestration of yolk proteins	742^{p}
fs(1)1163 female sterile (1) 1163	1-21		yolk protein-1 control, cis-acting	45 46 132 481
fs(2)B female sterile(2)Bridges	2-5.0		thymidylate synthetase (EC 2.1.1.6)	53
+α*Fuc*	3-35.5		α-fucosidase	653
+*Fum* (= *Fuh*)	1-(left of *cm*)	5F1-6D2	fumarase (fumarate hydratase)	271 323 635
+β*Gal*	2-20+	26A8	β-galactosidase (EC 3.2.1.23)	621
++*Gapdh*	(2R)	50D-51A2	glyceraldehyde-3-phosphate dehydrogenase	128 560 561
+*Gart* (=*ade2* or *ade3*? =*GART* = *ade8*)	(2L)	27C	glycinamide ribotide transformylase	180^{p} 585-6^{p} 647 661 676
+*Gdh*	3-81.7		glutamate dehydrogenase, NAD-dependent (EC 1.4.1.2-3)	526
Gdt3	2-20.5		temporal & tissue-specific cis-control for *Gpdh*	34 516 714
+*Gld* (= *Hex-1*= *Go*)	3-48	84CD	FADglucose dehydrogenase (EC 1.1.99.10)	54 55 524
β*Glu*	3-distal end	98F-100F	β-glucuronidase I & II activity	241
Go : see				
++*Got1*	2-75.0		glutamate oxaloacetate transaminase-1	161 525
+*Got2*	2-3.0 (or 4.8?)	22B1-4	glutamate oxaloacetate transaminase-2	159 161 460
glucose-6-phosphate dehydrogenase: see *Zw*				
+*Gpdh* (= α*Gpdh*)	2-20.5	25F5	*sn*-glycerol-3-phosphate dehydrogenase (EC 1.1.1.8), with cis systemic regulator	36-38 71 157 298-300 460 516 621-2 714 743

++*αGpo*	2-75.5	52D	α-glycerophosphate oxidase (EC 1.1.99.5), mitochondrial	298 300 653
+*Gpt*	1-42.6	11F1-12A2	glutamate pyruvate transaminase (EC 2.6.1.2)	247 414
+*Had*	1-54.4		hydroxy acid dehydrogenase (EC 1.1.1.45)	44 397
HDL gene family:				
++*H44D* ++*D44D* ++*L44D*	(2R)	44D	gene-specific mRNAs in 1st, 2nd, early 3rd larval instars & adult (Ca^{++}-activated secretory proteins?)	723p
histone genes:				
+*His1*	2-54	39D2,3-F1,2	histone 1	40 59 218 252
+*His2a*			histone 2A	289 316 351^{p}
+*His2b*			histone 2B	458 462 488 593^{p}
+*His3*			histone 3	661 670 732^{p}
+*His4*			histone 4	
+*Hex-A* (= *Hex-A,B*)	1-29.2	8D4-E	hexokinase-A	293 414
+*Hex-C* (= *Hex-3* = *Fk*)	2-73.5		hexokinase-C (fructokinase)	207 272 293 294 501
heat shock (HS) genes and related elements:				
heat shock cognates (hsc) - not HS induced, homology with *Hsp70*:				
Hsc1	(3L)	70C	500 b RNA transcript	474^{p} 538^{p} 661
Hsc2	(3R)	87D	375 b RNA transcript	
+*Hsc3*	(3R)	88E	400 b RNA transcript; 70K hsc4 protein	
heat shock protein (hsp) genes:				
+*Hsp22*	(3L)	67B	hsp22 protein	75^{p} 80^{p} 188^{p}
+*Hsp23*			hsp23 protein	197^{p} 219^{p} 255^{p}
+*Hsp26*			hsp26 protein	322 376 418^{p}
+*Hsp28* (= *hsp27*)			hsp28 protein	419^{p} 596^{p} 661 720^{p} 726^{p}
+*Hsp68*	(3R)	95D	hsp68 protein	10 187-8^{p} 355 376 661
+*Hsp70*	(3-51)	87A7(2 genes) 87C1(3 genes)	hsp70 protein, with cis control	9^{p} 10 51 81^{p} 133 150^{p} 171^{p} 179 187^{p} 188^{p} 196^{p} 198^{p} 199 200 201-2^{p} 216^{p} 251 254-6^{p} 258^{p} 281 283^{p} 322 357 375-6 395 399^{p} 442^{p} 464^{p} 468 518^{p} 636^{p} 648^{p} 652^{p} 659^{p} 661 664^{p} 679^{p}
+*Hsp83* (= *hsp82*)	(3L)	63BC1	hsp83 protein	10 187^{p} 188^{p} 255^{p} 302^{p} 376 442^{p} 577^{p} 661
other HS genes and related elements:				
Hsα *Hsβ* *Hsγ*	(2R) (3R)	42B, 87C1, & chromocenter	HS RNAs: αβ RNA αγ RNA	171^{p} 254^{p} 255^{p} 256^{p} 257^{p} 659^{p} 661
Hsr93D	(3R)	93D4-9	HS RNA mostly nuclear & not likely translated, with cis control	239 248 628 668

+*Hsp10F*	(1)	10F1	minor hsp's	255[p]
+*Hsp54E*	(2R)	54E1		
+*Hsp63F*	(3L)	63F1		
+*Hsp73D*	(3L)	73D1		
+*Hsp88B*	(3R)	88B1		
+*Idh1* (= *Idh-NADP*)	3-27.1 (bet. *h* & *th*)	66B-67C (66D1-67C)	NADP-isocitrate dehydrogenase-1 (EC 1.1.1.42), with *cis* control (?)	122 335 381 511
+*Idh2* (= *Idh*)	3-25.4 (bet. *jv* & *se*)		NADP-isocitrate dehydrogenase-2 (EC 1.1.1.42)	308 310
indirect flight muscle (IFM) myofibrillar protein genes: (see actin, muscle protein, TM genes)				172 667
Aberrant IFM Morphology Loci			**IFM Protein Affected**	
Bsh Bashed (= *Ifm(2)*- locus?)	2-51.3		several IFM proteins	
ewg erect wing	1-0.0	1A	80K & 90K proteins	
Flu Flutter	1-37.0	10F7-11D1	80K & 90K proteins	
flp flapwing	1-31	9B1-10A1	actin III (80K?), paramyosin (90K?), myosin light chains, 55K (54K?) proteins	172 253 479 540
gmp gumper	1-28	8E-9D	80K & 90K proteins	
gnd grounded	1-58		80K & 90K proteins	
hdp heldup	1-59.5		similar to *flp*	
Ifm(2)1	2-near 52		gene-specific array of IFM proteins, actin I & III, tropomyosin	172 479 667
Ifm(2)2				
Ifm(2)3				
Ifm(2)11	2-near 55			
Ifm(3)1	3-near 55			
Ifm(3)2				
Ifm(3)3				
Ifm(3)4				
Ifm(3)5				
Ifm(3)6				
Ifm(3)7				
int indented thorax (= *up*?)	1-43.5	12A1-7	similar to *flp*	
l(1)93p	1-near 21	7D1-6	80K & 90K proteins	172 540
mfd myofibrillar-defective	1-38	11A6-7	4 proteins absent (abn. phosphorylation?)	
rsd raised	3-95.4		similar to *flp*	172 240 253 479 540
sdby standby	1-right of *car*	19F-20F	80K & 90K proteins	172 540
sr stripe	3-62.0		80K & 90K proteins	
up upheld (= *wupB* ?)	1-43.5	12A1-7	similar to *flp*	172 287 465 472 540
vtw vertical wings	1-near 21	7D1-6	80K & 90K proteins	172 540

++-----	(3-50)	87B	22.5 IFM protein	33 483
++*Kf1*	(3R)	91B-93F	kynurenine formamidase-I	288
++*Kf2*	(2L)	25A-27E	kynurenine formamidase-II (EC 3.5.1.9)	288
l(1)ts403 lethal(1)temperature sensitive-403	1-42.0		control of heat shock proteins	110
l(2)37Bf lethal(2)37Bf right of *hk*	2-53.9,	37D	diphenoloxidase	690
l(2)amd lethal(2)α-methyl dopa	2-53.9+	37C1,2 (just left of *Ddc*)	α-methyl dopa hypersensitive (*Ddc* system?)	276 367 437-40 747
l(2)me lethal(2)meander	2-72		protease activity, isoacceptor $tRNA_4^{Glu}$ reduced	56 99 253 420 456 624
l(1)npr1 no puff regulator	1-0.0	2B5	trans control for *Sgs3*, *Sgs7*, *Sgs8*	662
+*Lap-A*	3-98.3		leucine aminopeptidase-A	23 113
+*Lap-D*	3-98.3		leucine aminopeptidase-D	23 113 350
larval cuticle protein (LCP) genes:				
+*Lcp1*	2-62	44D	L_3CP 1 (3rd instar)	127 366^p 463
+*Lcp2*			L_3CP 2 (3rd instar)	
+*Lcp3*			L_3CP 3 (3rd instar)	
+*Lcp4*			L_3CP 4 (3rd instar)	554-5^p 558 661
+*Lcp5*	3-		L_3CP 5 (3rd instar)	721-3^p
+*Lcp6*	3-		L_3CP 6 (3rd instar)	
+*Lcp8*	3-		L_3CP 8 (3rd instar)	
++*lpo*	3-57.2, .009cM left of *Aldox1*	89A	pyridoxal oxidase (low)	72 116 423 544
larval serum proteins (LSP) genes:				
+*Lsp1α*	1-39.5	11A7-B9	LSP-1α	47 342 365^p 477^p 522^p 629^p
+*Lsp1β*	2-1.9	21D2-22A1	LSP-1β	47 249^p 342 365^p 477^p 522^p 629^p 675 750^p
+*Lsp1γ*	3-(-1.41)	61A1-6	LSP-1γ	
+*Lsp2*	3-37(?)	68E3-4	LSP-2	2 3 249^p
lxd low xanthine dehydrogenase	3-34.5		modifier of Mo hydroxylases (AO, PO & XDX) & sulfite oxidase	73 103 143 253 423 459 493 710
lz lozenge	1-27.7	8D4-E1	monophenoloxidase	20 285 320 321
			diphenoloxidase	422 661
ma-l maroon-like (= *mal*)	1-64.8	19D1-3	controls incorporation of cyanolyzable sulfur into Mo hydroxylases (AO, PO, XDH)	63 77 100 115 117-21 126 143-4 423 492-3 459 684 739-40
map midgut activity pattern	2-80		trans control for *Amy-p* & *Amy-d*	1 95 546 548
+*Mdh1* (= *cMdh*)	2-37	31B-E	NAD-malate dehydrogenase (cytoplasmic) (EC 1.1.1.37)	4 158 297 298 414 415
+*Mdh2* (= *mMdh*)	3-62.6	90C-91A3	NAD-malate dehydrogenase (mitochondrial) (EC 1.1.1.37)	414 415

+*Men* (= *Mdh-NADP*)	3-51.7	87D1-2	NADP-malate dehydrogenase (malic enzyme: EC 1.1.1.40), with cis control)	125 135 308 414 417 511 744
m-Est (= *m-est*)	3-56.7		esterase-6 and leucine aminopeptidase modifier	68 69 503 649-50
male-specific lethal loci:				
mle maleless	2-55.8	41A-43A	control of X-linked enzymes (G6PD, 6PGD, FUM, -HAD)	26 129 386
msl1 male-specific lethal-1 (= *msl-1*)	2-53.3	36F7-37B8	control of X-linked enzymes (G6PD, 6PGD, FUM)	26 27
msl2 male-specific lethal-2 (= *msl-2*)	2-9.0	23E1-F6	control of X-linked enzymes (G6PD, 6PGD, FUM)	26 27 508
msl3 male-specific lethal-3 (= *msl-3*)	3-26		(control of X-linked enzymes?)	649 651
+*Mtn*	(3-49)	85E	metallothionein	657[P]
muscle protein genes: (see actin, IFM, TM genes)				
+*Mhc36B*	(2-52)	36B	myosin heavy chain	515[P] 661 697[P]
++*Mhc87E*	(3-52.3)	87E	myosin heavy chain (?)	33 483
++*Mhc88F*	(3-57)	88F	myosin heavy chain (?)	33 483
+*Mlc99E*	(3-101)	99E	myosin light chain	661
+----	(2L)	32CD	gene-specific, unidentified muscle proteins translated from abundant mRNAs	731[P]
+----	(2R)	46D-F & chromocenter		
+----	(2R)	47F-48D		
+----	(3L)	62CD		
+----	(3L)	63A-C		
+----	(3L)	69F		
+----	(3R)	88F		
++*N* Notch (complex)	1-3.0	3C7	choline DH(EC 1.1.99.1) dihydro-orotic acid DH (EC 1.3.99.9) α-glycerophosphate DH (EC 1.1.99.5) NADH DH & NADH oxidase (EC 1.6.99.3) succinate DH (EC 1.3.99.1) xanthine DH(O_2) (EC 1.2.99.1)	222 223 253 392 393 394 425 426 500[P] 612[P] 661 736
++*ninaE*	3-66	92AB	opsin (rhodopsin)	705
neither inactivation nor after potential-E				
oc ocelliless (associated with *In(2)7F1,2;8A1,2*)	1-23.1	7F1,2 to 8A1,2	stable position effect on shell protein-36 & shell protein-38	246 253 372[P] 373[P] 377
+*Odh*	3-49.2	86D1-4	octanol dehydrogenase (EC 1.1.1.73)	76 78 310 531
proteins-miscellaneous genes:				
+*P1*	(3L)	70CD	110K larval fat body protein P1	249[P] 675 720[P]
+*P6*	(3L)	70CD	29K larval fat body protein P6	249[P]
+ ----	(3L)	80C	26K embryonic, cytoplasmic protein	39[P]
+*Pgd* (= *6Pgd*)	1-0.63	2D3-4	6-phosphogluconate dehydrogenase (EC 1.1.1.44)	35 138 166-8 170 450 533

+Pgi	2-58.6		phosphoglucose isomerase	414 417
+Pgk	2-5.9	22D-23E3	3-phosphoglycerate kinase (EC 2.7.2.3)	60 416
+Pgm	3-43.4	72D1-5	phosphoglucomutase (EC 2.7.5.1)	185 402 403 416
+Phox (= *Bc?*)	2-80.6		phenol oxidase	19 575 695
pn prune	1-0.8	2D5-6	GTP-cyclohydrolase control	109 253 654
++pr purple	2-54.5	37B2-40B2	ramiopterin synthase sepiapterin synthase	98 235 253 398 449 549
++Pu Punch	2-97	57C1-8	GTP cyclohydrolase (EC 3.5.4.16)	253 654
pur1	1-left of *v*	9E1-3	purine 1	211 480
+r rudimentary	1-55.3	15A1	carbamyl phosphate synthetase(EC 2.7.2.9) aspartate transcarbamylase (EC 2.1.3.2) dihydroorotase (EC 3.5.2.3)	48 52 111-2 114 205-6 245 253 296 334 336 404 600 661 683 713^{p}
R3-55.4 (= $R^{3-55.4} = r^{3-55.4}$)	3-55.4		*sn*-GPDH trans control (α-GPDH modifier)	614 615
ras raspberry	1-32.8	9E1-3	GTP-cyclohydrolase control	109 253 454 480 654
red red Malpighian tubules	3-53.6	88A-C	GTP-cyclohydrolase control	253 654
+r-l rudimentary-like (= *ral*)	3-70	93B4-13	orotate phosphoribosyltransferase (EC 2.4.2.10) orotidylate decarboxylase (EC 4.1.1.23)	74 242 332 333
+Rbp49	(3R)	99D	ribosomal protein 49	408 494^{p} 661
+Rp^{II215} (= *AmaC4* = *l(1)L5* = *PolIII* = *Ubl*)	1-35.7	10C1-2	RNA polymerase II, 215K subunit (EC 2.7.7.6)	152 153 292 537 573 595^{p} 661 712^{p}

RNAs - unidentified gene products, developmentally expressed:

			Stage Expressed	
Embryonic poly(A)+ RNAs:				
	(2L)	25D	blastoderm-differential	637^{p} 735^{p}
(02 gene)	(2L)	31BC	maternal-differential	637^{p} 735^{p}
Deb-A	(2R)	48EF	developmental-embryonic	753^{p}
Deb-B	(2R)	48EF	developmental-embryonic	753^{p}
	(3L)	71A	gastrula-differential	
	(3R)	94F-95A	blastoderm-differential	637^{p}
	(3R)	99D	blastoderm-differential	637^{p}
	(3R)	99E1-3	blastoderm-differential	637^{p}
Larval Instar poly(A)+ RNAs:				
	(1)	4F-5A	late instar IV	746^{p}
	(3L)	67B	late instar III + pupa	720^{p}
	(3L)	71DE	late instar I	746^{p}
	(3L)	71DE	late instar II + III	746^{p}
Intermolt poly(A)+ RNAs:				
	(1)	3C7-D1	intermolt I	746^{p}
	(3L)	68C	intermolt II	746^{p}
	(3L)	68C	intermolt III	746^{p}
	(3L)	68C	intermolt IV	746^{p}
	(3R)	90BC	intermolt V	746^{p}

Adult poly(A)+ RNAs:				
	(1)	15AB	head-specific RNA for each locus (some with multiple genes per site)	642[P] 643[P] 661
	(2L)	28A, 28C, 32AB, 34F		
	(2R)	43AB,44CD, 46E,47E,48F 51B,57C		
	(3L)	66D,72BC,73D-F		
	(3R)	82F,92CD,99C,100B		
rRNA genes:				
(see *bb*)				
---- 5S RNA gene	2-95	56F	5S-rRNA	8[P] 28 214[P] 263[P] 324[P] 328 330 349 388-9 405[P] 432-3 458 495 587[P] 737-8[P]
rs rose	3-35.0		GTP-cyclohydrolase control	253 654
+*ry* rosy	3-52.0	87D12	xanthine dehydrogenase, with cis control (EC 1.2.1.37)	63-5 136-7 144 183 278 304 448 509[P] 579[P] 698[P] 727-8[P]
shell protein genes (chorion protein genes):				603
+*s15* (= *c15* = *A1*)	3-26.5	66D11-15	shell protein 15 (15K chorion protein)	163 369[P] 378 444 496 576[P] 647
+*s16* (= *c16* = *A2*)			shell protein 16 (16K chorion protein)	
+*s18* (= *c18* = *B1*)			shell protein 18 (18K chorion protein)	
+*s19* (= *c19* = *B2*)			shell protein 19 (19K chorion protein)	
+*s36* (= *c36* = *C1*)	1-23.1	7E11 to 7F1,2	shell protein 36 (36K chorion protein)	369[P] 371 372[P]
+*s38* (= *c38* = *C2*)			shell protein 38 (38K chorion protein)	377 378 444 647
+ ----	1-near *y*		70K shell protein	748
+ ----	1-between *y* and *cv*		100K shell protein	
(K254[TS])	1-17	5D5-6E1	defective shell (chorion) protein mutants	618
(K1212)	1-18.7	5D5-6C12		
(384)	1-20.5	7B8-C3		
(473)	1-20.5	7D10-8A5		
(K79)	1-bet. *ct-v*	8E-9B1		
(K451)	1-near *g*	12A6-D3		
(K1563[TS])	1-44.7	12D3-E1(?)		
(K499)	1-bet. *g-f*			
sdh	2-89		succinate dehydrogenase (EC 1.3.99.1), mitochondrial	243
salivary gland structural (SGS) protein genes for glue polypeptides:				
+*Sgs1*	2-13.9	25A3-D2	SGS-1	409
+*Sgs3* +*Sgs7* +*Sgs8*	(3-36)	68C3-5	SGS-3, SGS-7, SGS-8	2 231 478[P] 539[P] 565[P] 566 693[P]
+*Sgs4*	1-3.6	3C11-12	SGS-4	21 231-2 280[P] 295[P] 475 566 660[P] 661 674[P] 716[P]
+*Sgs6*	3-42.0	71C1-F5	SGS-6	491
snRNP genes: see U genes				

+*Sod*	3-34.6		superoxide dismutase	124 207 301 313
(= *To*)	(or 32.5?)		(tetrazolium oxidase)	460
+*Sodh*	3-64.5	91B-93F	NAD-sorbitol dehyroge-	41 42
(= *SoDH*)			nase, cytoplasmic	
sp	2-107.0	60B13-C5	phenol oxidase	253
speck				
su(b)	1-0.0	1B4-C4	β-alanine level	253 487
suppressor of black				
Su(b)	1-55.5	(15A1)	control of *r* gene	688
			products	
su(f)	1-65.9	20D or 20EF	affects processing or	253 580-1 706
suppressor of forked			translatability of	
			SGS-1, SGS-3, SGS-4	
su(r)	1-27.7		dihydrouracil dehydro-	15 111 383 384
suppressor of rudimentary			genase	
	1-0.0	1B11-13	suppresses *pr*, *s*, *sp* & *v*	178 203 204 269
suppressor of sable			($tRNA^{Tyr}$?)	406 427 517 624
				661
++*Sucr*	(2-37)	31CD-EF	sucrase	311
Tp1	(2-54)	39CD	small acidic temporal	559^{p}
(= *T1*)			protein (TI)	
+*Tpi*	3-101.3	99B-E	triosephosphate	414 416 505-6^{p} 661
			isomerase	731^{p}
+*Tm-c*			tropomyosin, cytoplasmic	
+*Tm1*	(3-57)	88F2-5	tropomyosin I, muscle	-506-7^{p} 661 731^{p}
+*Tm2*			tropomyosin II, muscle	
transfer RNA genes: see 547, 624 and E. Kubli in this volume				
++*Treh*	(2R)	55B-E	trehalase	312
(Troponin gene)	(2R)	49F	troponin C	
tubulin genes:				
(see *B2t*)				
+α*Tub67C*	(3L)	67C4-6	α-tubulin subunit	215^{p} 352
+α*Tub84B*	(3-47.8)	84B3-6	α-tubulin subunits	
			(2 genes)	
+α*Tub84D*	(3-48)	84D4-8	α-tubulin subunit	-215^{p} 315 352
+α*Tub85E*	(3-49)	85E6-10	α-tubulin subunit	
+β*Tub56C*	(2R)	56C	β-tubulin subunit	685
+β*Tub56D*	(2R)	56D4-12	β-tubulin subunit	677
+β*Tub97F*	(3R)	97F	β-tubulin subunit	677 685
tyr1	2-52.4		monophenoloxidase	250 285 361 422
tyrosinase-1	(= *tyr-1*)		(tyrosinase)	
			diphenoloxidase	
			(dopa oxidase)	
U-genes:				
U1	(1)	11B?		
	(2L)	21E	U_1 = $snRNA_2$	673^{p} 700^{p} 701
	(2R)	61A		
	(3R)	82E, 95C		
U2	(2L)	34AB, 38AB	U_2	498^{p} 707^{p}
U4	(2L)	40AB	U_4 = $snRNA_1$	
	(1)	14B		
U5	(2L)	23D, 34AB	U_5 = $snRNA_3$	-700^{p} 701
		35EF, 39B		
	(2R)	63A		
U6	(3R)	96A	U_6 = $snRNA_4$	
Ubl : see *RpII*				

++*v* vermilion	1-33.0	10A1	tryptophan oxygenase (Ec 1.13.1.12)	11 203 217 244 387 454
w white	1-1.5	3C1-2	2.7 kb RNA transcript; proximal cis control	519-20[p] 532[p] 570-1[p] 574[p] 602 606[p] 638-9[p] 608-1[p] 684 686[p] 692[p] 699[p] 751[p]
Xh^{abo}	1-(heterochromatin)	20C1-2	rDNA redundancy	317 353
yolk proteins (YP) genes: (see *fs(1)29*, *fs(1)1163*)				
+*Yp1*	1-29	8F-9A	YP-1	18[p] 45 193[p] 326-7 339[p] 424 473[p] 504 521 592[p]
+*Yp2*	1-29	8F-9A	YP-2	
+*Yp3*	1-44	12BC	YP-3	
Yp3R	1-near *Yp3*		YP-3 control, cis-acting	481
z zeste	1-1.0	3A3-4	represses expression of *w*, *bx*, *dpp*	253 567 598 602
+*Zw* Zwischenferment	1-62.9	18D	glucose-6-phosphate dehydrogenase (EC 1.1.1.49)	134 169 379-81 450 451 533 656[p] 661

+structural gene
++possible structural gene
[p]references with physical mapping data, i.e. molecular studies

Authors' Note

Three alphabetical lists of references are given with a line separating each list, reflecting map revisions with no attempt at integration. Some references have been deleted in the process of revision and some replaced by newer ones. We apologize for this time-saving approach and for any errors or omissions it may have engendered.

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Replicon properties of Drosophila genome. I. DNA fibre autoradiography of salivary gland cells held at the mid-part of S-phase.

^{3}H-TdR autoradiograms prepared from Drosophila polytene tissue reveal a heterogeneity in the cell population with respect to their replication cycle (Plaut et al. 1966; Mulder et al. 1968; Rodman 1968; Lakhotia & Mukherjee 1970; Kalisch & Haegle 1973; Chatterjee & Mukherjee

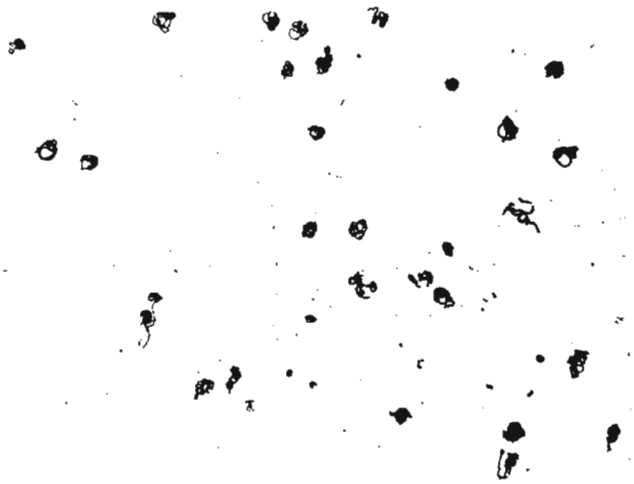

Fig. 1a. ^{3}H-TdR autoradiogram showing the accumulation of nuclei at the mid-part of S-phase.

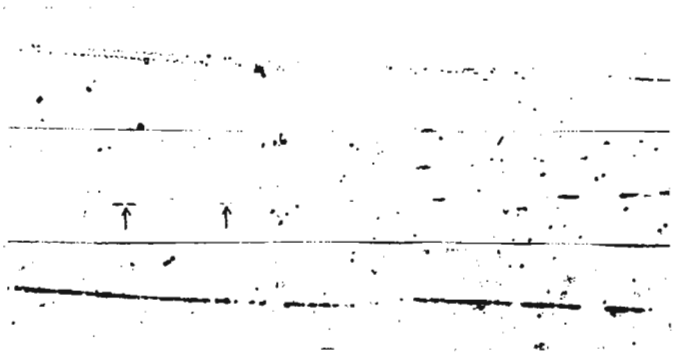

Fig. 1b. Different sizes/types of labelled segments obtained from the DNA fibre autoradiograms of polytene cells synchronized at the mid-part of S-phase. Arrows show the origins of post-pulse.

1975; Mukherjee et al. 1980). Studies on some molecular aspects of replication viz., initiation of replicons, their size, rate of fork movement, termination, etc., in a cell population demanded a synchronous (at least partial) population of cells, to avoid misinterpretations and laborious toil.

We, therefore, employed FdUrd block, cold thymidine release technique (Achary et al. 1981) and obtained more than 60% nuclei synchronized at the mid-part of the S-phase (Fig. 1a). DNA fibre autoradiography was done to study the said properties of Drosophila larval salivary gland polytene nuclear DNA synchronized at the mid-part of the S-phase.

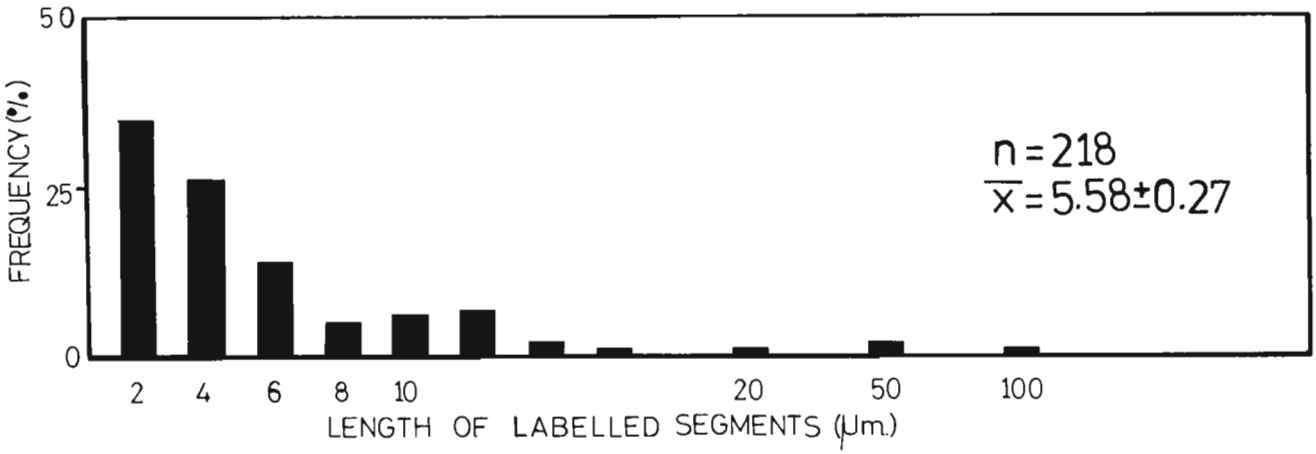

Fig. 2. Histogram showing the frequency distribution of different size labelled segments of DNA held at the mid-part of S-phase.

Basically the methodology of DNA fibre autoradiography involves lytic buffer (modified after Laughlin & Taylor 1979). The lysate is gently drawn over the slide, air dried and the processed for autoradiography.

Fig. 1b shows different types of labelled segments observed in these DNA fibre autoradiographic studies on the polytene nuclei synchronized at the mid-part of S-phase revealed a predominance of short labelled segments ranging from 2-6 µm with a mean of 5.58±0.27 µm (histogram). The finding suggests that the majority of the replicons are in the process of initiation rather termination in which case (latter) longer labelled segments would have been observed in good number. Works in our laboratory are in progress to substantiate our proposal.

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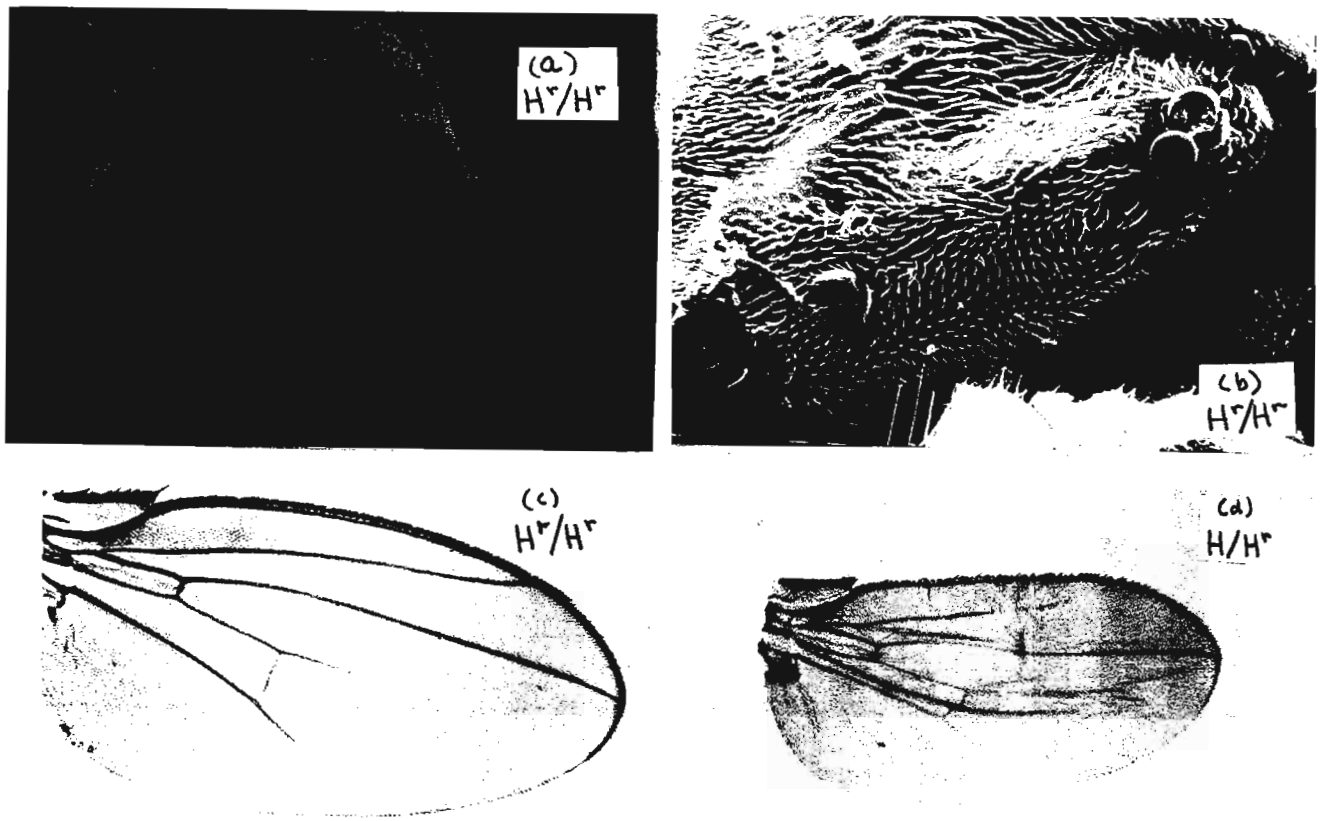
A new allele (H^r) at the Hairless locus of Drosophila melanogaster.

A mutant which produces suppression of a large number of macro and microchaetae has arisen spontaneously in a line selected for low dorsocentral and scutellar bristle number. This mutant is recessive and was found to map near ebony locus. An allelism test indicated that it is a new allele of the Hairless series (location III - 69.5). This allele was named Hairless-recessive and the H^r symbol is proposed to it.

Flies homozygous for H^r have almost all the bristles and hairs substituted with double or triple abnormal sockets (a and b). In wing, L IV and L V veins do not reach the margin (c).



Heterozygotes H^r/H die, probably at the pupa stage. Nevertheless there are some scapers which die a short time after eclosion; these individuals have an extreme Hairless phenotype: they have all the bristles and hairs suppressed or substituted with abnormal sockets, furthermore their wings are reduced and with abnormal L II, L IV and L V veins (d).

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Comparative genetics of neutron- and γ-ray-induced lethal b, cn and vg mutations in D.melanogaster.

It is a well-known fact that neutrons are more efficient than low-LET radiations for producing lethal visibles including those unaccompanied by detectable cytological changes. This fact was interpreted to mean (Muller 1954) that neutrons more frequently than low-LET radiations induce clusters of closely linked lethal and visible mutations which are then recorded as single genetic events. When this interpretation is correct, it can be expected that, in chromosome regions saturated by clusters of closely linked lethal and visible loci, neutrons must more often than low-LET radiations produce lethal visibles that complement to give viable visible combinations. However, if lethal visibles are a kind of the minute rearrangements with pleiotropic expression, such neutron-induced mutants will have lower frequencies of complementation for the lethal phenotype compared to lethal visibles induced by low-LET radiation. To test the alternatives the complementation patterns of 12 black, 13 cinnabar, and 11 vestigal lethal mutations induced by neutrons (0.1-0.85 MeV) or γ-rays (^{60}Co) and preserved by In(2LR)SM5 were first of all investigated through inter-se crosses between each of lethal mutations within the three regions of interest. Further, the extent of deficiencies supposed were determined by testing the survival of b lethal mutations in combinations with nub and j, of cn lethal mutations--with so and blo, and of vg lethal mutations--with sca, vg^C, vg^B, 1(2)C.

Results of the 328 inter-se as well as with reference markers crosses (in toto 16 γ-ray- and 19 neutron-induced lethal visibles were analyzed) are summarized by Figs. 1-3 (irradiation-induced mutations were named by the accepted alphanumeric code). As it can be seen, 80% neutron- as well as γ-ray -induced lethal visibles fail to complement, being deletions that extend for two, three, or more genic units neighbouring the specific loci of interest.

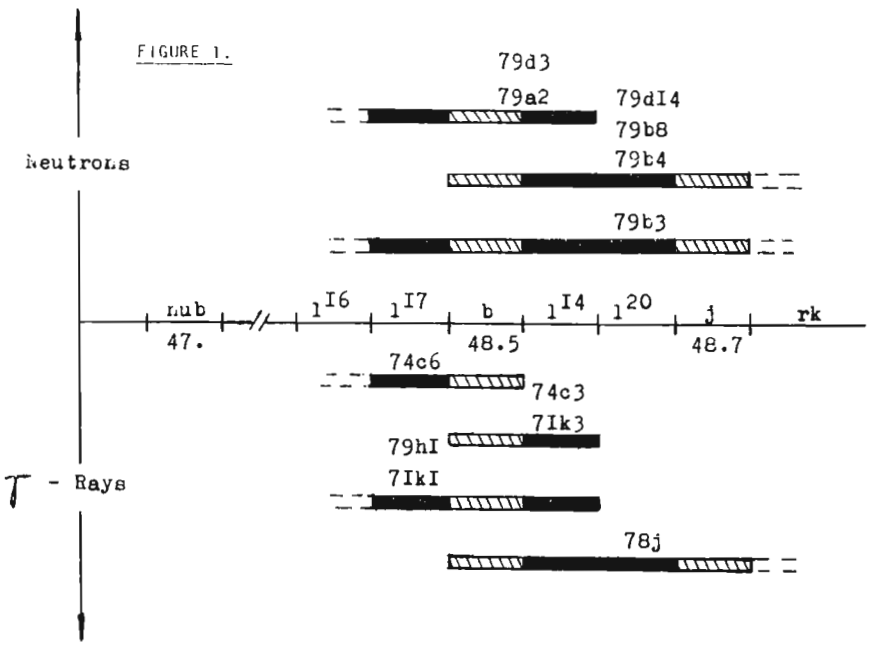

Fig. 1. Complementation map of neutron- and γ-ray-induced b lethal mutations of D.melanogaster as compared with genetic map (see for the latter Lindsley & Grell 1968; Woodruff & Ashburner 1979). Localizations determined by complementation patterns. 5 genetic units were defined. Black, affected units with lethal effects; hatched, units with visible (b or j) phenotype; dashed, further possible extension of the deletion.

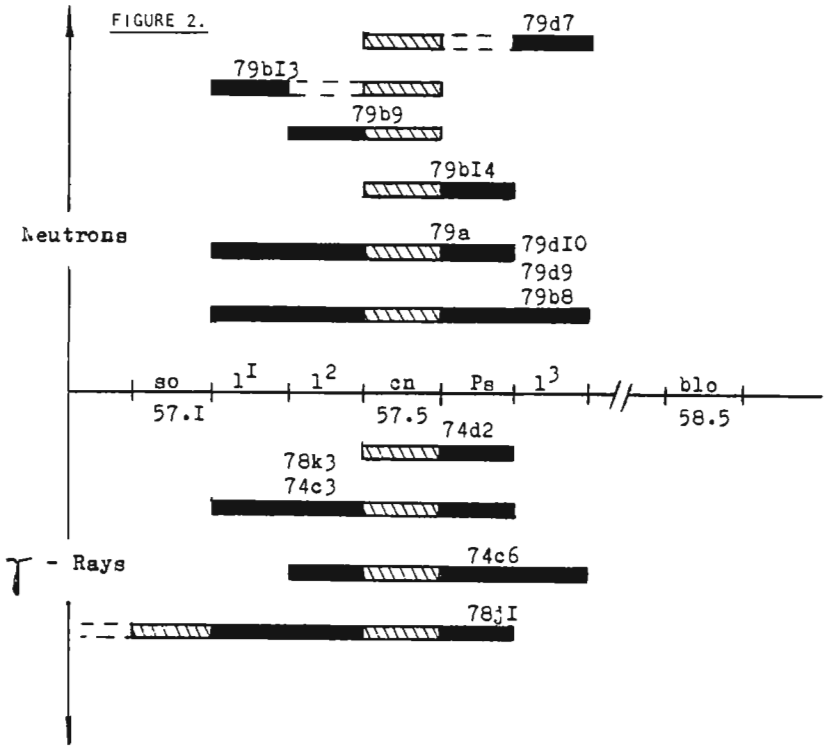

Fig. 2. Complementation map of neutron- and γ-ray-induced cn lethal mutations as compared with genetic map (see for the latter Lindsley & Grell 1968). 6 genetic units were defined. Black, affected unites with lethal effects; hatched, units with visible (so or cn) phenotype; dashed inside, inversion proposed; dashed outside, further possible extension of the deletion.

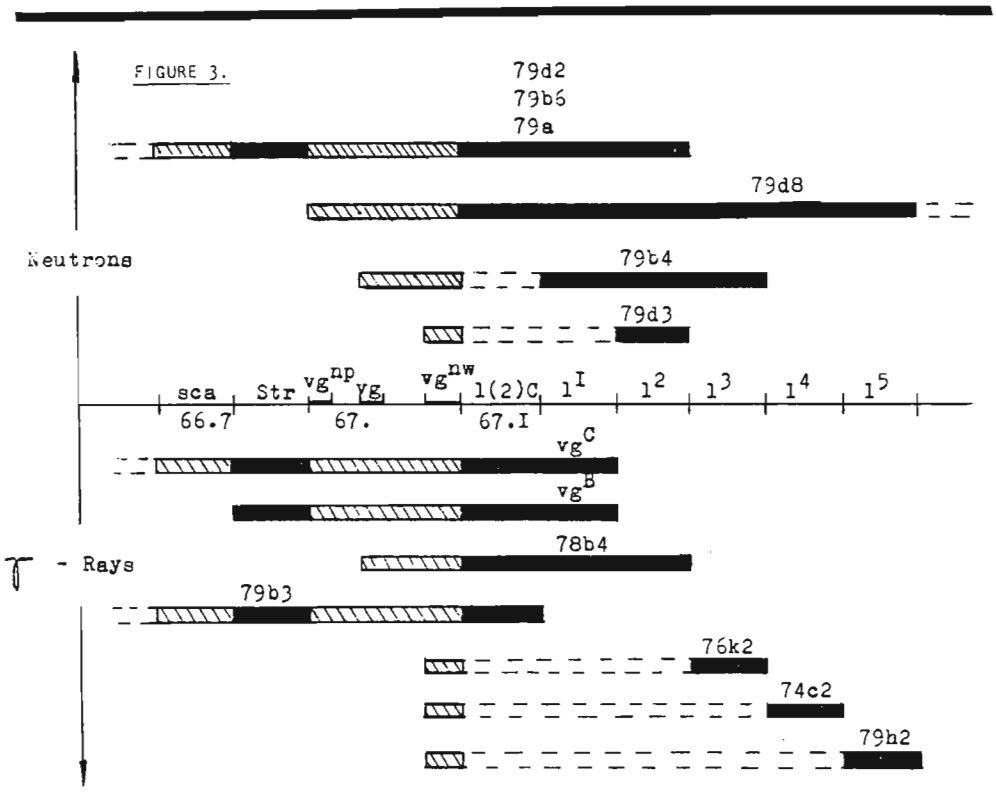

Fig. 3. Complementation map of neutron- and γ-ray-induced vg lethal mutations as compared with genetic map (see for the latter Lindsley &Grell 1968). Localizations determined by complementation patterns. 9 genetic units were defined. Black, affected units with lethal effects; hatched, units with visible (sca or vg pseudoalleles) phenotype; dashed inside, inversion proposed; dashed outside, further possible extension of the deletion.

The remaining lethal visibles appear to be a kind of inversions that complement to give rise to viable visible. It is important that after neutron irradiation lethal visible patterns for the black and cinnabar loci that do not mutate intragenically with recessive lethality are similar to that found for the vg locus mutating in this way. In the light of these data it is obvious that neutron- and γ-ray-induced lethal visibles are a kind of the minute rearrangements (deletions or inversions) with the equal size and position. Therefore, neutron-induced lethal mutations as well as mutations produced by low-LET radiation are qualitatively the same, and the differences in the genetic action of two radiations in question have a quantitative rather than a qualitative nature.

Grateful acknowledge is made to R.C. Woodruff, Bowling Green, Ohio, for supplying the nub, j, so, blo, sca, vg^B, vg^C, and 1(2)C stocks.

References: Lindsley, D.L. & E.H.Grell 1968, Publ.Carnegie Inst. 627:1-471; Muller, H.F. 1954, Radiation Biology (Ed: A.Hollaender, McGraw H.V., New York), vol. 1/1:496-507; Woodruff, R.C. & M.Ashburner 1979, Genetics 92:133-149.

Alonso, A. and A.Munoz. Universidad de Cordoba, ESPANA. Biometric characterization of some wing measurement in Drosophila melanogaster.

Although genetic populations developed tremendously through Hubby & Lewontin research (1966) and due to electrophoresis techniques which enabled gene allele manifestations to be individually visualized, it is well known that not all genetic manifestations can be understood in such terms. In the field of Quantitative Genetics, the auxiliary techniques are actually mathematical methods, biometrical to be exact. The research on heredity in the wing size of D.melanogaster published by Reeve & Robinson (1953) is very interesting, although these authors, as well as others usually use only one or two variables in their studies.

More recently, Alonso & Munoz (1982) have carried out multivariant analytical studies in order to localize discriminant traits. Of course, it is difficult to find concrete studies on wing size and form in D.melanogaster through use of multivariant analysis, but these can be found for other species (Lefebvre et al. 1974; Pereira 1972; Jolicoeur et al. 1960).

Using 100 males and 100 females in each of two laboratory populations, one maintained at 25°C and the other (a replica of the former) maintained at 30°C for one generation, we have carried out the fifteen measurements indicated in Fig. 1. The methodology employed was the analysis of principle components, based on the covariance matrix, and on the correlation to obtain the factorial matrix (using the logarithmical transformation of data). The methodology has been found adequate for the idea we have pursued from the outset, which is to say, for the detection of measurements denoting size and of those indicative of the wing form of the fly, as well as for a discriminating analysis between the natural groups formed in this study; males at 25°C, males at 30°C, females at 25°C and females at 30°C. In the component

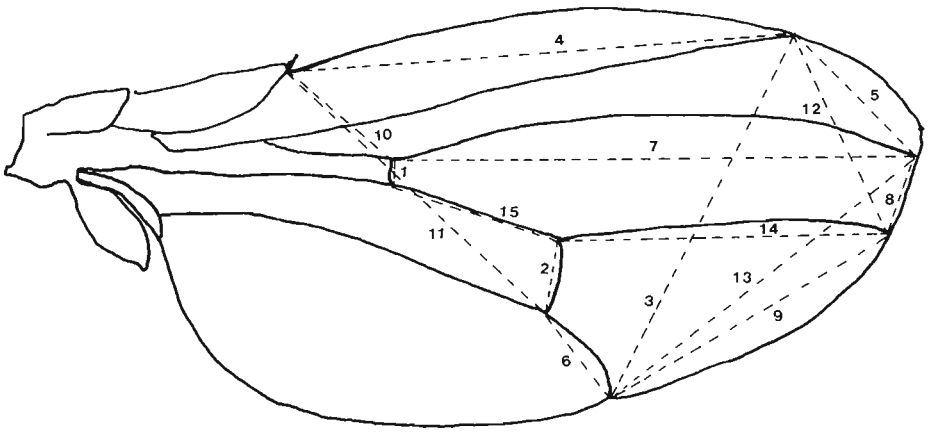

Fig. 1. Representation of the measurements taken on the Drosophila melanogaster wing.

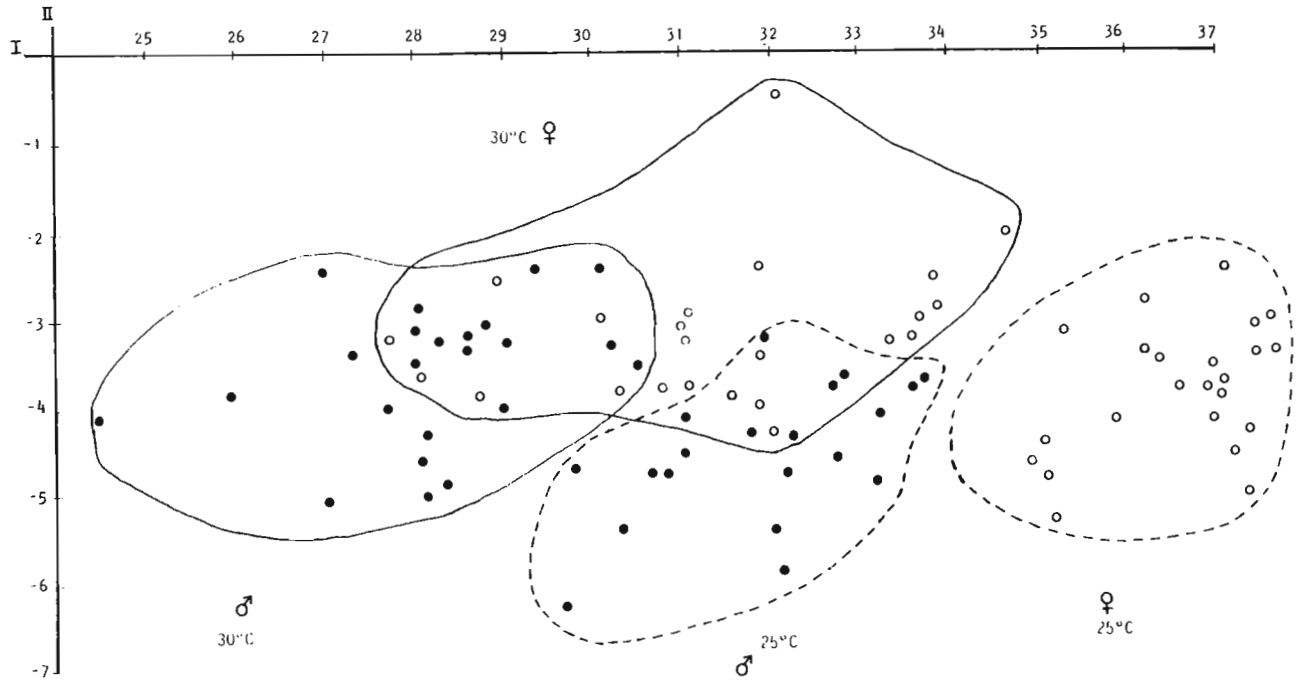

Fig. 2. Representation of the discrimination shown by the component principle analysis in the whole study, using only the first two components (I and II).

principle matrix, as the first component principle has a positive sign in all individuals and the second component is at times positive and others negative, the results confirm the fact that the first component indicates size and the second, form. From an examination of the factorial matrix, we deduce that the measurements denoting size are, in order of importance: 7, 3, 4, 13 and 14; the measurements indicative of form are 4, 15 (negative sign), and 1, 8 and 12 (positive sign) in such a way that when the former decrease, the latter increase and vice versa, thereby originating individuals with normal, lengthened or rounded wing forms as a result of the different interactions. The measurements 5, 9 and 11 have not been of use to indicate either size or form which is why they could be omitted in later studies.

Fig. 2. represents the relationship of the individuals with respect to the first two principle components (which absorb more than 80% of the total inertia); in this figure, the discrimination between the four established groups can be observed, although we must point out the somewhat ambiguous position of the females maintained at 30°C, the dysgenic temperature of these populations.

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Alonso, A., A.Rabasco and A.Munoz.
Universidad de Cordoba, ESPANA.

Allele frequencies distribution at the Est-C locus in wine cellar populations of Drosophila melanogaster.

The Est-C locus perhaps is one of the polymorphic allozimic loci which isn't studied as much as others, because the results can be erratic, at least in natural samples taken from different places and times. Since Beckman and Johnson (1964) described two alleles (the fast and the slow one) and its three correspondent phenotypes, many other alleles have been described in different populations. In the two, Japanese and North American populations of Drosophila melanogaster studied by Kojima et al. (1970) the genic frequencies for the fast allele were almost fixed (0.844 and 0.920, respectively) and the other alleles oscillating with frequencies of 0.104 and 0.074, and furthermore a very-fast allele with 0.005 frequency has been found in the Japanese population. In the natural Greek population studied by Trianthaphillidis and Christodoulou (1973), the genic frequencies for the three alleles were: 0.017 for the slow allele, 0.977 for the fast one and 0.06 for the v-fast allele.

Johnson & Schaffer (1973) analyzed this locus in twelve natural populations, in different places of the U.S.A. An average allelic frequency was found for the slow allele equal to 0.047, 0.897 for the fast one and 0.056 for the v-fast. Furthermore a fourth allele was detected in a population of Florida, called highly-fast, the allelic frequency was 0.005. Up to this moment, None of the revised authors find the recessive-null alleles. Girard & Palabost (1976) were the first in detecting the existence of a recessive allele in four of the fifteen populations taken from a wine cellar in South France. The allelic frequencies on the average were: 0.057 (Est-C^S), 0.883 (Est-C^F), 0.006 (Est-C^{VF}) and 0.024 (Est-C^O). However, Anxolabehere et al. (1975) studying the same populations of the paper of Girard & Palabost (1976) during three years didn't find the recessive allele or the v-fast allele, and the allelic frequencies were 0.042 for the slow allele and 0.958 for the fast allele.

Recently Trianthaphillidis et al. (1980) when studying in a natural population of the island of Corfu, once more found the three alleles which they had found in 1973, with genic frequencies of 0.026 (Est-C^S), 0.967 (Est-C^F) and 0.007 (Est-C^{VF}). Voelker et al. (1980) studying North Carolina populations and Langley et al. (1981) in London populations, found null alleles in this locus with frequencies 0.005 and 0.0049, respectively. In the latest reference revised (Singh et al. 1982) the authors detested four alleles (none of them recessive) in two american populations and one in Africa, while only the slow allele (and whatsmore the fast) was found in four different populations originally from America, France, Vietnam and Taiwan, which the allelic frequencies were 0.049 (Est-C^S) and 0.951 (Est-C^F). And the fast allele was fixed in both Canada and Australian populations.

As we can see, of all the authors revised by us, Girard & Palabost (1976, 1977), are the only ones that find null allele with significant frequency, when studying this polymorphic locus in the wine cellar populations.

We have studied this polymorphic locus in ten wine cellar populations, in the South Iberian Peninsula, using the starch-gel electrophoresis technique (Poulik 1957) with the modifications put forward by Beckman & Johnson (1964).

The results obtained can be seen in Table 1. The allelic frequencies have been calculated by Bernstein's method (Cavalli-Sforza & Bodmer 1971).

Table 1. Frequencies for Est-C^S, Est-C^F, Est-C^V and Est-C^O alleles in ten wine cellar populations.

Population	sample size	S	F	V	O	HET. obs.	HET. est.
C1	100	0.005	0.808	0.035	0.151	-	0.321
C2	100	0	0.995	0.005	0	0.01	-
C3	100	0	1	0	0	-	-
C4	100	0	0.985	0.015	0	0.01	-
C5	100	0.026	0.726	0	0.246	-	0.408
C6	100	0.016	0.834	0.035	0.113	-	0.286
C7	100	0	0.859	0.059	0.084	-	0.256
C8	100	0	1	0	0	-	-
C9	100	0.021	0.762	0.015	0.201	-	0.376
C10	100	0.011	0.924	0.017	0.053	-	0.153
Σ=1000		0.008	0.890	0.018	0.085	0.010	0.300

As we can observe, the percentage of the populations which have a fixed locus in the Est-C^F is higher than that of the other authors, although the genic frequency on an average of the ten populations does not differ at all from the others, as a result our fixations can have some sampling error.

The same as Girard et al. (1976, 1977), Voelker et al. (1980) and Langley et al. (1981), a null recessive allele has been found by us with frequencies higher than those from the French populations. But on the whole these allelic frequencies for the Est-C^0, have an insignificant value. All the last ones with lower values detected for other alleles, make this locus practically monomorphic.

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Alonso, A., A.Rodero and A.Munoz. Universidad de Cordoba, ESPANA. Study of seven wing measurements and of esterase-6 locus in Drosophila melanogaster.

Since 1966 when Harris in Europe and Lewontin & Hubby in America first applied, almost simultaneously, electrophoretic techniques in the study of allelic variants attempts to relate this allozymic polymorphism to biometrical characters began in laboratory species as well as in domestic ones. Genetic markers were tested for their usefulness in Zootechnics.

Electrophoresis provides us with data on an individual gene. That is, for each protein and variable enzyme, there is a variation associated with different alleles in the loci, and vice versa, each invariable protein corresponds to a monomorphic locus. They attempted to associate the qualitative polymorphism with biometrical traits which are polygenic and whose genes could not be individualized. In this way, Aguade (1974), Cuello (1974), Serra (1977) and Porras (1978) oriented their studies which yielded unsatisfactory results. This occurred because the authors tried to establish a linear relationship between two variation levels of differing complexity, in which the more complex could depend on the simpler, but not in a linear function. On the other hand, the allozymic polymorphisms used

TABLE 1. Frequency distribution of the seventh measurement.

25°C	CLASSES SIZE			30°C	CLASSES SIZE		
GROUP	≤6.20	6.25-6.75	≥6.80	GROUP	≤5.25	5.30-6.00	≥6.05
Est-6^{FF} males	28	11	1	Est-6^{FF} males	30	21	1
Est-6^{FF} females	0	11	21	Est-6^{FF} females	0	12	11
Est-6^{FS} males	35	9	0	Est-6^{FS} males	16	25	0
Est-6^{FS} females	0	16	30	Est-6^{FS} females	1	31	25
Est-6^{SS} males	4	6	1	Est-6^{SS} males	4	6	0
Est-6^{SS} females	1	3	7	Est-6^{SS} females	0	6	7
TOTAL	68	56	60	TOTAL	41	101	44

TABLE 2. Frequency distribution of the first measurement.

25°C	CLASSES SIZE					30°C	CLASSES SIZE				
GROUP	≤0.20	0.25	0.30	0.35	≥0.40	GROUP	≤0.20	0.25	0.30	0.35	≥0.40
Est-6^{FF} males	8	20	11	1	0	Est-6^{FF} males	8	30	4	0	0
Est-6^{FF} females	0	4	17	9	2	Est-6^{FF} females	1	7	13	2	0
Est-6^{FS} males	3	30	11	0	0	Est-6^{FS} males	10	22	9	0	0
Est-6^{FS} females	0	4	30	12	0	Est-6^{FS} females	3	11	35	7	1
Est-6^{SS} males	0	3	8	0	0	Est-6^{SS} males	2	6	2	0	0
Est-6^{SS} females	0	1	6	4	0	Est-6^{SS} females	1	1	8	2	1
TOTAL	11	62	83	26	2	TOTAL	25	77	71	11	2

had not been previously determined in the experiment outline, but instead by the techniques available in the laboratory at that moment.

Recently Pieragostini et al. (1979, 1981) have found a relationship between the Adh genotype and the body size of Drosophila melanogaster.

We have caught three natural populations of Drosophila melanogaster, they were pooled into one laboratory population equilibrated at the Est-6 locus. Afterwards, we maintained it in stable conditions of temperature and humidity for 24 generations without overlapping. The Est-6 locus was chosen because it codified a nonspecific enzyme which can act on exogenous substrates or on endogenous ones, and because it acts in the glucose metabolism and other systems. The measurements were taken according to Alonso & Munoz (1984) results in order to detect the size as well as the form of the wing. In the 25th generation, we separated two groups and one of them was submitted to 30°C. Table 1 shows the results of wing measurements no. 7 (Alonso & Munoz 1984) for different phenotypes and sexes at 25°C and 30°C, respectively, and Table 2 shows the same for the first measurement.

We have analyzed these data with a simple nested ANOVA, none of the analyses showing any differences between the esterase phenotypes in any of the seven measurements (the first 7 measurements of Alonso & Munoz, 1984) but all of them exhibiting differences between sexes and temperatures.

In the multivariant analyses (principal components) carried out, the individuals are always grouped according to sex and temperature and never according to the Est-6 phenotype.

In conclusion we do not detect any relationship between the wing size or form and the phenotype expressed by the Est-6 locus.

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Substrate specificity of esterases in D.pseudoobscura and D.melanogaster, with notes on the tissue localization of Esterase-5 in D.pseudoobscura.

Esterase-5 (Est-5) in D.pseudoobscura and Esterase-6 (Est-6) in D.melanogaster are apparently homologous loci (Abraham & Luchessi 1974). They reside on homologous chomosome segments and encode enzymes with similar catalytic properties. Est-5 maps to position 111.8, slightly distal to the visible mutant locus compressed (co) on the right arm of the X chromosome of D.pseudoobscura (Beckenbach 1981), and in D.melanogaster, Est-6 maps to III-36.8 (Wright 1963) close to the visible mutant locus gespleten (gs). The co and gs mutations produce similar phenotypic effects which further strengthens the case for homology. As part of a study of the determinants of esterase electrophoretic mobility, we have conducted tests to examine the substrate specificities and tissue localizations of these esterases. The results confirm that EST-5 and EST-6 resemble each other biochemically.

Homogenates (6 flies per 150 µl grinding solution: gel buffer with 5% sucrose and a dash of bromochlorophenol blue) were prepared from male and female D.melanogaster genotypically S/S, S/F and F/F for Est-5. For D.pseudoobscura samples were prepared from females with Est-6 genotypes of 100/100, 100/112 and 112/112 and from males with genotypes of 100/Y and 112/Y. Supernatants were run on polyacrylamide gels made by polymerizing 5% or 8% (w/v) solutions of acrylamide monomer and bisacrylamide cross linked (w/w ratio 19:1). The gel and electrode buffer was 87.7 mM Tris, 9.6 mM boric acid and 2.7 mM disodium EDTA pH 9.0 (at 25°C) after Keith (1983). All runs were carried out at 0°C.

A variety of chemical cocktails have been used in the past to detect the presence of enzymic gene products of Drosophila esterase loci. The enzymes (carboxyl esterases, E.C. 3.1.1.1) show a broad substrate specificity, but short chain aliphatic esters are usually found to make the best substrates. Both EST-5 and EST-6 hydrolyze β-napthyl esters more quickly than α-napthyl esters and short carbon chain aliphatic derivates (e.g., acetate) are preferred (see Narise & Hubby 1966, on EST-5 and Danford & Beardmore 1979, on Est-6). Nonetheless, EST-5 and EST-6 are routinely detected on gels by using α-napthyl acetate (α-NA) instead of or together with β-napthyl acetate (β-NA). Many different dyes which couple to the liberated napthol product have been used to visualize esterase activity on gels: Fast Red TR, Fast Garnet GBC, Fast Blue B, Fast Blue RR (these and all dyes and substrates used were provided by Sigma Chemical Co., St. Louis, MO, USA). Therefore, we designed a series of tests to examine the staining properties of these enzymes. After electrophoresis replicate gels of identical homogenates were stained in 0.1 M phosphate buffer pH 6.5 with 50 mg dye plus 20 mg of either only α-NA, only β-NA, or both α-NA and β-NA (substrates were added from 20 mg/ml solutions in acetone). These three substrate combinations were tested in the presence and absence of pronan-1-ol. The results are shown in Table 1.

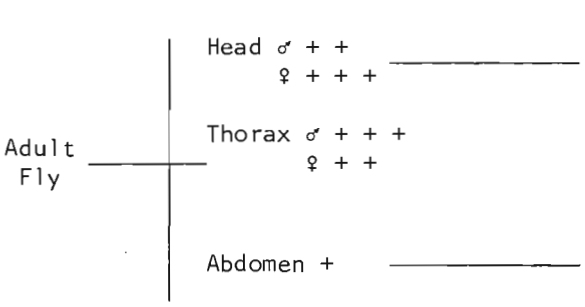
Table 1. Staining properties of the β-specific napthyl esterases EST-5 and EST-6.

Substrate	Propan-1-ol present?	Fast Red TR-napthol conjugate colour and intensity
α-NA	-	rust[1]
β-NA	-	orange[1]
α-NA + β-NA	-	orange
α-NA	+	rust; increased intensity
β-NA	+	orange; increased intensity
α-NA + β-NA	+	orange; increased intensity

1=with Fast Garnet GBC, the colours observed were brown and pink with α-NA and β-NA, resp.

No differences in staining properties were observed between sexes or genotype for either enzyme. The α-specific esterase Est-C from D.melanogaster, gives the same colour pattern except that the α-napthol-dye conjugate colour (rust or brown) predominates when α-NA + β-NA substrate mixtures are used.

Table 2. Tissue localization of Est-5 activity in D.pseudoobscura.



Eyes + +	Intensity of staining (visual estimate) is indicated by number of + symbols; ± indicates a trace of Est-5 activity. Sex-specific differences are indicated by ♂ and ♀ symbols.
Rest of head ±	
♂ Testes +	
♀ Ovaries +	
Gut ± (but + for a different esterase)	
Cuticle ±	
Malpigian tubules -	

In D.pseudoobscura samples run on 5% polyacrylamide gels, we observed a streak of esterase activity with slower mobility than EST-5. When 8% gels were used, the streaking was replaced by a concentrated band of activity. This locus stained rust coloured with α-NA plus β-NA substrate mixtures and Fast Red TR dye. Thus, we conclude that this represents an α-specific esterase (this zone did stain orange as expected with Fast Red TR and β-NA alone).

From these results we recommend the following: (i) For maximum detection of β-specific esterases, use β-NA plus Fast Red TR plus propan-1-ol. However, for ease of visual inspection and photography, rust or brown bands are more distinct than orange ones and thus α-NA plus Fast Red TR without propan-1-ol may be preferred. Depending on one's esthetic sense, other dyes such as Fast Garnet can be used, but the colour produced is somewhat faint.

(ii) To maximize information yield per gel, we recommend using both α-NA and β-NA plus Fast Red TR without propan-1-ol. This combination reveals all α- and β-specific esterases.

(iii) In order to pick out a favourite β-specific esterase from a background of α-specific esterases (e.g., EST-6 from EST-C in D.melanogaster) we suggest α-NA plus β-NA plus Fast Red TR (or Fast Garnet GBC) with propan-1-ol. The alcohol reduces the activity of the α-specific enzymes, and the presence of α-NA ensures that they come up a darker colour.

We studied the tissue distribution of Est-5 in D.pseudoobscura by dissecting out various organs from two adults carrying 112 allele only. Adult flies were divided into head, thorax, and abdomen. Organs within the abdomen were dissected and rinsed in saline. Eyes were excised from heads, and both eyes and eyeless heads were tested. Tissues were homogenized in 20 µl of grinding solution and 10 µl of supernatant loaded on gels. Whole flies used as controls were ground in 30 µl and 6 µl loaded on gels. The activity of EST-5 in the organ samples were judged visually by staining intensity (see Table 2).

Our results agree well with those of Lunday & Farmer (1983), especially with regard to the finding of high EST-5 activity within the eye of D.pseudoobscura. Our failure to find high EST-5 levels in saline-washed isolated abdominal tissues compared with levels in whole abdomens is also consistent with Lunday & Farmer's (1983) report that most EST-5 activity in D.pseudoobscura is present in the haemolymph. EST-6, on the other hand, has been shown to reside primarily in the anterior ejaculatory duct of male D.melanogaster (Sheehan et al. 1979).

Overall, the results of our substrate specificity experiment support the model of homology between Est-5 in D.pseudoobscura and Est-6 in D.melanogaster. The tissue localization of these enzymes, however, clearly demonstrates that these enzyme loci are now under different forms of developmental regulation.

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References: Abraham, I. & J.C.Luchessi 1974, Genetics 78:1119-1120; Beckenbach, A.T. 1981, DIS 56:23-24; Danford, N.D. & J.A.Beardmore 1979, Biochem.Genet. 17:1-22; Keith, T.P. 1983, Genetics 105:135-155; Lunday, A.J. & J.L.Farmer 1983, Biochem.Genet. 21:453-463; Narise, S. & J.L.Hubby 1966, Biochim.Biophys.Acta 122:281-288; Sheehan, K., R.C.Richmond & B.J. Cochrane 1979, Insect Biochem. 9:443-450; Wright, T. 1963, Genetics 48:787-801.

Baker, W.K., B.Kaeding and G.Jeppesen.
University of Utah, Salt Lake City.

Experiments designed to obtain evidence on selection for alpha-esterase haplotypes in laboratory populations of D.montana.

The gametic disequilibrium in natural populations among the 16 possible haplotypes of active (A) and null (0) alleles at the four closely-linked alpha-esterase loci in D.montana is attributed to natural selection (Baker & Kaeding 1981). We devised experiments to see if this selection could be demonstrated in laboratory populations.

In 1980 a control population cage was established from five ♂♂ and five ♀♀ from each of 75 isofemale strains collected in Utah the previous summer (1979). One year later (1981) this cage was sampled by crossing individual flies to homozygous null flies and electrophoresing their offspring to determine the two haplotypes in each sampled fly. Table 1 shows the observed numbers of the 16 different haplotypes in a sample taken from the Utah natural population in the summer of 1979 (data from Baker & Kaeding 1981), and the observed numbers in the sample taken from the control population cage after one year. The expected numbers (based on gametic equilibrium) of each haplotype and the ratio of observed/expected is also given. Table 2 provides the frequencies of the active alleles at the four loci observed in the natural population as well as the frequencies in the control population cage after a year. It can be seen from these two tables that gametic disequilibrium was observed in both the natural population and the control cage population although it was not as striking in the latter as in the former. It is also evident that the allelic frequencies were almost the same in the natural and laboratory populations although the frequency of active alleles at locus four was significantly lower in the population cage.

Now that we knew what the equilibrium allelic frequencies were in the laboratory and which haplotypes were favored and which disfavored under these conditions, it was possible to establish an experimental population cage in which the allelic frequencies were approximately the same as in the control cage population but in which the frequency of haplotypes in disfavor were in the majority. If selection were operating, the frequency of disfavored haplotypes should rapidly decrease with time in this experimental cage.

Table 1. (N = number of chromosomes)

Haplotype				Utah Nat. Population			Control Cage Pop.			Exper. Pop.	
1	3	2	4	Obs.	Exp.	Obs./Exp.	Obs.	Exp.	Obs./Exp.	N	Freq.
0	0	0	0	0	9.4	0	1	9.6	.10	480	.3125*
A	0	0	0	2	4.0	.50	1	3.8	.26		
0	A	0	0	13	11.1	1.2	13	15.1	.86		
0	0	A	0	8	11.5	.70	15	11.3	1.3		
0	0	0	A	5	5.1	.98	7	2.0	3.5		
A	A	0	0	18	4.7	3.8	7	5.9	1.2	96	.0625
0	0	A	A	22	6.2	3.5	1	2.3	.43	96	.0625
A	0	0	A	4	2.2	1.8	2	0.8	2.5		
0	A	A	0	18	13.5	1.3	23	17.7	1.3	288	.1875
A	0	A	0	5	4.9	1.0	14	4.4	3.2		
0	A	0	A	4	6.0	.67	4	3.1	1.3	192	.1250
0	A	A	A	0	7.3	0	1	3.6	.28		
A	0	A	A	0	2.7	0	0	0.9	0		
A	A	0	A	0	2.6	0	0	1.2	0		
A	A	A	0	1	5.8	.17	1	6.7	.15	384	.2500*
A	A	A	A	0	3.1	0	0	1.4	0		
				100	100.1		90	89.8		1536	1.0000

Table 2. (N = number of chromosomes sampled)

Date	Population	Frequency of Active Allele 1	3	2	4	N
1979	Utah, Natural	.30	.54	.55	.35	100
1980	Control Cage	.28	.61	.54	.17	90
Jun 82	Initial Exp. Cage	.31	.62	.50	.19	1536
Jul 82	Exp. 1st sample	.32	.74	.46	.17	100
Sep 82	Exp. 2nd sample	.41	.81	.64	.16	210
Dec 82	Exp. 3rd sample	.29	.58	.51	.24	252
Mar 83	Exp. 4th sample	.38	.62	.64	.27	254

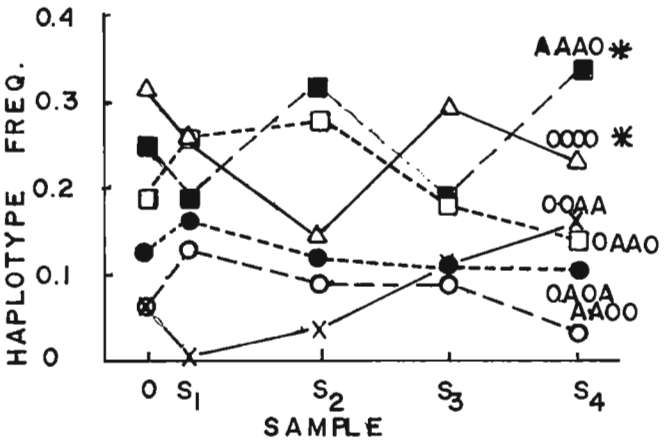


FIGURE 1.

The experimental cage was started with 768 flies comprised of equal numbers of ♂♂ and ♀♀ of the following genotypes: 96 00AA/AA00 + 192 0AA0/0A0A + 96 0AA0/0000 + 384 AAA0/0000. (The four genes are given in their order on the chromosome: 1, 3, 2, 4.) Table 1 gives the numbers of the six haplotypes used to initiate the experimental cage and an asterisk denotes a haplotype in extreme disfavor in both the control cage and the natural population. Thus, at a minimum, over 50% of the chromosomes in the experimental cage were putatively detrimental. The ratio of the six haplotypes initiating the experimental cage was arranged so that the allelic frequencies of active and null alleles at the four loci matched those of the control cage (see Table 2). Note that the cage was started from heterozygotes made from crosses of homozygous lines of the six haplotypes established previously from a population collected in Gothic, Colorado.

The experimental cage was set up in June 1982 as previously described, and in July F_1 larvae were removed, reared, and testcrossed to determine the relative frequencies of the six haplotypes among a sample of 100 chromosomes. Subsequent samples were taken in September 1982, December 1982, and March 1983. The haplotype frequencies observed in these samples are pictured in Figure 1, and the allelic frequencies observed are given in Table 2. There is no indication in these data of any consistent change in either haplotype frequencies or allelic frequencies over the period of experimentation. Certainly the detrimental haploytypes (asterisks) did not decrease in frequency. We estimate that the generation time in the cages was about a month, making a total of around nine generations between the first and the last sample. It was planned to take a last sample after an additional six months but a laboratory accident eliminated the population. These data provide no evidence of selection acting over the limited period of this experiment.

Supported by NSF Grant DEB-79-12336. References: Baker, W.K. & E.A.Kaeding 1981, Amer.Nat. 117:804-809.

Banerjee, I. & A.S.Mukherjee. University of Calcutta, India. Activation of potential initiation sites of DNA replication by Puromycin: evidence from fibre autoradiography.

In eukaryotes replicons occur in clusters (Edenburg & Huberman 1975). Such clusters are organized into replicon families and replicate at a given rate during DNA synthesis (S) phase (Van't Hof & Bjerknes 1978).

In the present study, DNA fibre autoradiography was used to determine the replication properties of polytene DNA at the level of initiation and chain elongation. We have used Puromycin which is a potent inhibitor of protein synthesis for monitoring the property. Our work is based on previous evidence supporting the idea that there are two different protein pools, one responsible for the control of DNA synthesis at the level of initiation and the

Table 1. Replicon properties.

Pre-treatment	Pulse time (^{3}H-TdR) (Sp.act.) (min.)	Mean length of labelled segments (µm±S.E.)	Average of means of length of labelled segments per 100 µm of stretch length (µm±S.E.)	Average No. labelled segments per 100 µm of stretch length (µm±S.E.)	Average of means of gap size per 100 µm of stretch length (µm±S.E.)	Average No. of gaps per 100 µm of stretch length (µm±S.E.)	Rate: fork movement µm/form/min.
Ringer	(Low) 10	4.34±1.08	5.83±0.66	9.90±0.54	5.80±0.97	9.72±0.49	0.434
Puromycin	(Low) 10	2.50±0.60	2.84±0.35	13.10±1.26	6.20±1.50	12.80±1.23	0.250
Ringer	(Low) 30	5.90±1.35	9.64±1.77	9.40±0.60	5.00±0.30	9.03±0.56	0.200
Puromycin	(Low) 30	2.60±0.64	3.42±0.33	16.75±1.56	4.35±1.26	15.90±1.58	0.086
Ringer	(High) 60	10.76±2.63	11.18±1.08	7.24±0.36	5.66±0.46	6.65±0.37	0.180
Puromycin	(High) 60	2.71±0.68	2.91±0.27	11.34±0.88	7.73±0.86	10.80±0.86	0.045
Ringer	(High) 90	12.60±1.94	20.00±2.20	5.33±0.37	8.19±0.76	4.92±0.36	0.140
Puromycin	(High) 90 (fed for 24 hours)	2.61±0.61	2.89±0.15	16.20±0.48	3.59±0.19	16.44±0.50	0.030
Puromycin	(High) 90 (fed for 48 hours)	2.52±0.58	2.69±0.14	20.65±0.53	2.50±0.09	20.80±0.52	0.028
Puromycin	(High) 90 (fed for 72 hours)	2.40±0.59	2.68±0.24	19.64±0.76	2.96±0.16	19.00±0.58	0.026
Ringer	(Low) 60 + (High) 60	13.80±0.68	18.66±2.07	5.14±0.37	5.03±0.32	5.03±0.32	0.115
Puromycin	(Low) 120	2.17±0.58	2.30±0.16	20.96±0.73	2.84±0.16	20.60±0.71	0.018
Ringer	(High) 60 + (Low) 120	20.50±3.60	19.90±1.47	4.20±0.19	3.92±0.19	3.92±0.19	0.114
Puromycin	(Low) 180	3.02±1.10	3.36±0.33	17.66±1.07	2.99±0.16	17.24±1.05	0.010

other, at the level of chain elongation (in preparation, and Mukherjee & Chatterjee 1984).

After Puromycin pre-treatment or incubation in Ringer (control), late third instar larval salivary glands from D.hydei were pulse-labeled in ^{3}H-TdR for different periods of time, starting from 10 mins to 180 mins. The glands were then lysed in lytic buffer (pH 10.8) containing proteinase K, passed through 5% chilled TCA, dehydrated, covered with AR10 stripping film or Ilford emulsion and exposed for a period of 4-8 months in light-tight bakelite boxes. After exposure, the slides were developed in D19b, fixed in X-ray fixer, dried mounted and observed under a Zeiss Photomicroscope III.

The data of all Puromycin treated and control slides are comprehensively pooled in Table 1. From the table, the following facts can be derived: (1) Mean length of labeled segments. In control, whereas a gradual increase in the mean length of labeled segments was observed, in Puromycin treated preparation, it remains more or less constant with increase in the length of pulse time.

(2) Average number of labeled segments/100 µm of stretch length. While in control, this number decreases, in Puromycin treated fibres the number increases (though fluctuating) with increase in the length of pulse time.

(3) Average size of the unlabeled gap and number of gaps/100 µm of stretch length. In control, the gap size remains more or less constant, whereas with Puromycin, the gap size, though fluctuating at the beginning, decreases gradually and then remains constant with increasing duration of pulse time. In control, the number of unlabeled gaps per 100 µm stretch decreases, while with Puromycin, the number tends to increase with increasing time.

(4) The rate of fork movement. In control, the rate is at first high, then drops down sharply and then gradually decreases with increasing pulse time. In Puromycin treated preparations, the rate though 2 to 10 folds less follows the same curvilinear regression as found in the control set.

It appears from the results that Puromycin induces a stage of replication found in early embryogenesis by activating the number of initiation sites, inducing clustering of replicons and reduced replicon size.

Furthermore, the results suggest that there may be two classes of replicon families as suggested by Hori (1979) and others. Puromycin inhibits the rate of fork movement in both types of replicon families.

References: Edenberg, H.J. & J.A.Huberman 1975, Ann.Res.Genet. 9:245-284; Hori, T. 1979, Jap.J.Genet. 55(1):41-54; Hori, T. & K.G. Lark 1974, J.Mol.Biol. 88:221-232; Van't Hof, J., A.Kuniyuki & C.A.Bjerknes 1978, Chromosoma (Berl.) 68:269-285.

Basden, E.B. Leyden Park, Bonnyrigg, Midlothian, Scotland. The Species as a block to mutations.

Mutants (phenotypes) of D.melanogaster and of a few other species of Drosophila have been described in detail. The number discovered since 1907 in melanogaster alone is many, many thousands and of every category.

There are no lists, however, of mutants that might be expected but are not found. One type will be discussed here, and for this purpose the species of Drosophila are grouped into two distinct divisions, viz: (1) The clear-wings. These are species whose wing-blades (including veins) are clear, hyaline, and quite unmarked. Clear wings include affinis, ananassae, funebris, hydei, melanogaster, pseudoobscura, subobscura, etc. (2) The marked-wings. Species whose wings bear a naturally pigmented spot or spots, or cloud, or pattern. Included here are hawaiian picture-wings, immigrans, robusta, quinaria-group, virilis-group, etc.

As far as is known there are no mutants (visible mutations) of any clear-wing species that have pigmented wing marks. Converesely, there are no mutants of marked-wing species that have unmarked wings. Excluded from clear-wing mutants are suffused general yellowing or darkening (as in yellow, black, dusky, ebony, sooty of melanogaster, and the shadowy smudge along the costa of subobscura at certain seasons), melanotic tumors, blood blisters, and developmental disturbances (e.g., black spotted wings (DIS 58:203), dumpy-oblique lethal vortex, and speck).

Wild-type marked-wings have one or more regular precise wing areas that are naturally and discretely pigmented in fully hardened flies. If the marks are multiple, any mutation would have to be assessed on the disappearance of all rather than on some of that particular type.

Thus it appears that at the species level there is a block to the apparently simple shift to or from pigmentation in the wings. In other words, the species is a block to some mutations. However, in a few species the male and female wings differ, one sex being clear-wing, the other marked-wing. Examples are D.tristis of the western palearctic and some species of the melanogaster-group. Evidently many clear-wing species do contain plenty of pigment in their bodies but it does not occur in discrete, localised spots in their wings. Yet two closely related species may belong to the opposite divisions. So where have all these intraspecific mutations gone?

Anyone has my consent to quote this note. I am grateful for information from M.Ashburner, H.Gloor, Oswald Hess, Claude W. Hinton, Costas B.Krimbas, Dan L.Lindsley, K.G. LUning, Dwight D. Miller, Toyohi Okada, D.Sperlich, Lynn H. Throckmorton, L.Craymer, and A-M.Jönsson(née Perje).

Bauer, S.J. York University, Downsview, Canada. Sex differences in pupation site choice in Drosophila melanogaster.

Pupation site choice (pupation height) was measured as the distance a larva pupated from the surface of the medium. Fifteen isofemale lines of Drosophila melanogaster were established from a natural population in the northern Toronto area and were tested for pupation height. Significant (F-19.55, $p<0.0001$) between line variation was found for this trait. From the 15 isofemale lines, two were chosen for further study: a low line and a high line. To determine whether there were sex differences for where larvae pupated, seven "strains" were tested: the two extreme lines, their two reciprocal crosses and backcrosses and the F_2 generation. A quantitative genetic analysis of pupation height in the isofemale lines will be published elsewhere. The present study reports a significant influence of sex on pupation height.

The method used was modified from Sokolowski and Hansell (1983). For each strain tested, 10 vials (11x2cm) containing 5.0 ml of a standard dead yeast-agar medium were each seeded with 10 closely-aged (±1.75 h) first-instar larvae. Care was taken to spill no medium on the walls of the vials. The vials were stoppered with standard-sized cotton balls and incubated at 24±1°C under conditions of 60% humidity with a light cycle of 12 hours light followed by 12 hours dark with the lights turned on at 8:00 a.m. Once the larvae had pupated and were close to emerging, the distance from the medium to a point between the two spiracles of each pupa was measured. The sex of each pupa was also recorded.

Table 1. Analysis of variance of pupation height for males and females of each strain of D.melanogaster.

Source of Variation	D.F.	F	P
Strain	6	17.83	0.0001
Sex	1	23.64	0.0001
Strain x Sex	6	0.62	0.7173
Residual	466		

Table 1 gives the results of an analysis of variance of pupation height for males and females for each of the seven strains tested. The effects of strain and sex were significant. In all strains, the mean male pupation height was higher than the mean female pupation height. There was no significant interaction between strain and sex. The results of an F_{max} test (Sokal & Rohlf 1969) showed that the variances were homogeneous ($F=2.64$, $p>0.05$).

The observed trend of sex differences in pupation site choice may be due to developmental differences between males and females. Casual observation showed that females tended to emerge before males and Alpatov (1930) found that males pupate before females. There also exist morphological differences between the sexes with females being somewhat heavier than males (Bakker 1959). Differences in pupation height between males and females may also have a genetic basis. When these trends are not taken into consideration, sex differences in pupation site choice in populations of Drosophila melanogaster could confound experimental results. For example, while selecting for an increase in pupation height, the sex ratio at each generation of selection will be heavily biased towards males.

References: Alpatov, W. 1930, Biol.Bull. Wood's Hole 58:85-103; Bakker, K. 1959, Ent. Exp. & Appl. 2:171-186; Sokal, R. & F.Rohlf 1969, Biometry, W.H.Freeman & Co., San Francisco; Sokolowski, M. & R.Hansell 1983, Behav.Genet. 13:267-280.

Belo, M. and D.A.Banzatto. Campus de Jaboticabal-UNESP, SP, Brasil. Association between Drosophila and yeasts. III. Attraction of males and females of D.ananassae.

The present experiment was carried out to confirm previous observations by Belo (1982) on the preference of D.ananassae (collected in Olimpia, SP, Brasil) for yeasts, which were classified according to the numbers of flies attracted in "most attractive," "intermediate" and "less attractive." Table 1 shows the frequencies of flies attracted for the yeast species in the attraction-box (Belo & Lacava 1980 & 1982).

The statistical analysis did not show any differences between males and females in their preferences for species of yeast, or for the interaction sex and yeasts, nor for the repetition within the sexes. The only detected difference was for the numbers of flies attracted to the yeasts. So in Table 1 the averages followed by the same letters are not statistically

Table 1. Percentage values for the flies attracted to yeast species, transformed in arc sin $\sqrt{x/100}$.

	Hansenula anomala	Pichia membra naefasciens	Saccharomyces chevalieri	Saccharomyces kluyveri	Torulospora delbrueckii
MALES	5.74	8.13	21.97	31.95	47.87
	9.97	11.54	25.10	45.00	30.00
	14.18	15.34	27.27	44.43	23.58
	5.74	5.74	21.97	60.00	18.43
FEMALES	8.13	5.74	30.00	36.87	36.87
	5.74	5.74	29.33	49.02	8.13
	12.92	18.43	41.55	30.00	22.79
	5.74	9.97	27.27	39.23	35.67
	8.52(a)	10.08(a)	28.06(b)	42.07(c)	27.92(b)

different, but are different otherwise (Tukey's test). Thus, eight tests using an association of yeast species different from that employed by Belo (1982) confirmed the previous finding: males and females did not differ in their preferences for the yeast species; also the "most attractive" species of yeast (Saccharomyces kluyveri, S.chevalieri and Torulospora delbrueckii) were more sought for by the flies than the "intermediate" ones (Pichla membranefasciens and Hansenula anomala). On the other hand, among the "most attractive" yeasts, S.kluyveri was more attractive to the flies than S.chevalieri and T.delbrueckii.

References: Belo, M. & P.M.Lacava 1980, DIS 55:146-147; Belo, M. & P.M.Lacava 1982, Naturalia (UNESP-Sao Paulo, Brasil) 7:35-45; Belo, M. 1982, Free-Docent Thesis (UNESP-Campus de Jaboticabal, SP, Brasil). [Work supported by CNPq-PIG IV]

Berry, T. and M.Snyder. University of Oregon, Eugene. Treatment for bacterial contamination.

Persistent bacterial contamination, presumably by Achromobacter, is a common problem. It is characterized by a brown discoloration of the medium along with a decreased yield. Hendrix & Ehrlich (DIS 40:99) have found a mixture of certain antibiotics added directly to the medium when it is made to be an effective measure against it.

We have modified, and, it appears, improved the effectiveness of their procedure somewhat by applying the antibiotics directly with an atomizer in addition to adding it to the medium. We make the antibiotic solutions as follows: (1) streptomycin solution: 13.8 grams of dihydrostreptomycin sulfate in 100 ml of water, and (2) penicillin/tetracycline solution: 4.25 grams of penicillin G potassium and 2.00 grams of tetracycline hydrochloride both in 400 ml of 95% ethanol.

Add three parts of (1) to one part of (2) in an atomizer. Mist each container with one spray immediately after the adults have been removed after 2-3 days of egg-laying. Development time may be delayed about two days at 25C, but the yield does not appear to be appreciably decreased.

Bhakta, R.K. and A.S.Mukherjee. University of Calcutta, India. Genetics and epigenetics of some sex-linked lethals in Drosophila melanogaster.

Twelve different sex-linked lethal mutations were recovered in Drosophila melanogaster. The $y^2w^ict^6f$ males were treated with X-ray. These males were crossed with yf:=/w^+.Y virgin females. The F_1 males were pair-mated with FM6/w^{rJ1} virgin females. If a lethal mutation was induced within the map-span of w^{rJ1} deficiency (3A2-3C2), females with $y^2w^ict^6f$/Df(1)w^{rJ1} genotype (yellow, white-ivory ♀) would not appear among the F_2 progeny, and the lethal mutation would be recovered from other F_2 females with y^2w^i 1 ct^6f/FM6 genotype (Fig. 1). The lethals thus isolated were tested with Df(1)w^{rJ2} and Df(1)62g18. The test revealed that the region 3A2-4 contains three lethals (viz., 1^{11},1^{15} and 1^{16}) and the region 3A5-8 contains other nine lethals (viz., 1^1 1^4, 1^7, 1^8, 1^9, 1^{13}, 1^{17}, 1^{18} and 1^{19}). Three complementation groups have been resolved by complementation tests (Fig. 2). They are: 1^1 group with seven lethals, 1^8 group with two lethals and 1^{11} group with three lethals.

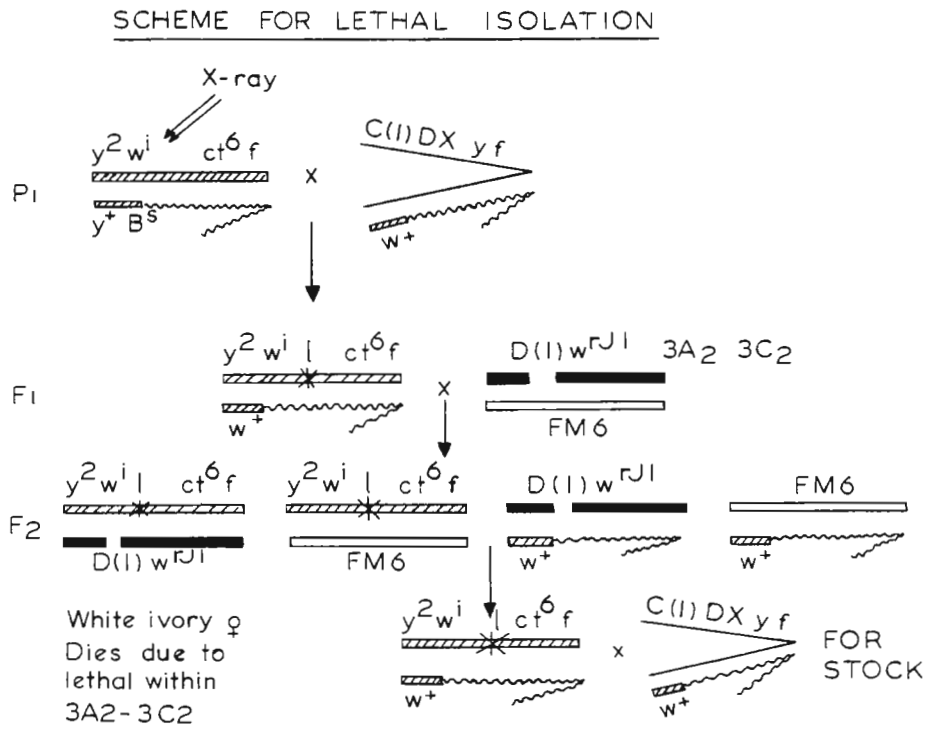


Fig. 1. Scheme for isolation of lethals in the X-chromosome against the deficiency w^{rJ1} spanning from 3A2-3C2.

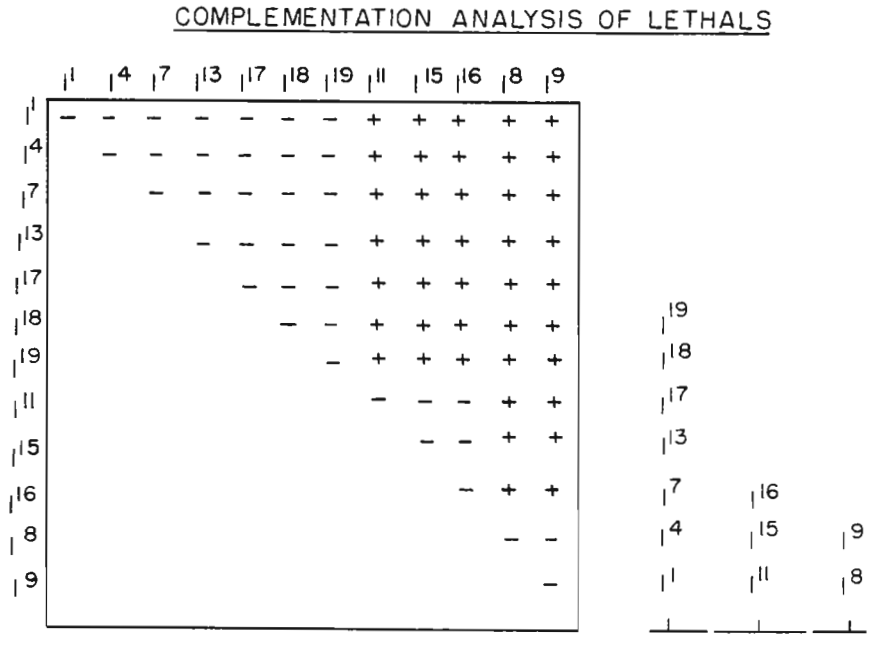
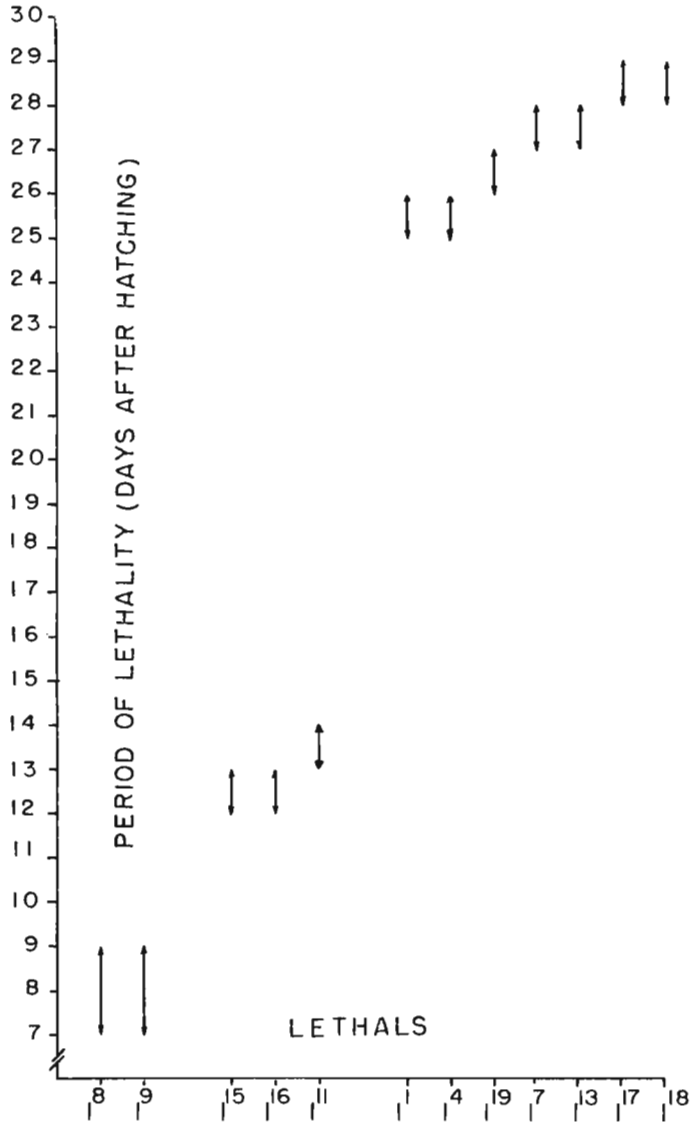


Fig. 2. Complementation test of the lethals to divide them into three groups (viz. 1^1, 1^{11} and 1^8).



To find out the time of lethal action and for the epigenetic characterization, the lethals were allowed to develop without cover. It was observed that they all die in larval condition, at different but specific age of their larval life. When the lethals were grouped on the basis of their duration of larval time, they could be divided into three groups which correspond with the respective complementation groups. the 1^{8} group survived up to 7-9 days after hatching, the 1^{11} group died within 12-16 days after hatching, but the 1^{1} group showed a very sharp deviation from others by their survival time up to 25-29 days after hatching (Fig. 3). The length and weight of the uncovered lethal larvae were measured for each lethal and from this study different lethals in different groups could be again divided into three groups as obtained from complementation study.

These studies suggest a clear correlation between the complementation group and the epigenetic integrity of the lethal mutants.

Fig. 3. Epigenetic study of the lethals by finding the period of lethality in uncovered condition.

Bock, I.R. La Trobe University, Melbourne, Australia. A matter of priority.

Wheeler (1981 The Genetics and Biology of Drosophila, Vol 3A) proposed a number of new synonymies; among them, D.hydeiodes was given as a synonym of D.nigrohydei.

The following statement is to be found on page 62 of GBD vol. 3b: "[Wheeler] does not recognize hydeoides and nigrohydei as distinct species. Since hydeoides has page priority in the publication in which both species were originally described, it, rather than nigrohydei, will be considered the valid name."

As the ignorance of ordinary taxonomic procedure revealed by this statement may not be confined to its author, it may be worthwhile to elaborate on the point concerned.

The International Code of Zoological Nomenclature governs the formation and emendation of all subspecific, specific, generic and family names within the animal kingdom. "The object of the Code is to promote stability and universality in the scientific names of animals, ..." (Code, Preamble), and adherence to the provisions of the various Articles ensures these simple objectives.

Nobody would dispute that synonymies are an inconvenience if not a curse, but given the level of activity on Drosophila species in many parts of the world, they cannot easily be avoided. In most cases, nomenclatural priority is simply established: the names

concerned have been published at different times and (with minor exceptions!--Code, Article 23) the "oldest available name" is the valid name of the taxon.

The situation is slightly more complicated where a single taxon has been described under two (or more) names in the same paper--or even in different papers within the same issue of a publication. The names in question are then "published simultaneously," and "their relative priority is determined by the action of the first reviser" (Code, Article 24). The name selected for conservation MAY be that with precedence of position in the work in which the species was described; but it need not be, and indeed the "first reviser" is advised (Code, Recommendation 24A) to select the name occurring later in the work if [for example] the species is already better-known by that name. As with all other provisions of the Code, there is no ambiguity; once the "first reviser" (Wheeler in the example cited above) has selected the name to be conserved and cited the other(s) as synonym(s), the matter is finalized.

Most significantly, there is no such thing as "page priority."

Stability in nomenclature will continue to be hindered as long as people merely follow their instincts or fancies in making taxonomic pronouncements. No more than simple adherence to the published rules of taxonomic procedure, on the other hand, is required for a universally stable system. Workers not wishing to bother themselves with details of the Articles of the Code can easily refer cases in doubt to a taxonomic specialist.

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Viability of Drosophila melanogaster reared on 'natural' food.

Though little is known about the natural breeding sites of Drosophila melanogaster, it is thought to breed mainly on rotting and fermenting fruit. It might therefore be desirable in certain experiments to use these natural food sources instead of artificial laboratory food media. This study was undertaken to measure the egg-to-adult survival of D.melanogaster reared on several kinds of natural food and in different conditions.

For the experiments females were allowed to lay eggs on 2% agar gels for 14 h after which the eggs were collected and cultured in the food vials, 100 eggs per vial. The experiment was performed on 5 different kinds of natural food: orange and grape (high sugar content), banana and potato (high starch content) and coconut (high fat content). The food was given to the flies in two ways: either big lumps of food were put into the vials (denoted as "pure food") or the food was ground in a blender and 400 grams were mixed with one liter agar solution (1.25% w/v) and this mixture was put into the vials (denoted as "agar-mixed food"). The latter procedure was tried because it allows easier handling of the food in experiments. Because fermentation may play an important role in nature the experiment was both performed with fresh food and with food that was seeded with some live bakers

Table 1. Mean egg-to-adult survival (%) on the different food media for both the "pure food" (A) and the "agar-mixed food" (B) experiment. Means are arranged according to decreasing survival. (All means sharing the same line are not significantly different at the 5% probability level (Tukey's test for multiple comparisons.)

A: "pure food"

orange +yeast	banana +yeast	coconut -yeast	orange -yeast	banana -yeast	coconut +yeast	grape +yeast	grape -yeast	potato +yeast	potato -yeast
91.2	69.9	66.1	49.6	44.9	21.4	3.2	0.7	no survival	no survival

B: "agar-mixed food"

banana -yeast	orange -yeast	orange +yeast	coconut -yeast	banana +yeast	potato -yeast	potato +yeast	coconut +yeast	grape -yeast	grape +yeast
71.7	55.6	45.6	22.1	20.0	14.7	7.7	3.3	2.0	1.4

Table 2. Analysis of variance of the data (after angular transformation) of the "pure food" (A) and "agar-mixed food" (B) viability tests. (*P < 0.05; **P < 0.001).
a=(Because there was no survival on potato food, this medium was not included in the analysis of variance.)

A				B			
Source	D.F.	Mean square	F value	Source	D.F.	Mean square	F value
Kind of food[a]	3	4986.8	80.3**	Kind of food	4	2265.2	62.6**
Fermentation/ no fermentation	1	284.2	4.6*	Fermentation/ no fermentation	1	1951.0	53.9**
Interaction	3	1357.5	21.9**	Interaction	4	764.4	21.1**
Error	32	62.1		Error	40	36.2	

yeast and kept fermenting at 25°C for three days before it was put into the vials. Each combination was replicated five times.

The results are shown in Tables 1 and 2. There are significant differences in survival between the different food sources: three of them, orange, banana and coconut, show a reasonably good survival, whereas the other two, grape and potato, do not. A population cage experiment further showed that potato's are a very poor food source for D.melanogaster as very low densities were obtained on this medium. The low survival on grapes was very surprising because it is well known that D.melanogaster is found in high densitites in vineyards where they breed on fermenting and rotting grapes. A possible explanation may be that this particular bunch of grapes, bought in a local grocery, was contaminated with a pesticide or a preservative which decreases larval survival. An additional experiment with another bunch of grapes showed a good survival of 50-70%.

A comparison between the "pure food" and the "agar-mixed food" vials shows that on the average the "pure food" vials gave higher survival rates ($F_{1,78}=4.94$, $P<0.05$). This may be explained by the fact that in the "agar-mixed food" vials the food source is diluted by the agar solution, making it harder for the larvae to gather enough food. Table 2 shows that fermentation had also a significant effect on viability. In the "pure food" vials fermentation gave rise to a higher average survival rate; this effect was reversed, however, in the "agar-mixed food" vials. The cause of this difference is unknown at the moment. Coconut seems to be an exception by showing a strong, three-fold, decrease in viability both in the "pure food" and the "agar-mixed food" vials, and this medium is responsible for the greater part of the significant interaction between the kind of food and fermentation/no fermentation, shown in Table 2. Coconut meat is well known for its high fat content and 6-8% of this total fat is octanoic acid. Leber-Bussching and Bijlsma (1983) showed that an interaction between octanoic acid and micro-organisms can cause a food situation that is lethal for D.melanogaster adults. It might well be that a similar situation arises when the coconut meat is left fermenting for three days, and that the decrease in viability is caused by this effect.

The results presented in this paper indicate that on the average the highest viability is obtained when D.melanogaster larvae are reared on pure fruit that has been kept fermenting for a few days, but one has to be aware of special circumstances and problems that can occur as found for coconut and grapes.

Reference: Leber-Bussching, M. & R.Bijlsma 1983, DIS 59:74-75.

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Locomotor activity and fitness of α-Gpdh and Adh genotypes in Drosophila melanogaster.

The functional significance of allozyme variants is still a matter of dispute: how much of this variation is selectively neutral?

For the α-Glycerophosphate dehydrogenase-1 (α-Gpdh-1 locus in D.melanogaster) a positive relation between biochemical activity and ability to fly was established: null mutants are unable to fly (1). Marked genotype-alcohol interaction is proved for alcohol-dehydrogenase (Adh) variants (2). We investigated the problem: can environmental alterations change the fitness relations of α-Gpdh and Adh variants? In this respect we studied the influence of temperature on "locomotor activity" and "mating success."

Drosophila stocks: "Groningen", four lines homozygous for Adh (first letter) and α-Gpdh (second letter) alleles: FF, FS, SF and SS. "Curacao" two lines homozygous for Adh alleles: F and S (both α-Gpdh F). "Haren", Adh like "Curacao", variable for α-Gpdh.

The flies tested were 4-5 days old and had been cultured at low density at 25°C on a sucrose-yeast-agar medium. They were kept two days at experimental temperature and hereafter tested (60 ♂♂ or ♀♀) in a "race-track" (Figure 1) for three minutes (4 replicates).

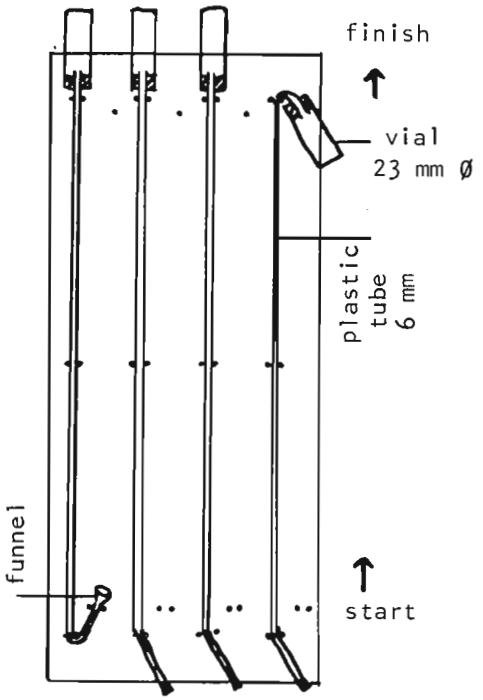

FIGURE 1.

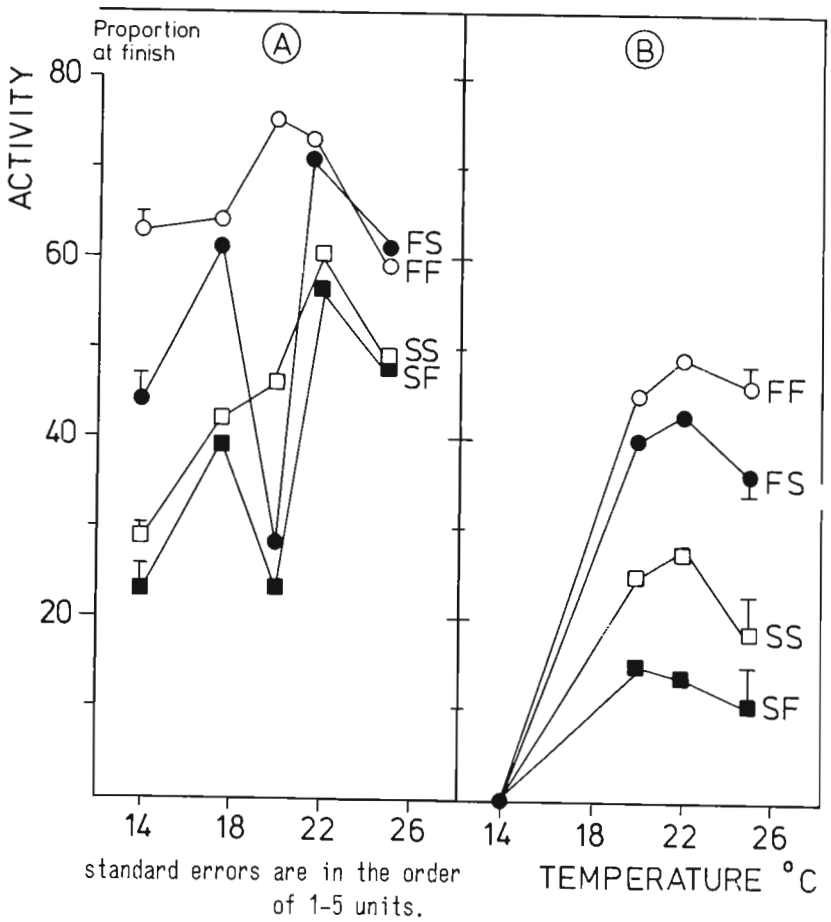

FIGURE 2. →

standard errors are in the order of 1-5 units.

Experiments. 1. Locomotor activity of the "Groningen" lines. The experiment was designed to investigate differences in locomotor activity between the four "Groningen" lines.

Figure 2 shows activity for ♂♂ (percentages, transformed to angles) at five temperatures in two independent experiments: A and B. The FF and FS lines are the most active genotypes in both experiments and at all but one (20°C, exp. A) temperature. The same holds for females but their activity is lower than in males, e.g., at 22°C: FF 32.3±1.0; FS 31.4±3.6 SF 13.8±1.9; SS 15.0±2.5 (compare with B).

So, from the "Groningen" population the two lines homozygous for the Adh-F allele are the most active lines.

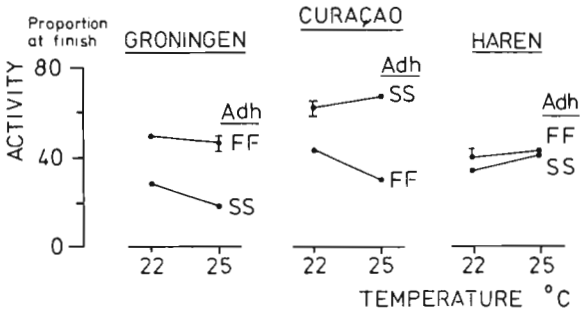

FIGURE 3.

Table 1. Number of females with progeny/male.

Temperature	Genotype			
	FF	FS	SF	SS
18°	1.0	1.5	1.4	1.4
20°	1.7	2.1	2.2	2.5
25°	3.2	3.4	3.4	3.4

(all means sharing the same line are not sign. different; x^2-tests; $P<0.025$).

2. Locomotor activity in other populations. To discriminate between effects of the Adh-gene and other genes associated with the Adh-locus, we measured activity of two other Adh strains with different geographical origin in the "race-track". Figure 3 shows a comparison of the three populations at two temperatures: In "Groningen": activity of FF exceeds activity of SS; in "Curacao" activity of FF exceeds SS; in "Haren" activity of FF equals activity of SS. The conclusion is justified that genes other than the Adh gene are responsible for the differences in locomotor activity.

3. Mating success in the "Groningen" lines. To examine the relation between activity and fitness, mating success of males (from experiment 1B) was measured.

Single males were presented (8 hours) to four females, one from each genotype. In Table 1 the number of fertilized females/male is shown for each of three temperatures. The most active genotype FF fertilized a lower number of females than the slow genotypes at all temperatures (sign. at 18° and 20°C). Since the number of offspring/♂/♀ with progeny was not significantly different for genotypes and temperatures, we can conclude that in this case activity is correlated negatively with mating success.

Discussion: Differences in locomotor activity were observed between allozyme genotypes in Drosophila melanogaster.

Activity of the Adh-F genotypes in the "Groningen" strain was higher than the activity of the Adh-S genotypes. There was no relation between activity and α-Gpdh-1 alleles (Figure 2). In a comparison of Adh variants from three geographically different strains, the results showed that not the Adh gene itself, but other "factors", associated with the specific Adh variants, must be responsible for the differences in activity between the strains (Figure 3).

To examine the relation between locomotor activity and "fitness", mating success of males was studied.

The number of females fertilized by the most active genotype FF was significantly lower than the numbers fertilized by the other genotypes at 18°C; at 20°C the FF and FS genotypes were both less successful than the two Adh-S genotypes; at 25°C there was no difference between the genotypes. So, at low temperature, it seems justified to state that: "slow and steady wins the race."

The Adh and α-Gpdh loci are localized on chromosome 2. It is known that genes for a number of behavioral characters--like locomotor activity, genotaxis and phototaxis--are localized on the X-chromosome of species of Drosophila (Table II, ref (3)).

Additional evidence for locomotor activity genes on the X-chromosome of D.melanogaster is presented in reference (4).

Factors which determine the differences in activity between the four "Groningen" genotypes must be genes different from these genes on the X-chromosome. In the "Groningen" population high fitness (mating success of ♂♂) was not combined with high locomotor activity ("FF" and "FS" genotypes) and high or low activity was not a pleiotropic effect of the Adh or α-Gpdh-1 alleles. It can also be said that the genes influencing flying ability (ref. 1) are different from the genes determining our vertical locomotor activity since the last are not related to the α-Gpdh-1 locus.

References: (1) O'Brien, S. & Y.Shimada 1974, J.Cell Biol. 63:864-882; (2) Bijlsma-Meeles, E. & W.vanDelden 1974, Nature 247:369-371; (3) van Dijken, F., M.P.Y.W.vanSambeek & W.Scharloo 1979, Behavior Genetics 9:563-570; (4) Wilson, R., B.Burnet, L.Eastwood & K.Connolly 1976, Genetical Research 28;75-88.

Botella, L.M. and J.L.Mensua. University of Valencia, Espana. Determination of the urea and uric acid content in D.melanogaster bred in non-crowded conditions.

In the course of the studies about larval development in crowded cultures of D.melanogaster, larval stop was detected by Mensua & Moya (1983), as a phenomenon which takes place at third instar of larval development. Further studies showed that urea (Botella et al. 1983a), as well as uric acid (Botella et al. 1983b) might account for the results usually observed in competition cultures. Both products have been shown to be present in D.melanogaster culture media. In order to investigate the relative quantities of these products in the bodies of stopped larvae, we have first studied the level of both products from 3rd instar on in larvae bred in non-crowded conditions. The method employed was as follows: seventy newly hatched larvae were seeded in 5 ml. of Lewis' medium. The culture was kept at 18.5±1°C in a thermoregulated room, at 70% relative humidity. At different days from the seeding day, larvae or pupae were extracted, washed in distilled water, dried on filter paper and weighed. Afterwards groups of ten larvae or pupae or adults (depending on the day of culture) were homogenized in 95 µl of Sodium Acetate 0.1 M, the homogenates were centrifuged at 4000 rpm for 5 minutes in a Beckman centrifuge and the supernatants recovered were subjected to quantitative analysis for determination of urea and uric acid contents following the method proposed by Lemar & Bootzin (1957) for urea and by Collins et al. (1959) for uric acid. A total of five replicas were made. The results obtained are shown in Table 1. As can be seen the content of uric acid in larvae of third instar decreases before pupation because in one

Table 1. Urea and uric acid levels at different stages of D.melanogaster development.

Days from the seeding day	Mean weight (mgrm./per individual)	Estimation of urea concentration* (mgrm./100ml./ mgrm.fresh weight)	Estimation of uric acid concentration* (mgrm./100ml./ mgrm.fresh weight
12 (3rd instar larvae)	1.70 ± 0.05	1.0 ± 0.1	5.3 ± 0.3
14 (1 day-old pupae)	1.40 ± 0.05	0.3 ± 0.1	3.7 ± 0.4
20 (7 day-old pupae)	1.60 ± 0.05	1.7 ± 0.5	4.3 ± 0.3
23 (excretion)	-	1.2 ± 0.7	17.0 ± 2.0
24 (adult)	1.00 ± 0.03	3.5 ± 0.7	17.0 ± 1.0

* These data were obtained with the following expression:

$$\text{Concentration} = \frac{(\text{Absorbance of each sample/standard absorbance}) \times \text{Standard concentration}}{\text{Mean fresh weight}}$$

The estimated concentrations will result from multiplying each result by the dilution factor. If we consider that each larva has an inner content about 1 microliter, then this factor would be approximately 1/10.

day old pupae the content is greatly diminished(5.3 in 3rd instar and 3.7 in one day old pupae). The figures which appear in Table 1 have been multiplied by a dilution factor (about 10) in order to obtain the quantitative values (see footnote in Table 1). During pupal stage a progressive accumulation of uric acid must occur owing to the fact that pupae cannot excrete into the media (uric acid content increases from 3.7 in one day old pupae to 4.3 in seven day-old pupae). For the last two phases analysis, mature pupae (ten day old) were incubated at 25±1°C for 20 hours allowing in this way the total emergence of all the adults. The analysis of the first excretion made by the recently emerged flies shows a high level of uric acid as can be expected after the completion of pupal stage in which external excretion does not occur. In the adult between 3 hours and 20 hours old, the concentration of uric acid is also high, which may be the result of a faster metabolic rate in the adult stage and at 25°C.

The urea content is kept at low but detectable levels throughout development, as a result we must admit that uricase acts in D.melanogaster breaking uric acid into urea.

References: Botella, L.M., A.Moya & J.L.Mensua 1983a, DIS 59:23-24; Botella, L.M., C.Gonzalez & J.L.Mensua 1983b, EDRC, Cambridge; Collins, P.F., H.Diehl & G.F.Smith 1959, Analytical Chemistry 31:1862-1867; Lemar, R.L. & D.Bootzin 1957, Analytical Chemistry 29:1233-1234; Mensua, J.L. & A.Moya 1983, Heredity 51:347-352.

Botella, L.M., A.Moya and J.L.Mensua. University of Valencia, Espana. Effect of butyrate on the development of D.melanogaster.

In the course of larval competition studies, larval stop in development was detected by Mensua & Moya (1983) by means of the overfeeding technique (Moya & Mensua 1983). In an attempt to find out the possible origin of this stop produced in crowded cultures, some natural waste products were assayed for their ability to reproduce the larval arrest in non-competitive conditions. Urea was first shown to delay larval development (Botella et al. 1983a), and this result was also confirmed for uric acid (main waste product of the Nitrogen metabolism in Insects).

Moreover both urea and uric acid were shown to be able to mimic the larval stop detected in overcrowded conditions (Botella et al. 1983b). Following the series of experiments with products which might reasonably reproduce the above results, to go more deeply into the mechanism of larval stop, Sodium Butyrate was assayed. The effect of Sodium Butyrate was assayed by adding this product in different concentrations (25 mM, 50 mM, 100 mM and 200 mM to Lewis' medium). Seventy larvae of an isogenic Oregon-R strain were seeded in 5 ml. of Lewis' medium (non-crowded cultures). The temperature was kept at 25±1°C. A total of five replicae were made at each dose, and a control of Lewis' medium without Sodium Butyrate was made. Table 1. shows the effects of Sodium Butyrate on survival and development time. As can be seen, there is an increase in development time with the Butyrate concentrations and survival decreases greatly from 0 to 200 mM.

Table 1. Effect of Sodium Butyrate over Mean Survival (S) and Mean Development Time (MDT).

Dose	S	MDT
0 (control)	56.6±2.7	13.51±0.14
25 mM	51.4±1.5	12.50±0.15
50 mM	34.2±4.3	14.75±0.49
100 mM	28.2±2.7	14.34±0.13
200 mM	6.6±1.4	17.21±0.28

Table 2. Mean survival (S) and Mean Development Time (MDT) in inner and outer population throughout overfeedings in crowded conditions (control) and for non-competitive media supplemented with 50 mM and 100 mM of Sodium Butyrate.

Overfeedings	S			MDT					
				Control		50 mM		100 mM	
	Control	50 mM	100 mM	Inner	Outer*	Inner	Outer**	Inner	Outer***
Control 5 ml.	61.8±0.8	54.0±1.4	31.4±2.9	-	13.7±0.1	-	15.4±0.1	-	18.2±0.1
8	61.0±2.2	45.0±5.1	39.8±3.6	14.5±0.5	16.5±0.2	15.2±0.2	15.3±0.1	-	17.9±0.1
10	50.8±4.8	52.8±2.5	27.2±1.0	14.4±0.2	18.4±0.1	14.7±0.1	16.3±0.2	-	18.7±0.1
12	54.5±2.7	51.6±0.4	35.4±1.6	15.2±0.1	20.5±0.1	15.9±0.1	18.1±0.1	16.8±0.3	19.2±0.1
14	36.2±4.2	52.2±1.9	37.6±1.1	15.6±0.5	22.4±0.3	15.7±0.1	21.0±1.0	18.1±0.1	20.9±0.1
16	34.2±2.0	55.6±2.3	29.6±1.1	14.1±0.1	25.0	16.3±0.1	-	18.8±0.1	23.1±0.9
0.5ml Control	19.8±2.2	-	-	17.3±0.2	-	-	-	-	-

* 1=7.97; b=1.05; R^2=0.998. ** a=7.31; b=0.94; R^2=0.970. *** a=12.42; b=0.63; $R^2$0.960.

Once the delayer effect of Butyrate had been shown, the following step was to compare the results of overfeeding experiments in crowded media (70 larvae in 0.5 ml. of Lewis' medium) with those of non-crowded (70 larvae in 5 ml. of Lewis' medium) supplemented with Sodium Butyrate. Only two doses of Butyrate were chosen for this kind of experiment: 50 mM and 100 mM which are those doses judged most suitable for the effect being sought. A total of five replicae were made. Table 2 shows the overfeeding in crowded media which served as control for the overfeeding in non-crowded media supplemented with 50 mM and 100 mM of Sodium Butyrate. The times of overfeedings were 8th, 10th, 12th, 14th and 16th day from the seeding day. In Table 2 larval stop is evident from the regression analysis. As regards total survival, the 50 mM concentration shows better survival than the crowded cultures, the opposite being true for the 100 mM concentrations. The regression of outer mean development over overfeedings shows larval stop in both concentrations, 50 mM and 100 mM, though the development at 50 mM is closer than 100 mM to crowded conditions.

Altogether the results reveal that Sodium Butyrate mimics quite accurately the result obtained in crowded cultures with respect to larval stop, delayed development and survival. Butyrate is known to inhibit cellular deacetylases of histones leading to an active state of chromatin (Weisbrod 1982). Thus, in one way or another the phenomenon of larval stop must be related to the regulation of gene expression, probably in relation to the genes responsbile for Juvenile hormone and Ecdysone production which are controlling all the development.

References: Botella, L.M., A.Moya & J.L.Mensua 1983a, DIS 59:23-24; Botella, L.M., C. Gonzalez & J.L.Mensua 1983b, EDRC, Cambridge; Mensua, J.L. & A.Moya 1983, Heredity 51:347-352; Moya, A. & J.L.Mensua 1983, DIS 59:90-91; Weisbrod, D. 1982, Nature 297:289.

Bouletreau, M., P.Fouillet, E.Wajnberg and G.Prevost. University of Lyon, France.

A parasitic wasp changes genetic equilibrium in D.melanogaster experimental populations.

Parasitism has long been suspected to be involved in genetic equilibrium and polymorphism of natural populations (Day 1974; Clarke 1979; Price 1980). However the lack of experimental evidence, at least for animal populations, makes this hypothesis rather speculative.

We compared the evolution of the allelic frequency at the sepia locus in experimental populations of D.melanogaster either free of parasites, or constantly kept under parasitic pressure by the larval endoparasite Leptopilina boulardi (Nørdlander 1980).

The cage populations were of the overlapping generations type, with a weekly introduction of four cups each containing 25 gm of fresh yeast medium (David & Clavel 1965), and a turnover based on a two weeks periodicity. Wild Drosophila and parasite strains originated from Tunisia. The mutant stock sepia has been kept under laboratory conditions for many generations.

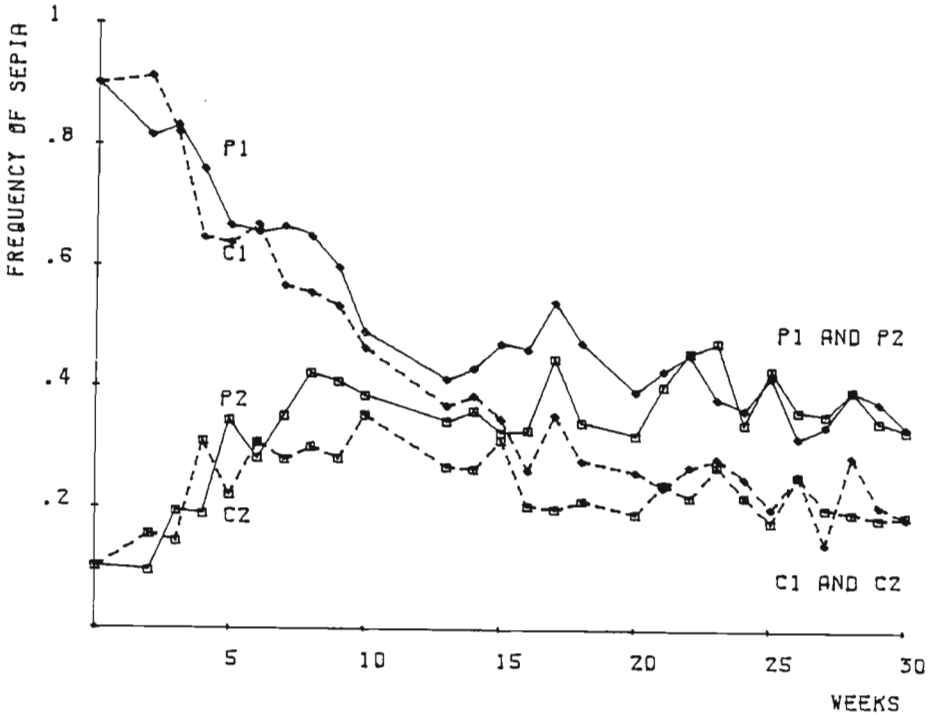

Fig. 1. Allelic frequencies at the sepia locus in control cages (C1 & C2) and in parasitized ones (P1 & P2).

Four cages were initiated simultaneously. The frequency of the recessive allele se was .9 in cage C1 (control) and P1 (parasitized); .1 in cages C2 and P2. Eggs were sampled weekly in each cage and after proper development, emerging flies were examined and the allelic frequency estimated by the square root of homozygous se/se frequency.

From the second week onwards, 200 Leptopilina boulardi couples were weekly introduced in cages P1 and P2. The biology of this wasp is very similar to that of its relative, L.heterotoma (=Pseudeucoila bochei). (see Van Lenteren 1976): females lay their eggs inside late 1st or early 2nd instars of D.melanogaster. Parasitized larvae grow up and pupate. At 25°C adult wasps emerge from the host's puparium on day 18 or 20 after
Since the developmental time of the parasite widely exceeds the fortnight's stay of cups in cages, no parasite could emerge inside cages and the weekly introduction of a new batch of adult parasites in cages P1 and P2 ensured a constant level of infestation allover the experiment.

Figure 1 shows the genetic evolution of the four experimental populations. Control populations C1 and C2 show a typical convergent evolution towards a .20 frequency equilibrium of the se allele, which is a classical value (Anxolabehere 1976). Parasitized cages P1 and P2 also show a convergent evolution. They reach their genetic equilibrium at the same time as control cages do, but the allelic frequency of se is much higher: .35. This striking difference obviously results from the presence of parasites in cages P1 and P2. The 1.8 fold increase in the equilibrium frequency of the less fitted allele demonstrates the possibility for a parasite, here a "parasitoid", to strongly affect the genetic makeup of the host population.

Further experiments are being carried out to clarify the underlying mechanisms. Preliminary results suggest that they are more complex than a trivial preference of the parasite for hosts of a given genotype.

References: Anxolabehere, D. 1976, Evol. 30:523-534; Clarke, B. 1979, Proc.Soc.Lond. 205:453-474; David, J. & M.F.Clavel 1965, Bull.Biol.Fr.Belg. 99:369-378; Day, P.R. 1974, Freeman San Francisco 238 pp.; Nørdlander, G. 1980, Ent.Scand. 11:428-453; Price, P.W. 1980, Princeton University Press 237 pp.; Van Lenteren, J.C. 1976, Neth.J.Zool. 26:1-83.

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Interspecific inhibition between D.melanogaster and D.simulans during oviposition process.

The purpose of this communication is to present a new oviposition behaviour observed in competition studies. One of the aspects studied in my doctoral thesis ("Competencia interespecifica entre D.melanogaster y D.simulans", unpublished) was to determine if the female fecundity of one of these species could be modified by the presence of virgin females of the other species. To achieve this, male and female virgins of both species were separately aged to 5 days. Then, groups of pairs of each species were mated and later the males were discarded. With these newly mated females, two experimental units were achieved. In experiment-1, four tests were simultaneously initiated with the following females per vial: Test-M, with 8 mated melanogaster females; Test-M(S), with 8 mated melanogaster females + 8 virgin simulans females; Test-S, with 8 mated simulans females; Test-S(M), with 8 mated simulans females + 8 virgin melanogaster females.

The females were allowed to lay eggs for 24 hours in vials filled with standard baker's yeast medium. Then, the 8 or 16 females were transferred to vials with fresh food. After 48 hours, the females were individually assessed for fertility. Any replication with dead or sterile females was discarded. The number of eggs laid throughout the period 0-24 hrs (first vial) and 24-48 hrs (second vial) were recorded and likewise, the number of pupae and adults produced. All experiments were carried out at 21.5°C and constant light. The results are shown in Table 1.

In D.melanogaster, no different fecundity was found between tests M and M(S), that is, the presence of virgin females of D.simulans did not affect the melanogaster oviposition process in a two day period. In D.simulans, however, a remarkable reduction of fecundity was apparent when virgin melanogaster females were present in the vial. This inhibition of laying makes the progeny of test S(M) 83% of the progeny obtained in test S. Undoubtedly, this inhibitory behaviour during oviposition must be originated through some effect derived from the presence of virgin females. A possible objection to this could be the different adult density of tests S and S(M) with 8 and 16 females, respectively. This was solved

Table 1. Results of experiment-I. Means and standard errors of the four tests described in the text, and comparison between M and M(S) and between S and S(M) by means of a student's "t".

		M	M(S)	P	S	S(M)	P
	Day-1	101.71± 8.92	84.66± 9.37	n.s.	81.20± 6.75	55.14±4.17	<0.001
Eggs	Day-2	49.14± 5.25	39.16± 3.11	n.s.	36.00± 4.21	31.86±6.06	n.s.
	Total	150.85±14.97	123.83±11.97	n.s.	117.20±10.14	87.00±6.81	<0.05
	Day-1	85.43± 6.77	80.00± 8.60	n.s.	61.20± 5.30	44.57±4.44	<0.05
Pupae	Day-2	45.29± 5.15	36.50± 2.67	n.s.	28.40± 4.15	26.43±5.81	n.s.
	Total	130.71±11.28	116.50± 9.61	n.s.	89.60± 8.01	71.00±5.44	n.s.
	Day-1	75.28± 7.77	73.83± 6.51	n.s.	40.00± 3.21	32.57±3.18	n.s.
Adults	Day-2	41.29± 4.52	34.50± 2.15	n.s.	24.00± 3.46	20.71±3.25	n.s.
	Total	116.57±12.15	108.33± 8.39	n.s.	64.00± 2.63	53.29±3.69	n.s.
	Replications	7	6		5	7	

Table 2. Results of experiment-II. Means and standard errors referred to the six tests described in the text, and multiple comparison between M,M(M) and M(S), and between S,S(S) and S(M) by means of the SNK-method (Sokal & Rohlf 1969).

	M	M(M)	M(S)	S	S(S)	S(M)
Eggs						
Day-1	84.11±2.68	76.25±6.22	63.13±3.45	70.00±5.90	74.71±6.44	28.25±3.75
	M(M) M	M(S)		S S(S)	S(M)	
Day-2	47.89±2.88	51.00±3.08	41.63±2.51	59.75±5.44	61.14±4.04	32.00±4.41
	M M(M)	M(S)		S S(S)	S(M)	
Total	132.00±4.64	127.25±7.35	104.75±5.15	129.75±9.16	135.85±7.53	60.25±7.29
	M M(M)	M(S)		S S(S)	S(M)	
Pupae						
Day-1	73.22±3.06	68.25±5.06	59.38±2.76	55.13±4.09	55.57±5.38	21.88±2.70
	M M(M)	M(S)		S S(S)	S(M)	
Day-2	43.22±2.71	45.63±3.46	39.75±2.70	48.50±4.53	47.43±3.56	29.00±3.32
	M(M) M	M(S)		S S(S)	S(M)	
Total	116.44±4.09	113.88±6.00	99.13±4.41	103.63±7.47	103.00±7.41	50.88±4.76
	M M(M)	M(S)		S S(S)	S(M)	
Adults						
Day-1	67.77±2.97	63.12±4.48	56.00±2.00	39.75±3.53	38.71±2.93	18.12±1.49
	M M(M)	M(S)		S S(S)	S(M)	
Day-2	40.88±2.26	42.87±3.40	36.75±2.18	34.37±3.55	33.43±2.94	23.50±2.89
	M(M) M	M(S)		S S(S)	S(M)	
Total	108.66±3.64	106.00±5.45	92.75±3.29	74.12±5.98	72.14±4.98	41.62±3.15
	M M(M)	M(S)		S S(S)	S(M)	
Replications	9	8	8	8	7	8

by means of experiment-II, carred out some weeks later with the same populations. In addition to the four tests previously described, two new tests were made: Test-M(M), with 8 mated + 8 virgin females of D.melanogaster; Test-S(S), with 8 mated + 8 virgin females of D.simulans; and therefore, with a 16-adult density per vial.

The methodology was the same as for experiment-I, and the results appear on Table 2. The mean values were compared by means of the SNK-method (Sokal & Rohlf 1969). Two means not joined by a horizontal line are different with a 95% probablity or greater.

Surprisingly enough, we can clearly observe an inhibition in the fecundity of D.melanogaster when virgin females of D.simulans are present. The productivity of test M(S) was 85% of the productivity of test M(M). The result cannot be imputed to a different adult density. Even more amazing was the response of D.simulans: the inhibition of fecundity in presence of virgin females of D.melanogaster S(M), was so large that the productivity obtained in 48 hrs only represents 56% of that obtained in absence of its sibling species, S(S). This is the first time an inhibitory behaviour during the oviposition process is described in the pair melanogaster-simulans. Some reports working with these species have demonstrated how the presence of eggs (Moore 1952; Eoff 1973) or larvae (Moth & Barker 1976) of one of these species can inhibit the normal laying of the other species. In my experiments, the virgin females do not lay eggs (ovules) and this is evident when comparing the egg-pupa viability of the different tests: when virgins are present in a test, the egg-pupa viability was the same or greater than when they are absent. Therefore, the simple presence of virgins was the factor causing an inhibitory response in oviposition. This could be related to species-specific visual or olfactory clues. Mainardi (1968) and Krause et al. (1980) have found a stimulating effect upon fecundity in D.melanogaster originated by the previous presence of males on the food which could be ascribed to a male pheromone. The different response of D.melanogaster found between experiments I and II carried out at different times, does not have a simple explanation, although it is closely related with several competitive results (inhibition-facilitation; mutual inhibition; mutual facilitation; no-interference) found in my doctoral thesis to study competition at different times.

In the present paper, the inhibitory effect of foreign females was smaller on the second day, and this suggests some type of female habituation.

A female behaviour causing such drastic decrease in the fitness of a species must have some biological meaning. The possibility exists that the female inhibition could be the result of a selective pressure acting to avoid the mixed development of both species. If the preadults of these sibling species are grown in the same food, the frequency of interspecific hybridization could be high since, first, the newly emerged adults have not developed to a full extent their sexual discriminative sense (Barker 1962; Manning 1967) and second, because the heterospecific pairing is more frequent when larvae of both species are developed in the same vial (Eoff 1973). If this supposition, under study at present time, were correct, then the above mentioned inhibitory behaviour could have an adaptative value.

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Charles-Palabost, L. & M.Lehmann.
University of Paris VII, France.

The effect of the temperature upon the variability of the gene pool in a Brasilian population of D.melanogaster.

It has been demonstrated in the following note that the genetic composition of the Porto-Alegre 1982 population has varied between September and November, probably in relation with climatic conditions. Temperature is one of the two major environmental parameters (the other is relative humidity) which have a biological significance for Drosophila (Alahiotis & Pelecanos 1980; Ayala 1968). Therefore, the effect of the temperature in this change has been examined.

The samples of September and November were kept at 18°C during eight months. Thus we are allowed to consider that September population was maintained in experimental conditions of temperature near to those of nature (21°C), while November population was maintained in experimental conditions of temperature very different of those in nature (27°C). Tables 1 and 2 give for each locus, the sample size (N), the genotypic frequencies and the X^2 values

obtained after comparison of experimental and freshly captured populations.

Table 1. Genotypic frequencies in the Porto-Alegre population freshly captured in September 1982 and in the sample maintained during eight months at 18°C.
* P < 0.05; ** P < 0.01

LOCI:	ACPH				ADH					EST-C				
Samples	N	FF	FS	X^2_1	N	FF	FS	SS	X^2_A	N	FF	FS	SS + rares	X^2_B
Sept. 82	100	0.96	0.04	5.21*	100	0.45	0.37	0.18	14.96**	100	0.90	0.08	0.02	4.27
Sept. 82 maintained at 18°C	100	0.87	0.13		100	0.60	0.38	0.02		100	0.85	0.15	-	

LOCI:	EST-6						α-GPDH					PGM				
Samples	N	FF	FS	SS	rares	X^2_3	N	FF	FS	SS	X^2_2	N	FF	FS	rares	X^2_2
Sept. 82	89	0.19	0.36	0.37	0.08	5.19	100	0.68	0.27	0.05	0.17	100	0.74	0.12	0.14	6.14*
Sept. 82 maintained at 18°C	100	0.11	0.42	0.44	0.03		99	0.66	0.28	0.06		100	0.87	0.04	0.09	

Table 2. Genotypic frequencies in the Porto-Alegre population freshly captured in November 1982 and in the sample maintained during eight months at 18°C.
* P < 0.05; ** P < 0.01.

LOCI:	ACPH				ADH					EST-C			
Samples	N	FF	FF+SS	X^2_1	N	FF	FS	SS	X^2_2	N	FF	FF+SS	X^2_1
Nov. 82	145	0.75	0.25	9.89**	143	0.41	0.35	0.24	12.26**	123	0.93	0.07	16.22**
Nov. 82 maintained at 18°C	100	0.91	0.09		100	0.46	0.47	0.07		100	0.74	0.26	

LOCI:	EST-6					α-GPDH					PGM					
Samples	N	FF	FS	SS	X^2_2	N	FF	FS	SS	X^2_2	N	FF	FS	rares (1)	rares (2)	X^2_3
Nov. 82	144	0.10	0.30	0.60	2.89	143	0.83	0.13	0.04	5.44	145	0.79	0.03	0.08	0.10	36.79**
Nov. 82 maintained at 18°C	100	0.04	0.31	0.65		100	0.72	0.25	0.03		100	0.63	0.30	0.04	0.03	

In the case of the population captured in September, the genotypic frequencies have varied highly significantly only for the Adh locus (X^2_2=14.96); the variations were more slight for Acph (X^2_1=5.21) and Pgm (X^2_2=6.14) loci. In November population, four tests were highly significant: X^2_1=9.89, X^2_2=12.26, X^2_1=16.22, X^2_3= 36.79, respectively, for Acph, Adh, Est-C, and Pgm loci.

Our data have shown that temperature has induced gene pool differentiation. Thus we can consider that seasonal fluctuations and especially temperature are responsible for the changes in the genetic composition of the Porto-Alegre population.

References: Alahiotis, S. and M.Pelecanos 1980, Genetika 12:209-217; Ayala, F.J. 1968, Science 162:1453-1459.

Charles-Palabost, L. and M.Lehmann.
University of Paris VII, France. Genic variation in a Brasilian population of Drosophila melanogaster.

The biochemical polymorphism of Drosophila melanogaster was investigated in natural populations of different geographical areas: Europa (Girard & Palabost 1976; Triantaphyllidis et al. 1980; David 1982), America (O'Brien & MacIntyre 1969; Berger 1970; Band 1975; Singh et al. 1982), Asia (Singh et al. 1982) and Africa (David 1982). Up to now, nothing was known about South American populations. Therefore we have examined a Brasilian population captured on a market at Porto-Alegre.

Table 1. Genotypic and allelic frequencies in the Porto-Alegre population.

(g) genotypes; (a) alleles; *P<0.05; **P<0.01.

Locus:		ACPH					ADH						
		(g)		(a)		X^2_1		(g)			(a)		X^2_2
Samples	N	FF	FS	F	S		N	FF	FS	SS	F	S	
Sept.	100	0.96	0.04	0.98	0.02	18.79**	100	0.45	0.37	0.18	0.63	0.37	1.18
Nov.	145	0.75	0.25	0.88	0.12		143	0.41	0.35	0.24	0.59	0.41	

Locus:		EST-C							EST-6						
		(g)			(a)			X^2_2		(g)			(a)		X^2_2
Samples	N	FF	FS	SS + rares	F	S	rares		N	FF	FS	SS	F	S	rares	
Sept.	100	0.90	0.08	0.02	0.94	0.05	0.01	1.09	89	0.27	0.36	0.37	0.37	0.58	0.05	16.04**
Nov.	123	0.93	0.06	0.01	0.96	0.04	-		144	0.10	0.30	0.60	0.25	0.75	-	

Locus:		α-GPDH						PGM								
		(g)			(a)		X^2_2			(g)				(a)			X^2_3
Samples	N	FF	FS	SS	F	S		N	FF	FS	rares (1)	rares (2)	F	S	rares	
Sept.	100	0.68	0.27	0.05	0.81	0.19	7.94*	100	0.74	0.12	0.07	0.07	0.86	0.09	0.05	7.17
Nov.	143	0.83	0.13	0.04	0.90	0.10		145	0.79	0.03	0.08	0.10	0.84	0.04	0.12	

Wild Drosophila melanogaster adults were collected in September and November 1982 and brought to the laboratory for genetic analysis. Starch gel electrophoresis was made immediately after the arrival of individuals. Six enzymatic loci were assessed: Acph (acid phosphatase; III - 101.4), Adh (alcohol dehydrogenase; II - 50.1), Est-C (esterase-C; III - 47.6), Est-6 (esterase-6; III - 36.8), α-Gpdh (α-glycerophosphate dehydrogenase; II - 20.5) and Pgm (phosphoglucomutase; III - 43.4). Table 1 gives for each locus, the sample size (N), the genotypic and allelic frequencies and the X^2 values after comparison between the two samples (September and November 1982).

The genotypic and allelic frequencies have varied between the collections of September and November, for three of the six loci studied: Acph, Est-6 and α-Gpdh. This change in the genetic pool of the population is associated with a variation in climatic conditions; in September the maximal mean temperature reaches 21°C and 27°C in November. Furthermore humidity appears to be very different between September (season of rains) and November (dry season). The relation between seasonal fluctuations and changes in the genetic composition of this population has been established experimentally (see previous Research Note).

According to the neutralist theory, migrations are responsible for the remarkable similarity in allozyme frequencies between populations of the same geographical origin. In the selectionist theory, this similarity is presented as resulting of identical selection pressures on populations which live in proximated biotops (Ayala et al. 1972a&b). In order to test these theories, results obtained for Porto-Alegre and French populations (from Girard & Palabost 1976) have been compared in Table 2.

Table 2. Allelic frequencies at six loci in French and Porto-Alegre populations. (N=sample size; *mean allelic frequencies obtained with 15 populations; for Pgm, see Singh et al. 1982).

Populations		ACPH N	ACPH F	ACPH S	ADH N	ADH F	ADH S	EST-C N	EST-C F	EST-C S	EST-C rares
French*		2688	0.99	0.01	2668	0.96	0.04	2625	0.88	0.09	0.03
Porto-Alegre	Sept.	100	0.98	0.02	100	0.63	0.37	100	0.94	0.05	0.01
	Nov.	145	0.88	0.12	143	0.59	0.41	123	0.96	0.04	-

Populations		EST-6 N	EST-6 F	EST-6 S	EST-6 rares	α-GPDH N	α-GPDH F	α-GPDH S	PGM N	PGM F	PGM S	PGM rares
French*		2640	0.27	0.71	0.02	2543	0.53	0.47	-	0.98	0.02	-
Porto-Alegre	Sept.	89	0.37	0.58	0.05	100	0.81	0.19	100	0.86	0.09	0.05
	Nov.	144	0.25	0.75	-	143	0.90	0.10	145	0.84	0.04	0.12

Despite seasonal changes in gene frequency at Porto-Alegre, allelic frequencies in the French populations and in the Brasilian population are very similar for three loci (Acph, Est-C, and Est-6). Of course, migrations of Drosophila melanogaster between France and Brasil are not possible and therefore cannot explain the homogeneity in allele frequencies observed at the Acph and esterases loci. Moreover how explain in this case the differentiation at the three other loci (Adh, α-Gpdh, Pgm)? Consequently, our results are not in agreement with neutralist theory. Another conclusion can be drawn: differentiation in allozyme frequencies are shown only for enzymes of energetic metabolism (especially Adh and α-Gpdh) which probably play in the adaptation a more important role than non-specific enzymes (Cavener & Clegg 1978).

The authors are very grateful to Drs. A. Brück and M. Napp (University of Porto-Alegre) who have sent the collections.

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Chaudhuri, G.K. and. A.S.Mukherjee.
University of Calcutta, India. Effect of α-Methyl-DL-Methionine on the replication of polytene chromosomes in Drosophila melanogaster.

The nucleoproteins of the eukaryotic cell contain acetyl, phosphoryl and methyl side chain groups which are metabolically active. It has been suggested that enzymatic control of side chain metabolism may be involved in the specific control of gene expression and in a more general way in such biochemical processes as may be involved in gene activity and cell division (Goodman & Benjamin 1972; Felsenfeld & McGhee 1982). In the present investigation possible effect of methylation using a methyl donor on DNA replication has been examined in Drosophila polytene chromosome.

In these experiments salivary glands from late third instar larvae of Drosophila melannogaster (Oregon R^+) were dissected out in buffered Drosophila Ringer (pH 6.8) and incubated for 20 mins in α-Methyl-DL-Methionine (obtained from Sigma Chemical Co., USA) at a concentration of 10^{-3}M. The control sets were incubated for the same period in Ringer. The glands were then incubated for 20 minutes in ^{3}H-thymidine (conc. 400 μCi/ml, sp. activity 12,700 mCi/m mole, obtained from Bhabha Atomic Research Centre, Trombay, Bombay, India). The squash preparations of chromosome were processed for autoradiography using Kodak AR 10 stripping film, exposure time being 24 days.

Table 1. Data on the frequency of labelled chromosomes in control and α-Methyl-DL-Methionine treated salivary glands of male and female Drosophila melanogaster.

	^{3}H-TdR Pulse time(min)	No. of nuclei	Number of labelled nuclei			Number of unlabelled nuclei
			Early patterns (DD-1C)	Mid patterns (2C-3C)	Terminal patterns (3C-3D-CL)	
CONTROL ♂	20	649	16 (2.46%)	115 (17.72%)	402 (61.93%)	116 (17.87%)
α-Methyl-DL-Methionine Treated ♂	20	894	3 (0.33%)	45 (4.99%)	554 (70.13%)	292 (22.26%)
Control ♀	20	518	29 (5.49%)	87 (16.48%)	275 (52.08%)	127 (22.16%)
α-Methyl-DL-Methionine Treated ♀	20	1065	2 (0.19%)	24 (2.27%)	735 (70.2%)	304 (28.81%)

Results of the two sets of experiments are presented in Table 1. Data revealed that the frequency of initial and mid labelling patterns (termed DD-1C and 2C-3C) is drastically decreased. In contrast, the frequency of terminal patterns (marked by discontinuous labelling on dark bands) is increased in both sexes. These results suggest that methylation does take place in this system and inhibits the initiation of replication, since it has been suggested earlier that both DD-1C and 2C-3C labelling patterns include successive stages of initiation in Drosophila polytene chromosomes (Mukherjee 1982; Mukherjee & Chatterjee 1983).

Interestingly the frequency of labelling of the 20 sites on the autosome 2R is remarkably similar in the two sexes for both control and α-Methyl-DL-Methionine treated preparations, whereas, for the 45 replicating sites in the X-chromosome, the frequencies of labelling are distinctly different in the two sexes. They are much less in the male than in the female. The sites 3C, and 7E, 11A and 12DE are exceptions. When the mean silver grain numbers on each X chromosome segments (1A-12DE) and the autosomal segment 56AB to 60F are compared in the control and treated sets and the ratios of grains X/A are analyzed, it becomes evident that the inhibition is more drastic in the male X chromosome than in the female X (Table 2).

Table 2. Summary of grain count data in control and α-Methyl-DL-Methionine treated glands.

Samples (No. of nuclei)	Mean grain no. on the segment 1A-12DE of X (±SE)	Mean grain no. on segment 56AB to 60F of 2R (±SE)	X/A ratio (±SE)
Control ♂ (32)	204.5 ± 4.72*	123 ±1.24	1.66 ± 0.06*
Treated ♂ (28)	124.14 ± 6.84	113.2 ±2.33	1.09 ± 0.03
Control ♀ (24)	215.3 ± 6.57*	121 ±0.77	1.77 ± 0.1 *
Treated ♀ (30)	140.4 ± 6.61	105.5 ±5.33	1.33 ± 0.043

* $P < 0.05$

These results suggest firstly that methylation - demethylation may indeed be involved in initiation of DNA replication and secondly, X chromosomal organization in male and female may determine a differential methylation in the two sexes. Further works are in progress to find out the amount and distribution of methylating sites on the X and autosomes.

References: Goodman, R.M. & W.B.Benjamin 1972, Chromosoma; Felsenfeld, G. & J.McGhee 1982, Nature 296:602; Mukherjee, A.S. 1982, Current Science; Mukherjee, A.S. & C.Chatterjee 1983, J.of Cell Science.

Choi, Y., Y.M.Ha & S.K.Kim. Yonsei University, Seoul, Korea. Inversions in a natural population of D.melanogaster.

Chromosomal inversion polymorphisms of D. melanogaster at the Anyang, a large vine growing area (middle region of Korea) have been examined. Samples of wild 1,235 individuals were taken in late August, respectively for each year of 1981, 1982, 1983. Nineteen different inversions were found from the natural population, distributed one in X-, five in II- and thirteen in III-chromosome. A list of various inversions is presented in Table 1. Eight of them proved to be polymorphic, cosmopolitan type, rests rare endemic. Most of them were single paracentric, except two overlapping inversions in III-R. The relative frequency of various gene arrangements is given in Table 2. A few low frequencies of endemics are presented as a group of rare endemic in each chromosome arm. Homogeneity tests between the year to year samples do not show the statistically significant differences, except two cases in III-R arms (two out of twelve comparisons). In general, the chromosomal polymorphisms of this species seem to be temporary stable for the period of 1981-1983, and approximately in equilibrium at the present population. Furthermore, we found some evidence of non-random-association of inversion for the case of 3RP-3RC, 3RP-3RMo, and 3RC-3RMo. Non-random-associations between the overall inversions of 3L-3R, and 2L-3L could be detected.

Table 1. Inversions from the Anyang natural population of D.melanogaster in Korea.

Chromosome-Arm	Symbol	Breakpoint	Type
X	In(1) a	4C;11C/D	new endemic
II-L	In(2L) t	22D;34A	common cosmopolitan
	In(2L) K	22A;26B	rare cosmopolitan
	In(2L) b	32D;3F	new endemic
	In(2L) c	23D/F;27C	new endemic
II-R	In(2R)NS	52A;56F	common cosmopolitan
III-L	In(3L) P	63C;72E	common cosmopolitan
	In(3L) M	66D;71D	rare cosmopolitan
	In(3L) d	65A;67C	new endemic
	In(3L) e	71B;80C	new endemic
	In(3L) f	64E;76A	new endemic
III-R	In(3R) P	89C;96A	common cosmopolitan
	In(3R) C	92D;100F	rare cosmopolitan
	In(3R) Mo	93D;98F	rare cosmopolitan
	In(3R) KI	88D;94A	recurrent endemic
	In(3R) g	87F;91F	new endemic
	In(3R) h	93F;97B/C	recurrent endemic
	In(3R) oa	90B/C;97C-92D;100F	new endemic overlapping
	In(3R) KL	93D;98F-89C;96F	recurrent endemic overlapping

Table 2. The relative frequencies of standard and cosmopolitan inversions from the Anyang natural population of D.melanogaster, 1981-1983.

Year	1981	1982	1983	Average	Homogeneity test
2L St	0.9675	0.9400	0.9357	0.9490	1) n.s.
t	0.0281	0.0550	0.0590	0.0462	2) n.s.
K	0.0021	0.0050	0.0027	0.0032	3) n.s.
rare	0.0021	0	0.0027	0.0016	
2R St	0.9199	0.9250	0.9008	0.9158	1) n.s.
NS	0.0801	0.0750	0.0992	0.0842	2) n.s.
					3) n.s.
3L St	0.9654	0.9575	0.9759	0.9660	1) n.s.
P	0.0260	0.0425	0.0214	0.0300	2) n.s.
M	0.0021	0	0	0.0008	3) n.s.
rare	0.0065	0	0.0027	0.0032	
3R St	0.8052	0.8050	0.8686	0.8243	1) P<0.001
P	0.1429	0.0525	0.0509	0.0858	2) P<0.001
C	0.0346	0.0900	0.0617	0.0607	3) n.s.
Mo	0.0130	0.0375	0.0107	0.0202	
rare	0.0043	0.0150	0.0080	0.0089	
N*	462	400	373	1,235	

(total number of chromosomes examined

N.S.: non-significance between: 1) 1981 & 82; 2) 1981 & 83; 3) 1982 & 83.

References: Ashburner, M. & F. Lemeunier 1976, Proc.R.Soc.Lond.B. 193:137-157; Inoue, Y. & T.K.Watanabe 1979, Jap.J.Genetics 54:69-82; Knibb, W.R., J.G.Oakeshott & J.B.Gibson 1981, Genetics 98:833-847; Mettler, L.E., R.A.Voelker & T.Mukai 1977, Genetics 87:169-176; Stalker, H.D. 1980, Genetics 95:211-223; Zacharopoulow, A. & M.Pelecanos 1980, Genetica 54:105-111.

Chung, Y.J. Ewha Woman's University, Seoul, Korea. Biochemical genetic study of Drosophila populations in Korea.

Enzyme polymorphism has been extensively investigated from various places by many authors. In order to establish a biochemical genetic system in Drosophila populations in Korea, six enzyme (ADH, α-GPDH, MDH-1, MDH-2, ACPH and ME) alleles of natural populations of D.melanogaster from 10 localities (Kangreung, Seoul-Seungsudong, Seoul-Wangsibri, Seoul-Sinchon, Seoul-Seungsandong, Jeonju, Pusan, Mokpo, Jeju-Hanrim and Jeju-Moseulpo) in Korea were analyzed by means of starch gel electrophoresis.

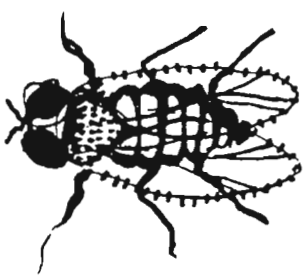
The results obtained were as follows: (1) the natural populations of D.melanogaster from 10 localities in Korea showed polymorphism as to ADH, α-GPDH, MDH-1, ACPH and ME alleles. But MDH-2 alleles were found to be monomorphic. (2) Heterozygosity was calculated to be 48.40% for α-GPDH and 28.00% for ADH allele. The rest of enzymes showed a low heterozygosity below 10%. (3) The FF genotypes of ACPH and ADH alleles were most frequently distributed through the Korean Natural populations of D.melanogaster, whereas the SS genotypes of MDH-1, MDH-2 and ME alleles were most frequently involved in all populations, and the commonest genotype of α-GPDH alleles were found to be the FS genotype. (4) The F gene frequency was found to be higher than the S gene in ACPH, ADH and α-GPDH, whereas S gene frequency was found to be higher than the F gene in MDH-1, MDH-2 and ME alleles. (5) The predominance of the FF genotype and the F gene of ADH alleles was weakened in the second year experiment compared to first year's but the genotype and gene frequencies of α-GPDH alleles showed no difference between the first and the second year experiment. However, heterozygosity revealed still high values in average (ADH:0.3048; α-GPDH:0.4645). (6) This investigation is desired to extend the other localities in Korea so that a biochemical genetic system of Drosophila populations in Korea may be established more robustly.

References: Chung, Y.J.&K.S.Lee 1972, J.Kor.Res.Inst.Bet.Liv. 9:123-132; Chung,Y.J., Y.S.Han & Y.L.Chung 1982, Kor.J.Zool. 25:123-129.

Coyne, J.A. University of Maryland, College Park, Maryland USNA.
Report of J.A. Coyne.

Burgundy (bg) is a sex-linked recessive mutant of D.mauritiana that confers a dark ruby eye color with no pseudo-pupil. The mutant, available from the Bowling Green Stock Center, is one of only two described in this species (Woodruff 1980). Because of the importance of D.mauritiana in studies of the genetics of speciation, mapping of this and other markers is essential. Burgundy females of D.mauritiana were crossed to white (w, 1-4.1) males of the sibling species D.simulans. The hybrid females are fertile and heterozygous for both markers. These were crossed to wild-type D.simulans males, and the male offpspring scored for recombination between w and bg (the two species are homosequential and the hybrid females show free recombination). Since w bg males almost certainly have white eyes, there are only three phenotypes among the backcross males; the numbers scored were 362 white, 342 burgundy, and 20 wild-type. I assumed that 20 of the white males were actually w bg. A rough estimate of recombination between the loci is thus 40/724 or 0.055±0.017. An approximate location for burgundy is thus either 1-9.6 or near 1-0, the base of the X chromosome. Crosses of burgundy females to similar eye-color mutants of D.melanogaster in this region showed that the mutant is allelic with prune (pn, 1-0.8). Burgundy should thus be regarded as identical to prune.

Reference: Woodruff, R.C. 1980, DIS 55:217.



Craymer, L. California Institute of Technology, Pasadena, California USNA. Automating saturation screens with crisscross lethals.

Large scale identification and isolation of chromosomes which carry lethals in a region specified by a deficiency can be a tedious operation. Muller's idea of using crisscross lethals in mutation studies (1953 DIS 27:104-106) can be adapted for saturation screening to simplify the scoring and balancing steps. The idea is to recover a mutagen-treated chromosome over a chromosome which carries two lethals, ℓ_1 ℓ_2, and mate to Df, ℓ_1/Bal, ℓ_2 where Df is a deficiency for the region of interest and Bal is a balancer chromosome. The lethals ensure that all of the F_2 progeny will carry the treated chromosome and that there will usually be two classes of progeny: */Df, ℓ_1 and */Bal, ℓ_2. If, however, the treated chromosome carries a lethal in the region of interest, then only the */Bal, ℓ_2 progeny will survive. Balanced stocks of induced lethals are thus automatically recovered by this procedure, and the scoring process can be simplified by having either the Df or Bal chromosome marked with a dominant visible mutation which can be recognized without etherization. The following screen has been used by M. Crosby to recover lethals in the 68A to 69A region:

P_1 In(3R)C, Sb ℓ(3)a/Ser Dr^{Mio} females are crossed to treated males

F_1 In(3R)C, Df(3L)vin^5, Tb ℓ(3)a/TM3, Sb Ser females are crossed to single */In(3R)C, Sb ℓ(3)a males

F_2 Look for cultures with no Tb adults; these cultures carry lethals in the deficiency region.

As originally envisioned, the */Ser Dr^{Mio} males were also to be tested, using Df(3L)vin^5, Dr^{Mio}/TM3 Sb Ser females for the screening cross. Unfortunately, Ser/TM3, Sb Ser is not lethal (M. Crosby, personal communication) and this half of the screen had to be abandoned. Tb and Dr^{Mio} were chosen for the screen since they could be easily scored through the sides of a vial: Tb, because it could be scored in pupal cases, and Dr^{Mio}, because it is easily scored in adults. At the time this screen was designed, no third chromosome balancer existed which carried an adequate dominant visible; TM6B with either D^3 or Tb (with a lethal) should be useful for other such screens, and Cy can be used for the dominant visible in second chromosome screens.

Craymer, L. California Insitute of Technology, Pasadena, California, USNA. A procedure for constructing isogenic stocks.

Isogenic stocks are useful in many experiments where it is critical that there be little or no background genetic variation. Muller's triple balancer scheme (1936 DIS 6:7) is commonly used, but this method has drawbacks. Females which are simultaneously heterozygous for efficient first-, second-, and third-chromosomal balancers have seriously reduced fertility, and the individual balancer chromosomes have reduced efficiencies because of the interchromosomal effect of heterologous rearrangements on recombination. The following scheme circumvents these problems, although it does require two generations more than the triple-balancer scheme.

P_0 C(1)M4, y^2; If; Sb/TM6B, h D^3 e females x + male.

P_1 C(1)M4, y^2; If; Sb/TM6B, h D^3 e and
FM3, v^{0f}/y sc $l.z^9$ v f; Sp bw^D/SM5 females
x +; +/If; +/Sb male [1 male to 3 or 4 of each type of female]

P_{2a} C(1)M4, y^2; If; Sb/TM6B, h D^3 e females
x +; +/If; +/TM6B, h D^3 e males

P_{3a} Like P_{2a}

P_{3b} +/+; +/+; Sb/TM6B, h D^3 e females x +; +/If; +/TM6B, h D^3 e males

P_4 +; +; TM6B, h D^3 e/+ females x males

P_5 +; +; + isogenic stock.

Some comments are in order concerning the balancers listed. FM3, SM5, and TM6B are the most efficient balancers available for their respective chromosomes. FM3/+; SM5/+; TM6B/+ females could be used in a triple-balancer isolation--perhaps 2-5% of the offspring would carry a recombinant chromosome, with an average of about 2% of the genome substituted per recombinant (these figures are educated guesses based on some observations of multi-chromosomal balancer combinations)--although some problems might arise from the reduced fertility and viability of individual females. In the suggested scheme, with double-balancer heterozygotes used in one generation and single-balancer heterozygotes used in a later generation, it is unlikely that as many as 0.5% of the offpspring of the double-balancer cross would be recombinant for the balanced chromosomes and very unlikely that recombinant third chromosomes be recovered from the crosses involving TM6B/+ females.

Chromosome substitutions can also be carried out without going through a triple-balancer intermediate. For X-substitutions, the scheme is

P_0 $+_a$; $+_a$; $+_a$ females x SM6/In(2LR)bw^{V1}; TM6B, h D^3 e/In(3R)Mo, Sb sr males

P_{1a} $+_a$; $+_a$; $+_a$ females x $+_a$; SM6/+; TM6B, h D^3 e/+ males

P_{1b} $+_a$; $+_a$; $+_a$ females x $+_a$; In(2LR)bw^{V1}/+; In(3R)Mo, Sb sr/+ males

P_2 $+_a$; In(2LR)bw^{V1}/+; In(3R)Mo, Sb sr/+ females [from P_{1b}] x $+_b$; $+_b$; $+_b$ males

P_3 $+_a$; SM6/+; TM6B, h D^3 e/+ females [from P_{1a}]
x $+_a$; In(2LR)bw^{V1}/$+_b$; In(3R)Mo, Sb sr/$+_b$ males

P_4 $+_a$; SM6/$+_b$; TM6B, h D^3 e/$+_b$ females x males

P_5 $+_a$; $+_b$; $+_b$ stock

This series of crosses thus substitutes the $+_a$ X-chromosome into the $+_b$ stock. SM5 would be preferable to SM6 in this series of crosses, since SM5 has the better balancing properties; however, SM6 is listed because the SM6/In(2LR)bw^{V1}; TM6B, h D^3 e/In(3R)Mo, Sb sr stock currently exists.

To substitute a $+_b$ third chromosome in to a $+_a$ background:

P_0 $+_a$; $+_a$; $+_a$ females x SM6/In(2LR)bw^{V1}; TM6B, h D^3 e/In(3R)Mo, Sb sr males

P_{1a} $+_a$; $+_a$; $+_a$ females x $+_a$; In(2LR)bw^{V1}/$+_a$; TM6B, h D^3 e/+

P_{1b} $+_a$; $+_a$; $+_a$ females x $+_a$; SM6/+; In(3R)Mo, Sb sr/+ males

P_2 $+_a$; SM6/+; + females [from P_{1b}] x $+_b$; $+_b$; $+_b$ males

P_3 $+_a$; $+_a$; TM6B, h D^3 e/+ females [from P_{1a}] x $+_a$; SM6/+; In(3R)Mo, Sb sr/$+_b$ males

P_4 $+_a$; SM6B/$+_a$; TM6B, h D^3 e/$+_b$ females x males

P_5 $+_a$; $+_a$; $+_b$ stock

The series of crosses for substituting a second chromosomes is rather similar to the third chromosome substitution crosses.

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Synthesis of a ring third chromosome and its use in inserting markers into In(3R)C.

Despite the early successes of Muller (1918 Genetics 3:422-499) and Sturtevant (Carnegie Inst.Wash.Publ. 421:1-27), it is difficult to insert markers into In(3R)C through double crossovers even with the aid of the interchromosomal effect of heterologous rearrangements. After scoring on the order of 100,000 flies in unsuccessful attempts to insert Tubby into In(3R)C with the aid of C(1)M3 and CyO, I decided to abandon the direct approach and look for a more practical method. I have now accomplished this marker insertion with the aid of a ring third chromosome.

The Upper portion of Figure 1 shows the synthesis of the ring, R(3C)S1, from In(3LR)P88+(3R)C and a structurally normal chromosome. In(3LR)P88+(3R)C/+ females are mated to LS(3)P88/DS(3)P88 males (see Genetics 99:75-97 for a description of the LS and DS notation) to recover the LS(3)P88/DS(3)P88, IN(3R)C--+ constellation. R(3)S1 is generated by a crossover in the 92E-100F regions of the DS(3)P88, In(3R)C--+ chromosome; the ring is duplicated for 89C to 92D and deficient for terminal chromatin in 61A and 100F. The duplication provides a convenient marker for identifying putative ring derivatives. It can be used to cover a deficiency that would otherwise cause lethality.

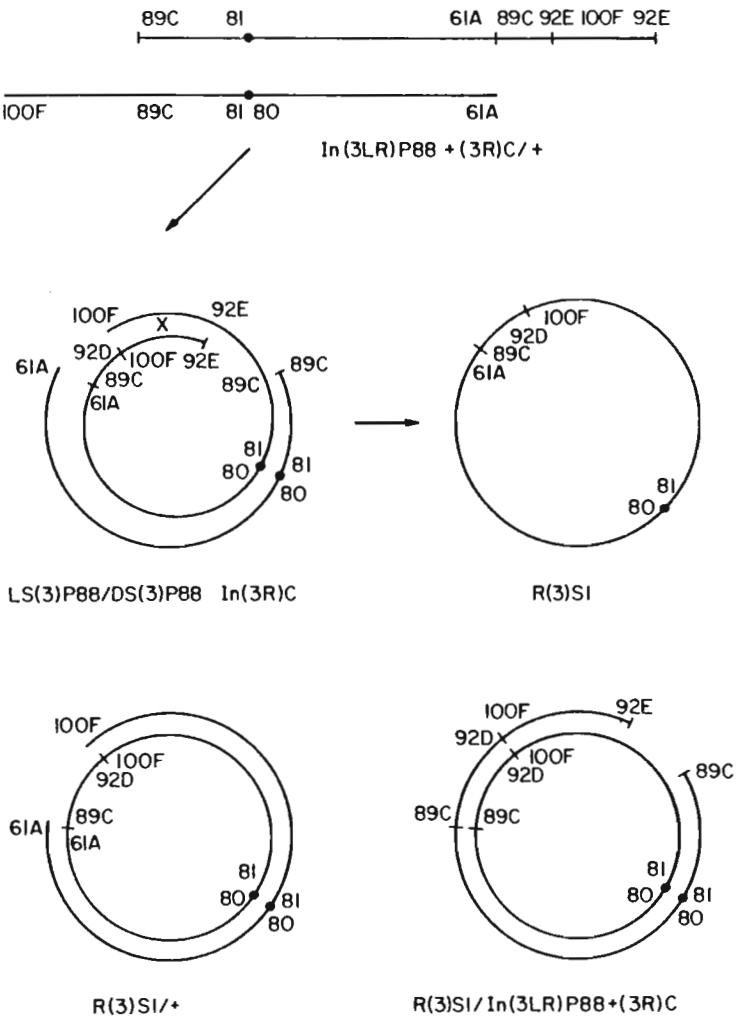

Fig. 1. Synthesis of R(3)S1 and the predicted pairing configurations for R(3)S1/+ and R(3)S1/In(3LR)P88+(3R)C.

To recover the ring, LS(3)P88/DS(3)P88, In(3R)C, bx^{34e} e--Tb ca females were mated to Df(3R)P47/Dp(3:3)MRS, Sb--+ males (Df(3R)P47 extends from 89D to 92A, while Dp(3:3)MRS is derived from Tp(3)MRS and has 87D to 93C inserted at 71B-C). Sb^+ offspring were then presumably R(3)S1/Df(3R)P47. Most of the Sb^+ offspring proved to be sterile, but it was possible to recover and maintain an R(3)S1, Tb ca chromosome. The ring structure was verified cytologically from metaphase figures from larval ganglia, and genetic confirmation was provided by transferring both Df(3R)P47 into the ring from a standard sequence chromosome and Tb ca from the ring into In(3LR)$Ubx^{UL}P88^{R}$+(3R)C.

The lower portion of Figure 1 illustrates the expected configuratio of R(3)S1 heterozygotes for either a standard sequency of In(3LR)P88+(3R)C chromosome. Double crossovers readily occur in females of either genotype so that markers can be transferred from a standard sequence chromosome to R(3)S1 to In(3LR)P88+(3R)C or from the inversion to the ring to the structurally normal chromosome. (Actually, In(3LR)$Ubx^{UL}P88^{R}$ is used instead of In(3LR)P88 since In(3LR)P88/R(3)S1 is lethal; In(3LR)$Ubx^{UL}P88^{R}$ also has the advantage of carrying sbd^2 as a marker.)

Newly recombined R(3)S1 chromosomes commonly induce sterility. R(3(S1/Df(3R)P47 progeny, identifiable as having minor phenotypic abnormalities characteristic

of the ring, from the ring synthesis are usually sterile or viturally so; moreover, those animals which are virtually sterile--at most 2 progeny--have sterile or nearly sterile progeny. R(3)S1, Df(3R)P47+ progeny from R(3)S1/Df(3R)P47 mothers also show this pattern of sterility. The few crossovers which are completely fertile show no indication of sterility in subsequent generations.

Because of this sterility problem and because of the lowered viability of R(3)S1+ due to hyperploidy, it is desirable to selectively recover ring-bearing progeny when inserting markers into the ring from a standard sequence chromosome. This can be accomplished by crossing R(3)S1, Df(3R)P47/marker females to Df(3R)P47/Dp(3;3)MRS, Sb--+ males to recover recombinant R(3)S1 chromosomes (with or without the marker) over Df(3R)P47 and later test for presence of the marker. To transfer markers from the ring to In(3R)C, it is convenient to recover an $In(3LR)Ubx^{UL}P88^{R}+(3R)C$, sbd^2 chromosome with the marker from R(3)S1, marker/$In(3LR)Ubx^{U}$ +(3R)C, sbd^2 ss Ubx^{U} mothers: sbd^2 provides a marker for the inversion, and absence of the Ubx^{U} phenotype identifies a crossover.

Stocks of (1) R(3)S1, Df(3R)P47, ca/In(3R)C, Sb cd Tb ca,
(2) Df(3R)P47/Dp(3;3)MRS, Sb--+,
and (3) $In(3LR)Ubx^{U}+(3R)C$, sbd^2 ss Ubx^{U} cd/$T(2;3)ap^{Xa}$ are available from the Pasadena stockcenter.

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Transferring markers to or from autosomal inversions.

Markers can be transferred from a standard sequence chromosome into a large pericentric inversion by double crossing over. For inversions of moderate length--on the order of 10 numbered divisions--such double crossovers can be exceedingly rare and are prohibitively rare for small inversions. It is, however, possible to transfer markers from one inversion to another through a sequence of selected single crossovers. $InA^{L}B^{R}$/InB females are crossed to $InB^{L}A^{R}/InA^{L}B^{R}$ males to selectively recover recombinant $InA^{L}B^{R}$ and $InB^{L}A^{R}$ chromosomes (it is assumed that $InA^{L}B^{R}$/"+" and $InB^{L}A^{R}$/"+" are lethally aneuploid genotypes). InA and InB are then reconstituted by crossing $InA^{L}B^{R}/InB^{L}A^{R}$ females to structurally normal males. The reconstituted InA and InB chromosomes are frequenctly double crossover chromosomes, so that markers may be transferred from one inversion to the other via this sequence of crosses.

Transferring markers from a structurally normal chromosome to moderate length or smaller inversions is accomplished by first transferring the markers into a large inversion, then transferring the markers from the large inversion to the smaller one. As an example, the following sequence of crosses was used to insert se, h^2, rs^2, and th into In(3L)P:

P_1 C(1)M4, y^2; In(3L)C90/se h^2 rs^2 th st cp in ri p^{p} females were crossed to se h^2 rs^2 th st cp in ri p^{p} males.

C(1)M4 is present in this cross to increase crossing over. In(3L)C90 is a large pericentric inversion with 62B and 80 breaks. In(3L)C90, se h^2 rs2 th st was recovered in the P_2. A balanced stock of C(1)M4, y^2; In(3L)C90, se h^2 rs^2 th st/In(3L)P, Me h D^3 was then constructed.

P_4 C(1)M4, y^2; In(3L)C90, se h^2 rs^2 th st/In(3L)P, Me h D^3 females were crossed to $In(3L)C90^{L}P^{R}+(3R)P18$, Ubx $e^4/In(3L)P^{L}C90^{R}$ males,

to recover $In(3L)C90^{L}P^{R}$, se h^2 rs^2 th st/$In(3L)P^{L}C90^{R}$ (recognizable as being Me^{+} and Ubx^{+}) and $In(3L)P^{L}C90^{R}$, se h^2 $D^3/In(3L)C90^{L}P^{R}+(3R)P18$, Ubx e^4. These two genotypes were crossed to each other to produce a

C(1)M4, y^2; $In(3L)C90^{L}P^{R}$, se h^2 rs ^{2}th st/$In(3L)P^{L}C90^{R}$, se h^2 D^3 stock.

P_6 C(1)M4, y^2; $In(3L)C90^{L}P^{R}$, se h^2 rs^2 th st/$In(3L)P^{L}C90^{R}$ females were crossed to: th st cp in ri p^{p} males.

A few $th_2st^{+}{}_2$offspring (In(3L)P, se h^2 rs^2 th) offspring were produced and a stock of In(3L)P, se h^2 rs^2 th was then established.

The In(3L)P, D^3 combination used in the above synthesis was derived in a somewhat similar manner; h was inserted into In(3L)P by a rare double crossover, and In(3L)P, Me h D^3 was constructed from these chromosomes and In(3L)P, Me.

Large paracentrics exist for all major autosomal arms--In(2L)DTD27 (21B; 40), $In(2R)bw^{VDe1}$ (41; 59), In(3L)C90, and In(3R)P110 (81F; 99). Stocks of $In(2L)NS^{L}DTD27^{R}/In(2L)DTD27^{L}NS^{R}$ and $In(2L)Cy^{L}DTD27^{R}/In(2L)DTD27^{L}Cy^{R}$ have been constructed in addition to the $In(3L)C90^{L}P^{R}/In(3L)P^{L}C90^{R}$ complex. These stocks were derived by applying the methods which I have described for deriving crossover products of pericentric inversions (Genetics 99:75-77, 1981).

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Differential puffing activity in two E chromosomal arrangements of D.subobscura.

A comparison of the E chromosome puffing patterns of the different gene arrangements were carried out in order to investigate the possible effect of inversions on gene expression. Two strains of Drosophila subobscura were used: H271 which is homozygous for E_{st} arrangement and Ra121 which is homozygous for $E_{1+2+9+12}$ arrangement. The puffing patterns of late third instar larvae and different aged prepupae were analyzed. The prepupal samples were taken at 0, 4, 10 and 18 hrs after the eversion of the anterior spiracles. 20 individuals were analyzed per developmental stage and strain. Five nuclei were observed from each of the individuals analyzed. For the average degree of puffing activity two criteria were taken into account: (a) size of puffs, and (b) frequency of appearance of each puff at every stage analyzed. The puffs and breakpoints of $E_{1+2+9+12}$ inversion were located using the standard salivary gland chromosome map of Kunze-Mühl and MUller (1958). The breakpoints of $E_{1+2+9+12}$ arrangement are the following: E_1 58D/59A-62D/63A, E_2 58D/62D-64B/64C,

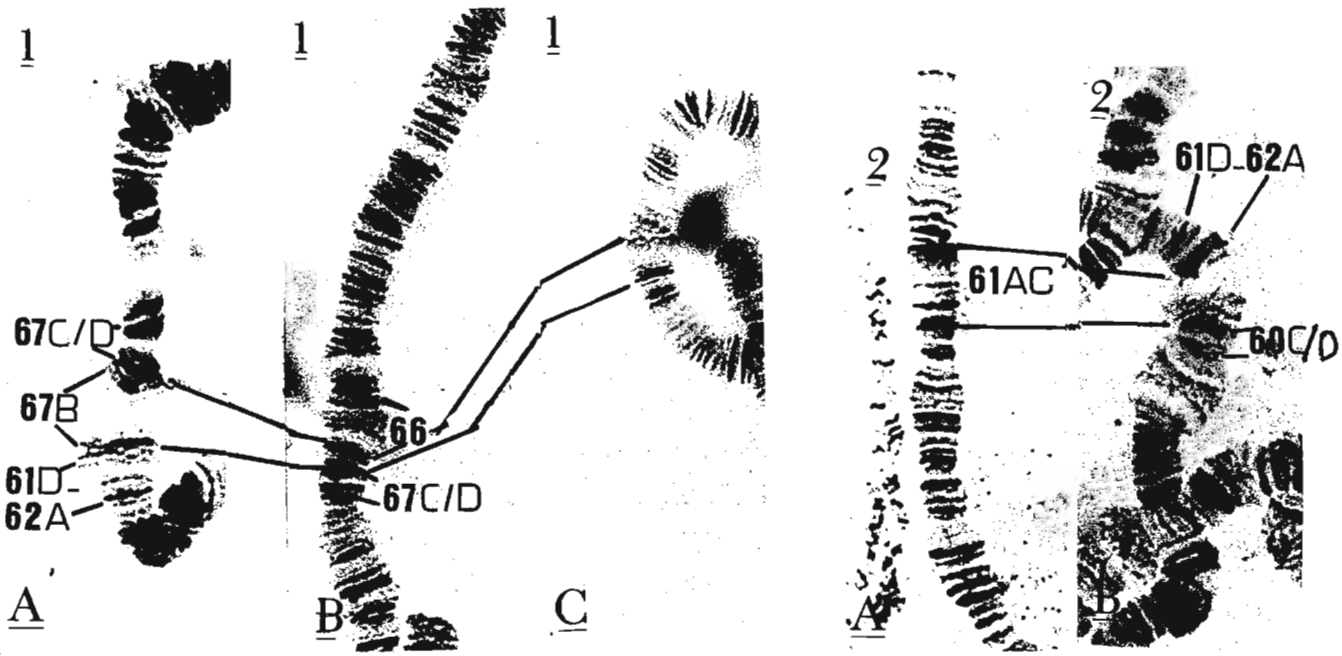

Figure. E chromosomes of D.subobscura: (1A) $E_{1+2+9+12}$ arrangement of Ra121 strain (18h prepupa). (1B) E_{st} arrangement of H271 strain (4h prepupa). (1C) E_{st} arrangement of H271 strain (0h prepupa). (2A) $E_{1+2+9+12}$ arrangement of Ra121 strain (0h prepupa). (2B) E_{st} arrangement of H271 strain (18h prepupa).

E_9 58D/64D-68B/68C, E_{12} 61C/61D-67A/67B. All individuals were dissected in Ringer Drosophila solution (pH 7.2). Salivary glands were fixed in ethyl alcohol: acetic acid (3:1), and were stained in lacto-aceto orcein (acetic orcein 80%, lactic acid 20%). All experiments, cultures and cytological preparations were carried out in a thermoregulated room at 19±1°C.

Several puffs show a similar pattern of activity in E_{st} and $E_{1+2+9+12}$ arrangements, for instance 68DE,69B, 70A and 70BC. Other puffs show quantitative differences or differences in the timing of activity. Finally, a few puffs display strong differences. Among this group can be included the large puffs 67B and 61AC. In the $E_{1+2+9+12}$ arrangement 67B is active in third larvae, regresses by 0h prepupae and after puparium formation increases its activity throughout the prepupal period (Figure 1A). In spite of this, 67B is an occasional puff in the E_{st} chromosome. It only appears actively in the third instar and at the beginning of prepupation and always at low frequency (Figures 1B and 1C).

Another striking difference is the size of 67B in both chromosomes. In $E_{1+2+9+12}$ arrangement 67B is a large puff as can be seen in Figure 1A. The size of this puff varies little thoughout its time of activity. In spite of this, 67B is a small puff in E_{st} chromosome. Its maximum size can be observed on Figure 1C. 61AC shows complementary behaviour (Fig. 2A and 2B). It is a large puff that maintains its activity throughout the prepupal period in E_{st}, and a small occasional puff in $E_{1+2+9+12}$ chromosome. It is important to emphasize the location of these puffs on the E chromosome. Both puffs are located at the boundaries of E_{12} inversion. In the E_{st} chromosome (Fig. 1B and 2B), 67B is located between the active locus 66 and the occasional puff 67C/D, and 61AC is located between the occasional loci 60C/D and 61D-62A. In the $E_{1+2+9+12}$ chromosome 67B is located between 67CD and 61D-62A. It is interesting to note the behaviour of this last site. 61D-62A shows activity both in E_{st} and in $E_{1+2+9+12}$, whereas in E_{st} it is close to 61AC and is never very large (Fig. 2B), in $E_{1+2+9+12}$ it is close to 67B and is always very large (Fig. 1A). It is obvious that 67B and 61AC show the greatest differences in puffing activity between E_{st} and $E_{1+2+9+12}$ arrangements. Both are located at the boundaries of the E_{12} inversion, and not only do they change their position in the chromosome but also the sites close to both are different in the two arrangements. It is possible that these differences in gene activity at puff level can be due to position effect.

References: Kunze-MUhl, E. & E.MUller 1958, Weiter Untersuchungen Uber die chromosomale Strukturtypen bei Drosophila subobscura, Coll.Z.indukt.Abstamm.-Vererb.Lehrer 87:65-84.

Di Pasquale Paladino, A. and P.Cavolina.
University of Palermo, Italy. Caffeine effect on tumor manifestation in the tu-pb stock of D.melanogaster.

Table 1. Complete development in medium containing various concentrations of caffeine.

	% ♀♀ tu	N.♀♀	% ♂♂ tu	N.♂♂
Control	32.82	1301	7.22	1149
Caffeine 500 ug/ml	29.13	1253	5.73	1238
Caffeine 1000 ug/ml	17.61*	1221	3.68*	1060
Caffeine 0.01 M	7.23*	166	1.45*	207

* $P < 0.05$ (compared to control)

We have now investigated the effect of caffeine added to the nutrient medium of developing tu-pb larvae. Statistical analysis of results obtained after complete development of larvae on medium containing various concentrations of caffeine (Table 1) demonstrates that a dose of 500 ug/ml does not exert any influence upon tumor incidence, while concentrations equal to 1000 ug/ml and 0.01 M (corresponding to 1984 ug/ml) exert a significant inhibitory effect upon tumor appearance in adult insects. 0.01M concentration was chosen for experiments involving exposition of larvae to caffeine medium for a limited period of time. Results are shown in Table 2.

Experiments involving egg deposition onto normal medium, followed by transfer onto caffeine medium: tumor incidence is significantly decreased, if compared to control, only in the 48h group. This finding suggests that caffeine is active only during the first stages of development.

Experiments involving egg deposition onto caffeine medium followed by transfer onto normal medium: tumor incidence was found to be lower in groups of larvae transferred from

caffeine to caffeine medium than in those transferred from caffeine to normal medium. The latter groups, however, do not significantly differ from larvae completing their development on normal medium. A significant decrease of tumor incidence can be therefore shown only if caffeine is present in medium since the beginning of development and for the whole larval life. Such decrease should not be ascribed to different survival rates, since survival patterns do not always correspond to tumor incidence patterns.

This work was supported by a grant from Ministero Pubblica Istruzione (60%).

Table 2. *(P < 0.05) (compared to control)

Period of larval life treated		treated larvae	survival rate	% ♀♀ tu	N. ♀♀	% ♂♂ tu	N. ♂♂
Deposition onto normal medium:							
48 h	To normal	621	47.6	18.7	160	4.4	136
	To caffeine	1184	30.8	11.1*	252	0.7	276
72 h	To normal	272	53.6	13.5	74	2.7	72
	To caffeine	666	55.7	15.4	194	1.7	177
96 h	To normal	203	78.8	15.4	71	2.2	89
	To caffeine	401	62.1*	16.7	137	3.5	112
120h	To normal	426	66.9	21.3	145	1.4	140
	To caffeine	377	64.1	23.1	108	0.7	134
Deposition onto caffeine medium:							
24 h	To normal	1249	33.8*	23.3*	231	7.8	192
	To caffeine	1434	24.4	13.6	190	4.3	161
48 h	To normal	947	28.2*	21.5*	130	8.6	138
	To caffeine	1069	22.6	13.3	127	1.7	115
72 h	To normal	628	42.6*	35.9*	139	3.1	129
	To caffeine	716	18.8	9.2	76	0.0	59
96 h	To normal	402	64.1*	34.2*	105	6.5	153
	To caffeine	525	50.6	14.1	141	0.8	125
120h	To normal	480	52.5	29.7*	138	1.6	118
	To caffeine	540	50.3	16.5	121	0.0	151
Development in normal medium		986	43.7	27.4	215	6.4	216

Di Pasquale Paladino, A. and P.Cavolina. University of Palermo, Italy. Further investigations on the tu-pb melanotic tumor mutant of D.melanogaster.

In the search for understanding the mechanism of tumor manifestation in the tu-pb mutant, a peculiar case of melanotic tumor manifestation in Drosophila melanogaster (Di Pasquale Paladino & Cavolina 1982; Di Pasquale Paladino & Cavolina 1983), we have undertaken an analysis of factors that may in some way affect this character.

Results of temperature shift experiments, which are carried out in order to determine the temperature-sensitive period, are summarized in Fig. 1. Shift-down experiments show that tumor manifestation is inhibited when temperature is shifted during the early stages of larval development. Tumors appear, although with a very low frequency, when flies had been left at 23.5°C until the 72nd hr of development. Percent tumor incidence typical of the strain is attained when development is completed at 23.5°C. In shift-up experiments a detectable decrease of tumor incidence is found only when larvae are left at 18°C also during the late stages of development. Tumor incidence is found to decrease also when temperature shift corresponds to the 144th hr of development.

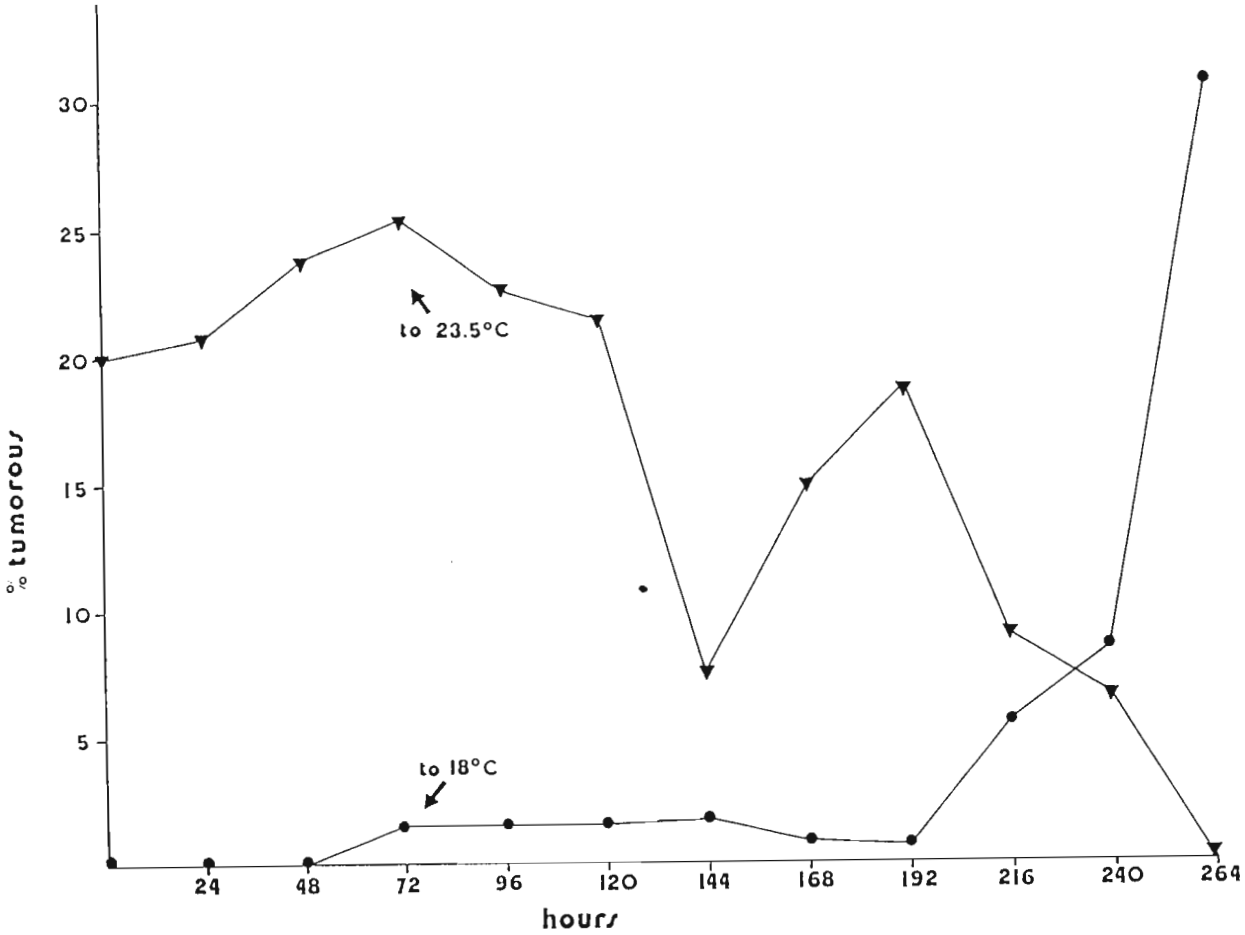

Figure 1. Results of the temperature shift studies.

i	♀♀	$O/C(1)RM,y^2su(w^a)w^a$; +/+ ; +/+ X ♂♂ tu-pb/tu-pb
ii	♀♀	$Y/C(1)RM,y^2su(w^a)w^a$; tu/+; tu/+ X ♂♂ tu-pb/tu-pb
iii	♀♀	$Y/C(1)RM,y^2su(w^a)w^a$; tu(or +)/tu ; tu/tu X ♂♂ bb/YSX.YL;In(1)EN,y ; +/+ ; +/+ (selected fenotypically tu)
iv	♀♀	$O/C(1)RM,y^2su(w^a)w^a$; tu(or +)/+ ; tu/+ X ♂♂ tu-pb/tu-pb
		1/2 of XO males and XXY females are: tu(or+)/tu ; tu/tu

Figure 2. Crosses made to obtain XX/Y females and X/O males carrying the tu-pb genes.

Table 1. Tumor incidence in XX/Y, X/Y and X/O individuals.

	% ♀♀ tu	N.♀♀	% ♂♂ tu	N.♂♂
progeny from the (ii) cross: ♀ XX/Y and ♂ X/Y	11.1	216	2.24	223
progeny from the (iv) cross: ♀ XX/Y and ♂ X/O	8.77	729	1.46	953

The different degree of penetrance in either sex is a constant feature of tu-pb. Number of experiments were carried out to elucidate the possible relationship between male or female genotype, sexual phenotype and tumor manifestation. No difference of percent tumor incidence was observed between XY and XO males (χ^2=0.27) when combinations of sex chromosomes were altered (Fig. 2 and Table 1). Percentage of individuals with tumors, however, is higher in females (even with XXY genotype). Percent difference between sex is not modified. Tumor incidence in female individuals with XX genotype, but phenotypically transformed into males by the transformer gene and having gene combination yielding tu-pb manifestation was checked by a series of crossings described in Fig. 3. The results shown in part 1 of Table 2 show that tumor incidence in male individuals and in female individuals transformed into males attains the same values, since penetrance is low in both cases. On the other hand, as is shown by results summarized in part 2 of Table 2, tumors become manifest in a remarkably higher percentage in females than in males, in individuals carrying the same recombinant chromosome tra tu-pb, in which the heterozygous gene transformer is not manifest.

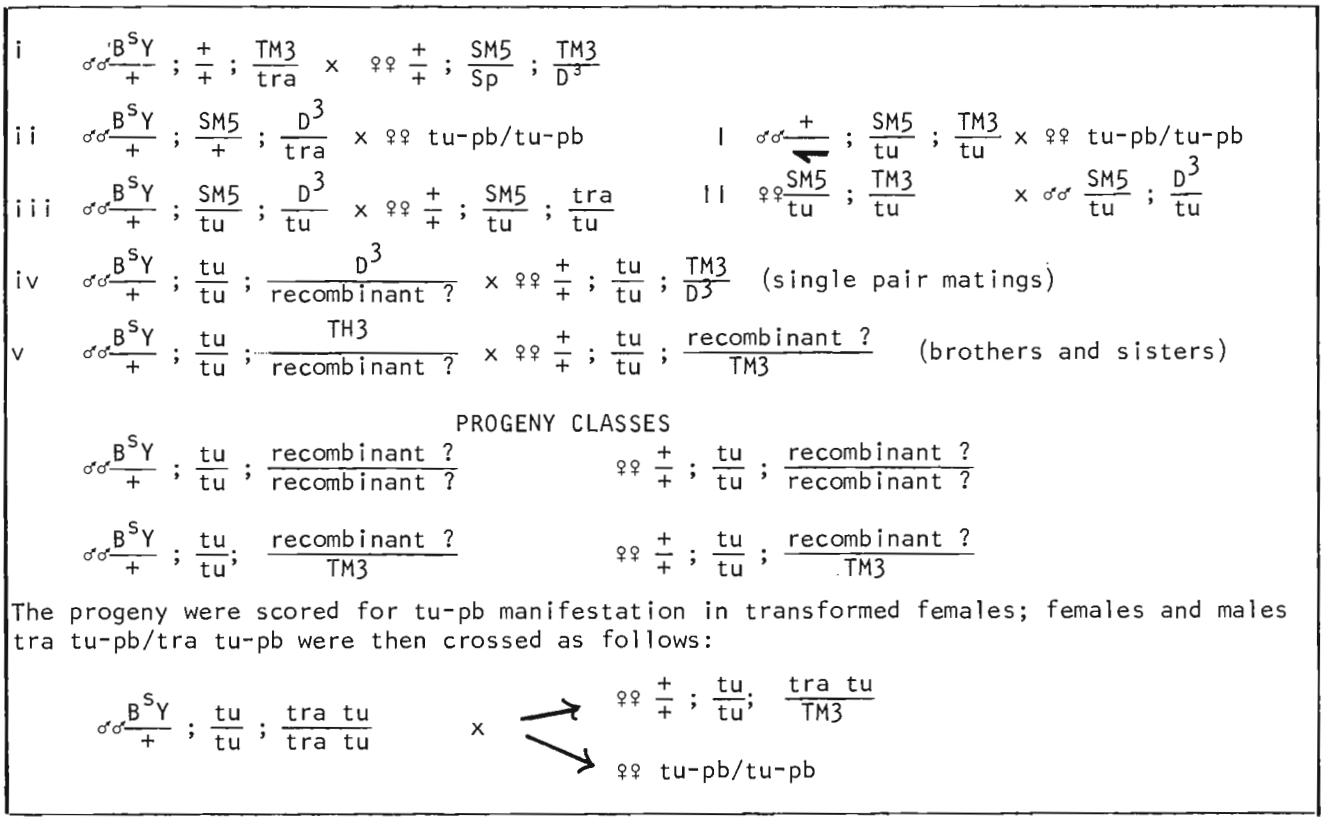

Figure 3. Matings made to obtain tu-pb tra recombinant chromosomes.

These results suggest that temperature-sensitive period starts when pupae are almost near emergence and that temperature during emergence appears to be decisive for the manifestation of this character. Temperature-sensitive period coincides more or less exactly with the stage in which melanotic masses become evident. There is probably some relationship between the different incidence of tumors in the two sexes and the male or female phenotype, while such difference seems to be independent of the sexual genotype. Tumor manifestation is particularly low in phenotypically male individuals.

This work was supported by a grant from Ministero Pubblica Istruzione (60%).

References: DiPasquale Paladino, A. & P.Cavolina 1982, Atti A.G.I. 28:155-158; DiPasquale Paladino, A. & P.Cavolina 1983, DIS 59:31-33.

Table 2. Tumor manifestation in individuals carrying a tra tu-pb recombinant 3rd chromosome.

1. Tumor incidence in homozygotes tra,tu-pb/tra,tu-pb from the cross:

♀ $B^{S}Y/+$; tu/tu ; tra,tu-pb/tra,tu-pb x ♂ +/Y ; tu/tu ; TM3/tra,tu-pb

transformed females		males	
% tu	N.	% tu	N.
8.09*	184	8.42**	190

* $\chi^2=86.032$; $P<0.01$

2. Tumor incidence in heterozygotes tra,tu-pb/tu-pb from the cross:

♀ $B^{S}Y/+$; tu/tu ; tra,tu-pb/tra,tu-pb X ♂ tu-pb/tu-pb

females		males	
% tu	N.	% tu	N.
52.04*	269	12.50**	224

** $\chi^2=1.394$; $0.20<P<0.30$

Dubucq, D., E.Depiereux and A.Elens. Universitaires Notre Dame de la Paix, Namur, Belgium. Phototactism and temperature.

The data here presented concern the phototactical behavior of Drosophila flies, assayed according to Benzer (1967) and to Kekic (1981), at three temperatures: 20°C, 25°C and 30°C. In both methods, the negative as well as the positive responses to light are determined. In the Benzer "counter-current" method the flies are submitted, moreover, to repeated mechanical stimuli: the most "sluggish" flies remain in the "0.0" test tube, the most phototactic flies concentrate in the "0.5" tube. In the Kekic maze the most phototactic flies go to the right

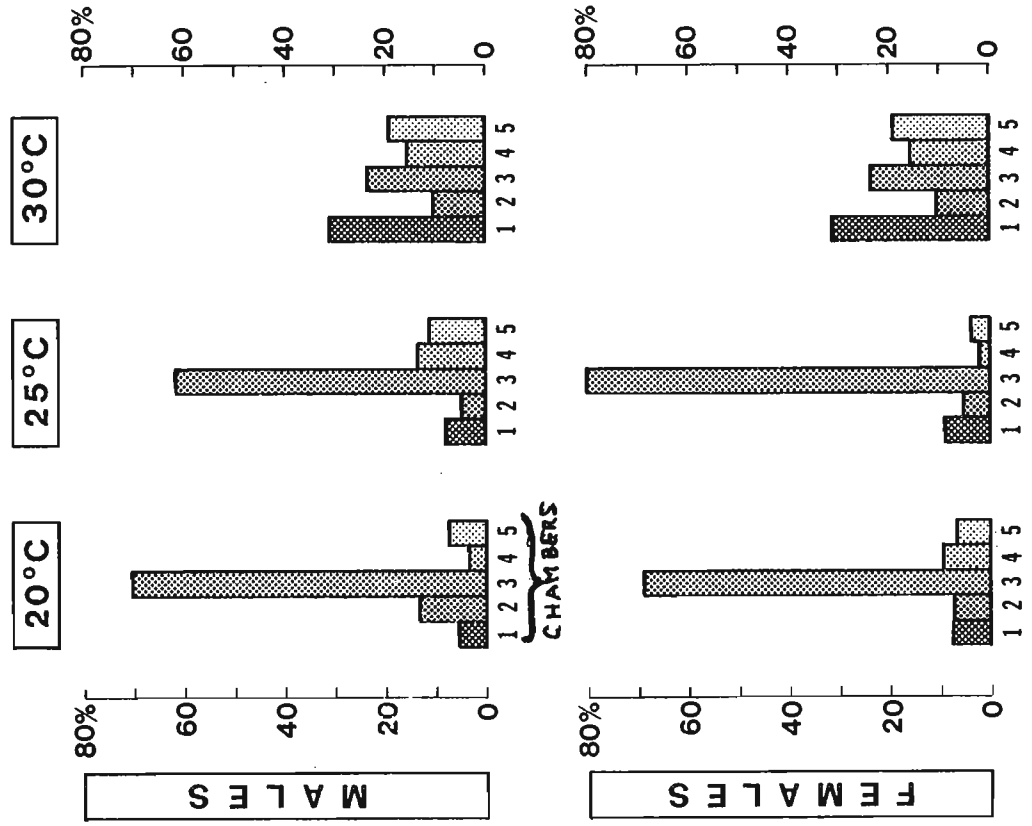

Fig. 1. Distribution of the flies after 1 hr tests in the Kekic maze (in%).

Chambers	Light, lux
1	500
2	800
3	2,000
4	4,500
5	6,500

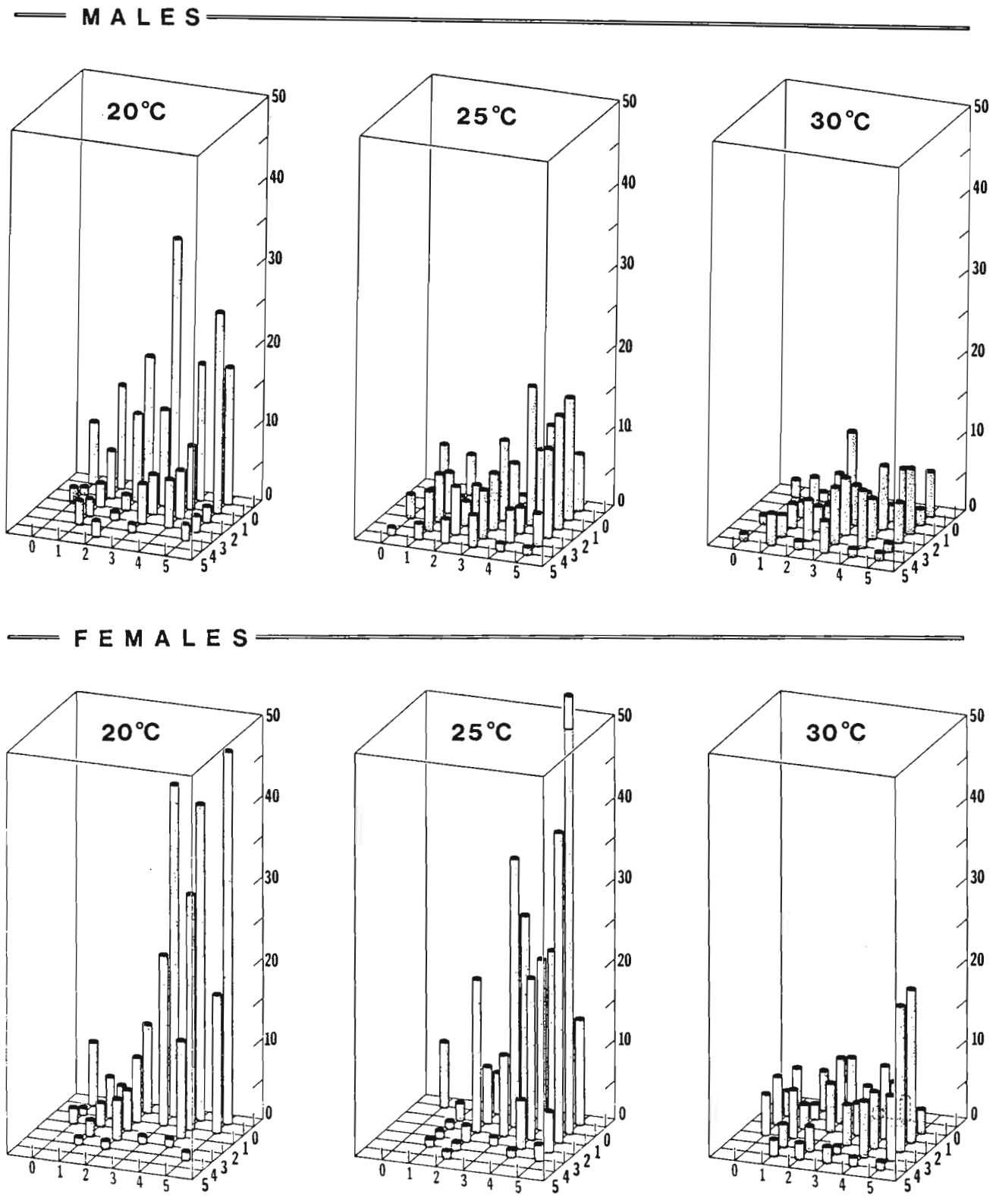

Figure 2. Distribution of the flies after a test for positive and for negative phototactism, according to Benzer (one minute runs). Ordinate: number of flies in the test tube. Front view abscissa: no. of positive responses (toward light). Side view abscissa: no. of negative responses (from light).

and the negatively phototactic go to the left; the most sluggish flies remain in the central chamber ("start"). The strain here studied is a "wild" one, called "Namur", and known as polymorphic for some enzyme variants. The tests have been done at the same time for both methods at the same temperature, with three repetitions for each temperature, according to a "latin square" design. Males and females were tested separately.

The Fig. 1 shows the results obtained using the Kekic maze. The Fig. 2 shows a three-dimensional representation of the final distribution of the flies in the different tubes after the tests done according to Benzer. In both methods, the phototactical behavior of the males and of the females differ significantly (with a probability of 0.0005, as shown by a X^2 test).

The influence of temperature is evident: at 30°C, the dispersion of the flies in the different test tubes or chambers is much more marked (the differences seem to be highly significant: probability of 0.0005 in the X^2 test).

The simplest explanation of it seems to be that the flies are more active at 30°C than at a lower temperature.

Perhaps a selection procedure for phototactism should give better results if the flies are tested at 30°C rather than 25°C.

References: Benzer, S. 1967, Proc.Nat.Acad.Sci. 58:1112; Kekic, V. 1981, DIS 56:178.

Duttagupta, A. and S.Banerjee. University of Calcutta, India. In vivo synchronization by Aphidicolin and Ricin in Drosophila.

Larval salivary glands of Drosophila contain an asynchronous cell population. They are in array of a replicating types, covering the whole of the S-phase. In our previous publication (Achary et al. 1981), we reported the usefulness of 5'-Flurodeoxyuridine in in vivo synchronization. In this report we present the results of our similar experiments with Aphidicolin and Ricin.

Aphidicolin is a tetracyclic deterpene tetraol, obtained from a fungus (Cephalosporium aphidicola). It is a specific inhibitor of DNA polymerase α with no effect on DNA polymerase β and γ (Ikegami et al. 1978). It binds to all eukaryotic DNA polymerase α reversibly (Huberman 1981). Ricin (Ricinus communis) a highly toxic plant protein, is also a potent inhibitor of DNA polymerase α (Bhattacharyya et al. 1979).

Early third instar giant female larvae of Drosophila melanogaster were fed on 1 ml (1M) sucrose containing 24 µg/ml Aphidicolin for 24, 48, 72, 96, 120 and 168 hrs and RIcin (1 mg/ml) was fed for 48 hrs only. Autoradiograms were prepared from the larval salivary gland. The frequency of labelling patterns was scored according to the classification of Chatterjee & Mukherjee (1975).

It can be observed from Table 1 that there was a net increase of 3C-3D types of nuclei (mid part of the S-phase), which reached its peak at 48 hrs, where 77% synchronized cells could be obtained. This then gradually declined as the feeding progressed. The frequency of DD-1C-2C (early patterns) remain more or less unchanged. Similarly Ricin produced about

Table 1. Frequency percent within the labelled nuclei.

Patterns	Aphidicolin (24 µg/ml)						Ricin(1mg/ml)
	24 hr	48 hr	72 hr	96 hr	120 hr	168 hr	48 hr
DD	3.07	-	0.68	0.50	-	1.23	-
1C	-	-	-	0.50	-	1.23	-
2C	1.53	0.61	1.37	2.50	2.05	2.46	-
3C	16.92	27.60	28.96	16.00	23.28	27.77	29.26
3D	49.23 (66.15)	49.68 (77.28)	46.19 (75.15)	47.50 (63.50)	45.20 (68.48)	27.15 (54.92)	45.12 (74.38)
2D	6.15	8.58	4.82	4.00	6.84	1.85	13.41
1D	23.07	12.88	15.17	26.00	21.91	38.27	12.19
CHL	-	0.61	2.75	3.00	0.68	-	-

74% synchronization within 48 hrs of Ricin feeding. Further work is in progress to obtain an increased frequency of in vivo synchronization, and to chase them in the later part of the same S-phase.

We gratefully acknowledge Dr. A.H. Todd of Imperial Chemical Industries and Dr. M.R. Singh of Indian Institute of Chemical Biology for the chemicals and the University Grants Commission (Sanction letter No. UGC/2961/Jr. Fellow (Sc) dated 13.3.80) for financial support.

References: Achary, P.M.R., K.Majumder, A.Duttagupta & A.S.Mukherjee 1981, Chromosoma 82:505-514; Battacharyya, P., I.Simet & S.Basu 1979, Proc.Natl.Acad.Sci. USA 76:2218-2221; Chatterjee, S.N. & A.S.Mukherjee 1975, Ind.J.Expt.Biol. 13:452-459; Ikegami, S., T.Taguchi, M.Ohashi, M.Oguro, H.Nagano & Y.Mano 1978, Nature 275:458-460.

Duttagupta, A., P.C.Das and P.K.Dutta. University of Calcutta, India. Genetic fine structure of Giant (gt) locus in Drosophila melanogaster.

The present paper concerns the genetic fine structure of the Giant (gt) locus (1.0-0.9) in Drosophila melanogaster, a locus responsible for an extra round of replication with concomitant increase in larval polyteny (Judd et al. 1972) and recently reported involvement in embryonic morphogenesis (Honisch & Campos-Ortega 1982). Recessive lethals were isolated in the region 3A1-4 according to the scheme outlined in Figure 1. Out of a total of 9055 chromosomes tested against Df(1)62g18 according to the scheme outlined in Figure 1, only 32 were found to be recessive lethals. The putative lethals were tested for allelism first against the mutant gt and then with two other alleles of gt, viz., gt^{x11} and gt^{E6}. In case, these recessive lethals were allelic to the mutant gt, then the heterozygous female class would be absent or its frequency would be low; such lethals were designated as an allele of gt. Allelism test against gt w^a showed that 10 out of the 32 lethals gave a very few or no survivors in heterozygous condition with gt. While test of allelism against alleles gt^{x11} and gt^{E6} revealed that the alleles were non-complementing all the 10 lethals that were allelic to gt and interestingly also non-complements certain lethals isolated against

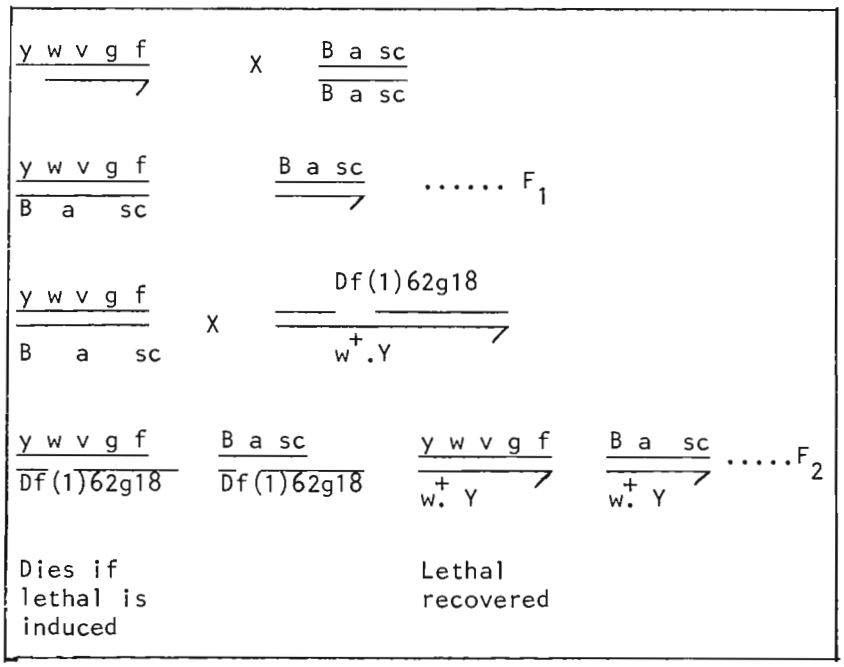

Fig. 1. Screening protocol for isolation of recessive lethals in the region 3A1-4. Males with chromosome markers y w v g f were fed with 0.025M EMS according to the method of Lewis & Bacher (1968).

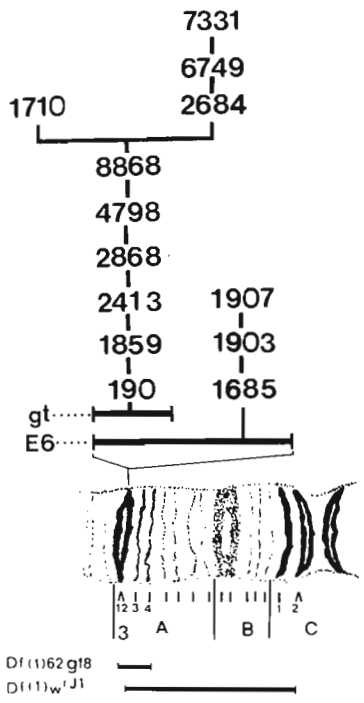

Fig.2

Df(1)62g18, but were complementing to gt. Inter se complementation analysis of the 10 lethals that were allelic to gt, revealed the presence of two complementation groups. Our finding has been summarized in Figure 2.

Inclusion of the mutant gt in the band 3A1 by Judd et al. (1972) was based on the complementation of this locus with $Df(1)w^{rJ1}$ and its non-complementation with three other deficiencies, Df(1)65j26, Df(1)X12 and Df(1)62g18, and in all of which the chromomere 3A1 was missing or reduced. The ten mutants that were found to have reduced the viability of the ℓ/gt heterozygote, partially or completely indicate that these lethals impaired seriously the functional unit of the gt allele in heterozygous condition, the survival of the ℓ/+ flies but not of the ℓ/gt class, indicates the presence of a decisive factor that acts in the trans heterozygotes, i.e., in the ℓ/gt, in trans dominant fashion. The isolation of 3 more lethals viz., 1685, 1903 and 1907 which complement gt but greatly reduces the viability in combination of gt^{x11} and gt^{E6}, points towards a difference between the alleles gt^{x11}, gt^{E6} and gt. This leads to the assumption that the functional areas of the alleles gt and E6 differ slightly. It therefore seems probably that the ten lethals which non complement gt, lie in that part (probably distal) of band 3A1 which is not covered by Df(1)62g18 and where gt locus has been mapped. This observation is specially significant in view of the findings by Judd et al. (1972) that the chromomere 3A1 is not deleted in $Df(1)w^{rJ1}$, but is reduced or missing in Df(1)62g18. The three other lethals viz., 1685, 1903 and 1907 seems to be larger lesions that probably span the whole chromomere 3A1 or further right to the proximal region of 3A1 and represents the true extent of this gt locus.

Further work on allelism test of these lethals against two other alleles of gt viz., gt^{13z} and gt^{Q292}, are in progress.

We gratefully acknowledge Drs. B.H.Judd, University of Texas; T.C.Kaufman, Indiana University and T.A.Grigliatti, University of British Columbia, for stocks and the University Grants Commission (Sanction No. F.23-457/75 (SR-II) dated 30.6.1977) for financial support.

References: Honisch, S. & J.A.Campos-Ortega 1982, DIS 58:76-77; Judd, B.H., M.W.Shen, & T.C.Kaufman 1972, Genetics 71:139-156; Lewis, E.B. & F.Bacher 1968, DIS 43:193.

Duttagupta, A. and A.DuttaRoy. University of Calcutta, India. Induction of a new Minute mutation in the second chromosome of Drosophila melanogaster.

During the course of saturation of a deficiency Minute mutation [$M(2)\text{-}z^{B}$; 24E1-2; 24F7-8)], we recovered some flies with Minute phenotype. Their genetical behaviour showed that these new Minutes failed to complement the deficiency, but complement M(2)-z mutation (for details see our report in this volume). When these Minute mutants are crossed with our seven complementing lethal alleles (DuttaRoy et al. 1984), it was observed that all these 7 groups kill this Minute as trans heterozygote. Analysis of polytene chromosome revealed that this new Minute allele bear a deletion for 24E region only (Fig. 1). A lethal allele of dumpy ($dp^{1\text{-}DG83}$) non-complemented this new Minute. This Minute, therefore, unlike others in this region is deleted for section 24E only.

Figure 1.

Reference: DuttaRoy, A., P.K.Manna & A.K.Duttagupta 1984, J.Biosci. (in press).

Duttagupta, A. and A.DuttaRoy. University of Calcutta, India. Reassessment of breakpoints of a deficiency on 2L of Drosophila melanogaster.

The deficiency M(2)-z^B was originally isolated by Bridges. The breakpoints of the deficiency has been mentioned as 24E2-F1; 25A1(2) (Lindsley & Grell 1968). In our cytological preparation (stock obtained from Mid-America Drosophila Stock Center at Bowling Green, Ohio USNA), we found that the distal breakpoints as mentioned is alright; we however observed that the proximal breakpoint is between 24F7-8 and 25A1(2). The band 25A1(2) remains intact in this deficiency (Figure 1).

Figure 1.

The deficiency M(2)-z^B noncomplements three alleles, namely dumpy (dp), dwarf-24F(dw-24F) and M(2)-z. Broderick & Roberts (1982) assigned M(2)-z at 25A1(2) by making use of a series of duplications. Since different Minute mutations instead of being additive show complementary genetic effects among each other (Schultz 1929), the failure of M(2)-z to complement DfM(2)-z^B awaits a proper explanation.

We recently isolated 45 mutations in DfM(2)-z^B locus (38 lethals and 7 new Minutes). None of these mutations can kill M(2)-z in heterozygous condition (DuttaRoy et al. 1984). We therefore can assume that (i) the deletion in M(2)-z^B may involve some interband between 24F7-8 to 25A1(2). The same interband may have a lesion in M(2)-z that does not allow the recovery of the trans-heterozygote M(2)-z/M(2)-z^B, (ii) M(2)-z might be acting as a polarity mutation or (iii) M(2)-z^{B+} and M(2)-z^+ region may involve together in the synthesis of a molecule with a dimeric structure. Therefore, mutation in both the loci seriously impedes some essential function of the fly and leads to lethality.

References: Broderick, D.J. & P.A.Roberts 1982, Genetics 102:71; DuttaRoy, A., P.K. Manna & A.K.Duttagupta 1984, J.Biosci. (in press); Schultz, J. 1929, Genetics 14:366.

Duttagupta, A. and A.K.Ghosh. University of Calcutta, India. Effect of cadmium chloride on the polytene chromosome of Drosophila salivary gland.

It is well known that different heavy metal ions stabilize or labilize the ordered conformation of DNA molecules in vitro. They may either promote the reversible unwinding and rewinding of multiple stranded helix. We have studied the effect of $CdCl_2$ (a heavy metal salt) on the polytene chromosome of Drosophila.

Second instar larvae of Drosophila ananassae were fed with $CdCl_2$ salt (1×10^{-6} molar dissolved in sucrose solution) for 48 hrs. The larval salivary gland chromosomes were then prepared by squas technique. In case of control, larvae were fed with only sucrose solution from 2nd instar for 48 hrs.

Out of 103 treated nuclei observed, 95 chromosome arms showed asynapsis, of which 19 were X-chromosomal and 76 autosomal arms. In case of control of the 103 nuclei observed only 31 chromosomes show such asynapsis, of which 9 were X-chromosomal and 22 autosomal arms (see Table).

	Total number of nuclei observed	Number of asynapsed chromosomes	X-chromosome	Autosome	% of asynapsis
Control	103	31	9	22	6.00
Treated	103	95	19	76	18.40

The data, therefore, reveals that in treated nuclei chromosomes asynapsis is 3 times more than that of control nuclei. The high percentage of asynapsis in treated nuclei is probably due to some ionic disturbance.

Duttagupta, A., A.Kar and A.DuttaRoy. University of Calcutta, India. A deficiency Minute mutation that acts as an enhancer of position-effect variegation.

$M(2)\text{-}z^{B}$ is a deletion spanning the polytene chromosome section 24E1-2; 24F7-8. We tested the effect of this deletion on brown-variegation (bw^{V1}). Level of Drosopterin pigment was measured following the methods of Reuter et al. (1983) by making bw^{V1} heterozygous with $M(2)\text{-}z^{B}$ chromosome. We have already reported the analysis of 38 lethal mutation in this region (DuttaRoy et al. 1984). Until now fourteen such lethal alleles have been tested with bw^{V1} (Fig. 1). Our analysis revealed that $DfM(2)\text{-}z^{B}$ act as a definite enhancer of brown-variegation where the quantity of pigments dropped down to less than half of the $bw^{V1}/+$ level. Some of the lethal alleles which behaved as point mutations also showed some reduction. The enhancement was not as pronounced as it was observed in case of deficiency $M(2)\text{-}z^{B}$. Work is in progress to see the effect of rest of the alleles.

Reference: Reuter, G. & J.Szidonya 1983, Chromosoma 88:277.

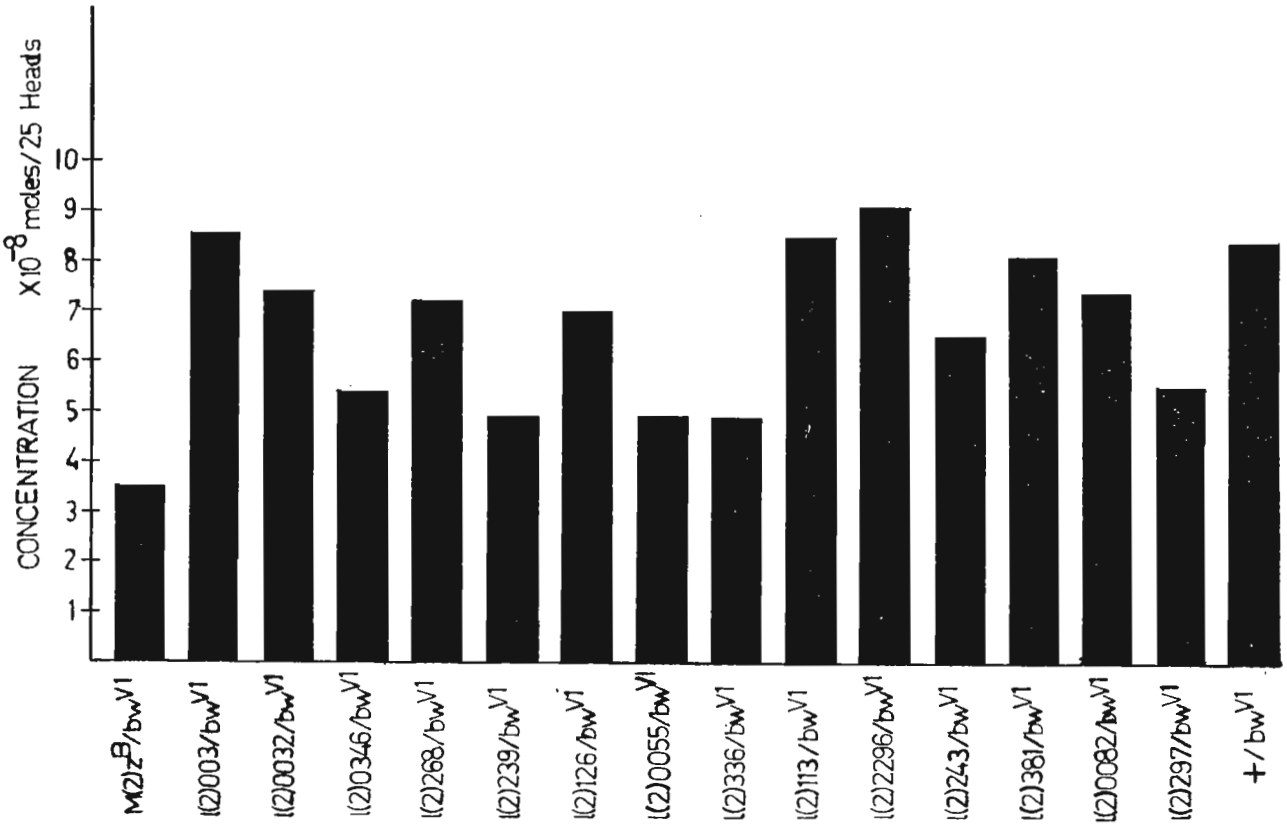

Figure 1.

Duttagupta, A.K., D.Mutsuddi and M.Mutsuddi. (Das). Univ of Calcutta, India. Conservativeness in the regulation of replication in three related species of Obscura group.

The two arms of the X chromosome (XL and XR) of Drosophila pseudoobscura and D.persimilis have different phylogenetic origin. The XL is homologous to the X and XR is homologous to 3L of D.melanogaster (Sturtevant & Novitski 1941; Muller 1950). The works of Abraham & Lucchesi (1973) and Mukherjee & Chatterjee (1976) have shown that in D.pseudoobscura, both the X chromosomal arms (XL and XR) are hyperactive and early replicating. Interestingly, in the same species, Chatterjee et al. (1976) for the first time have reported that the C element (3rd chromosome) replicates earlier than the remaining autosomes in both the sexes. In our present investigation, we have examined the replicative and transcriptive behaviour of the two X chromosomal arms (XL and XR) as well as the 3rd chromosome in the other species, D.persimilis and the replicative behaviour of these three chromosomal elements (XL, XR and 3rd chromosome) in interspecific hybrids of D.persimilis and D.pseudoobscura.

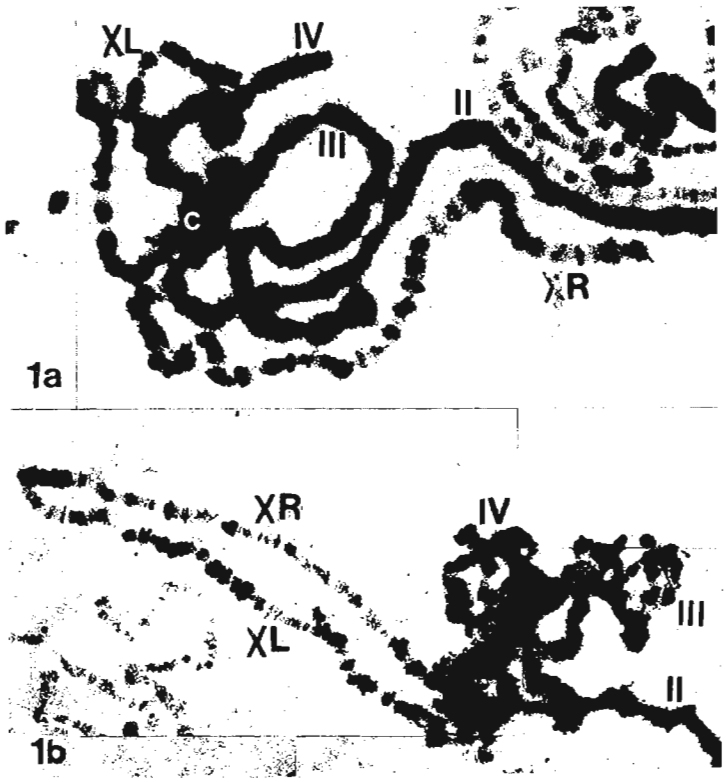

Fig. 1. Autoradiograms showing ^{3}H-TdR labelling on the two X chromosomal arms (XL and XR) in comparison to the pattern on the autosomes (a) D.persimilis male and (b) hybrid male. A = autosome.

Fig. 2. Autoradiogram showing ^{3}H-TdR labelling the XR, 3rd chromosome and 4th chromosome of D.persimilis male nucleus. 3rd chromosome representing the distinct asynchronous replication pattern with 4th chromosome.

The results of our investigation reveal, that, like that of D.pseudoobscura, both the X chromosomal arms (XL and XR) in D.persimilis are both hyperactive and early replicating in male (Fig. 1a) and the 3rd chromosome, irrespective of sex, is also characterized by its early replicating property than the remaining autosomes (Fig. 2). In the hybrid male nuclei, both the X chromosomal arms (XL and XR) as well as both the homologue of the C element, derived from persimilis and pseudoobscura, maintain their respective replicating property (Fig. 1b). Moreover, the frequency of ^{3}H-TdR labelling of each replicating unit on XL, XR and both the homologue of the C element (3rd chromosome) exactly corresponds to those of

their corresponding sites of the respective chromosomal elements of the parental species. Such autonomous 'parental' behaviour of each chromosomal arm in the hybrids and the early replicating property of the C element (3rd chromosome) in D.pseudoobscura, D.persimilis as well as in D.miranda (X_2, Das et al. 1982), as mentioned earlier (Mutsuddi et al. 1984), indicate the presence of inbuilt genetic control of replication that is conserved in individual chromosome during evolutionary process.

This work is financially supported by a U.G.C. junior reseach fellowship to Mausumi Mutsuddi (Das).

References: Abraham, I. & J.C.Lucchesi 1973, Genetics 74:52; Chatterjee, S.N., S.N. Mandal & A.S.Mukherjee 1976, Chromosoma (Berl.) 54:117-125; Das, M., D.Mutsuddi, A.K.Duttagupta & A.S.Mukherjee 1982, Chromosoma (Berl.) 87:373-388; Mukherjee, A.S. & S.N.Chatterjee 1976, J.Microscopy 106:199-208; Muller, H.J. 1950, The Harvey Lecture Series 43:165-229; Mutsuddi (Das), M., D.Mutsuddi, A.S.Mukherjee & A.K.Duttagupta 1984, Chromosoma (Berl.) 89:55-62; Sturtevant, A.H. & Novitski 1941, Genetics 26:517-541.

Duttagupta, A.K., D.Mutsuddi and M.Mutsuddi (Das). University of Calcutta, India.

Unequal diameter of the homologous chromosomal elements in the hybrids of D.mulleri and D.arizonensis.

The salivary gland chromosomes of the hybrids, produced from the cross Drosophila mulleri females to D.arizonensis males, are being investigated. In hybrid females, a certain proportion of nuclei represent a beautiful situation of coexistence of two homologue with distinct differential diameter for all the chromosomal elements in the same nucleus. While one homologue is much wider, other is distinctly thin, being almost half or about one third to that of the former. Interestingly, irrespective of diameter, the staining intensity is equal in both the homologue and they show considerable good pairing in most of the homologous sites. Study of ^{3}H-TdR labelling pattern of the salivary gland chromosomes in these hybrid females reveal that the replication pattern of all the homologous sites are similar between these two homologue (Fig. 1a-d) and to those of the corresponding sites of their respective parental species.

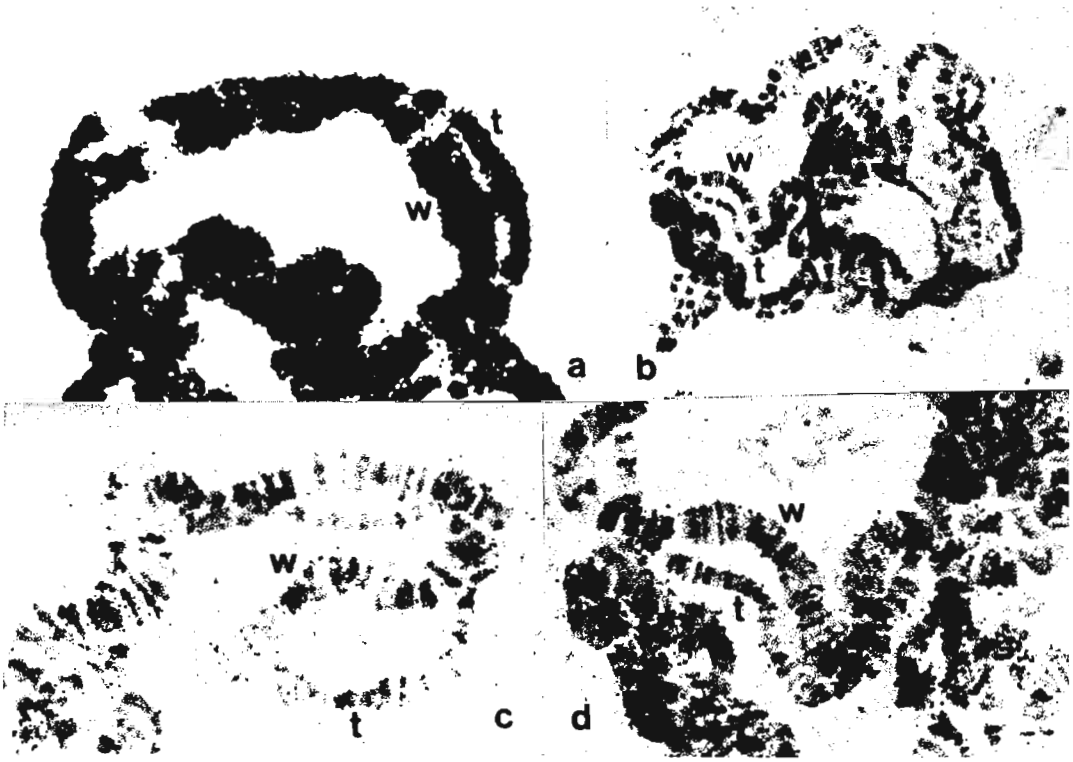

Fig. 1. Autoradiograms showing similar ^{3}H-TdR labelling patterns on the two homologue with differential diameter in hybrid females of D. mulleri and D. arizonensis. Such synchrony in replication cycle (a) very early terminal stage, (b) very late terminal stage; (c) and (d) represent magnified portion of (b).

w - wider homologue t = thinner homologue

In the hybrid male nuclei, though the unusual increase of the X chromosome and micro-chromosome are evident and also have been reported earlier (Bicudo & Richardson), the coexistence of two homologue with such differential diameter for all chromosomal elements in the hybrid females is undoubtedly unique. Howver, the similar staining intensity and synchronous pattern of replication between these two homologue suggests that the unusual increase in diameter between these two homologue is probably due to similar chromatin condensation between them but additional polyteny in one homologue over the other.

This work is supported by a U.G.C. junior research fellowship to Mausumi Mutsuddi (Das).

References: Bichudo, H.E.M.C. & R.H.Richardson 1977, Proc.Natl.Acad.Sci(Wash) 74:3499-3502.

Duttagupta, A.K., M.Mutsuddi(Das) and D.Mutsuddi. Univ. of Calcutta, India.

Effect of transforming mutants on the X chromosomal replication pattern in Drosophila melanogaster.

In Drosophila melanogaster, sex determination is under the control of X chromosome/A autosome ratio (Bridges) as well as wild type alleles of the sex-transforming mutants (Baker & Ridge). With the help of such sex-transforming mutants and by changing the sexual physiology of the flies, attempts have been made to study the role of altered sexual physiology on X chromosomal gene expression (Muller; Komma; Smith & Luchesi). In our present study, we have examined the ^{3}H-TdR labelling pattern of the salivary gland chromosomes in changed physiological conditions with a view to determine the effect of such sex-transforming mutants on the X chromosomal replication pattern. Five such mutants viz., sex-combless (sx), double sex (dsx), double sex dominant (dsx^{D}), intersex (ix) and transformer-2 (tra-2) were used in our present study and DNA replication pattern have been examined in 6 genotypic conditions vix., sx/Y, dsx/dsx; XY, dsx/dsx; XX, dsx^{D}/+; XX, ix/ix;XX tra-2/tra-2;XX.

Autoradiograms reveal that generally while the X chromosomes in sex-combless males (sx/Y) and male intersexes (dsx/dsx; XY) are early replicating (Fig 1a,b) than the remaining autosomes, the X chromosome in pseudo-males (tra-2/tra-2;XX) and three types of female interesexes.

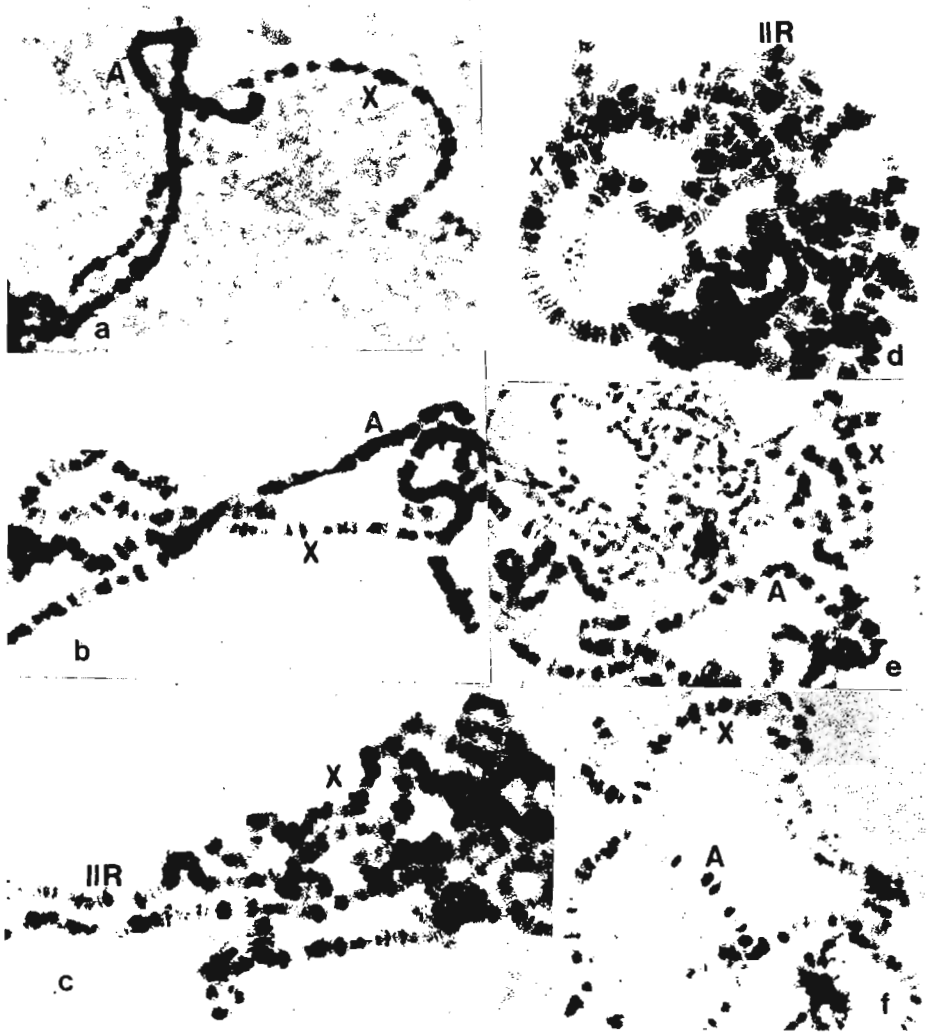

Fig. 1. Autoradiograms showing ^{3}H-TdR labelling on the X chromosome in comparison to the pattern on the autosome in
(a) dsx/dsx;XY,
(b) sx/Y,
(c) ix/ix;XX,
(d) tra-2/tra-2;XX,
(e) dsc/dsx;XX and
(f) dsx^{D}/+;XX.
X = X chromosome,
A = autosome.

(dsx/dsx;XX, dsx^D/+;XX and ix/ix;XX) replicate in a synchronous manner along with the autosomes (Figs. 1c-f). However, the detailed sitewise analysis of the ^{3}H-TdR labelling reveal that, in each condition, there are significant reproducible alterations for some replicating sites on the X chromosome. Among these six experimental conditions, the number of altered sites are maximum (eleven) in pseudomales (tra-2/tra-2;XX), minimum (one) in sexcombless males (sx/Y) and in remaining cases it is two in dsx/dsx;XY and seven in all the three female intersexes (dsx/dsx;XX, dsx^D/+;XX and ix/ix;XX). Our results, however, fail to establish a positive correlation between the changed sexual physiology and the altered labelling frequencies on the X chromosomes. At the same time, significant change in labelling frequency of some autosomal sites in pseudomales (tra-2/tra-2;XX) and male and female intersexes (dsx/dsx;XY and dsx/dsx;XX) indicate that the effect of these sex-transforming mutants are not only limited to X chromosomal replication.

This work is supported by a U.G.C. junior research fellowship to Debasish Mutsuddi.

References: Baker, B.S. & K.A.Ridge 1980, Genetics 91:383-423; Bridges, C.B. 1932, In: Sex and Internal Secretions, Williams & Williams: 55-63; Komma, D.J. 1966, Genetics 54:497-503; Muller, H.J. 1950, Harvey Lecture Series 43:165-229; Smith, P.D. & J.C.Lucchesi 1969, Genetics 61:607-618.

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X-chromosome replication in Drosophila.

The majority of the existing evidences on the replicative and transcriptive activity of the salivary gland chromosomes in sex-specific lethals (Belote & Lucchesi 1980; Lucchesi & Skripski 1981; Ghosh et al. 1981), different karyotypic conditions (Maroni & Plaut 1973; Annaniev & Gvozdev 1975; Lucchesi 1977) as well as different Drosophila species (Mukherjee & Beermann 1965; Lakhotia & Mukherjee 1970, 1972; Abraham & Lucchesi 1973; Mukherjee & Chatterjee 1976; Das et al. 1982) points to an intriguing relationship between relative diameter, level of transcriptive activity and duration of replication of the X chromosome. Still today, several models have been proposed (Mukherjee 1974; Lucchesi 1977; Davidson & Britten 1979; Mukherjee 1982) to explain the regulatory

Fig. 1. Autoradiograms showing ^{3}H-TdR labelling on X chromosomes in comparison to the pattern on the autosome in (a) 1.62 X chromosomal segment (b) 2.15 X chromosomal segment and (c) in metafemale (3X;2A). A=autosome, X=X chromosome. Arrow indicating the break points.

mechanism involved in X chromosome transcription, but the regulation of other two process is still obscure. The present investigation have been undertaken to explore the regulatory mechanism involved in X chromosome replication.

In our present study, different X chromosomal segmental aneuploids and hyperploids (see Stewart & Merriam 1975; Maroni & Lucchesi 1980) were constructed from different X;Y translocation stocks (T(X;Y) B44, J8 and B 29, Stewart & Merriam 1975). Metafemales (3X;2A) were produced by crossing attached-X females (C(1)RM, y pn/Y) to wild type males. Replication pattern of the salivary gland chromosomes were studied after pulse labelling with ^{3}H-thymidine (specific activity: 17,400 μCi/mM, BARC, Trombay, India; cons. 500 μCi/ml, exposure time 20 days).

Our results reveal that in individuals with 1.50 and 1.62 X chromosomal segments, each part of X chromosome (regardless haplo or diplo) is puffy, pale stained and early replicating (Fig. 1a). On the other hand, in all the aneuploid and hyperploids (from 1.85 to 2.85 X chromosomal segments), each part of X chromosome (regardless haplo, diplo or triplo) is narrower, intensely stained and exhibits synchronous pattern of replication with the autosomes (Fig. 1b). Interestingly, in metafemales (3X;2A), where per gene transcriptive activity is even lower than that of their diploid sisters (Lucchesi et al. 1974), individual (haplo) X chromosome is equal in diameter with that of individual (haplo) autosome and the X chromosome always replicate synchronously with the autosomes (Fig. 1c). Therefore, from all the above results, we would like to propose: (1) X chromosomal replication pattern, like transcription, does not depend upon X/A ratio, and (2) X chromosomal replication pattern always depend upon the relative diameter of the individual X chromosome, i.e., condensation or decondensation of X chromatin materials.

This work is supported by a U.G.C. minor research project to Debasish Mutsuddi.

References: Abraham, I. & J.C.Lucchesi 1973, Genetics 74:52; Ananiev, E.V. & V.A. Gvozdev 1975, Chromosoma (Berl.) 49:233-241; Belote, J.M. & J.C.Lucchesi 1980 Nature 285:573-575; Das, M., D.Mutsuddi, A.K.Duttagupta & A.S.Mukherjee, Chromosoma (Berl.)1982 87:373-388; Davidson, E.H. & R.J.Britten 1979, Science 204:1052-1059; Ghosh, S., A.K.Duttagupta & A.S. Mukherjee 1981, Proc.V Cell.Biol.Conference, Bangalore:20; Lakhotia, S.C. & A.S.Mukherjee 1970, J.Cell.Biol. 47:18-33; _____ & ____ 1972, Proc.Zool.Soc.(Cal.) 25:1-9; Lucchesi, J.C. 1977, Amer.Zool. 17:685-693; Lucchesi, J.C., J.M.Rawls & G.Maroni 1974, Nature 248:564-567; ____ & T.Skripski 1981, Chromosoma (Berl.) 82:217-227; Maroni, G. & W.Plaut 1973, Chromosoma (Berl.) 40:361-377; ____ & J.C.Lucchesi 1980, Chromosoma (Berl.) 77:253-261; Mukherjee, A.S. 1974, The nucleus 17:183-199; ____ 1982, Current Science 51:205-212; ____ & W.Beermann 1965, Nature 207:785-786; ____ & S.N.Chatterjee 1976, J.Microscopy 106:199-208; Stewart, B.R. & J.R.Merriam 1975, Genetics 79:635-647.

Duttagupta, A. and I.Roy. University of Calcutta, India. Isolation of nascent DNA from polytene chromosomes of Drosophila melanogaster.

The polytene chromosomes of Diptera offer a possibility to reveal whether ^{3}H-TdR labelling pattern of the chromosome could be correlated with the size of the replication unit (Lonn 1980). The aim of the work presented here was to investigate whether nascent DNA fragments can be selectively released from polytene chromosomes during cell lysis. If so, the size of the nascent DNA could give some insight into what particular replication pattern may be mean at the replicon level.

Third instar larvae of Drosophila melanogaster (giant) were used as the experimental material. Salivary glands were labelled with ^{3}H-thymidine (77.2 Ci/m mole) for four hours and then transferred to a neutral non-denaturing lytic buffer. After 12 hours in lytic buffer at 20°C the lysate was transferred directly on to a polyacrylamide gel (5% concentration). The gel was run at 20 volts for four hours at room temperature and then stained with ethidium bromide. On visualization with a U.V. lamp a single band 6 mm from the top of the gel well was observed. The gel was sliced into 3 mm pieces and each piece was subjected to elution buffer individually, overnight in a 37°C water bath. The DNA was precipitated out with ethanol and lyophilized. Scintillation fluid (4% omniflouer in toluene) was added and the count taken on a Packard Scintillation Counter.

On taking the count two distinct peaks (Table 1) were obtained, with none in between. The first peak corresponded to the band visualized with U.V. The second peak corresponded to a position, right at the bottom of the gel, slightly smaller than the first one.

Table 1. Results of scintillation count of DNA isolated from the larval salivary gland of giant Drosophila melanogaster.

POSITIVE ID	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
TIME (min.)	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
GREEN CPM	52	63	175	108	44	37	49	51	44	52	63	37	23	72	140

Since lysis has been carried out in a neutral non-denaturing buffer and great care has been taken to avoid artefactural shearing, we have good reason to believe that the second peak corresponds to nascent DNA.

Reference: Lönn, U. 1980, Chromosoma 77:29-40.

Eggleston, P. University of Liverpool, Great Britain. Correlation in the induction and response of SF and GD sterility.

The occurrence of specific genetic aberrations in the progeny of certain outcrosses in Drosophila melanogaster is well documented. The abnormalities, which include reduced egg hatchability (SF sterility) and reduced egg production (GD sterility) have been referred to collectively as "hybrid dysgenesis." It has been argued that two independent interactive systems contribute to the hybrid dysgenesis syndrome (Kidwell 1979). These are the I-R system (usually detected by the presence of SF sterility) and the P-M system (usually detected by the presence of GD

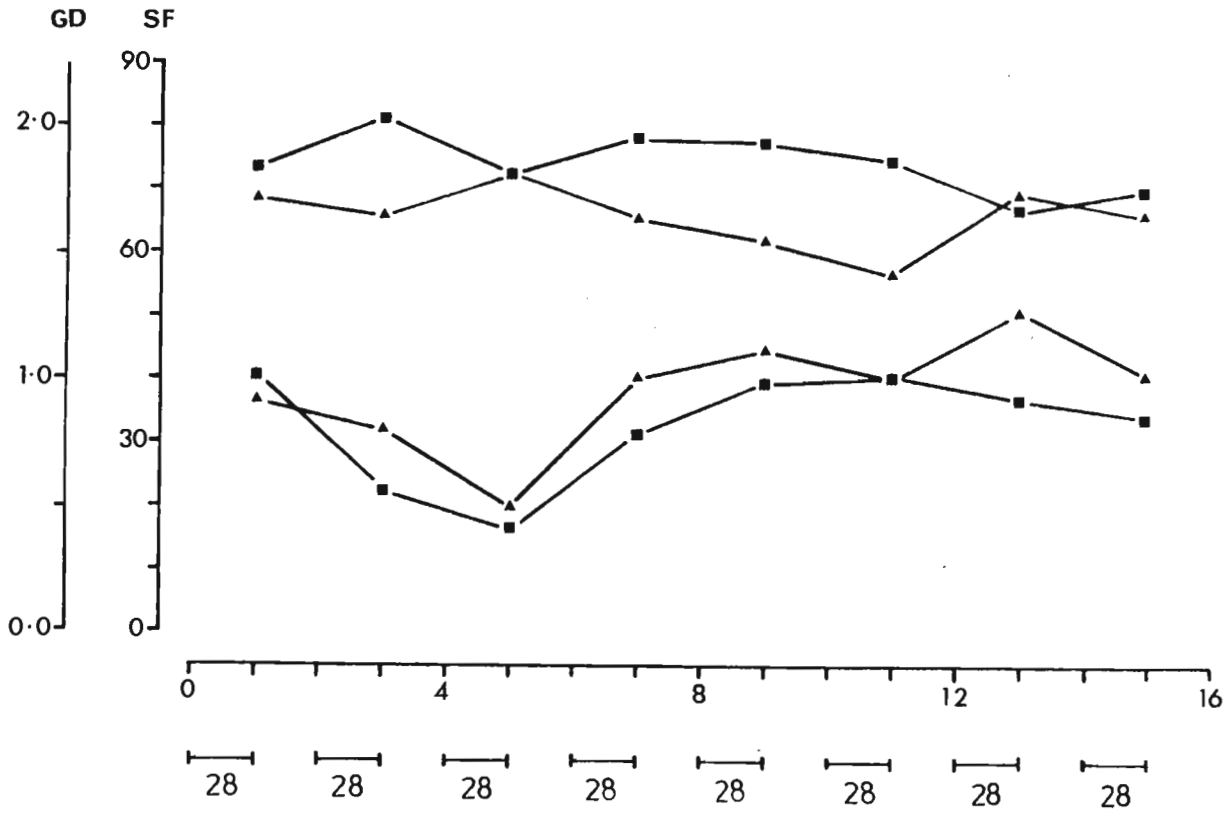


Fig. 1. Mean percentage egg hatchability or SF score (■) transformed to angles and mean egg production or GD score (▲) transformed to square roots of the number of eggs laid per female per hour for both Cross A and Cross B. The x-axis shows developmental age at 18°C and the onset and duration of the 28°C pulse treatments is indicated by the black bars.

sterility). The two systems as described, however, have remarkable similarities and there is experimental evidence to suggest that any apparent independence may well be an artefact of the measurement techniques used (Eggleston & Kearsey 1980).

Dysgenic traits tend to occur in only one of a pair of reciprocal crosses, namely that in which the female carries a specific cytoplasmic state which mobilises a family of genetic elements present within the male genome (Cross A). The subsequent transposition, excision and integration of these inducing elements is thought to be resposible for the various aberrations observed in the progeny. The reciprocal cross (Cross B) produces normal progeny. It is generally held that all dysgenic traits display a sensitivity to developmental temperature in that their expression is negligible at 18°C but maximised at about 28°C. The following experiment reveals a strong correlation in the induction of SF and GD sterility and in their response to changes in developmental temperature. The extent of this correlation suggests that the two traits are causally dependent.

Reciprocal crosses were made between a marker stock known to have a reactive cytoplasm (y bw st (A)) and a wild type inbred line from the Texas population (TEX 1) which was known to carry inducing elements. Approximately 10,000 eggs were collected from the parents of each cross over a period of a few hours in order to maximise developmental synchrony. These were transferred at a density of 100 per vial to 80 vials for each of Cross A and Cross B. All 160 vials were individually randomised and incubated at 18°C. Every two days, 10 replicate cultures were sampled from each cross and placed at 28°C for 24 hours. Afterwards they were returned to complete their development at 18°C. Each culture received only one pulse treatment so that eventually, for each cross, eight sets of 10 replicate cultures had been subjected to a 28°C treatment at successive developmental stages. The effect of this high temperature pulse on sterility induction could therefore be monitored throughout the life cycle. Upon emergence, the female progeny were assessed for egg production and hatchability as described by Eggleston & Kearsey (1980).

The response of each cross is shown in Figure 1 where mean egg hatchability (SF score) and mean egg production (GD score) are plotted against the developmental age at which the 28°C treatment was administered. As might be expected, the developing Cross B progeny were unaffected by exposure to high temperatures, returning an overall mean hatchability of 92.7% and an overall mean egg production of 2.69 eggs per female per hour. An analysis of variance revealed no significant differences between the eight sets of Cross B progeny for either trait (Table 1a,b). The response of the developing Cross A progeny, however, is evident from the substantially reduced SF and GD scores (Figure 1). It can be seen that the 28°C treatment brings about a clear reduction in the Cross A egg production and hatchability regardless of the developmental stage at which it occurs. Certain stages, however, particularly the early larval stages (days 1-5), have a higher sensitivity as indicated by the greater depression in SF and GD scores. This increased sensitivity is maximal towards the end of the second larval instar (day 5) after which it declines, resulting in a slight increase to both hatchability and egg production. Differential sensitivity throughout the life cycle is reflected in the analysis of variance which reveals highly significant differences between the eight sets of Cross A progeny for both SF and GD score (Table 1a,b).

Table 1. (a) Analysis of variance of the SF scores. The percentage egg hatchability for each replicate culture was determined from a sample of 25 eggs.
(b) Analysis of variance of the GD scores. The egg production of each of the replicate cultures was scored on two occasions.

			Cross A			Cross B		
	Item	df	MS	F	P	MS	F	P
a)	Between Sets	7	806.64	7.30	<0.001	224.68	2.04	>0.05
	Within Sets	72	110.54			109.91		
	Total	79						
b)	Between Sets	7	1.11	7.16	<0.001	0.30	1.71	>0.05
	Between Occ.	1	0.57	3.66	>0.05	0.03	0.15	>0.05
	Sets x Occ.	7	0.07	0.44	>0.05	0.23	1.33	>0.05
	Error	144	0.15			0.17		
	Total	159						

Perhaps the most striking feature of this experiment is the extraordinary similarity in the observed SF and GD scores for Cross A throughout the life cycle. The correlation between the two characters is strong (r=0.82) suggesting that, on average, 67% of the GD variance is directly dependent on the variation among SF scores (r^2=0.67). The correlation would be even stronger if the sharp increase in egg production seen during the pupal phase (days 11-13) could be excluded. This increase in egg production is not matched by an increase in hatchability and a similar effect has been reported previously (Eggleston & Kearsey 1980). It seems likely that this phenomenon is due to a temperature shock occurring during meiosis. Such shocks are known to increase both recombination and DNA replication (Grell 1972) and this may result in an increased egg production. This effect would appear to be independent of the hybrid dysgenesis syndrome since it occurs to an equal extent in the developing Cross B progeny. The results of this and similar experiments reveal a remarkable similarity in the response of SF and GD sterility to changes of developmental temperature. Such a degree of similarity would be unlikely to occur if the two traits were under independent genetical control. It may well be that SF and GD sterility and therefore I-R and P-M hybrid dysgenesis are, in fact, causally dependent and that the same nuclear-cytoplasmic interaction is responsible for all of the dysgenic traits which can be induced in a cross of this kind.

References: Eggleston, P. & M.J.Kearsey 1980, Heredity 44:237; Grell, R.F. 1972, Genetics 73:87; Kidwell, M.G. 1979, Genet.Res. (Camb.) 33:205.

Ehrman, L. and D.Baumann-Meringolo. State University of New York, Purchase, New York USNA. Courtship followed by rare D.pseudoobscura male matings.

To determine if the rare male advantage (observed in eight species of Drosophila [see Ehrman & Probber 1978; Meringolo et al. 1982]) is the result of the females' preference for males of a type different from tnat by which they are first courted (Spiess & Schwer 1978; Spiess 1982) D.pseudoobscura of the CH and AR strains were tested. (These are highly inbred and were originally reported by Ehrman et al. 1965.)

A profoundly modified direct observation mating chamber was used; the chamber is divided into two equal compartments by a removable, rotatable barrier made of fine wire mesh. Groups tested were of the following composition: 13 females plus 39 males--13 minority and 26 majority types. Males were marked (in half of the groups the rare type was marked, and in the other half, the majority type male was marked) by placing a small drop of white liquid paper on the dorsal thorax. First, with the barrier in place, the 13 females plus 13 males (either rare or majority) were placed in one half of the chamber, with the remaining 26 males in the other half of the chamber. The males were allowed to court but not to mate. After 15-20 min, the barrier was removed, all the files were allowed to mingle, and matings were scored (Table 1).

Table 1. Type of D.pseudoobscura male preferred when females were courted by rare type first, and when females were courted by majority type first. (NM = no mount) (Within each group of females tested, half the time the rare males were CH and half the time they were AR males.)

AR Females		
AR female w/rare males first, then majority introduced:		
AR x rare	33	31.7%
AR x majority	49	47.1
NM	22	21.2
AR female w/majority males first, then rare introduced:		
AR x rare	35	33.7%
AR x majority	41	39.4
NM	28	26.9

CH Females		
CH female with rare males first, then majority introduced:		
CH x rare	29	27.9%
CH x majority	55	52.9
NM	20	19.2
CH female with majority males first, then rare introduced:		
CH x rare	31	29.8%
CH x majority	51	49.0
NM	22	21.2

Table 1 (contin.):

All Females Combined:

Female w/rare male first, then majority introduced:			Female w/majority male first, then rare introduced:		
w/rare male	62	29.8%	w/rare male	66	31.7%
w/majority male	104	50.0	w/majority male	92	44.2
NM	42	20.2	NM	50	24.1

In sum then, with a constant 2 Common:1 Rare male ratio, and with a total of 324 observed matings after observed courtships, when the females were courted by rare males first, they mated with the rare males 37.3% of the time; when courted by majority type first, they mated with rare males 41.8% of the time. Although increases in rare male advantages occur when majority males courted first, a rare male advantage is still maintained when rare males initially court. In addition, note that both AR and CH females award about the same magnitude of rare male advantage. Results therefore indicate that the type of male which courts first influences the subsequent degree of rare male advantage, at least in these strains.

References: Ehrman, L. & J.Probber 1978, Amer.Scient. 66:216; Ehrman, L., B.Spassky, O.Pavlovsky, & Th.Dobzhansky 1965, Evolution 19:337-346; Meringolo, D.B., R.Silibovsky, & L.Ehrman 1982, in: Genetics, Development and Evolution of Drosophila (ed: S.Lakovarra), Plenum Press; Spiess, D.B. 1982, Behavior Genetics 12:209-221; Spiess, D.B. & W.A.Schwer 1978, Behavior Genetics 8:155-168.

Engeln, H. Institute fur Genetik, Freie Universitat Berlin, FRG. Oviposition site preferences in different populations of Drosophila melanogaster.

As Parsons (1978) pointed out habitat selection plays an important role in the evolutionary strategies of organisms and in influencing their fitness in nature. In this context oviposition site preference is an important behavioural trait and has been studied already by several authors (e.g., McKenzie & Parsons 1972; Richmond & Gerking 1978; Fogleman 1979; Krause et al. 1980). For an optimal survival of larvae it is necessary that Drosophila females choose optimal conditions at oviposition sites. One important factor pointing to the quality of the food composition is the amount of ethanol in it. For example the sibling species Drosophila melanogaster and D.simulans differ in their adaptations to environments containing ethanol and occupy different ecological niches when competing in the same area; D.simulans prefers medium without ethanol and is less tolerant to ethanol than D.melanogaster (McKenzie & Parsons 1972). Since differences between species are existing the question arises whether there are different adaptations within one species concerning ethanol preferences.

Three samples of different Drosophila melanogaster populations were used in this experiment (Table 1). The first one (+K, +T, Da, Ma, Pa) involved laboratory populations collected from places located far away from each other (Europe, Africa, South-America). The second one (UI, UII, MP) consisted of fresh captured populations collected from three habitats within Berlin, Germany. The third group (I1, I23, II5, M4, M19) involved five single female lines derived from the Berlin populations of the second sample.

Fifty 3-4 days old non-virgin females were anaesthetized with carbon dioxide for about 5 seconds and then put into a glass cylinder of 11cm diameter and 5.5cm height. Each cylinder contained 4 food copus (3.5cm diam), two of them filled with standard medium (cornmeal, agar, molasses) and two filled with medium including ethanol of 9% by volume (prepared after McKenzie & Parsons 1972). Because the number of eggs laid per time unit varied between strains, flies were allowed to lay eggs for 1-2.5 hours to receive egg densities that were countable. After this period the flies were removed and eggs were counted. Experiments were carried out at light intensities between 250-650 lux and at a temperature of 25±1°C.

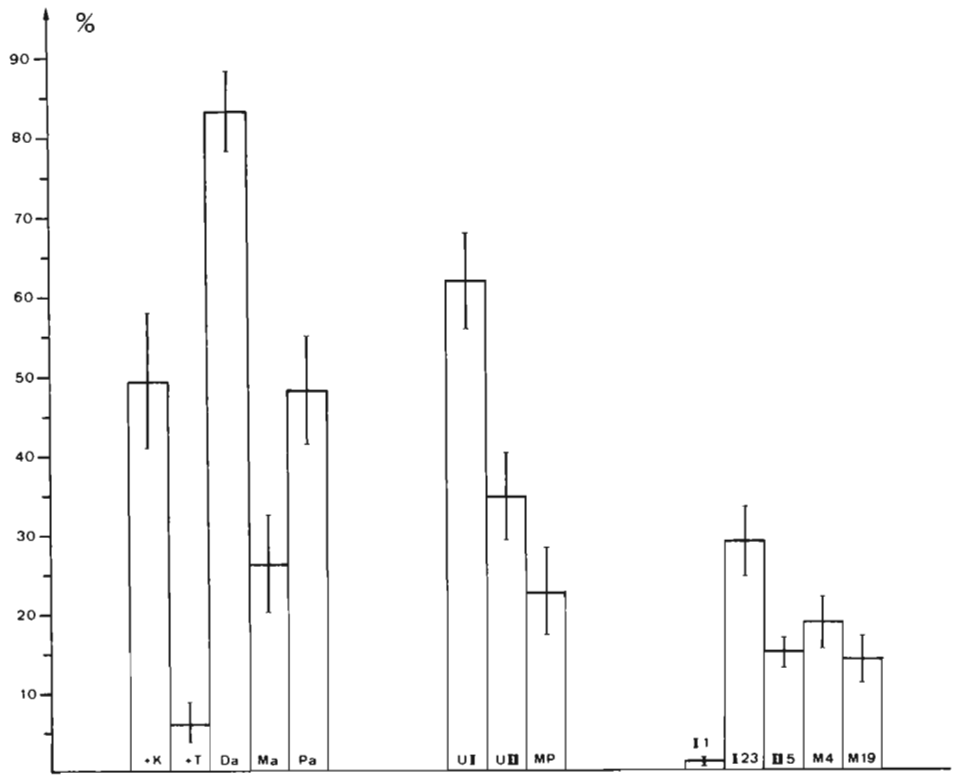
Of each strain the mean percentages of eggs laid on ethanol medium were calculated from 8 replicates each with two glass cylinders. A statistical analysis was performed using the Student-Newman-Keuls-test (SNK-test). The greatest differences exist between laboratory populations originated from places located far away from each other, but there were found also significant differences between populations and between single female lines collected

Table 1. Origin of strains, mean percentages of eggs laid on ethanol medium and homogenous groups by SNK-test.

SNK-test	strain	ethanol(%)	number off eggs	origin, year of capture	
laboratory	+T	5.9	3090	Berlin, Germany	1976
populations	Ma	26.2	3515	La Mancha, Spain	1972
	Pa	48.1	3105	Paramaibo, So.America	1976
	+K	49.2	4076	Berlin, Germany	1930
	Da	83.1	4429	Benin, Africa	1972
fresh	MP	22.7	5924	Berlin-Dahlem	1978
captured	UII	34.8	7646	B.-Wannsee, forest	1978
populations	UI	62.0	7339	B.-Wannsee, garden	1978
single	I1	1.1	3814	UI	
female	M19	14.1	3731	MP	
lines	II5	15.0	2286	UII	
	M4	18.8	5990	MP	
	I23	29.3	7368	UI	

distances: UI-UII 750m; UI-MP 12.5km.

within Berlin (Table 1, Figure 1). There is also considerable variation within strains (see standard error). Krause and coworker (1980) found that females prefer to lay eggs at places being scented by males and del Solar & Palomino (1966) reported that females tend to deposit eggs at sites where eggs are already placed by other females. This could partly explain the differences between replicates within strains.



The great amount of variation between strains was surprising. Richmond & Gerking (1979) obtained preferences for ethanol medium (values >88%) in 4 strains of Drosophila species. McKenzie & Parsons (1972) on the other hand got values of 52% and 54% in two strains of D.melanogaster. Furthermore McKenzie & Parsons (1974) found that populations of D.melanogaster collected in a vineyard were more resistant to ethanol than populations captured outside the vineyard. So it can be concluded that there exists a great amount of variability within the species D.melanogaster concerning oviposition site preferences. As ethanol containing habitats and those without ethanol often will be found in the

Fig. 1. Mean percentages and standard errors of eggs laid on ethanol medium.

neighbourhood, the variability may be a consequence of adaptation to local environment conditions. This suggestion is supported by McKenzie & Parsons (1974) cited above who found that within their vineyard population those strains most closely associated with alcohol in the environment in the cellar were more resistant than strains captured outside the cellar. The considerable difference between the single female lines I1 and I23 derived from the same origin population suggests that there is enough genetic variance within the same population to develop into divergent lines. Single female lines can be viewed as founder populations and a single female being driven to an unoccupied habitat could establish a new population differing in its ethanol preference compared with the origin population. This might be a first step to speciation and so the result of our experiment supports the theory of speciation via founder effect (Mayr 1942; White 1978).

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Falk, R. and S.Baker. The Hebrew University, Jersualem, Israel. Production of centric-autosomal-Y translocations.

The availability of stocks with rearranged autosomes, such that one autosomal arm is attached to its homologue (compound arm) and the other arm is free, e.g., C(2L)/F(2R) and F(2L)/C(2R) stocks, makes the screening for translocations between chromosome-Y and the centric heterochromatin of autosomes (centric autosomal-Y translocations: CAYT) a straight forward procedure. Males with a Y-chromosome marked at both ends ($\underline{B}^S Y^{L\circ} Y^S \underline{y}^+$) and a marked chromosome 2 (dp b cn bw) were irradiated and then mated to females with C(1)DX,yf X-chromosomes and a dominantly inverted marked chromosome-2, In(2LR)CyO, $\underline{dp}^{\ell v1}$ Cy pr cn^2. All y^+ B^S Cy cn^2 daughters were mated either to C(2L)RM/F(2R)bw males or to F(2L)dp/C(2R)RM,cn males. No progeny were expected from the great majority of these daughters, which were C(1)DX,y f/B^SY y^+; CyO/dpb cn bw. Only daughters that carried centric-autosomal translocations with the Y-chromosome--i.e., they were C(1)DX y f/O; CyO/$T_{Y;2}$ $\underline{y}^+$ $\underline{B}^S$ -- or with chromosome-4 were fertile (unless gametes of rare autosomal non-disjuntion in both parents happend to complement each other in the zygote). Since newly induced translocations were expected only rarely, it was not necessary to mate the F_1 females individually, and up to 10 females were mated to the appropriate males in some culture bottles. The results of four translocation-induction experiments are given in Table 1.

Table 1.

Expt. No.	X-ray dose to ♂♂	No. F_1 ♀♀	Translocations recovered with tester: F(2L)dp/C(2R)RM,cn No. ♀♀ tested	F(2L)dp/C(2R)RM,cn fertile cultures	F(2L)dp/C(2R)RM,cn CAYT	C(2L)RM/F(2R),bw No. ♀♀ tested	C(2L)RM/F(2R),bw fertile cultures	C(2L)RM/F(2R),bw CAYT
I	3500R	30	14	1	0	16	1	0
II	3500R	48	25	3	0	23	3	1
III	3000R	930	-	-	-	930	8	5
IV	3000R	2410	2410	16	11	-	-	-

In each experiment about 1000 irradiated males were mated to an excess of females for 6 days in 25 culture bottles. Flies were transferred twice to fresh culture bottles. In Expt. I and II F_1 females were mated individually. In Expt. III most females were mated in groups of 4-10 per culture bottle. In Expt. IV all females were mated in groups of 10 per culture bottle.

About 0.7% of the F_1 paternal gametes irradiated with a dose of 3000R carried the expected centric autosomal translocations, all with the Y-chromosome. It appears that with the slightly higher dose of 3500R as many as 10% of the irradiated sperm carried CAYT, and that the increase in radiation dose caused also a steep increase in the frequency of many other chromosomal aberrations, that resulted in dominant lethality, thus allowing the recovery of only a few F_1 daughters.

Farmer, J.L. Brigham Young University, Provo, Utah USNA. Expression of ey in Drosophila pseudoobscura.

The eyeless mutation (ey) was found in a wild population by Bryant (1980). When I obtained the stock from the center at Austin, Texas, the penetrance of ey was extremely low compared to the value reported by Bryant (1980). Since no other laboratory had the stock, I tried to increase the penetrance by selective breeding. Single-pair matings and close inbreeding of progeny did produce a few flies which were unilaterally eyeless, but when these progeny were interbred, they had a very low fertility and a stock could not be established. Backcrosses of the unilaterally eyeless flies with their wild-type sibs produced a few progeny with the same phenotype, but they also were infertile in crosses with each other.

In an attempt to overcome the infertility, I outcrossed the unilaterally eyeless flies with a vigorous wild-type stock which carried the TL inversion (obtained from W.W.Anderson). As expected, the F1 was all wild-type and had a high fertility. (The fertility remained high through all succeeding generations.) Unexpectedly, the F2 was also all wild-type. The F3 produced a few eyeless flies (approx. 5%) from both single-pair matings and from mass matings.

After four generations of intensive selection and closeinbreeding of only completely eyeless flies (no facets and no detectable pigment below the integument in the normal position of the eyes) penetrance was higher. At 25°C about 5 to 10% are completely eyeless, about 5 to 10% are nearly completely eyeless (ranging from a single facet to a small number of facets on one or both sides or patches of pigment beneath the integument without facets), with the remainder about equally divided between unilaterally eyeless flies (with the same range of expressivity noted above) and wild-type flies. The unilaterally eyeless flies have one eye that is morphologically completely normal except that in many flies the color is duller than wild-type, as though the drosopterins were reduced.

The eyeless phenotype seems to be due to a major gene with modifiers, although further crosses would have to be done to verify that hypothesis.

If eyeless flies are allowed to lay eggs for a short time in a bottle, their progeny eclose in the order: completely eyeless first, wild-type last, other phenotypes in between but strongly overlapping each of the first two phenotypes.

The penetrance of ey is greatly enhanced at 18°C, approaching 100% completely or nearly completely eyeless flies.

The ey stock called SHB-5 which is currently maintained at the Mid-America Drosophila Stock Center (Bowling Green) is the stock which I derived from the crosses described above.

Reference: Bryant, S.H. 1980, DIS 55:212.

Fogleman, J. University of Denver, Colorado USNA. The ability of cactophilic Drosophila to utilize soaked soil as larval substrates.

Both rearing records and aspiration records indicate a very high degree of host plant specificity among the cactophilic Drosophila of the Sonoran Desert (Fellows & Heed 1972) with little species overlap (Heed 1978). Recently, investigations into the ecology of D.mettleri have shown that it utilizes a greater variety of substrates than had been previously thought. In addition to its normal substrates of soil which has been soaked by saguaro or cardon rot exudate, D.mettleri can tolerate the alkaloids in senita cactus that have been shown to be toxic to all other species tested except the resident species, D.pachea (Kircher et al. 1967; Fogleman et al. 1982). Field experiments have demonstrated that D.mettleri will use soil which has been soaked with senita rot juice as a breeding substrate when available. D.mettleri has also been reared from organpipe soaked soil (Fogleman at al. 1981).

D.mettleri is a rather unusual drosophilid in that it is one of only two species that have been reported as utilizing soaked soils as breeding substrates. The other species, D.heedi, is found in a xeric region on the island of Hawaii (Kaneshiro et al. 1973). The closest relative to D.mettleri is D.eremophila which has been found associated with cactus (Opuntia pads) but never reared from them (Heed 1977). The similarity between mettleri and eremophila in both adult and larval morphology has led to the speculation that eremophila is also a soil breeder. The experiments reported here were designed to answer two questions: (1) can D.eremophila (like mettleri) utilize soaked soil as a larval substrate, and (2) do any of the other cactophilic Drosophila endemic to the Sonoran Desert (nigrospiracula, mojavensis, and pachea) possess the capability of utilizing soaked soil substrates.

Experiments were performed in plastic petri dishes (100 x 15 mm). Cactus and soaked soil substrates were produced as follows: frozen cactus tissue was thawed, homogenized in a blender, autoclaved, and innoculated with a suspension of 10 of the major cactophilic yeasts plus the bacterium, Erwinia carnegieana. After one week, petri plates were filled with 25gm of the necrotic tissue and the remainder was filtered to provide the juice used to soak soil samples. Forty grams of soaked soil per plate (15% moisture by weight) was approximately the same volume as 25gm of cactus. The weight of each substrate plate was recorded and maintained throughout the experiment by periodic addition of more juice. 100 first instar larvae (24 hr old) of one of the 5 Drosophila species were transferred to each plate (5 replicates/test except where noted). The results are shown in Table 1.

Table 1. Average viability (in percent ± standard deviation) of five species of cactophilic Drosophila.

	DROSOPHILA SPECIES				
SUBSTRATE	NIGRO.[1,2]	METTLERI	EREM.	MOJ.	PACHEA
AGRIA	***	24.6±7.2	54.6±13.9	73.0± 5.7	***
AGRIA SOIL	***	1.9±3.9	0.3± 0.9	0.1± 0.3	***
ORGANPIPE	***	71.8±8.1	76.8± 6.5	77.2±13.0	***
ORGANPIPE SOIL	***	3.0±2.1	4.8± 2.8	4.8± 5.2	***
SAGUARO	68.4±9.8	78.0±4.1	76.4± 7.2	---	***
SAGUARO SOIL	0.4±0.4	30.0±4.7	11.0± 2.8	---	***
SENITA	***	15.8±3.5	-0-	***	71.4±6.0
SENITA SOIL	***	7.8±4.3	3.4± 1.4	***	0.6±1.9[1]

1= ten replications/test. 2= data from Fogleman et al. 1982.
--- = test not performed-viability unknown.
*** = test not performed-viability presumed to be zero based on previous studies.

From the data in this table, the average viability of a species in its typical cactus substrate is about 75%. Less than 5% viability can probably be considered a negative response. As such, it seems reasonable to conclude that mojavensis, pachea, and nigrospiracula cannot utilize soil which has been soaked by rot juice from their normal cactus host. Also, agria soaked soil appears to be a particularly inhospitable substrate. It is important to point out, however, that the viabilities measured in the soil substrates might be generally depressed, perhaps due to overcrowding. A previous report of the viability of mettleri in saguaro soil (lab experiments, 10 reps) was 57% (Fogleman et al. 1982) compared to the 30% measured here. In addition, mettleri has been reared from both senita soil and organpipe soil and yet the measured viabilities are very low. Therefore, conditions used in these experiments may have been more stringent than those in nature, and the date in Table 1 should be viewed as reflections rather than estimates of viabilities in natural substrates.

A two way ANOVA using arcsin transformed viability data of mettleri and eremophila on the various substrates showed that these two species are signficantly different ($F=161.09$; $df=7.74$; $P<0.001$). A highly significant species x substrate interaction was also evident. Nevertheless, one can compare the mean viability of an individual species. e.g., mettleri or

eremophila, on one substrate to the mean viability of all species averaged over all soil substrates (excluding mettleri-saguaro soil and the individual case involved in the comparison). These comparisons indicate that the viabilities of mettleri in senita soil and eremophila in saguaro soil are significantly higher than the overall average viability in soils (mettleri: $F=4.683$; $df=1.13$; $P<0.05$ -- eremophila: $F=21.514$; $df=1.13$; $P<0.001$). This supports the statement that eremophila, like mettleri and unlike the rest of the cactophilic Drosophila, can use certain soaked soil substrates.

Substantiation of the use of soil substrates by eremophila could take the form of collections of soaked soils from under Opuntia and other cacti in areas of Mexico where eremophila is common and/or laboratory experiments which test the behavioral preference of eremophila for ovipositing on soaked soil versus cactus.

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Gartner, L.P. University of Maryland, Baltimore, Maryland USNA. The effects of 4000 and 8000 R of X-irradiation on Drosophila life span.

Numerous studies have demonstrated that high doses of ionizing radiation have deleterious effects on Drosophila life span (see review: Ducoff 1975). Interestingly, the amount of radiation necessary for such effects vary among the various laboratories. Some studies, using doses as high as 40 kR were unable to demonstrate life span shortening (Blair & Baxter 1970; Sonnenblick & Gartner 1967; Gartner 1973a; Westerman & Parsons 1972), while others, utilizing doses in the 20 kR range, reported considerable life span shortening (DeReggi 1975; Giess & Planel 1977). Still others claim that low and very low doses of radiation increased life span of Drosophila (Strehler 1959, 1962, 1964; Nothel, 1965; Lamb 1964, 1965), and these claims have been seriously questioned (Sonnenblick & Grodis 1963; Blair & Baxter 1970; Atlan, Miquel & Welch 1970). The purpose of the present investigation was to examine the effects of very low doses of X-irradiation on adult Drosophila melanogaster.

Oregon-R strain of Drosophila melanogaster was housed in a mass bred situation in pint sized milk bottles, on a corn meal-molasses-agar medium (Gartner 1973b), at 20°C. Experimental populations, derived from young parents, were placed in shell vials, five males and five females per vial. Flies were irradiated on the fourth day of imaginal life with 250 kVcp X-rays (15 mA, HVL-0.94mm Cu + 3mm Al) at 1332.5 R/min, for a total exposure of 0, 4000 and 8000 R. Subsequent to radiation, the flies were monitored daily, five days per week, and deaths were recorded by sex. Flies were transferred to fresh media on a weekly basis.

The results of the present investigation demonstrate that neither 4000 R nor 8000 R of X-irradiation have an appreciable effect on Drosophila imago life span. The unirradiated females of this report had a mean longevity of 90.5 days, while the irradiated females lived for 90.1 (4000 R) and 84.3 days (8000 R). Control males had a mean life span of 81.0 days, while their irradiated counterparts lived for 81.7 and 77.8 days, at 4000 R and 8000 R, respectively. Although females have a consistently longer average life span than males, the maximum length of life was the same for both sexes at all exposures.

Hence, the present study can offer no suggestions as to the prima cause of radiation induced life span prolongation, if indeed such exists. Instead, it must argue against any such life span lengthening effects, and must urge that further studies be initiated to investigate the matter to a greater extent by utilizing other insect species.

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TABLE 1. LIFE SPAN OF FEMALE IMAGOES

Dose (R)	N	Mean Life Span Days ± S.E.	Signif.	Maximum Life Span Days
0 R	47	90.5 ± 4.55	NS	138
4000 R	44	90.1 ± 4.24	NS	134
8000 R	37	84.3 ± 5.03		129

TABLE 2. LIFE SPAN OF MALE IMAGOES

Dose (R)	N	Mean Life Span Days ± S.E.	Signif.	Maximum Life Span Days
0 R	38	81.0 ± 6.24		138
4000 R	41	81.7 ± 5.84	NS	134
8000 R	33	77.8 ± 5.62	NS	127

TABLE 3. LIFE SPAN OF COMBINED POPULATION

Dose (R)	N	Mean Life Span Days ± S.E.	Signif.	Maximum Life Span Days
0 R	85	85.8 ± 3.75		138
4000 R	85	85.9 ± 3.60	NS	134
8000 R	70	81.1 ± 3.71	NS	129

Gauger, A. and G.Schubiger. University of Washington, Seattle, Washington USNA.

A method to screen for Df(3R)P9 homozygotes.

Drosophila melanogaster embryos that are homozygous for a deletion of the bithorax complex Df(3R)P9 die in late embryogenesis or early first instar (Lewis 1978). These animals have an abnormal pattern of ventral denticle belts indicating that all the segments from the anterior metathorax to the seventh abdominal segment have been transformed to mesothorax (Lewis 1978). This transformation is of great interest to both geneticists and developmental biologists. However, to date there has been no method to recognize the homozygous (Df(3R)P9 phenotype until after the cuticle has differentiated. We have observed that Df(3R)P9 embryos incubated at 18°C fail to complete germ band shortening (GBS). Thus this morphological criterion can be used to select for the homozygous mutant class.

We crossed flies of the genotype Df(3R)P9/Dp(3;3)P5;Sb inter se, and collected eggs on agar plates for 30 min or less at 25°C. We then transferred the eggs to 18°C and incubated them until the embryos had begun GBS (about 18 or 19 hr after collection). This corresponds to stage 10 (Bownes 1975) of embryonic development. They were then dechorionated on double-stick tape, covered with paraffin oil (Baker), and observed under a dissecting scope. A total of 258 embryos were observed. 199 (77%) of the embryos we observed developed normally, completing GBS, head involution and dorsal closure (Turner & Mahowald 1979). However 59 (23%) of the embryos never completed GBS, even though other developmental processes such as head involution were normal (Figure 1, Table 1). We separated those embryos with incomplete GBS from those with complete GBS and allowed both classes to continue embryogenesis at room temperature. It should be noted here that the two classes are indistinguishable prior to and during GBS, so we waited until after the normally developing embryos had completed GBS to separate out the incomplete GBS class.

Most of the complete GBS class continued to develop and hatched normally. We assumed that all of the larvae that hatched successfully were heterozygotes and phenotypically wild

Figure 1. Morphology of Df(3R)P9 complete and incomplete GBS embryos. Df(3R)P9 embryos of the same age were dechorionated and observed by microscope. The majority of embryos (A) completed GBS, while approximately one quarter (B) exhibited incomplete GBS. (x 960)

Table 1. Assortment of embryos with respect to germ band shortening and ventral belt pattern.

Class	Ventral Belt Pattern	
	Wild Type	Df(3R)P9
Complete GBS	197	2
Incomplete GBS	4	55

type. Those that failed to hatch, as well as some of the hatched larvae, were mounted for phase contrast microscopy in lactic acid and ethanol (Lewis 1978), and their denticle belt pattern examined. Only 2 of the complete GBS class examined had the Df(3R)P9 phenotype; these may have been overlooked in the bulk screening procedure. We also mounted all of the incomplete GBS animals. All but 3 of these embryos failed to hatch. 55 out of 59 expressed the Df(3R)P9 phenotype; the other 4 animals had a wild type belt pattern. These could have been incorrectly identified during the screening procedure if they were younger than the other embryos.

This screening method should only be performed on embryos incubated at 18°C, since the results are not as clean when they have been incubated at 25°C. However, incomplete GBS has also been observed in another Df(3R)P9 stock that is marked with multiple wing hair. This indicates that the incomplete GBS phenotype may be used to screen for Df(3R)P9 homozygotes in all Df(3R)P9 stocks.

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Gerasimova, T.I.[1] and Yu.V.Ilyin[2] [1]Institute of Molecular Genetics, USSR Academy of Sciences, [2]Institute of Molecular Biology, USSR Academy of Sciences. The role of the mobile element mdg4 in the formation of unstable cut mutations in Drosophila melanogaster.

Earlier, an unstable ct^{MR2} allele was obtained in a cross of Oregon-R females and MRh12/Cy males under hybrid dysgenesis (Gerasimove 1981) (the MRh12/Cy genome contains multiple copies of the P-element). This mutation was characterized in the homozygous stock by a high frequency of reversions, the occurrence of new unstable visible and lethal mutations and super-unstable ct mutations, as well as the emergence of new mutations at other loci in the X-chromosome (Gerasimova 1981, 1982). In the present study, we look into the molecular nature of the mobile element integrated at the cut locus in the ct^{MR2} mutant. To this end, we have carried out in situ hybridization on crushed salivary-gland chromosomes of ct^{MR2} larvae, using the standard procedure (Ilyin et al. 1978). In our hybridization assays, we used plasmid DNA (labeled with ^{3}H and ^{125}I) containing the following elements: mdg1, mdg2, mdg3, mdg4 (Tchurikov et al. 1981), copia (Finnegan et al. 1978) obtained from D.Finnegan, fb elements obtained from S.Potter (Potter et al. 1980), and P-element from G.M.Rubin (Rubin & Spradling 1982). The cut locus is known to be located in the 7B region of the X-chromosome. Mdg4 is the only one of the above-listed elements that hybridizes with the 7B region in the ct^{MR2} mutant. This is a typical mdg which contains direct and inverted repeats at the edges. The total length of mdg4 is 7 kb. Its structure has been revealed earlier by Yu.V.Ilyin. In situ hybridization was performed with different subfragments of mdg4. The picture was the same: the label was invariably found in 7B. At the same time the original Oregon stock, whence ct^{MR2} was derived, does not contain mdg4 in the 7B region (or anywhere in the X-chromosome).

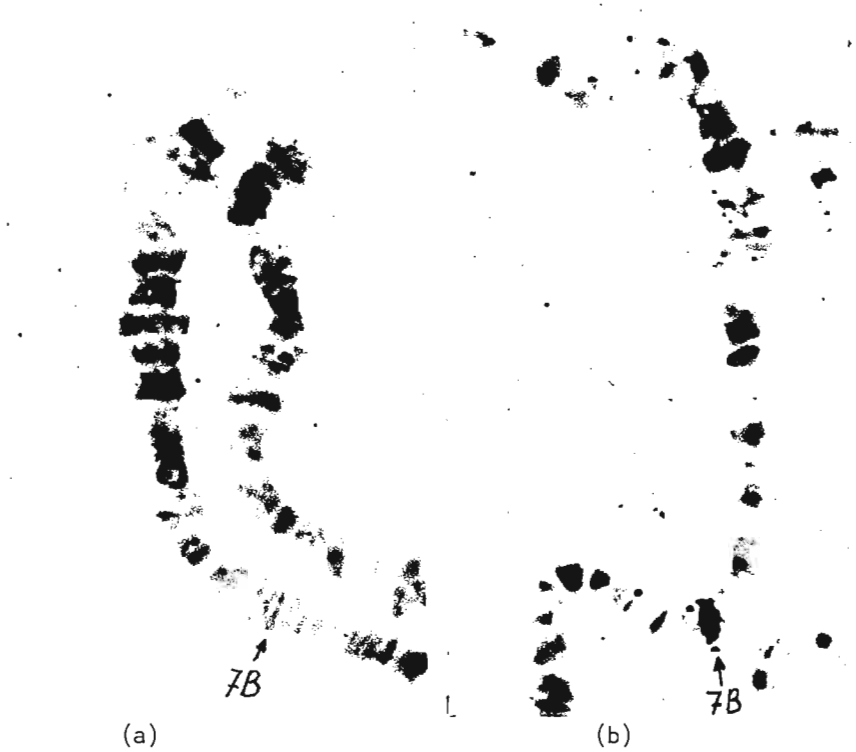

Figure 1. Results of the in situ hybridication DNA mdg4 with polytene chromosomes: (a) stable revertant $ct^{+}sn^{17}$, (b) unstable ct mutant - ct^{pn10}.

To test the connection between mdg4 and the instability of the ct^{MR2} allele, we carried out the hybridization of mdg4 and a wide range of different mutants and revertants derived from ct^{MR2}. In all our tests (nine altogether) of stable reversions, mdg4 proved to have disappeared from the 7B region (Fig. 1a), whereas in unstable ct alleles differing from ct^{MR2} in phenotype, including the unstable ct lethals (a total of 12 different mutations were analyzed) mdg4 was retained in the 7B region (Fig. 1b). These results prove the connection between the unstable ct^{MR2} mutation and the integration of mdg4 at the cut locus. The emergence of new ct mutations is probably due to the displacement of mdg4 within the cut locus. The point is that the new mutations affect different parts of the locus, including its "regulatory" and "structural" regions (Gerasimova 1981, 1982). The occurence of new mutations cannot be attributed to legitimate recombination, since all types of unstable ct mutations and ct^{+} revertants emerged in clusters, i.e., at the premiotic stage. Never in all our assays did we observe mdg4 in other remote loci of the X-chromosome, even when it "left" the 7B region. Mdg4 is probably transferred to long distance with a lower probability. Another possibility would consist in the existence of a limited number of sites preferred by mdg4. Indeed, mdg4 is an mdg of few copies. In Oregon-R, it is only present in two copies in the autosomes, and in the chromocentre.

The instability of the ct^{MR2} mutation was maintained in a homozygous stock for 1.5 years, 50 generations. The frequency of reversions to the wild type remained the same: 1.5×10^{-3}. Then, however, the reversions died down dramatically to a near-zero level. One of the possible explanations would be the loss of full P-elements, which seem to be responsible for the synthesis of transformation enzymes. Therefore, we crossed ct^{MR2}/ct^{MR2} females (two copies of the P-element) to MRh12/Cy males (multiple copies of the P-element). The reversion frequency went up as far as 8×10^{-3} in the progeny. Here, again mdg4 disappeared from the 7B region. Thus, the migration of mdg4 in the ct^{MR4} allele can be controlled.

The existence of such a genetically chracterized system along with a cloned mobile element offers good opportunities for the study of mdg transposition mechanisms and their effects on the target genes.

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Gerasimova, T.I.[1] and Yu.V.Ilyin.[2] [1]Institute of Molecular Genetics, [2]Institute of Molecular Biology, USSR Academy of Sciences. Transpositions of various mobile elements and their relation of unstable mutagenesis in Drosophila melanogaster.

It has been shown earlier that the instability of ct^{MR2} is due to the integration of the mobile element mdg4 in the 7B region, i.e., the region of the cut locus (Gerasimova 1981). The formation of stable ct^{+} revertants in a homozygous ct^{MR2} stock was often accompanied by new unstable mutations in other X-chromosome loci: y, w, cm, sn, g, m, with a frequency of $1\text{-}8\times10^{-4}$. While mdg4 left the region of the cut locus, it was not detected elsewhere in the X-chromosome. Hence the emergence of new unstable mutations involves the integration of other mobile elements in the loci concerned. This was tested for the P-element. To that end, the polytene chromosomes of ct^{MR2} larvae and stable revertants carrying new mutations were subjected to in situ hybridization with the ^{3}H-DNA of plasmids containing mdg4 and the P-element obtained from D.Rubin (Rubin et al. 1982). The standard hybridization procedure was used (Ilyin et al. 1978). The results are listed in Table 1. The X-chromosome of the ct^{MR2} mutant has a hybridization site for the P-element only in 17C. Yet, the 17C region does not contain the P-element itself, only the flanking sequences cloned together with the P-element (Rubin et al. 1982). The formation of stable revertants involves the removal of mdg4 from the 7B region and the appearance of new copies of the P-element in the X-chromosome. Some new mutations are associated with the integration of the P-element in the loci concerned. For instance, the revertants carrying mutations in the sn locus, $ct^{+}sn^{MR2}$ and $ct^{+}sn^{110}$, were characterized by the appearance of the P-element in 2-4 new sites, including the 7D region which corresponds to the sn locus (Fig. 1). For a revertant with a w^{1} mutation, the P-element appeared in the 3C region, again corresponding to the locus concerned. Thus, the excision of mdg4 from the 7B region in the case of stable ct^{+} revertants is often accompanied by a mobilization of the P-element. Its integration in the regions of the w and sn loci is the cause of these unstable mutations. However, in the case of other unstable mutations: y, cm, g, m, the P-element was not detected in the relevant regions. The emergence of these mutations is probably associated with the transposition of other mobile elements that are activized at the same time as mdg4 and the P-element.

Table 1. Results of in situ hybridization of ^{3}H-DNA of the P-element and mdg4 with X-chromosomes of the ct^{MR2} mutant and its derivatives.

Mutations	Hybridization sites for the P-element	for mdg4
ct^{MR2}	17C	17B*
$ct^{+}sn^{MR110}$	3A; 7 D; 17C	-
$ct^{+}sn^{+}$	4F, 6F, 9B, 17C	-
$ct^{+}sn^{MR2+}$	2F, 7D, 9CD, 10F, 17C	-
$ct\ w^{MR1}$	1B, 1DC, 3C, 5F, 17C	-
$cm^{MR1}\ ct^{MRpN1}$	17C	7B
$cm^{+}ct^{+}\ sn^{17}$	3A, 7D, 9B, 13D, 17C	

*= 3C, 7B, 7D = sites of localization w, ct and sn loci.

Fig. 1. Results of in situ hybridization with polytene chromosome $cm^{+}ct^{+}sn^{17}$ mutant. 7D = the site of the localization of sn locus.

Earlier a stock was described (a derivative of ct^{MR2}) carrying two unstable alleles, $cm^{MR1}\ ct^{MRpN1}$, and characterized by a 70% simultaneous reversion of both alleles at the carmine and cut loci (Gerasimova 1983). Almost half of the double $cm^{+}ct^{+}$ revertants carried sn mutations. This was accompanied by the excision of mdg4 from the 7B region and the integration of the P-element in 7D (the region of the sn locus). The nature of the mobile element in the cm locus (GE) is unknown, but it is neither mdg4 nor the P-element.

Thus at least three mobile elements are simultaneously mobilized in this case. Different mobile elements seem to have different locus specificities: mdg4 prefers the cut locus, the P-element prefers w and sn.

The above results show that the activization of mobile elements conforms to the "all-or-none" principle. Various mobile elements are mobilized with a frequency of 10^{-3}-10^{-4}, leading to the reversion of some mutations and to mutagenesis in other genes. This is often accompanied by the appearance of new copies of the P-element in the X-chromosome.

What triggers off these transposition processes? Hardly the P-element, for it is present in all cells, while transposition occurs in one out of 1000-10000 cells. The processes may be genome-controlled, as suggested by the cm^{MR1} ct^{MRpN1} stock, where the transposition events involving a number of mobile elements are enhanced by an order of magnitude as compared with the ct^{MR2} stock. The activization of mobile genetic elements is certainly important in evolution, since it is capable of causing spontaneous changes in the genome and ensuring its rapid rearrangement.

The authors would like to thank Prof. N.F.Maysoedov for his interest and support, Prof. G.P.Georgiev for a discussion of the results, Mrs. N.V.Knizhnikova for technical assistance.

References: Gerasimova, T.I. 1982, Molec.Gen.Gent. 184:544; Gerasimova, T.I. 1983, DIS 59:37-38; Ilyin, Y.V., N.A.Tchurikov, E.V.Ananiev, A.P.Ryskov, G.N.Yenikolopov, S.A. Limborska, N.E.Maleeva, V.A.Gvozdev & G.P.Georgiev 1978, Cold Spring Harbor Symp.Quant.Biol. 42:959; Rubin, G.M., M.G.Kidwell & P.M.Bingham 1982, Cell 29:987.

Ghosh, M. University of Calcutta, India.

Nucleolar chromatin thread in different species of Drosophila.

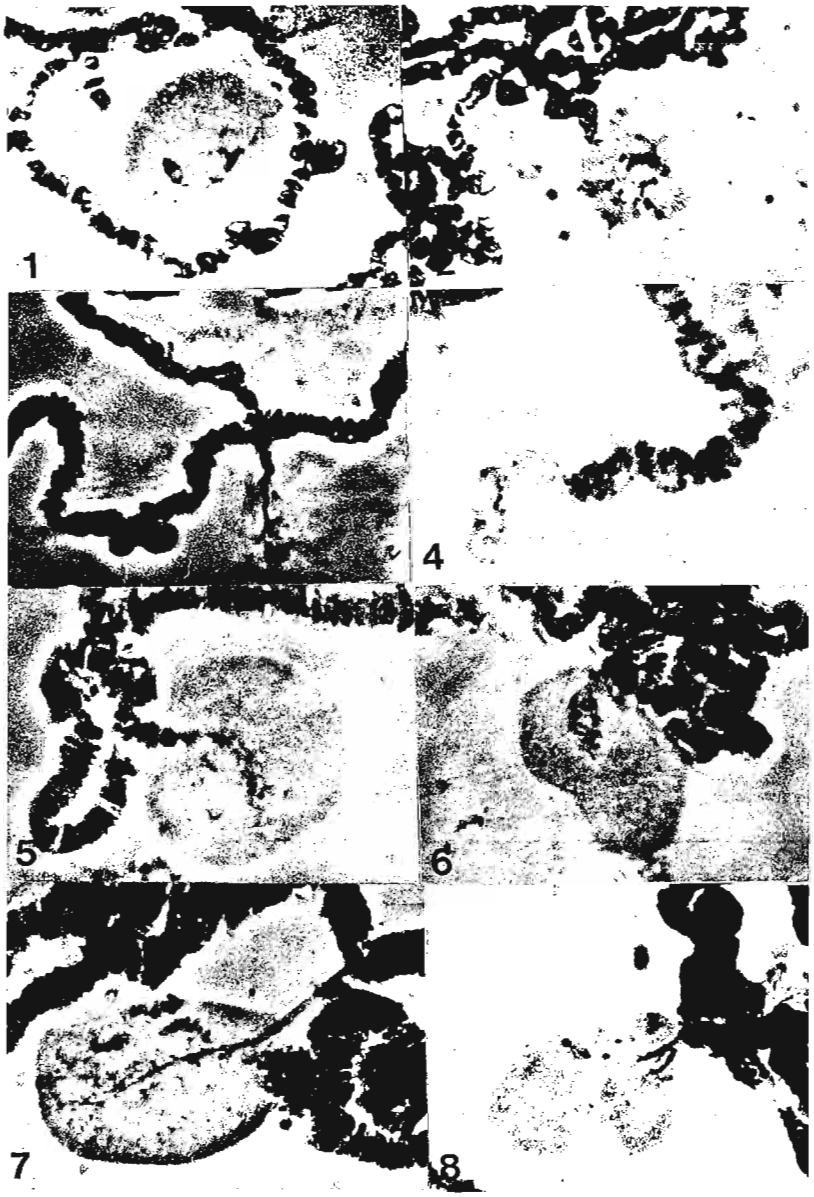
It has been reported earlier (Ghosh & Mukherjee 1982) that the nucleolus of Drosophila salivary glands exhibit a variation in morphological conformations of nucleolar chromatin thread (NCT) in different cytological preparations. That such intranucleolar structures are DNA materials have also been confirmed by Feulgen staining, Acridine Orange and Hoechst 33258 fluorescence. Futhermore, these NCT structures have been classified into four principal types (Ghosh & Mukherjee 1982).

Results on the treatment of nucleolar chromatin thread with various chemical agents viz. NaOH, HCl, DNase, 2,4 Dinitrophenol, heat treatment followed by acridine orange (AO) fluorescence revealed that both chromosome and NCT exhibit identical AO fluorescence. These findings suggest that the general organization of both chromosome and nucleolar chromatin is similar (Ghosh & Mukherjee 1982).

In the present investigation it has been revealed that in all the species (altogether 14 species reported in this issue) of Drosophila studied, 4 main types of NCT are manifested as has been reported earlier (Ghosh & Mukherjee 1982). In addition, some interesting features of the NCT have also been noted. These features are as follows.

In some preparations NCT appears as puff. These puffs resemble the puffs of chromosome (Fig. 1). Sometimes specific chromosome ends as a large puff, i.e., nucleolus with NCT (Fig. 2). The nucleolus itself also appears as a puff from the terminal portion of a chromosome (Fig. 3), showing the nucleolus with Type 2 NCT. It is also interesting enough to note that in Drosophila melanogaster the X-chromosome of some nuclei ends at its terminal (proximal) portion as a large puff within which a clear doublet is present. The terminal large puff appears as a nucleolus with NCT (doublet band). The doublet of the NCT is similar to that with some doublets of chromosome (Fig. 4). The banding pattern, i.e., bands, interbands are also observed in the NCT (Fig. 5).

The NCT sometimes appears as a small puff, i.e, chromosomal small band ends in a small puff in the nucleolus (Fig. 6). The NCT of Drosophila sometimes appears as banded structures i.e., NCT with regular bands and interbands as they are found in the chromosomes. Such regions can be clearly distinguished as dark and light bands and interbands of NCT. In many nuclei of different species of Drosophila a clear connection has been marked between the chromosomal band(s)and the nucleolus, i.e., NCT is continuous with the chromosomal band(s) (Figs. 7 & 8).



FIGURES. Photomicrographs showing the NCTs in different species of Drosophila. NCTs appear as: (1) puffs; (2) chromosome ends as a puffy structure with Type 1 NCT; (3) one chromosomal band is continuous as NCT; (4) chromosome ends as a puff in which NCT is present as a doublet; (5) NCT differentiated as bands and interbands; (6) NCT as a small puff; (7 & 8) NCT appears to originate from one band of the chromosome.

Fig. 1 = D.nasuta; 2 = D.melanica; 3 = D.virilis; 4 = D.melanogaster; 5 = D.pseudoobscura; 6 = D.ananassae; 7 = D.hydei; and 8 = D.simulans.

In general it appears from our data that (1) in all species of Drosophila studied (altogether 14 species) 4 main NCT types are found as reported earlier (Ghosh & Mukherjee 1982); (2) the structural organization of both chromosomes and NCT are similar as revealed by the presence of bands, interbands and puffs in both chromosomes and nucleolus.

Reference: Ghosh, M. & A.S. Mukherjee 1982, Cell and Chromosome Res. 5(1):7-22.

Ghosh, M. and A.S.Mukherjee. University of Calcutta, India. Evolutionarily related species and their NCT structures.

Nucleolus of eukaryotes is now well known as a distinct body which is either round or oval in shape. There are some reports (Rodman 1969; Lettré et al. 1968) that the nucleolar chromatin thread or NCT (DNA) remains embedded within the nucleolar mass. A species specific nucleolar chromatin structure in polytene nuclei of Drosophila has also been reported by Barr and Plaut (1966a,b). Rodman (1969) reported variable conformations of nucleolar chromatin (DNA) present in Drosophila melanogaster. Earlier we have reported that there are 4 major types of nucleolar chromatin threads (NCTs) in the nucleolus of Drosophila hydei (Ghosh & Mukherjee 1982a,b). In the present investigation we have examined the NCT in 14 species of Drosophila viz., D.melanogaster, D.simulans, D.willistoni, D.insularis, D.melanica. D.miranda, D.pseudoobscura, D.persimilis, D.virilis, D.nasuta, D.malerkotliana, D.bipectinata, D.ananassae and D.hydei to find out the evolutionary relationship in the NCT structure, if any.

Analysis of the data reveals that (1) the 4 major types of NCTs are present in all the species studied, (2) the NCTs of slaivary gland nuclei are not species specific, (3) the frequencies of different NCTs in the closely related species are more or less equal and,

(4) the differences in frequency of NCT types is observed between distantly related species (Figs. 1-3).

References: Barr, H.J. & W.Plaut 1966a, J.Cell Biol. 31:10A; Barr, H.J. & W.Plaut 1966b, J.Cell Biol. 31: 17-22; Ghosh, M. & A.S.Mukherjee 1982a, DIS 58:66-67; Ghosh, M. & A.S.Mukherjee 1982b, Cell and Chromosome Res. 5(1):7-22; Lettré, R., N.Paweletz, S.Ghosh & W.Siebs 1968, Mater.Med.Nordmark 20(11):30-38; Rodman, T.C. 1969, J.Cell Biol. 42:575-582.

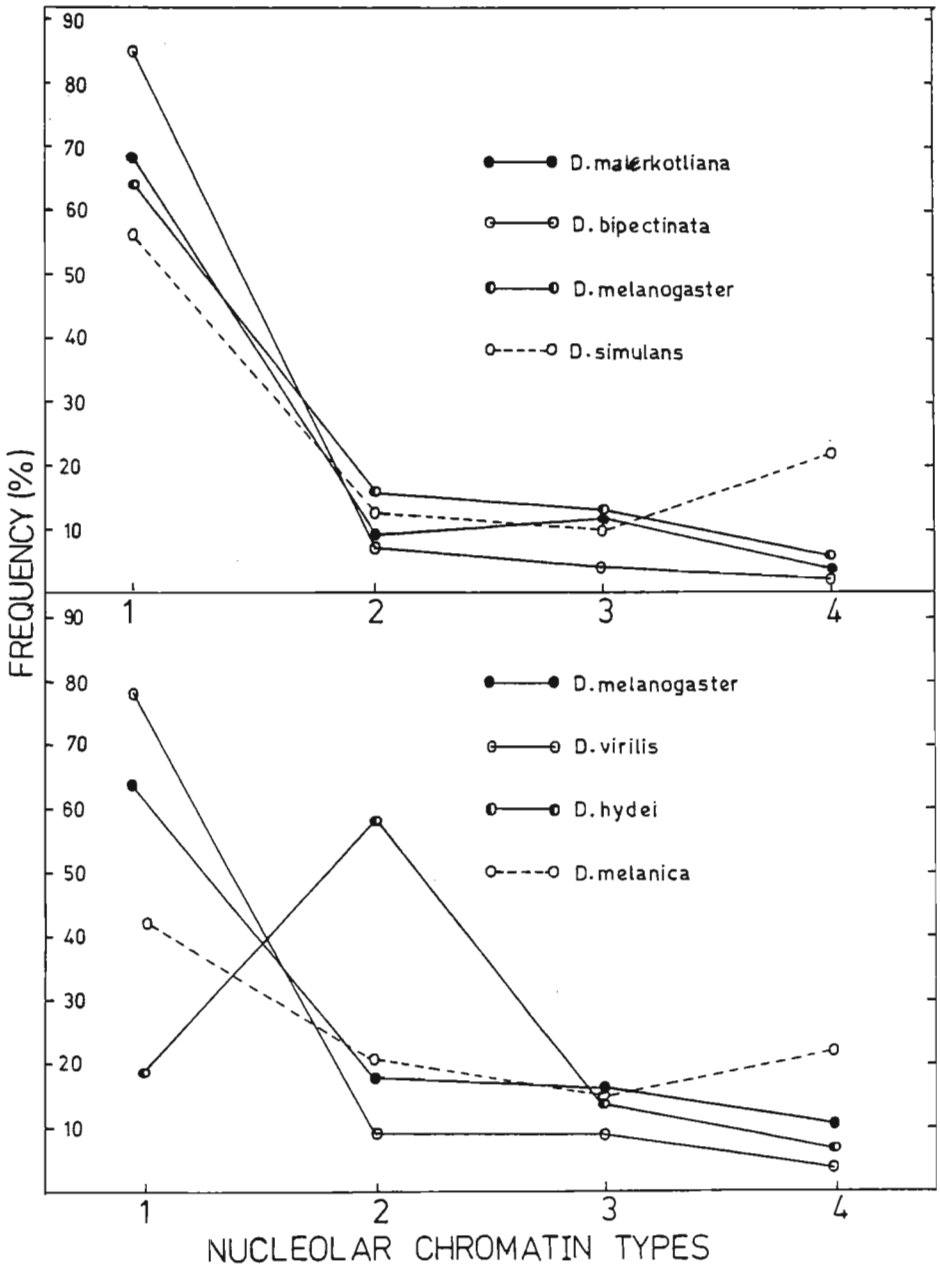


Figure 1. Comparative frequencies of different NCT types in closely and distantly related species of Drosophila.

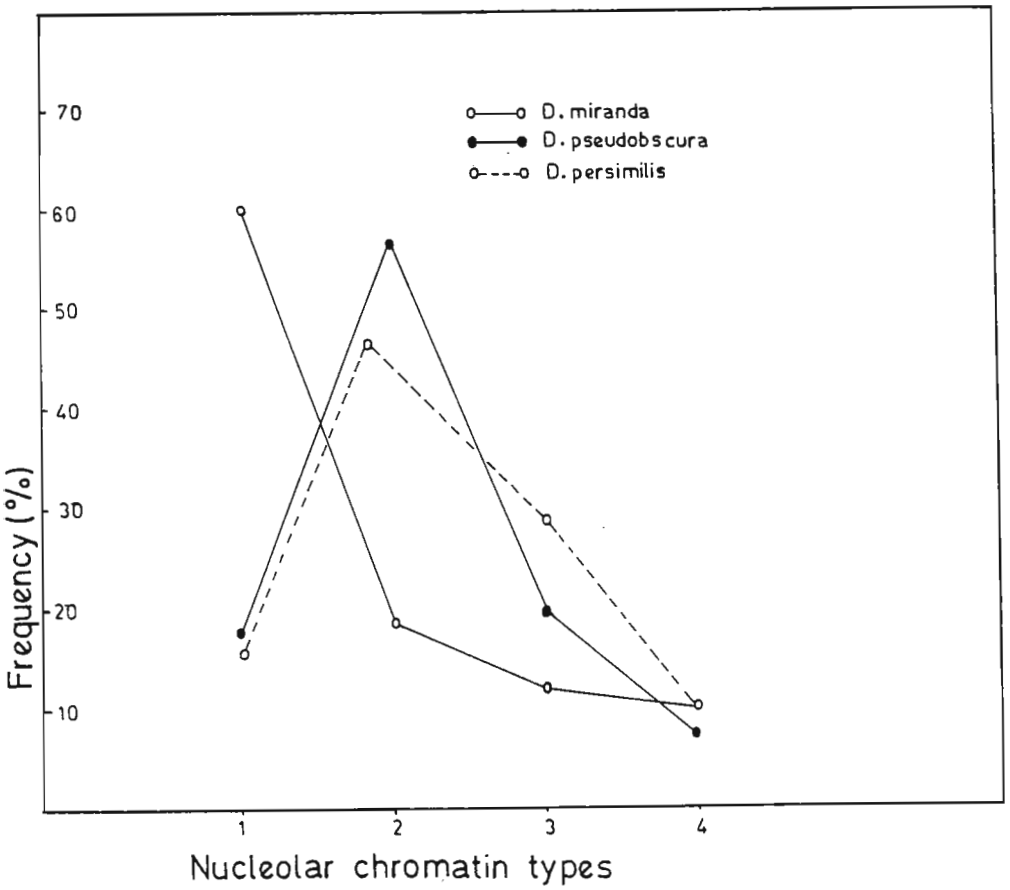


Fig. 2. Frequencies of different NCT types in three species of closely related species of Drosophila.

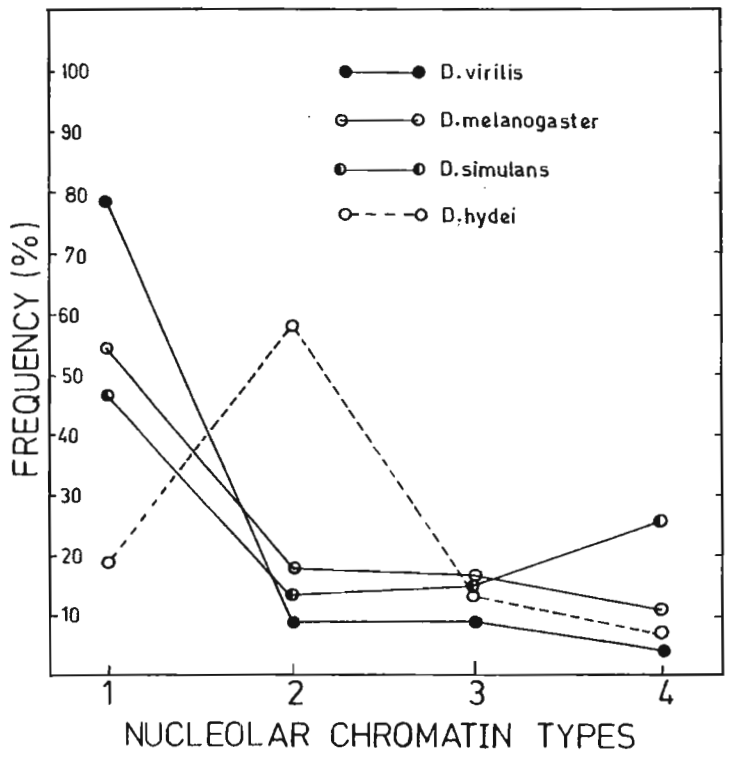
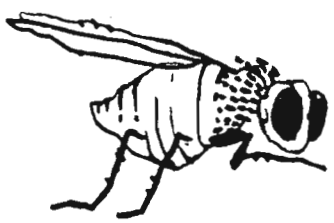


Fig. 3. Frequencies of different types of NCT in different distant related species of Drosophila.



Ghosh, M. and A.S.Mukherjee. University of Calcutta, India. Nucleolar chromatin thread (NCT) in hybrids of Drosophila.

been performed with the hybrids of Drosophila and some important conclusions have been drawn from these investigations. Patterson & Stone (1952) reported that there are numerous examples of interspecific hybridization in the genus Drosophila where the sex ratio is normal. Ohno (1969) from his study demonstrated that the dominance for NOR activity ordinarily is the same in the reciprocal crosses, although a strong maternal influence is often found. Mitrofanov and Sidorova (1981) reported that there are also deviations from Haldane's rule (1922) in interspecific Drosophila hybrids. Based on these above mentioned ideas our present investigation was performed to examine the NCTs in hybrids obtained from crosses between different species of Drosophila.

For this purpose four species of Drosophila viz., Drosophila melanogaster, D.simulans, D.pseudoobscura and D.persimilis were selected and four sets of crosses were designed. Cross I: virgin females of D.melanogaster X males of D.simulans; Cross II: virgin females of D.simulans X males of D.melanogaster; Cross III: virgin females of D.pseudoobscura X males of D.persimilis; Cross IV: virgin females of D.persimilis X males of D.pseudoobscura.

Cytological squash preparations following aceto-carmine aceto-orcein staining method of salivary gland cells of the hybrids from each cross were made.

From the cytological preparations it is evident that all four principal types of NCTs are present in the F_1 hybrids as they are in the parents. A maternal influence is observed in the production of NCTs in most of the F_1 hybrids examined. It is clear from the data (Fig. 1) that when parental flies were D.melanogaster ♀ and D.simulans ♂, all females of F_1 do not show the frequency of NCT as in the D.melanogaster female. On the other hand, when the parental female was D.simulans only male offsprings are generated and they show the frequency of NCT as in D.simulans. But in the cases of crosses III and IV, i.e., D.pseudoobscura (♀,♂) X D.persimilis (♂,♀) progenies of both sexes were obtained unlike the crosses I and II and there also a maternal influence is observed (Fig. 2).

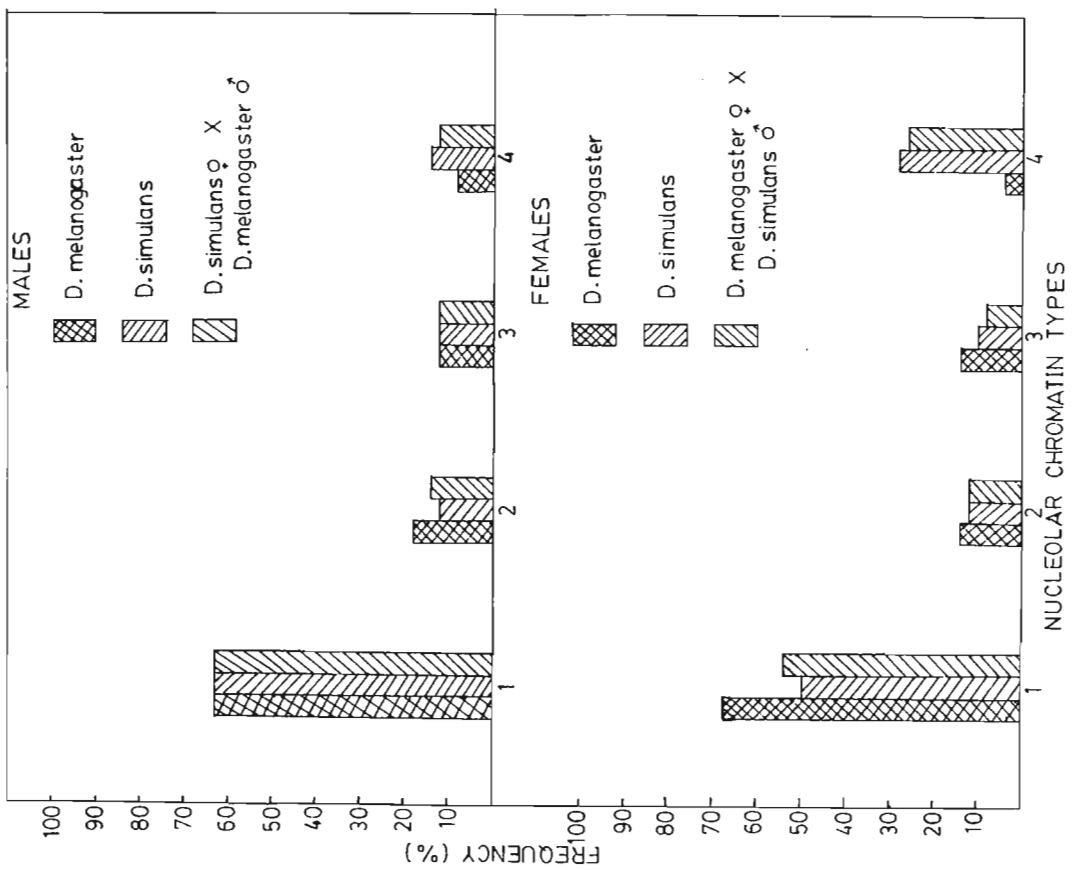


Fig. 1. Histogram showing the frequency distribution of different NCT types in male and female F_1 hybrids obtained from crosses between D.melanogaster (♂,♀) and D.simulans (♀,♂).

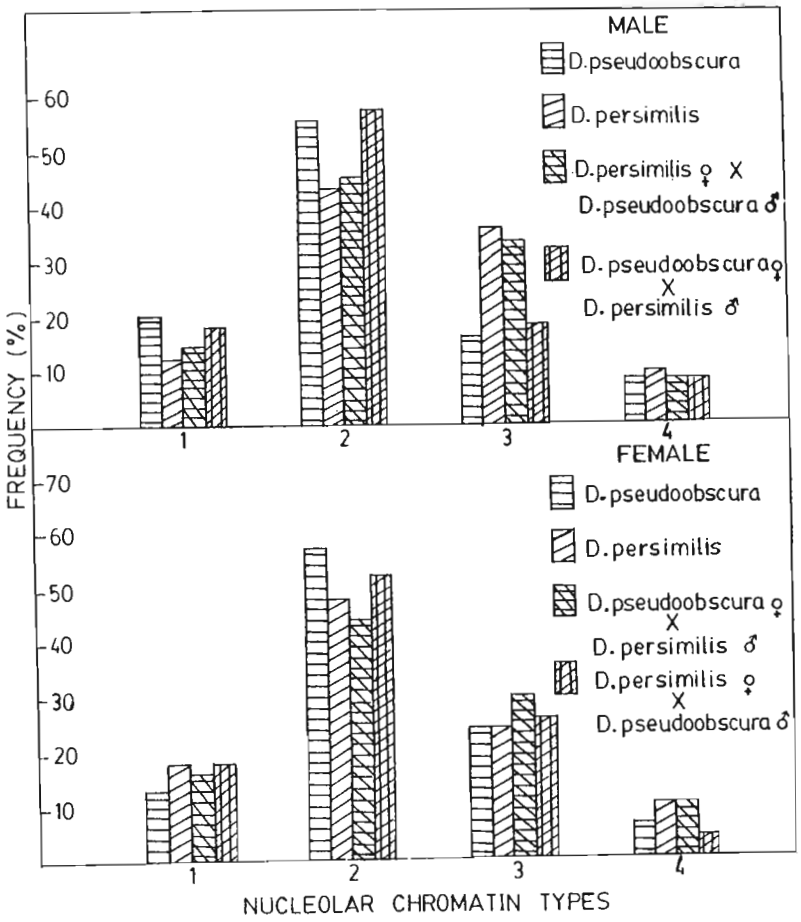


Fig. 2. Histogram showing the frequency distribution of different NCT types in male and female F_1 hybrids obtained from crosses between D.persimilis (♂,♀) and D.pseudoobscura (♀,♂).

Therefore, from the data it is revealed that (1) in all the cases 4 major types of NCTs are present, (2) only hybrid females in cross I and males in cross II were obtained but there is no variation in the NCT structures; only variation in frequency was observed, (3) both males and females were obtained in the hybrid progenies from crosses III IV, (4) a maternal influence over the frequencies of NCTs was observed in a general way.

References: Haldane, J.B.S. 1922, J.Genet. 12:101-109; Mitrofanov, V.G. & N.V.Sidorova 1981, Theor.Appl.Genet. 59:17-22; Ohno,S. 1969, in: Heterospecific genome interaction (V.Defendi, ed.), Wistar Institute Press, Philadelphia, pp. 137-150; Patterson, J.T. & W.S.Stone 1952, in: Evolution in the Genus Drosophila, The MacMillan Co., New York, pp. 386-387.

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Evidence for the presence of telomeric material in the junction of 2L and 2R in entire compound two chromosomes.

The entire compound autosomes, in which chromosomes arms are attached end to end to form a double length chromosome arm, exhibit a genetic behavior as well as a cytological appearance in the polytenes that suggests that no essential genes have been lost in their manufacture. This has given rise to the speculation (Novitski et al. 1981, Am.J.Human Genet. and 1981 Genetics) that the distal break on one arm (to which the proximal break on another

has become joined) may have occurred in the telomere region.

To test this supposition, a telomere-specific clone was obtained from Prof.Ed Strobel at Purdue. This plasmid which contains a 1.25 kb fragment of the cDm 356 repeat unit inserted into pBR 322 was hybridized in situ with polytene chromosomes of a line carrying compounds of both the second and the third chromosomes. Hybridization at the junction between the tip of 2L and the base of 2R was obvious (see Figure), suggesting that some telomeric material may still be present at this juncture.

On the other hand, in our relatively few good preparations, we did not see any clear cases of hybridization at the corresponding juncture of the 3L tip and the base of 3R. This may mean that there was no such hybridizing material present interstitially on the third chromosome, or that with our techniques it was not demonstrable.

Gonzalez, F. University of Valencia, Espana. Non-effect of light conditions as a selective force for an eye colour mutant of Drosophila melanogaster.

Analyzing natural populations of Drosophila melanogaster a greater amount of eye colour mutants was found among flies captured in a cellar than among those captured in a close vineyard (Najera & Mensua 1982). To investigate the possible effect of light intensity on eye mutant alleles, the following experience was carried out.

Two isofemale strains captured in a cellar near Requena (Valencia) were used. One strain (2/63) had normal eyes whilst the other (2/54A) was the eye mutant 'cardinal' (cd: 3-75.7). Both strains had been kept in the laboratory for 4 years at 25±1°C and 60±5% relative humidity in 250 ml bottles supplied with 50 ml of food.

Three light environments were chosen to simulate light conditions existing in the cellar where flies were captured: (i) normal (fluorescent) laboratory light, (ii) semi-darkness, covering the cultures with red and blue filters simultaneously, and (iii) complete darkness, using a black box.

Three different cultures were initiated with the following gene frequencies:

	allele 2/63 (+/+)	allele 2/54A (cd/cd)
Culture A	0.5	0.5
Culture B	0.2	0.8
Culture C	0.8	0.2

Two replicates for each initial composition and light environment were made.

Cultures were kept for six months in the above mentioned conditions by a weekly serial transfer system. After this time, gene frequencies were estimated according to the method of Cotterman (1954). Table 1 shows the results obtained averaging the two replicates, as no significant differences between them were observed.

Table 1. Gene frequencies of cardinal allele.

Light regime	Culture A	Culture B	Culture C
Normal	0.3936	0.4497	0.3917
Semi-darkness	0.4858	0.3787	0.4354
Darkness	0.3949	0.4510	0.3983

Table 2. Analysis of variance of results shown in Table 1.

Source of variation	d.f.	SS	MS	F
Initial compos.	2	5.92×10^{-4}	2.96×10^{-4}	0.1274^{ns}
Light conditions	2	8.22×10^{-4}	4.11×10^{-4}	0.1768^{ns}
Error	4	9.29×10^{-3}	2.32×10^{-3}	
Total	8	1.07×10^{-2}		

ns = non-significant

Table 2 shows the analysis of variance performed with these results. As can be seen, there are no significant differences either among light environments or among initial compositions. Thus, it can be affirmed that after six months all the populations have approached to an equilibrium point, with a frequency for the allele 2/54A around 0.42.

This result leads to the conclusion that light intensity is not a factor responsible for the greater presence of eye colour mutants in the inner of a cellar than in its outer.

References: Cotterman, C.W. 1954, Estimation of gene frequencies in nonexperimental populations, in: Statistics and Mathematics in Biology (ed: O.Kempthorne et al.), Iowa State College Press;

Najera, C. & J.L.Mensua 1982, Analisis de la variabilidad de mutantes que afectan a la sintesis de pigmentos oculares de Drosophila melanogaster en poblaciones naturales, XVIII Jornadas de Genetica Luso-Espanolas, Granada, Spain.

Gonzalez, F. and J.Ferre. University of Valencia, Espana. Non-dependence of the eye pigmentation of Drosophila melanogaster on light conditions.

Two different studies to test the possible dependence of pteridinic pigment accumulation in the eyes of Drosophila melanogaster under different light conditions have been carried out.

Fly extracts (40 heads and 5 male bodies) were subjected to two-dimensional thin layer chromatography on cellulose plates. Quantitative estimation of the fluorescence of the separated pteridines was performed in a Perkin-Elmer model MPF-44B spectrophotometer with a thin layer chromatography plate scanner attachment. Neodrosopterin, drosopterin, isodrosopterin, aurodrosopterin, sepiapterin, pterin, biopterin, 7,8-dihydro-acetylhomopterin and xanthurenic acid were measured.

A first study intended to find whether light conditions affected the pigmentation during the development of the eye. Oregon-R flies reared at 25°C were kept in the dark from the 1st larval instar. A control in normal (fluorescent) light was reared simultaneously.

Adult flies were analyzed at 9 and 30 days after eclosion. No difference between flies kept in the dark and control flies was found. Thus, light has no appreciable effect either on the synthesis of eye pigments during the pupal stage or on the amount of pigments retained by the adults.

An eye colour mutant isofemale strain (cd: 3-75.7) captured in a cellar, was reared in different light environments to study the possible selective effects of light upon genes affecting the amount of eye pigments. Three different environments were chosen to simulate light conditions existing in the cellar where flies were captured: (i) normal fluorescent light, (ii) semi-darkness, covering the cultures with red and blue filters simultaneously, and (iii) complete darkness, using a black box.

Cultures were kept at 25±1°C, 60±5% relative humidity, in 250 ml bottles supplied with 50 ml of food. They were maintained by a weekly serial transfer system. After 8 months, nine days old flies were analyzed. No differences among the chromatographic patterns of the three cultures were found. This suggests that selective pressure, if it exists, is too weak to be detected under these conditions.

Gonzalez, A. and J.L.Mensua. University of Valencia, Espana. Inversions in two natural populations of Drosophila melanogaster from cellar and vineyard.

Data about inversions found in two natural populations of Drosophila melanogaster from the locality of Requena (Valencia, Spain) are presented.

The populations studied come from two sites with relatively different environments, above all in regard to alcohol concentration and temperature: inside a cellar and an area of vineyards located 4 Km away from the cellar. Both populations were captured in late October (after the grape harvest).

The possibility of association between lethal chromosomes and inversions in them, was also studied.

One-hundred-and-sixty-one third chromosomes were analyzed for inversions (86 from the cellar and 75 from the vineyard).

Of these 161 chromosomes, 38 from the cellar and 40 from the vineyard were lethal-carrying chromosomes.

For the analysis of inversion, crosses were made with "rucuca" stock which is homozygotic for the standard arrangement in the third chromosome.

Table 1 shows the total frequencies of inversions for the two populations studied. A significantly lower frequency of inversions at the 5% level was observed in the cellar compared to the vineyard.

The types and frequency of inversions per chromosomic arm from the cellar and vineyard populations is shown in Table 2. In accordance with Inoue and Watanabe (1969) the category of the inversions is also mentioned, taking into account their geographic location and frequency.

Table 1. Total inversion frequencies of the third chromosome in cellar and vineyard populations.

POPULATION:	CELLAR	VINEYARD
No. of Chromosomes analyzed:	86	75
No. of Chromosomes carrying inversions:	12	21
Total frequencies of inversions (%):	13.95±2.73	28.00±3.53

t=2.21; g.l.=159; P<0.05

The two common cosmopolitan inversions (3R)P and (3L)P show lower frequency in the vineyard than in the cellar.

The inversion (3R)P was found in association with the inversion (3L)P in two third chromosomes from the cellar and one from the vineyard.

In this work an inversion [(3R) 86E-92F] which not previously been described is shown in Figure 1. This inversion was found in the cellar population.

A significant association between lethal chromosomes and chromosomes carrying inversions was not observed in either of the two populations studied (cellar X^2=0.563, d.f. 1, 0.5>P>0.4; vineyard X^2=0.450, d.f. 1, P=0.5).

References: Inoue, Y. & T.K.Watanabe (1979), Japan.J.Genet. 54:69-82.

Table 2. Frequency of the different types of inversions of the third chromosome in cellar and vineyard populations.

ARM	INVERSION		CELLAR(N=86)	FREQUENCY(%)	VINEYARD(N=75)	FREQUENCY(%)	CATEGORY
3L	In(3L)P	63C; 72E	5	5.8	6	8.0	common cosmopolitan
	In(3R)P	89C;96A	7	8.1	13	17.3	common cosmopolitan
	In(3R)C	92D;100F	0	0	3	4.0	rare cosmopolitan
3R	In(3R)Mo	93D;98F	1	1.1	0	0	rare cosmopolitan
	In(3R)	86E;92F	1	1.1	0	0	unique endemic

N = Number of chromosomes analyzed

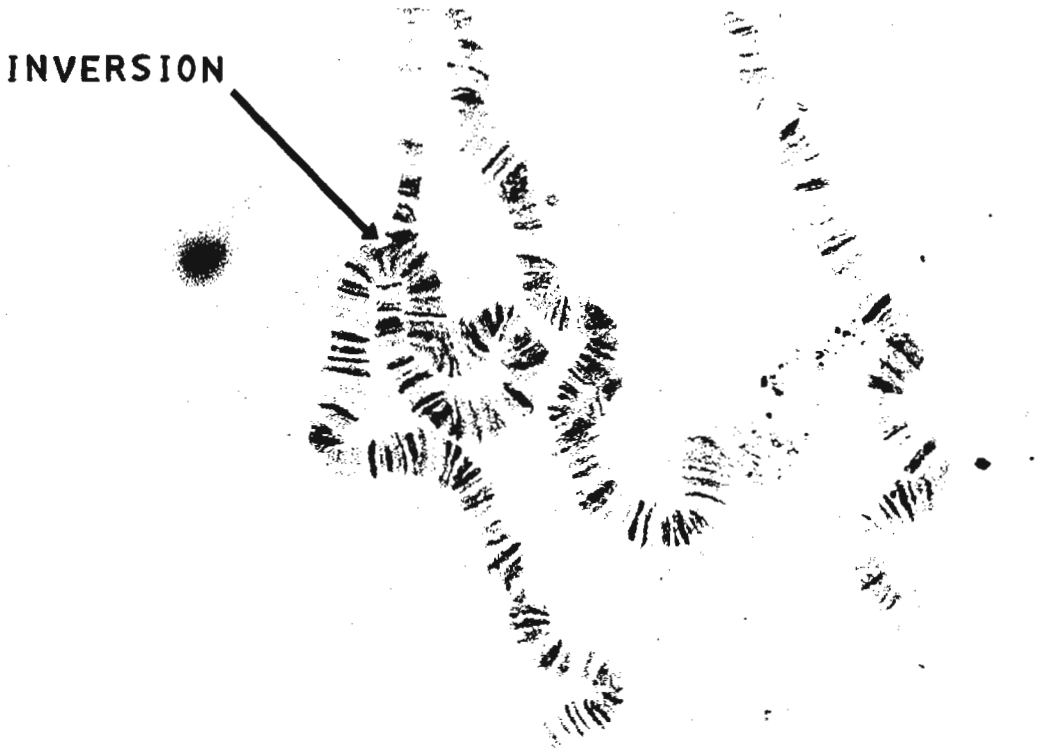

Figure 1.

In(3R)
86E-92F

Greenberg, R.M. and P.N.Adler, University of Virginia, Charlottesville, Virginia USNA.

Protein synthesis in homoeotically transformed imaginal discs.

We have examined the pattern of abundant protein synthesis in various homeotic imaginal discs. The mutants used were: bithorax (bx^3), which replaces anterior haltere structures with anterior wing; postbirthorax (pbx), transforming posterior haltere to posterior wing; and engrailed (en^1), in which posterior wing structures take on characteristics of anterior wing. Mutant stocks used were bx^3/bx^3, red pbx/TM9, and cn en^1/CyO, $1(2)513^{DTS}$. All mutations are listed in Lindsley & Grell (1968) with the exception of TM9, a balancer third chromosome containing a dominant temperature-sensitive lethal. Homozygous cn en^1 larvae were obtained by crossing the balanced stock inter se and pulsing the embyros and early larvae for two days at 30°C. Because the engrailed phenotype is not expressed strongly at 30°C (Lawrence & Morata 1976), larvae were raised at 25°C following the 30°C pulse. Although non-engrailed larvae survived with this protocol, they developed more slowly than cn en^1 wing disc morphology differed subtly, but nonetheless perceptibly, from wild-type. Discs were dissected, labelled with ^{35}S-methionine, and their proteins runon 2D gels as described in Greenberg & Adler (1982).

Neither bx^3, nor pbx haltere discs differed in their patterns of abundant protein synthesis from bx^3 or pbx wing discs. Since we also find no reproducible differences between wild type wing and haltere discs, it stands to reason that these two mutations do not alter the wild type patterns of abundant protein synthesis.

Wild type wing and haltere discs synthesize a protein (RG38) with a nonuniform, homologous spatial distribution (Greenberg & Adler 1982). Thus, RG38 is synthesized preferentially in presumptive anterior, dorsal, and proximal regions of the wing disc and in anterior, dorsal haltere disc. Because bx^3 and pbx change the size of particular disc regions (i.e., a bx^3 anterior haltere disc is larger than wild type), these mutations might also be expected to result in a concomitant alteration in the level of RG38 synthesis. We have, however, observed no such result.

Since en^1 directly affects the pattern of wing disc derivatives, it might also be expected to alter the pattern of RG38 synthesis. Wing discs from cn en^1 larvae were labeled as usual and cut into anterior and posterior fragments. Examination of 2D PAGE protein patterns revealed no difference from wild type. Thus, it appears that, although en^1 causes major changes in fly morphology, the mutation does not affect pattern as assayed by RG38 level of synthesis. A trivial explanation for this result may reside in the fact that only more distal derivatives of the wing exhibit the engrailed phenotype. Since only low levels of RG38 synthesis can be found in the presumptive distal region of wild type wing discs, an anterior/posterior transformation there would be swamped by the much larger amounts of RG38 in other wing disc regions; the change would be undetectable.

We thank Margaret MacQueen for expert technical assistance. Dr. T.R.F.Wright & Dr. Michael Russell generously provided Drosophila stocks. RMG was supported by NIH training grant #HD07192. PNA was supported by a research career development award (NIH KHD00361), a grant from NIH (HD11763), and a grant from NSF (PCM 8203205).

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Hara, T. and H.Kurokawa. University of Tsukuba, Sakura-mura, Japan. Analysis by a partial chromosome substitution for interspecific difference in male 6th sternite bristles between D.auraria and D.biauraria.

Male flies of D.auraria have about 14 bristles on the 6th sternites, while those of D.biauraria have generally none. Backcross experiments were made by using mutant markers of D.auraria chromosomes. It was found that both X- and A-chromosomes were obviously responsible for the bristle manifestation (Hara & Kurokawa 1983).

A chromosome assay was performed by using a wild strain of D.biauraria and mutant strain of D.auraria being homozygous for y(X), cn(A), cu(B). The A-chromosome had a major effect which manifested 8.6 bristles on an average and the X-chromosome had a moderate one being conformable to 4.1 bristles. The effect of the B-chromosome was a little, corresponding to 1.1 at the most. Interactions between the effects of different chromosomes were negligibly small in all combinations.

References: Hara, T. & H.Kurokawa 1983, Jpn.J.Genet. 58:497-504.

Harshman, L.G. University of California, Davis, USNA. The incidence of ovarian dysgenesis in Drosophila simulans.

The P-M syndrome of Drosophila melanogaster is a set of progeny aberrations that can be induced by crossing males (P) that are recently isolated from the field with lab-stock females (M). The reciprocal cross does not show the response (Kidwell 1977), which typically includes male recombination, increased mutation rate, and a failure of the germline to develop (ovarian dysgenesis in females). In Drosophila simulans it has been observed that crosses within and between strains collected in France during the 1970's produced a pattern of ovarian deterioration and transmission ratio distortion which indicated the presence of a system like P-M (Periquet 1981).

This note describes a study designed to characterize the pattern of ovarian dysgenesis in Drosophila simulans by crossing lab stocks with recently isolated lines. The lab stocks used were fa rb, y w , f^2 nt pm st e, and A. The markers fa, pm, rb, st, w, and y were described by Sturtevant (1929), and A, f^2, and e are known to have been in culture for at least fifteen years. The f^2 nt pm st e stock was constructed in the last few years. Little else is known about the history of the strains but presumably they are comparable to old stocks in Drosophila melanogaster. Ten samples of recently isolated lines were collected from July to September 1982, no more than nine months before the experiment. They include isofemale lines from nine California populations, which ranged from San Diego 350 miles north to Patterson, and isofemale lines from Belmont, Massachusetts. A total of 32 isofemale lines, from two to five per population, were employed in the study.

The crosses were made by confining two males with a virgin female in a vial of standard cornmeal medium. The ovarian dysgenesis phenotype of Drosophila melanogaster is temperature-dependent (Engels & Preston 1979), consequently all simulans crosses were initiated and maintained at 28°C. After eclosion adult progeny were transferred to fresh media for two to four days. Thereafter, approximately twenty females were dissected from each cross and scored for the presence of an ovary or ovaries that were so deteriorated that no normal eggs were present.

In the course of the survey female progeny from 190 matings were examined. In crosses between males from recently isolated lines and lab stock females 32 of 1891 offspring (1.69%) had one or two deteriorated ovaries. In the reciprocal cross 41 of 1531 offspring (2.68%) had a dysgenic phenotype. None of the lab stocks showed a strong propensity to produce ovarian deterioration, and none of the populations sampled had particularly reactive isofemale lines. It is possible that these laboratory strains or isofemale lines from the regions of collection are neutral and unreactive. However, it appears that Drosophila simulans does not have a direct analog to the P-M system of Drosophila melanogaster.

Acknowledgements: I would like to thank Fred Cohan and Gail Simmons for technical advice, numerous isofemale lines, and helpful comments.

References: Engels & Preston 1979, Genetics 92:161; Kidwell et al. 1977, Genetics 86:813; Periquet 1981, Heredity 46:255; Sturtevant 1929, Carnegie Institute of Washington Publ. No. 399.

Heiskanen, A., P.Jokela, M.Laitinen, M-L. Savontaus and P.Portin. University of Turku, Finland. Effect of temperature on the intraindividual variation of sternopleural bristles in D.melanogaster.

The within-individual or intraindividual component of variation is entirely of environmental origin. It can be measured by multiple measurements within individual, and is therefore called repeatability (Falconer 1981). The within-individual variation is due to special environment in contrast to common environment which together with genetic variation causes variance between individuals. Repeatability can be studied by measurements following each other in time or by measurements of variation between symmetrical or metamerically repeated organs such as sternopleural bristles and abdominal sternite bristles respectively. The within-individual source of variation has also been called developmental noise by Suzuki et al. (1981) since it is principally of stochastic origin the genotype and the common environment being identical for example for bilaterally symmetric organs of the individual. As the cause of the developmental noise Suzuki et al. (1981) suggested the random distribution of rare biologically active molecules such as vitamins the mean concentration of which can be less than one molecule per cell. Such molecules can cause differences between symmetric organs because their distribution is determined largely by chance. Already Astauroff (1930) is studying variation in bilaterally symmetric organs concluded that the differences between the sides of the animal are not due to the environment of the animal but result from a specific quality of the developmental process of the character itself.

Reeve and Robertson (1954) studied the sources of variation in the numbers of sternite chaeta of D.melanogaster, and found out that the special environment caused as much as 58% of the variation, while additive genetic variation was 33%, non-additive genetic variation 6%, and the variation caused by the general environment only 3%. Thus the great majority of the environmental component of the variation was due to special environment (developmental noise).

We investigated the variation of sternopleural bristles of D.melanogaster in the crossbred Canton-S stock, and in the inbred Samarkand stock (256 generations of sib mating) in different temperatures (20°C and 29°C). We counted the numbers of sternopleural bristles on both sides of the flies separately in 75 females and males raised in 20°C and 29°C. The variances are given in Table 1. Since the genetic variance is nonexistent in the inbred stock, we could divide the variance in the crossbred stock into components as follows. The within-individual variation gives the effect of the special environment, and the rest of the variance was divided into genotypic and common environmental variance. The environmental variance was obtained by substracting the between individuals variance of the inbred stock from the between individuals variance of the crossbred stock. The components of the variance are given in Table 2 for the crossbred stock in both temperatures. It can be seen that the variance caused by the special environment is significantly greater in 20°C ($F=2.08^{**}$). Thus the developmental noise is higher in higher temperature. It is suggested that this is caused by increased movement of small biologically active molecules in the higher temperature.

Table 1. The variances in numbers of sternopleural bristles in an inbred (Samarkand) and crossbred (Canton-S) stock in different temperatures. The number of females and males examined in each series was 75.

	20° C			29° C		
	♀	♂	mean	♀	♂	mean
SAMARKAND (inbred)						
Within individuals	0.76	0.85	0.80	1.47	1.44	1.45
Between individuals	2.02	1.87	1.95	2.84	2.38	2.61
Total variance	2.78	2.71	2.75	4.30	3.82	4.06
CANTON-S (crossbred)						
Within individuals	1.01	1.11	1.06	2.55	1.86	2.21
Between individuals	3.72	4.64	4.18	6.78	5.01	5.90
Total variance	4.73	5.76	5.24	9.33	6.88	8.10

Table 2. The sources of variance in the crossbred Canton-S population in different temperatures. N = 150 in each series.

	Proportion of variance (%)	
Source of variance	20°C	29°C
Genotypic	42.5	40.6
Common environment	37.1	32.2
Special environment	20.3	27.2
Total	100.0	100.0

References: Astauroff, B.O. 1930, Z.Indukt.Abstamm.u.Vererbungsl. 55:183-262; Falconer, D.S. 1981, "Introduction to Quantitative Genetics" Longman Group Ltd.; Reeve, E.C.R. & F.W. Robertson 1954, Z.Indukt.Abstamm.u.Vererbungsl. 86:269-288; Suzuki, D.T., A.J.F.Griffiths & R.C.Lewontin 1981, "An Introduction to Genetic Analysis" Freeman & Co.

Herforth, R.S.[1], H.L. Carson[2] and L.Chang.[2]
1 Augsburg College, Minneapolis, Minnesota USNA. 2 University of Hawaii at Manoa, Honolulu, Hawaii USNA. A new arrival to the Hawaiian Islands: Drosophila cardini.

In March of 1983 a female Drosophila was collected in Honolulu near Leahi Hospital. The collection site was at the southeast corner of Kilauea Avenue and Makapuu, near property occupied by Kapiolani Community College. The fly was caught in a banana trap hung from branches of what appeared to be the cactus Cereus undatus. This female died without laying any eggs, but her external characters indicated that she was probably a member of the cardini group. These characters included: clouded crossveins; black bands on the posterior edge of yellowish-brown abdominal tergites, with the black in the lateral areas not reaching the lateral edge of the tergites and extending anteriorly and medially in the posterior tergites; brilliant orange eyes; and reddish-brown mesonotum, scutellum and pleurae. Another female was collected from the same site in August of 1983, again using a banana bait. This female was successful in laying eggs, and an isofemale line was established. Examination of males of this line showed that they lacked a protuberance on the anteroventral margin of the labellum and possessed anal plates with one or two long anteriorly directed bristles on the anteromedial corner of the plates. These features are characteristic of D.cardini Sturtevant (Stalker 1953).

Since the cardini group consists of about 16 sibling and near-sibling species, the metaphase chromosome group was determined by brain smears. This showed the presence of 6 pairs of chromosomes, including 5 pairs of acrocentrics and one pair of microchromosomes. D.cardini is the only member of the species group which has this somatic metaphase figure (Futch 1962; Heed & Russell 1971). The chromosomal and morphological features thus lead to the conclusion that these flies are members of Drosophila cardini Sturtevant. This species has been found in Florida, Mexico, Central and South America, and the West Indies but has never before been recorded from the Hawaiian Islands. In fact no other species of cardini group has been found here. D.cardini thus represents a new arrival to the Hawaiian Islands.

Acknowledgement: These flies were collected while the first author was on leave at the Arbovirus Program, Pacific Biomedical Research Center, Univ. of Hawaii, Honolulu, USNA HI.

Ref: Futch,D.G. 1962, Univ.Texas Publ. 6205:539-554; Heed,W.B. & J.S.Russell 1971, Univ.Texas Publ. 7103:91-103; Stalker,H.D. 1953, Ann.Ent.Soc.Amer. 56:343-358.

Holliday, M. and J.Hirsch. University of Illinois, Urbana-Champaign, Illinois. Excitatory (Pavlovian) Conditioning.

Using a variation of Nelson's (1971) procedure for conditioning the blow fly Phormia regina, we demonstrate excitatory conditioning of the proboscis extension reflex in D.melanogaster.

The flies came from a population established by crossing the Berlin line with another produced by mixing nine Austin, TX inbred lines obtained from Birmingham, England. They were virgin males and females, 44-48 hr old, 36 hr food deprived and water satiated, when conditioned on the automated stimulating apparatus (Vargo, Holliday & Hirsch 1983; Holliday, Vargo & Hirsch 1983).

As outlined in Figure 1, the conditioning procedure presents for 5 sec to the foretarsi a 0.5-M NaCl conditioned stimulus (CS), followed after a 0.5-sec interval by a 0.25-M sucrose unconditioned stimulus (US) for 5 sec (also accessible to the proboscis for 2-3 sec), itself followed after a 170-sec interval by a distilled H_2O intertrial stimulus (ITS) for 5 sec, which, in turn, is followed after a 175-sec interval by the start of the next trial. Thus, the intertrial interval (ITI) is 6 min. The ITS serves to discharge any residual sucrose induced excitatory state (CES, Dethier, Solomon & Turner 1965) which in D.melanogaster can last at least 10 min (Vargo & Hirsch 1982a,1982b). With a 6-min ITI, it is important to discharge CES in order to avoid confounding non-associative excitation with associative responding (conditioning) to the CS.

For 111 flies Figure 2 presents average results combined from four experiments. Over nine trials, responding (1) to the CS increases significantly (regression coefficient: B=3.7,

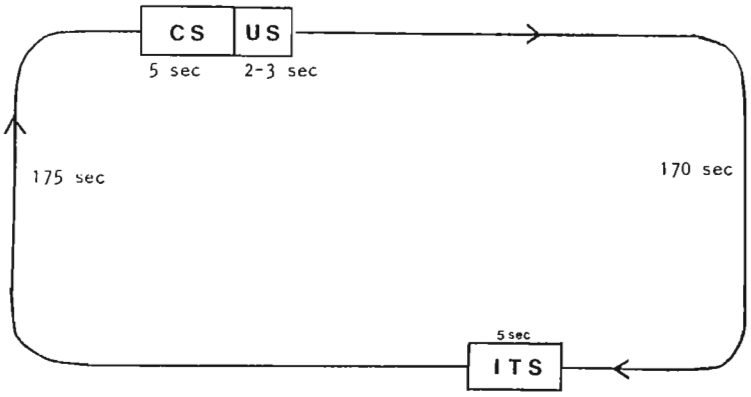

Fig. 1. Conditioning stimulus schedule.

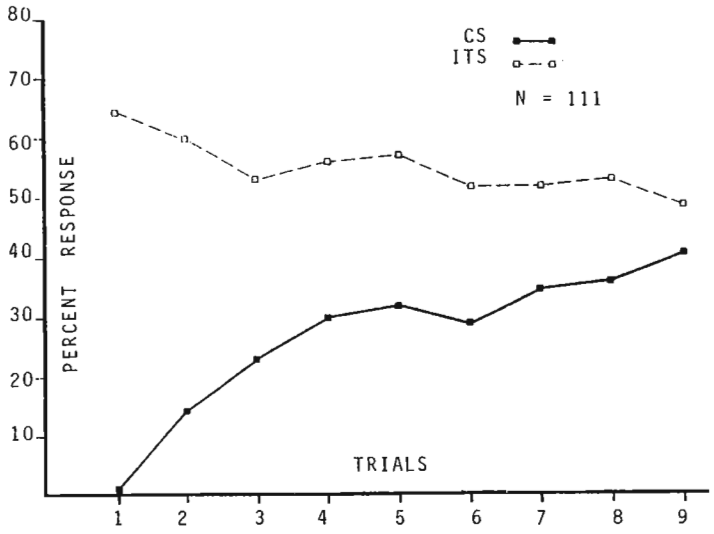

Fig. 2. Conditioning: Percent response over trials to CS and ITS for 111 flies (including the 22 flies from the paired group in Figure 3).

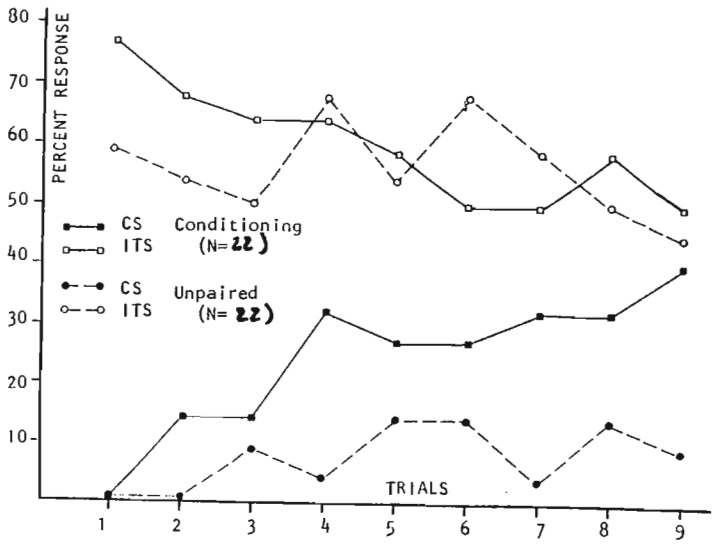

Figure 3. Percent response over trials to CS and ITS for an unpaired control and a paired conditioning group. (N = 22 per group).

$p<0.05$), thus showing conditioning, but (2) to the ITS decreases significantly though less steeply ($B=1.58$, $p<0.05$).

To demonstrate the effectiveness of the ITS for discharging CES, a control group was tested on the same day as a conditioning group--an unpaired CS was positioned to precede the US by 90 sec on the assumption that such a separation would prevent conditioning, which requires CS-US contiguity. Figure 3 shows the effect of any residual post-ITS CES on the conditioned response to be minimal. Over trials, the unpaired group (N=22) shows a much smaller increase in responding than does the paired (conditioning) group (N=22), with a significant difference between the two (Kolmogorov-Smirnov two-sample test, two-tailed probability: $Z=1.41$, $p<0.037$). Additional control experiments have demonstrated that neither sensitization nor pseudoconditioning has a significant influence on the conditioned response.

This unique procedure permits train/testing 18 individuals in a 54-min session and yields a complete response record of the conditioning of each individual.

References: Dethier, V.B., R.L. Solomon & L.H.Turner 1965, J.Comp.& Physiol.Psych. 60:303-313; Holliday, M., M.Vargo & J.Hirsch 1983, DIS 59: 140-141; Nelson, M.C.1971, J.Comp.& Physiol.Psych. 77:353-368; Vargo, M. M.Holliday & J.Hirsch 1983, Behav. Res.Meth & Instrumen. 15:(1)1-4; Vargo,M. & J.Hirsch 1982, J.Comp.& Physiol.Psych. 96:452-459.

Acknowledgement: This work was supported by a Grant for research training in Institutional Racism (MH15173), from the National Institute of Mental Health; a Biomedical Research Support Grant (NIH RR 7030-1-5-21332), from the National Institute of Health; a Grant for research in Behavior-Genetic Analyses (BNS-83-00353), from the National Science Foundation.

Kambysellis, M.P., P.Hatzopoulos, and E.M. Craddock. New York University (New York) and State University of New York (Purchase) New York USNA. Rapid in vivo incorporation of radioactive amino acids into vitellogenin proteins of Drosophila grimshawi.

In our studies on the timing of vitellogenin protein synthesis in D.grimshawi (Kambysellis, Hatzopoulos & Craddock 1984), we have found that by feeding the flies for a brief time (1-5 minutes) with radioactive amino acids, sufficient label is introduced into the flies to permit in vivo incoporation into proteins for several days. In representative experiments outlined here, groups of five mature 18 day old females were starved for 15 minutes, and then introduced into a feeding chamber which consisted of a plastic shell vial (10 cm long, 3.5 cm in diameter, Connecticut Valley Biological Supply Co., Inc.) into which was inserted a piece of Kimwipe paper (2x1.5cm) four layers thick. This paper was saturated with 100 µl of a 20% sucrose solution containing a mixture of radioactive amino acids, namely 25 µCi each of ^{3}H-aspartic acid (s.a. 10.0 Ci/mmole), ^{3}H-serine (s.a. 16.8 Ci/mmole), ^{3}H-glycine (s.a. 15.0 Ci/mmole), ^{3}H-lysine (s.a. 68.0 Ci/mmole) and ^{3}H-leucine (115.2 Ci/mmole), and 100 µCi ^{35}S-methionine (999.8 Ci/mmole). The paper was then placed in the middle of the vial which was lain on its side as is usual for culturing the large Hawaiian Drosophila. Once introduced into the vial, the flies are attracted immediately to the sucrose-amino acid mixture and feed continuously for about one to one and a half minutes. The same feeding chamber can be used to feed up to ten groups of flies in each experiment. Following feeding (pulse), the flies were placed either on regular Hawaiian Drosophila medium (Wheeler & Clayton 1965) or in empty vials for the chase period. At the end of variable periods of chase, the groups of five flies were anaesthetized, their hemolymph collected (Kambysellis 1984), and the fat bodies and ovaries dissected, homogenized in 50 µl of 50 mM Tris-HCl pH 8.2, 0.25M NaCl buffer, and centrifuged for one minute in an Eppendorf centrifuge. The aqueous phase

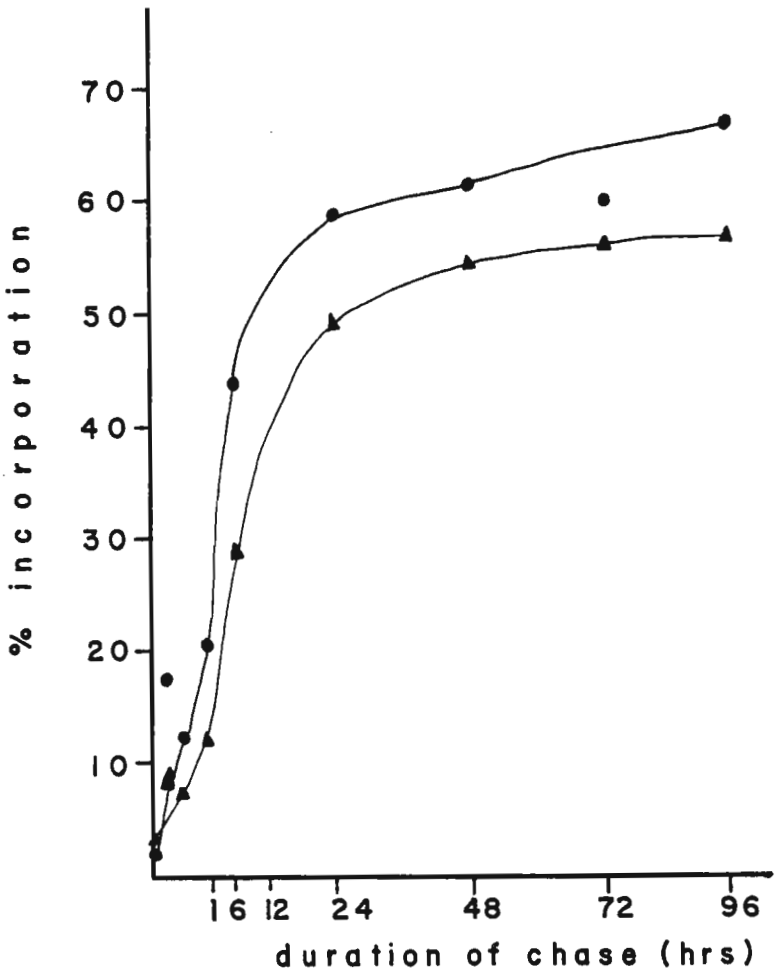

Fig. 1. Incorporation of radioactive amino acids into TCA insoluble proteins. Percentage of incorporation was determined by dividing the number of TCA precipitable counts in a particular sample by the total cpm in that same sample. Notice that the scale on the horizontal axis has been expanded for the first hour of the chase to disclose the four observations during that period.

Table 1. In vivo incorporation of ^{3}H- and ^{35}S-amino acids into proteins in adult female D.grimshawi. TCA precipitable counts in aliquots of tissue homogenates from groups of five 18-day old female flies fed radioactive amino acids for 1 min, followed by variable periods of chase, with the flies (a) maintained on food, or (b) starved for the chase period.

	(a) flies on food			(b) flies starved		
Chase period	hemolymph (1µl≅1 fly)	fat body (one)	ovaries (one pr)	hemolymph (1µl≅1 fly)	fat body (one)	ovaries (one pr)
0'	2,760	826	1,104	333	376	428
15'	1,816	521	2,143	2,047	754	1,639
30'	1,224	1,058	3,289	1,520	890	2,123
60'	1,740	1,117	4,802	3,963	2,575	6.090
6 hr	64,307	26,831	108,023	34,119	20,142	102,489
24 hr	75,602	48,344	248,557	-	27,106	127,111
48 hr	47,323	64,132	245,044	45,164	53,514	334,700
72 hr	-	45,885	251,302	-	-	-
96 hr	-	31,375	227,406	-	-	-

between the lipid and precipitated material was removed for analysis. Aliquots from each sample were assayed for: (a) total cpm; (b) incorporation of radioactive amino acids into TCA insoluble proteins; and (c) the presence of specific proteins on 7-12% T gradient SDS polyacrylamide gels.

As shown in Table 1, sufficient radioactivity was incorporated into proteins in all three tissue samples almost immediately after feeding. The low initial incorporation, which represents about 2% of the total radioactivity found in the tissues at that time (Fig. 1) steadily increased to reach 50% incorporation by 12 hr chase in the ovaries and 24 hr chase in the fat body (fig. 1). Maximum incorporation into TCA insoluble material was reached by 24 hr in hemolymph (Table 1) and 48 hr in the fat body of flies maintained on food for the chase. Incorporation into the ovary showed a different pattern, due to the uptake and storage of yolk proteins. The slow incorporation during the first hour of chase was followed by a sharp increase in the next 6 hr, reaching a maximum 24 hr and maintaining a plateau until 72 hr, after which a decrease was observed. In the flies maintained for the chase period in dry vials, where the precursor radioactive amino acids were not diluted by feeding on non-radioactive medium, incorporation continued to increase in all three tissues until the death of the flies between 48 and 72 hr of chase.

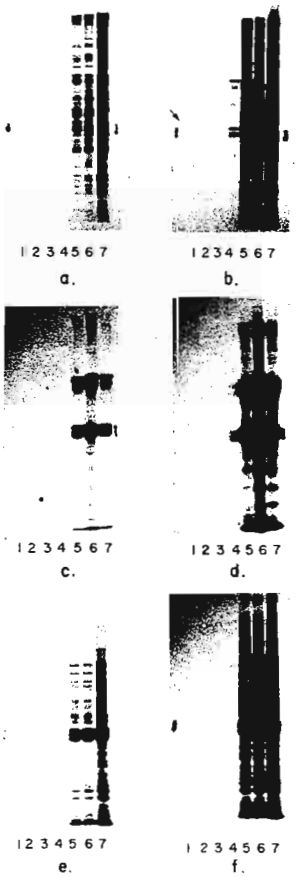

Fig. 2. Fluorographs of SDS-PAGE showing labeled proteins in fat body (a,b), hemolymph (c,d), and ovary homogenates (e,f). Samples were taken at various intervals after feeding the flies for one min with radioactive amino acids. The durations of the chase period are as follows: no chase (lane 1), 15' (lane 2), 30' (lane 3), 60' (lane 4), 6 hr (lane 5), 24 hr (lane 6) and 48 hr (lane 7). (a,c,e) represent 4 days exposure and (b,d,f) 13 days exposure of the X-ray films. The arrow points to the vitellogenin proteins. Black dots indicate the positions of the three yolk proteins present in control egg homogenates.

Fluorographs of the proteins resolved by gradient SDS-PAGE show that in the fat body (Fig. 2a,b), amino acids were incorporated into vitellogenin proteins almost immediately after the pulse, and by one hour of chase (lane 4), the three newly synthesized vitellogenin proteins and another unrelated protein of about 75,000 daltons were the predominant labeled proteins. By 6 hr, scores of other proteins had also been synthesized, some of which subsequently became even more abundant in this tissue than the vitellogenin proteins. Incorporation of labeled amino acids into proteins continued for four days with only a slight decrease in intensity.

In hemolymph (Fig. 2c,d), the first proteins to be detected in trace quantities at the 15 min chase were the three vitellogenins, followed by two larger proteins in the range of 75,000 daltons. These serum proteins together with the vitellogenins rapidly increased in quantity after one hour of chase, and reached a maximum by 24 hr. They remained as the predominant hemolymph proteins throughout the chase.

In the ovaries (Fig. 2e,f), the appearance of the vitellogenin proteins was similar to that in the hemolymph. They were detected following 15 min chase and by 6 hr represented the predominant class of egg proteins. Several other less abundant proteins showing a pattern of electrophoretic mobility similar to that of proteins in the fat body were also found after the 6 hr chase. Whether these are identical proteins in the two tissues is not known.

It is of interest to notice the quantitative differences in the three vitellogenins between tissues. In the fat body, the V_2 protein is synthesized in lower quantitites than the V_1 and V_3 proteins, while in the hemolymph and ovary all three proteins are present in roughly equimolar amounts. This observation supports the suggestion that the vitellogenins synthesized in the D.grimshawi ovary (Kambysellis, Hatzopoulos & Craddock 1983) are secreted into the hemolymph prior to their sequestration by the oocyte. Experiments to document this assumption are now in progress.

Supported by NIH grant AG 01870 and NSF grant PCM-7913074.

References: Kambysellis, M.P., P.Hatzopoulos & E.M.Craddock 1983, Genetics 104:s39; Kambysellis, M.P. 1984, DIS 60: ; Kambysellis, M.P., P.Hatzopoulos & E.M.Craddock 1984, W.Roux's Archiv. submitted; Wheeler, M.R. & F.E.Clayton 1965, DIS 40:98.

Kekić, V., M.Andjelković and G.Bächli.
University of Belgrade, Yugoslavia, and University of Zurich-Irchel, Switzerland.

Studies of Drosophilidae (Diptera) in Yugoslavia. V.Collections from Mljet.

At the end of August and beginning of September 1982, we collected Drosophilidae ssp. from two localities on the Adriatic island of Mljet: Pomena and St.Mary.

Pomena is characterized by an abundant vegetation of aleppo pine (Pinus halepensis), holm oak (Quercus ilex) and other kinds of plants characteristic of those forests. At this place we also studied the dispersal rate of D.subobscura, see Taylor et al. 1984. St.Mary is a small island about 300 m in diameter, in a bay of Large Lake on Mljet; it is an island about 300 m in diameter, in a bay of Large Lake on Mljet; it is an island within an island. Here a Benedictine Monastery, St.Mary, was built in the twelfth century and has since been renovated to become a small hotel. Although this location is much drier than Pomena, several species of pine (Pinus pinea, Pinus halepensis), cypress (Cupressus sempervirens), bay (Laurus nobilis) and olive (Olea europea) are found here, as are many cactic (especially Opuntia ficus indica). The ground is covered with characteristic grasses and rocks. In both localities the flies were caught by sweeping nets over fermenting mixed fruit (watermelon, grape and apple) baits exposed on open plates. Flies were preserved in alcohol and brought to the laboratory for identification. In Pomena they were collected only in the evening; at St.Mary they were collected in the mornings as well.

The results are shown in Table 1. It can be seen that in the relatively "wilder" habitat, Pomena, subobscura flies are six times as numerous as melanogaster/simulans flies. This ratio is reversed at St.Mary, where many more melanogaster/simulans flies were collected, the ratio being 1:2.5.

The ratio between sibling species, melanogaster:simulans, is also different in these two localities: at Pomena it is 1:4.3, while at St.Mary it is 1.2:1.

Table 1. Drosophila found in two collection locations on Mljet, Yugoslavia.

Species	Pomena evening		St. Mary evening		St. Mary morning	
	males	females	males	females	males	females
Drosophila:						
D.cameraria		2				
D.funebris			1	1		1
D.hydei						1
D.melanogaster	51	73	183	323	193	322
D.phalerata	1	2				2
D.simulans	233	303	164	200	233	240
D.subobscura	1077	2995	172	304	72	20
D.testacea		1		2	1	
Acletoxenus:						
A.formosus	1					
Leucophenga:						
L.maculata	2	2				
Scaptomyza:						
S.pallida	2	1	1	2	1	1
TOTAL	1367	3379	521	832	500	587

Table 2. Summary of Drosophila found from Adriatic collection sites in Yugoslavia.

Species	Localities			
	Mljet	Brioni	Kupari	Porec
Drosophila:				
D.ambigua				1
D.busckii		1		
D.cameraria	2			
D.funebris	3	3		8
D.helvetica				39
D.histrio				5
D.hydei	1	24	7	
D.immigrans		8	48	63
D.littoralis				1
D.melanogaster	1145	136	178	512
D.obscura				1
D.phalerata	5			58
D.repleta			5	
D.simulans	1373	443	1135	204
D.subobscura	4640	178	1957	2811
D.testacea	4		3	149
D.transversa			1	1
D.tristis				1
Acletoxenus:				
A.formosus	1			
Amiota:				
A.flavopruinosa			1	
Leucophenga:				
L.maculata	4			
Scaptomyza:				
S.pallida	8			3
TOTAL	7168	793	3335	3857

The sex ratio of the dominant Drosophila species in these localities is also interesting--especially of D.subobscura. In the evening collections of Pomena only 26.5% males were found, while at St.Mary 36% and 78% males were found in the evening and morning collections respectively.

In Fig. 1, the localities in Yugoslavia in which Drosophilidae fauna have been studied up to now are identified. Comparing the Adriatic sites, Table 2, it can be seen that Drosophila fauna of Mljet more closely resembles that of the North-Adriatic island Briono (Kekić & Marinkovic 1979) than that of Kupari or of Poreć (Bächli & Kekić, in press).

References: Kekić, V. & D.Marinkovic 1979, Aquilo Ser. Zool. 20:118-128; Taylor, C.E., J.R.Powell, V.Kekić, M.Andjelkovic & H.Burla 1984, Evolution, in press.

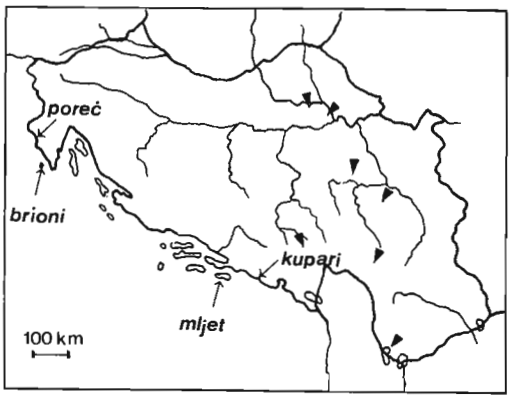

Fig. 1. Collection localities in Yugoslavia.

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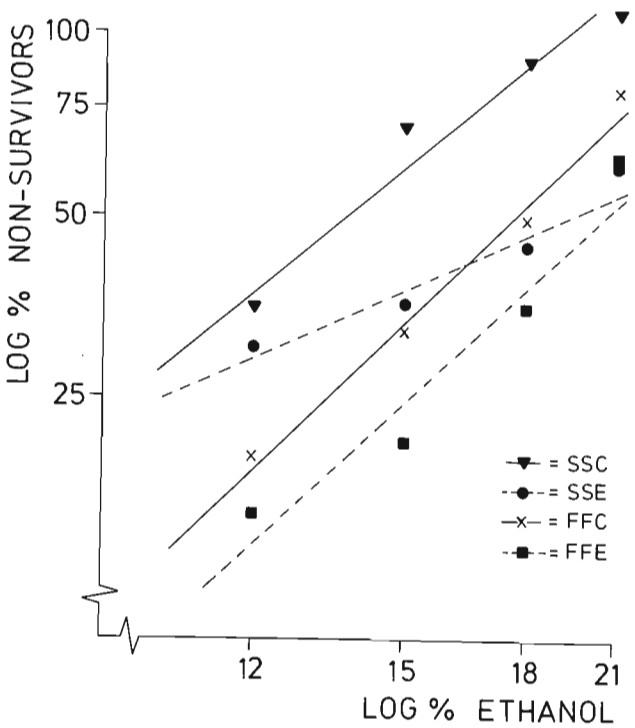
Adaptation of Drosophila melanogaster after long term exposure to ethanol in relation to the alcohol dehydrogenase polymorphism.

Strains of Drosophila melanogaster, either homozygous for the Adh^S allele or for the Adh^F allele were kept for a long time on food supplemented with ethanol. After 100 generations the survival of these strains (SSE and FFE) was compared with the survival of the control strains (SSC and FFC). Both in the juvenile and in the adult life stages the ethanol tolerances, expressed as median lethal doses (LD50) showed a significant increase (Table 1). In both life stages, however, no consistent relation with the in vitro ADH-activity was found. These findings are in contrast with those of McDonald et al. (1977), who reported increased ADH-activity and ADH-amount in a strain selected for increased tolerance to ethanol by exposing it to ethanol vapor. Perhaps the difference between their and our results can be explained by the difference in the employed selection procedure.

Table 1. Median lethal doses (LD50) of ethanol (% v/v) for the survival of adapted and the control strains; 95% confidence intervals given in parentheses.

Adh-genotype	adult survival		egg-to-adult survival	
	control strain	adapted strain	control strain	adapted strain
SS	23.9(23.1-24.7)	27.2(26.3-28.1)	12.6(11.9-13.3)	18.4(16.1-20.9)
FF	28.3(27.4-29.3)	30.2(29.2-31.2)	16.8(15.7-17.9)	20.2(18.5-22.0)



EGG-TO-ADULT SURVIVAL FOR DIFFERENT Adh-GENOTYPES. RELATIVE TOLERANCE TO ETHANOL

It will be clear that the strongest selection takes place during the pre-adult life stage and consequently adaptation will mainly be induced during this life stage. Therefore it appeared promising to plot relative resistance of the selected and unselected strains for egg-to-adult survival.

Figure 1 shows that the curves of the two control strains (SSC and FFC) are not quite parallel, but their regression coefficients do not differ significantly as may be expected for 2 strains derived originally from the same population (Van Delden et al. 1978). The FFC strain will survive better on ethanol supplemented food compared to the SSC strain, because of the difference in ADH activity (Van Delden et al. 1975, 1978) and therefore the FFC curve lies below the SSC curve.

The curve for the ethanol selected F strain (FFE) goes parallel to that of the FFC strain but has been shifted to the right; this is an indication that, in this case, adaptation has been induced by the Adh-locus only. In case of the SSE strain, however, the curve has not only been shifted to the right, but itsslope has been altered too. Therefore also other loci are involved in the process of adaptation. The SSE and FFE curves show an intersection point at 21% ethanol and it can be

extrapolated that at higher ethanol concentrations SSE would survive better than FFE.

The overall conclusion is, that by keeping the strains on ethanol supplemented food, both the SSE and the FFE strains have increased their tolerance to ethanol significantly. The mechanism of this adaptation remains unclear because no consistent relations between ADH-activity and survival were found. It is clear, however, that the adaptation has not been realized in the same way for the S and the F strains. Furthermore, adaptation for the SSE strain has been relatively better than for the FFE strain.

References: VanDelden, W., A.C.Boerema & A.Kamping 1978, Genetics 90:161-191; VanDelden, W., A.Kamping & H.vanDijk 1975, Experientia 31:418-419; McDonald, J.F., G.K.Chambers, J.David & F.J.Ayala 1977, Proc.Natl.Acad.Sci USA 74:4562-4566.

Knoppien, P. University of Groningen, The Netherlands. No evidence for rare male mating advantage in Drosophila melanogaster for strains raised at different temperatures.

Rare male mating advantage, which can be defined as frequency-dependent male sexual fitness with an advantage for the rare type, has become a widely discussed phenomenon (Bryant et al. 1980; Spiess 1982). There is some evidence that the rare male effect can occur among strains which only differ phenotypically (Dal Molin 1979; Grant et al. 1980). It has been shown that the rare male effect can be induced by different raising temperatures in Drosophila pseudoobscura (Ehrman 1966) and in Drosophila persimilis (Spiess 1968). In this paper it will be asked whether rare male mating advantage also occurs in Drosophila melanogaster for strains which differ only in raising temperature. The relevance for rare male mating advantage of a difference in male mating success, which was found between the strains, will be discussed.

All flies used were homozygous Fast for the alcohol dehydrogenase-locus, and derived from the Groningen base population (VanDelden et al. 1978). The flies for the mating experiments were raised as larvae and stored either at 20°C or at 29°C. Parents of these flies laid eggs in bottles, for 5 days at 20°C, or for 3 days at 29°C; each bottle contained 15 pairs. Composition of the food and methods for collecting and storing virgin flies are described by Pot et al. (1980). For each run of an experiment 50 pairs were used at a particular ratio of types (type is here defined as a group of flies raised at a particular temperature). Type frequency was varied simultaneously for both sexes. All mating experiments were done at 25°C, and lasted 30 minutes. Copulating pairs were removed from the mating chamber, while the type of each individual was recorded (see Pot et al. 1980 for further details). Flies were marked either with a minimal amount of red or green fluorescent dust for identification alternating the color between runs. To minimize possible effects of day to day variation in mating success on the frequency-dependent effect, experiments were conducted for all three ratios at the same day, varying the sequence in which the runs were done. Six runs were performed for each ratio at successive days. Virgin flies were six days old in three of these days and 12 days old in the other three days.

Differences in mating success were determined according to a method proposed by Pot et al. (1980). Following this method a mating chance ratio r was defined as follows. Let a be any given male of type A, any given male of type B, present in the mating chamber at a given moment.

$$\text{Then } r = \frac{P(a \text{ is the next male to mate})}{P(b \text{ is the next male to mate})} .$$

For statistical tests to determine whether r differs from unity, and to test whether r differs from one experiment to another, we refer to Pot et al. (1980).

The results are summarized in Table 1. For females no difference in mating success was detectable between flies raised at low and high temperature ($P>0.1$). On the contrary males raised at low temperature have a significant higher mating success than males raised at high temperature ($P<0.001$). It is suggested that this is the case because low raising temperature enhances size in Drosophila melanogaster. Large size generally enhances mating success for Drosophila species (Ewing 1961; Ehrman 1966). Differences between r-values were tested for each combination of ratios in order to detect any possible frequency-dependent effect. None of these tests gave significant results, nor for males nor for females.

It is suggested by some authors that differences in mating success between strains can give rise to a rare male effect (Bryant et al. 1980; Ewing 1978). According to Bryant et al.

Table 1. Differences in mating success for Drosophila melanogaster flies raised at 20°C (A) and 29°C (B), depending on frequency.

Frequency of type A	# Runs	Matings (♀ x ♂) AxA	AxB	BxA	BxB	r♀	r♂
0.1	6	3	12	26	142	0.82±0.22	3.16±0.66
0.5	6	63	15	59	42	0.77±0.12	3.77±0.62
0.9	6	152	6	19	1	0.98±0.23	3.56±1.39

(1980) this is due to the fact that when males of the more successful strain are rare, they have to compete with only a few other successful males, which would imply one-sided rare male mating advantage in favor of the more successful strain. In a model applying truncation selection (Ewing 1978), it is predicted that rare male mating advantage will occur when strains differ in mating success. In accordance with this prediction rare male mating advantage was found for the strains used by Ewing (1978) when they differed in size, and consequenty in mating success, whether size differences were genotypically or phenotypically determined. It is shown in this paper as well as by the results of Pot et al. (1980), who found no rare male effect for alcoholdehydrogenase variants of Drosophila melanogaster, which differed considerably in mating success, that even a large difference in mating success does not necessarily imply rare male mating advantage.

References: Bryant, E.H. et al. 1980, Genetics 96:975-993; Dal Molin, C. 1979, Amer. Natur. 113:951-954; Ehrman, L. 1966, Anim.Behav. 14:332-339; Ewing, A.W. 1961, Anim.Behav. 9:93-99; Ewing, A.W. 1978, An investigation into selective mechanisms capable of maintaining balanced polymorphisms, PhD Thesis, Porthmouth, Polytechnic; Grant, B. et al. 1980, Evol. 34:983 -992; Pot, W. et al. 1980, Behav.Genet. 10:43-58; Spiess, E.B. 1968, Amer.Natur. 102: 363-379; Spiess, E.B. 1982, Amer.Natur. 119:675-693; Van Delden, W. et al. 1978, Genetics 90:161-191.

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Use of zeste suppression in a chromosome carrying a white duplication to facilitate the scoring of Minute mutations.

In 1977, Huang published a report on the induction of Minute mutations by MMS and MNNG. He stated that the method, requiring only one generation, would be a favourable alternative to the sex-linked recessive lethal test, for routine testing of chemical compounds. It seems a tedious, job, however, to score objectively small numbers of Minute mutations among large numbers of flies. The study of Persson (1976) showing that several Minutes act as suppressors of zeste in a particular duplication of white suggests the possibility of scoring Minute mutations as eye colour changes. Based on this, we attempted an experiment in which, after treatment with the chemical mutagen methyl methanesulfonate (MMS), F_1 flies were scored for eye colour changes, and afterwards checked for a Minute phenotype.

It appeared to be critical which duplication of white was used. It was observed in a pilot experiment that, among several duplication stocks obtained from the Umea stock center, the "Dp(1:1)w^{rG}, y ac z" (Persson used this indication in his original paper) and the (probably identical) "Dp(1:1)3C1, y ac z" (no. 91) did not show any z suppression effect with several 2nd and 3rd chromosome Minutes, whereas "Dp(1:1)3A6-3C2, y ac z" (no. 90) and "Dp(1:1)3A6-3C2, y^2 ac z" (no. 23) did. For the mutation experiment chromosome no. 90 was selected.

The test scheme used was as follows: Berlin-K male flies were treated with 1 mM MMS for 24 hours, and subsequently mated with virgin females heterozygous for the white duplication chromosome no. 90 and the Basc chromosome. In both the treatment and the control group 20 culture bottles were set up each containing 5 treated males and 10 females. y ac Males (carrying the duplication chromosome) were scored for non-zeste eyes. A normal sex-linked recessive lethal test was run concurrently.

Table 1. Classification of red-eyed males in the 1 mM MMS treatment series (3069 y ac males counted).

	Minute complete	Minute mosaic	Minute$^+$	Total
red eye complete	2	4	1	7
red eye mosaic	-	18	8	26
Total	2	22	9	33 (1.1%)

In the control series, 1283 y ac males were counted, all of which had zeste eyes. In the MMS treated series 3069 y ac males were scored. The results (Table 1) show that in this group a sizeable number of red-eyed males was observed, most of which showed a Minute phenotype as well. Interestingly, the majority were mosaics, having only one red eye or red sectors in one or both eyes, the Minute phenotype often being expressed only by the missing of verticals or aristae. 29 Retests were fertile. Of these, only 2 showed transmission of the red-eyed and Minute phenotypes. Among these was one of the two flies showing the complete phenotype for both red-eye and Minute. The other one was mosaic for both eye colour and Minute. The preponderance of mosaics among MMS-induced mutations is in accordance with date of Lee (1976) on mosaics among induced visibles and with the ratio of mosaic versus complete recessive lethals being relatively high for MMS (Vogel & Natarajan 1979).

The frequency of induced Minutes is quite similar to the rates found by Huang (1977) with MMS at a recessive lethal induction of 16-22%. (This author does not mention the occurrence of mosaics). If the number of Minute loci on chromosomes 2 and 3 is taken as 30-40 (Huang 1977) and the number of loci on the X-chromosome mutable to recessive lethal as 800 (Abrahamson et al. 1980), our figures for induced Minutes (1.1% for all red-eyed males, 0.8% for the red-eyed males also showing Minute) and the simultaneoulsy obtained recessive lethal frequency of 21.4% are not far apart on a per locus basis. This would suggest that indeed most induced Minutes act as suppressors of zeste and can be scored as eye-colour changes.

In conclusion, the principle of scoring Minute mutations by the more objective criterion of the eye-colour change appears to wrok. However, in our experience the test is not likely to take less time than the regular sex-linked recessive lethal test. This applies also when large sample sizes are needed for the detection of weak mutagens. Moreover, performing a brood pattern analysis with individual pairings would be more cumbersome because the numbers of treated males have to be considerably larger than in the case of the recessive lethal test.

References: Abrahamson, S. et al. 1980, Envir.Mutag. 2:447-453; Huang, S.L. 1977, Mutation Res. 44:145-148; Lee, W.R. 1976, in: M.Ashburner & E.Novitski, eds, The Genetics and Biology of Drosophila melanogaster, V.1c:1299-1341; Persson,K. 1976, Hereditas 82:111-120; Vogel, E. & A.T.Natarajan 1979, Mutation Res. 62:51-100.

Krimbas, C.B. and M.Loukas. Agricultural College of Athens, Greece. Further addition to the Greek fauna.

One male of Drosophila subsilvestris was captured in Karpenissi on June 18, 1981, in a collection of 1199 Drosophila flies. It is the first time subsilvestris is recorded in Greece. The male was identified by crosses with virgin females of other European obscura group species: it produced repeatedly offspring only with subsilvestris virgin females. The other flies of this collection were 1169 D.subobscura, 17 D.obscura, 1 D.ambigua, 1 D.helvetica, 3 D.immigrans and 8 D.cameraria. Until now 23 species of Drosophila have been recorded from Greece.

Kuhn, D.T., D.F.Woods[2] & D.J.Andrew[3].
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2 University of California, Irvine;
3 University of California, San Diego
USNA. Mapping of tuh-3 between pbx and fl in D.melanogaster.

The tumorous-head mutant gene (tuh-3) was mapped in partial aneuploids and by recombination studies distal to pbx (3.58.8+) and presumably is one of the most posterior bithorax-complex genes (Kuhn et al. 1981; Kuhn and Woods 1982). These studies did not tell us how distal tuh-3 was to pbx. We report here the results of our mapping tuh-3 between postbithorax (pbx) and fluted (fl). We mapped fl to 3-59.7+. Female tumorous-head flies were mated to sbd^2 bx^3 pbx fl/ TM1 males. F_1 females were backcrossed to sbd^2 bx^3 pbx fl/ TM1 males. Male offspring that were fl only were isolated for further testing. Twenty males were shown to carry a 3rd chromosome in which a cross-over occurred somewhere between pbx and fl. Identity matings were necessary to determine presence or absence of tuh-3. From sbd^2 bx^3 pbx tuh-3^+ fl/ sbd^+ bx^+ pbx^+ tuh-3 fl^+ females, cross-over bearing males that were either tuh-3 fl or tuh-3^+ fl were selected. The recombinant chromosomes were balanced over TM1.

Table 1. Localization of tuh-3 by analyzing cross-overs between pbx and fl in 3R.

Strain	Tumorous-head defect				Male genital disc defect			
	with	without	total	% penetrance	with	without	total	% penetrance
pbx tuh-3^+ fl / X → pbx^+ tuh-3 fl								
pbx^+ tuh-3 fl^+								
#3	65	38	103	63.1	32	70	102	31.4
#6	74	28	102	72.5	78	25	103	75.7
#11	69	30	99	69.7	80	21	101	79.2
#17	54	51	105	51.4	36	66	102	35.3
#23	83	20	103	80.6	55	70	125	44.0
#25	59	38	97	60.8	85	19	104	81.7
#26	67	32	99	67.7	24	83	107	22.4
#47	74	34	108	68.5	35	64	99	35.4
#49	76	51	127	59.8	54	47	101	53.5
#50	61	18	79	77.2	2	37	39	5.1
#81	70	46	116	60.3	59	42	101	58.4
#86	93	9	102	91.2	53	49	102	52.0
#90	47	16	63	74.6	72	55	127.	56.7
#92	68	36	104	65.4	42	68	110	38.2
#94	74	26	100	74.0	59	48	107	55.1
Totals	1034	473	1507	68.6	766	764	1530	50.1
pbx tuh-3^+ fl / X → pbx^+ tuh-3^+ fl								
pbx^+ tuh-3 fl^+								
#10	0	100	100	0.0	0	100	100	0.0
#21	0	111	111	0.0	0	118	118	0.0
#57	0	108	108	0.0	1	44	45	2.2
#73	0	102	102	0.0	0	106	106	0.0
#96	0	131	131	0.0	0	101	101	0.0
Totals	0	552	552	0.0	1	469	470	0.2

#57/Df(3R)P9 uncovers tuh-3

Two different genetic tests were needed to detect presence of tuh-3. The mutant gene acts as a semidominant in the presence of an X-linked maternal effect gene, symbolized tuh-1h, to cause growths of abdominal and/or genital tissue in the head. Tuh-3 acts as a simple recessive in the presence of a second maternal effect gene, symbolized tuh-1g, which causes the defect to switch from the head to the posterior of the fly where internal and external genitalia may be completely absent. The maternal effect genes tuh-1h (head defects) and tuh-1g (genital defects) are naturally occurring alleles. The semidominant head defect phenotype was detected among F_1 progeny resulting from mating males of each cross-over strain to females that carried an attached X-chromosome homozygous for tuh-1h and a 3rd chromosome gene that enhances the penetrance and expression of the head defects. The recessive phenotype was found by mating the appropriate males to females possessing an attached X-chromosome homozygous for tuh-1g and backcrossing the attached X-chromosome bearing female offspring to the males from each strain being tested.

Table 1 shows that 15 of the 20 cross-overs between pbx and fl occurred distal to or right of tuh-3. The remaining 5 cross-overs were between pbx and tuh-3. Tuh-3 was mapped much closer to pbx at 58.8+ than to fl at 59.7+. This places tuh-3 at about 59.0+. However, it must be emphasized that an exact placement of tuh-3 was not possible because the viability of flies with recombinant chromosomes was quite low. A total of 93 males was originally selected as potential cross-over types. Of these, 40 either were sterile or did not survive even light etherization. The 15 tuh-3 fl cross-over types showed both the head defect with tuh-1h and the genital defect with tuh-1g. The tumorous-head phenotype was seen in 68.6% of the flies examined with average penetrance ranging from a low of 51.4% to a high of 91.2%, while the genital disc defect was found in 50.0% of the males examined with a low of 5.1% penetrance to a high of 81.7% penetrance. The five tuh-3^+ fl strains of flies lacked a mutant phenotype when confronted with either maternal effect gene. Strain #57 had 1 of 45 males showing the trait. However, when the tuh-3^+ fl chromosome was made hemizygous for tuh-3^+ by placing it with Df(3R)P9, which uncovers tuh-3, none of the 100 males tested showed the phenotype.

This research was supported by NIH Grant AG 01846.

References: Kuhn,D.T., D.F.Woods & D.J.Andrew 1981, Genetics 99:99; Kuhn, D.T. & D.F. Woods 1982, DIS 58:96.

Larochelle, C., J.Côté, and F.Garcin. Laval University, Quebec, Canada. The ethanol metabolic pathway in D.melanogaster and D.simulans.

The two cosmopolitan sibling species D.melanogaster and D.simulans are able to use environmental ethanol as source of energy; however, D.melanogaster exhibits better capacity to handle high ethanol concentrations than D.simulans (Parsons et al. 1979).

This differential tolerance to ethanol could be explained at the biochemical level by a much higher alcohol dehydrogenase (ADH) activity in D.melanogaster than in D.simulans. Nevertheless the ADH-mediated oxidation of ethanol generates acetaldehyde, a highly toxic product. For fly survival it is essential that acetaldehyde be rapidly oxidized into acetate.

In most animal species this critical step involves and NAD^+-dependent aldehyde dehydrogenase (ALDH). In Drosophila, aldehyde oxidase (ALDOX) a flavine enzyme using many aldehydes as substrates, was assumed to be the active enzyme (see Courtright 1967; Dickinson 1970, 1971). Recently we have provided evidence that ALDH is present in Drosophila and that its physico-chemical properties are very similar to those of other animal species (Garcin et al. 1981,1983).

In these experiments we have carried out a comparative study of ADH, ALDH, and ALDOX activities in D.melanogaster and D.simulans. We show that ADH and ALDH activities are directly correlated with the level of tolerance to ethanol and acetaldehyde whereas ALDOX activities are inversely correlated.

Drosophila melanogaster collected from Colmar (France) and Drosophila simulans collected from Villeurbanne (France) were kindly made available by Prof. J. David Laboratoire de Biologie et de Genetique Evolutive Gif-sur-Yvette (France). The flies were grown in low density populations on Drosophila medium (Carolina medium 4-24) and maintained in an incubator providing a constant temperature (25±1°C) and an 18 hr light/6 hr dark photoperiod with fluorescent light. Adult flies, 5 to 6 days old were frozen in liquid nitrogen, homogenized and centrifuged as described in Garcin et al. 1983. The resultant supernatants were kept

Table 1. Specific activities of ADH, ALDH and ALDOX in D.melanogaster and D.simulans.

	D.melanogaster	D.simulans
ADH mU/mg prot.	74.0±4*	23.0±0.8
ALDH mU/mg prot.	25.7±1.5	11.3±0.6
ALDOX U/mg prot.	0.74±0.02	1.41±0.13

* each value represents the mean ±SD of 4 independent experiments.

Table 2. Ethanol and acetaldehyde toxicity in D. melanogaster and D.simulans.

	D.melanogaster	D.simulans
Ethanol LC_{50}	11.3	4.2
Acetaldehyde LC_{50}	2.6	1.5

at -80°C until assayed. ADH and ALDH activities were determined spectrophotometrically by monitoring the formation of NADH at 340 nm (see Garcin 1979; Garcin et al. 1983). ALDOX activity were determined according to Dickinson (1971). Protein concentrations were measured according to the method of Bradford (1976) using serum albumin as the standard. Enzymatic specific activities are expressed in Units (or milliUnits) per mg. protein.

Table 1 shows the results obtained in the two sibling species for the activities of ADH, ALDH and ALDOX. These data were obtained from four independent experiments. ADH and ALDH in D.melanogaster are respectively threefold and two fold higher than those in D.simulans. In contrast ALDOX activity is twofold higher in D.simulans.

For comparison purposes we present in Table 2 in vivo data obtained in previous experiments on ethanol and acetaldehyde toxicity in the two sibling species. It can be seen from the concentrations inducing 50% lethality in the population, (LC 50s) that D.melanogaster is much more tolerant to both agents than D.simulans.

Thus it appears from our biochemical data that both dehydrogenases(ADH and ALDH) play a significant biological role for the expression of alcohol and acetaldehyde tolerance in the two species. The data on ALDOX activity confirm our previous hypothesis that this enzyme is possibly not involved in the ethanol metabolic pathway. Though the biological role of ALDOX is not yet precisely known, our data suggest that the higher ALDOX activity in D.simulans could confer to this species an adaptive advantage over D.melanogaster in environments where other aldehyde substrates are present in large concentrations.

References: Bradford 1976, Analyt.Biochem. 72:248; Courtright, J.B. 1967, Genetics 57: 25; Dickinson, W.J. 1970, Genetics 66:487; ____ 1971, Devl.Biol. 26:77; Garcin 1979, in Metabolic effects of Alcohol (Avogaro, Sirtoli & Tremoli, eds), Elsevier-Amsterdam; Garcin, F., J.Cote & S.Radouco-Thomas 1983, Comp.Biochem.Physiol. 75B:205; Parsons et al. 1979, Aust.J.Zool. 27:767; Garcin,F., S.Radouco-Thomas, T.Cote & C.Radouco-Thomas 1981, Prog. in Neuropsychopharmacol. 5:619.

Latorre, A., R.deFrutos & L.Pascual. Universidad de Valencia, Espana. Loci activity in three A chromosomal arrangements of Drosophila subobscura.

The authors who have studied the patterns of puffing activity in several Drosophila strains carrying different chromosomal aberrations found that, in general, chromosomal arrangements do not affect puffing (see revision of Ashburner & Berendes 1978). A great similarity in the puffing patterns of species carrying different inversions was also obtained by Moriwaky & Ito (1969). However, deFrutos & Latorre (1982) found some differences in the puffing patterns of two different U chromosomal arrangements.

In the present work, patterns of puffing activity of the sex chromosome of three strains are statistically compared. The strains studied were: H271, from a locality near of Helsinki (Finland), which is homozygotic for the A_{st} arrangement; Ra121, from Las Raices, Canary Islands (Spain), which is homozygotic for the A_2 arrangement and R225, from Ribarroja, Valencia (Spain), which was fixed in homozygous for the A_1 chromosomal arrangement. The study was carried out at the 0h. prepupa stage, which coincides morphologically with the eversion of the anterior spiracles. A total of 50 preparations were prepared per strain. Of these, only the females were analyzed. Thus, a total of 33 preparations for A_{st} chromosome,

Table 1. Mean values and standard error (based on two repetitions) of the number of active loci in the A chromosome at 0h. prepupa stage in H271, Ra121 and R225 strains.

Puffs	H271	Ra121	R225
1C	0.020±0.020	0.000±0.000	0.000±0.000
2C	0.005±0.005	0.155±0.005	0.095±0.005
4A	0.025±0.025	0.230±0.100	0.000±0.000
5D	0.535±0.055	0.340±0.050	0.530±0.000
6E-7A	0.005±0.005	0.060±0.010	0.025±0.015
8E/9A	0.025±0.025	0.000±0.000	0.005±0.005
9B	0.160±0.020	0.060±0.050	0.025±0.025
9D	0.010±0.010	0.150±0.060	0.020±0.020
10AB	0.310±0.030	0.300±0.077	0.310±0.051
11D	0.045±0.045	0.085±0.045	0.120±0.040
12	0.440±0.150	0.395±0.045	0.175±0.085
13A	0.005±0.005	0.115±0.085	0.060±0.020
13BC	0.285±0.045	0.340±0.150	0.735±0.025
13E	0.030±0.010	0.135±0.095	0.035±0.005
14B/C	0.095±0.085	0.000±0.000	0.025±0.015
14CD	0.005±0.005	0.120±0.090	0.015±0.015
15B/C	0.010±0.010	0.025±0.025	0.000±0.000
15DE	0.195±0.005	0.140±0.070	0.050±0.050
16B	0.275±0.015	0.370±0.140	0.105±0.045

Table 2. Analysis of Variance.

Source of Variation	SS	d.f.	MS	F
Strain	0.0277	2	0.0139	0.79 ns
Puff	2.3840	18	0.1324	7.57 *
Strain x Puff	0.6300	36	0.0175	3.24 *
Error	0.3052	57	0.0054	

ns: not significant; * $P < 0.001$

29 for A_2 chromosome and 32 for A_1 chromosome were analyzed. In each preparation five nuclei were observed. The obtention of the cultures as also the dissection of the salivary glands and the method of chromosome squash preparation used were identical to those described by deFrutos & Latorre (1982). In each locus only the presence (+) or absence (-) of activity were considered, and the results are given in frequencies out of the total of chromosomes analyzed.

On considering as a puff all the loci found active even once among the total chromosomes studied, a total of 19 puffs were found in A_{st} chromosome, 17 in A_2 chromosome and 16 in A_1 chromosome. The list of puffs is given in Table 1. In A_2 chromosome only 16 puffs were taken into account, because puff 12D was not analyzed. This locus is found in an active state as a consequence of the A_2 inversion (Stumm-Zollinger 1953) that divides the 12 region into 12AC and 12D. In the A_{st} and A_1 chromosomal arrangements the whole region becomes active (puff 12) whereas in A_2 chromosomal arrangement activity was found at the two loci (12AC and 12D) and their moments of activity did not always coincide in the same chromosome. That is to say, it is possible that through the effect of the inversion a differential activity in 12 region was originated. A more detailed study of this region, in order to compare the results in a greater number of strains, is being carried out.

In this species the low activity of the A chromosome in 0h. prepupa is remarkable, and differs from that obtained in the autosomes. A chromosome is the only one that presents a peak of activity in third instar with a decrease of activity in 0h. prepupa (Pascual et al. Genetica, in press).

To compare the results among the strains, two-way analyses of variance with repetition were carried out. Repetitions were obtained taking two sets of fairly equal number of preparations at random. The frequency of appearance of each puff is calculated in each repetition. Mean values and errors are given in Table 1. In Table 2 the results of the variance analysis are shown.

Differences were not found between the chromosomes of the three strains. On the other hand, as was hoped, there do exist differences among puffs. Nevertheless, the existence of a significant interaction strain x puff must be interpreted. That is to say, while a similarity in the total activity of the three strains at 0h. prepupa exists, and the greater or lesser total activity of the puffs is independent of the strain, there are puffs which are more active in some strains than in others and puffs which are less active in some strains than in others.

Further analysis is required before it can be understood whether the existence of the interactions are due to a specific effect of the inversions more than to a strain effect.

References: Ashburner, M. & H.D.Berendes 1978, The Genetics and Biology of Drosophila, V.2b:315-395, Acad.Press; deFrutos, R. & A.Latorre 1982, Genetica 58:177-188; Moriwaky, D. & S.Ito 1969, Jap.J.Genet. 44:129-138; Stumm-Zollinger, E. 1953, Z.Vererblehre 85:382-407.

Lichtenstein, P.S.[1,2], M.Emmett[1], L.Dixon[2], and A.J.Crowle[1]. [1]Webb-Waring Lung Institute, University of Colorado Health Sciences Center and [2]University of Colorado at Denver, Colorado USNA. Developmental changes in activity of peroxidase isozymes of D.melanogaster.

Recent studies have demonstrated developmental and age-related changes in total peroxidase activity of D.melanogaster (Armstrong et al. 1978; Poole 1983). Peroxidase commonly exists in multiple isozyme forms throughout the plant and animal kingdoms. Therefore, the purpose of this investigation was to demonstrate and characterize the presence of peroxidase isozymes in D.melanogaster as well as to correlate the total age-related activity changes to the individual isozymes.

The peroxidase substrates DAB (diaminobenzidine) and Hydrogen peroxide produce an insoluble brown product which can easily be trapped in an agarose gel yielding high resolution of peroxidase activity (Graham & Karnovsky 1966). The peroxidase specificity of the stain was demonstrated in two ways: 1. horseradish peroxidase (HRP) and Drosophila extract yielded the brown insoluble product in the gel, whereas neither catalase nor polyphenol oxidase produced any color formation, and 2. both the catalase inhibitor, 3-amino-triazole (Samis et al. 1972; Bewley & Lubinsky 1979), and the polyphenol oxidase inhibitor, phenylthiourea (Dickinson & Sullivan 1975; Smith & Shrift 1979), failed to prevent the color formation by HRP or Drosophila extract.

Electrophoresis of singly fly extracts (1 fly/20 ul) in 1.4% agarose/1.0% dextran gels using a Tris Glycine discontinuous buffer and stained with DAB/H_2O_2 at pH 4.4 (Herzog 1973) yielded 2 to 4 peroxidase isozymes (depending on the developmental stage). Though these zymograms indicated that the transition between isozymes was a continuous process throughout the developmental stages, certain predominant patterns could be correlated to each stage as seen in Figures 1 and 2.

The transition from larva (3rd instar) to early pupa (untanned, newly pupated) was associated with a shift from isozymes 3 and 4 to isozymes 1 and 2. As the pupa tanned (middle pupa), there was a large loss of activity in all isozymes, and only small residual activity from isozyme 1 and/or 3 was detected. In the late pupal stage (darkly pigmented with wing and eye development), there was an increase in total activity, though not as intense as in the larval and early pupal stages. The activity was mostly due to isozymes 3 and 4. Newly emerged flies (0-6 hr) also produced observable activity, though staining more intensely in female flies and again shifting toward isozymes 1 and 2. The above data agreed well with the total peroxidase activity changes found by Armstrong and Poole.

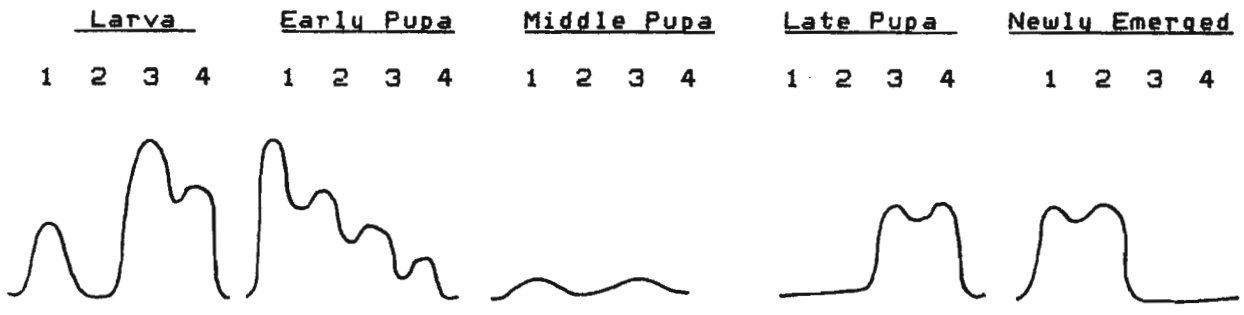


Fig. 1. Densitometric scan of prominent patterns for each stage. Peak height is correlated to amount of isozyme activity.

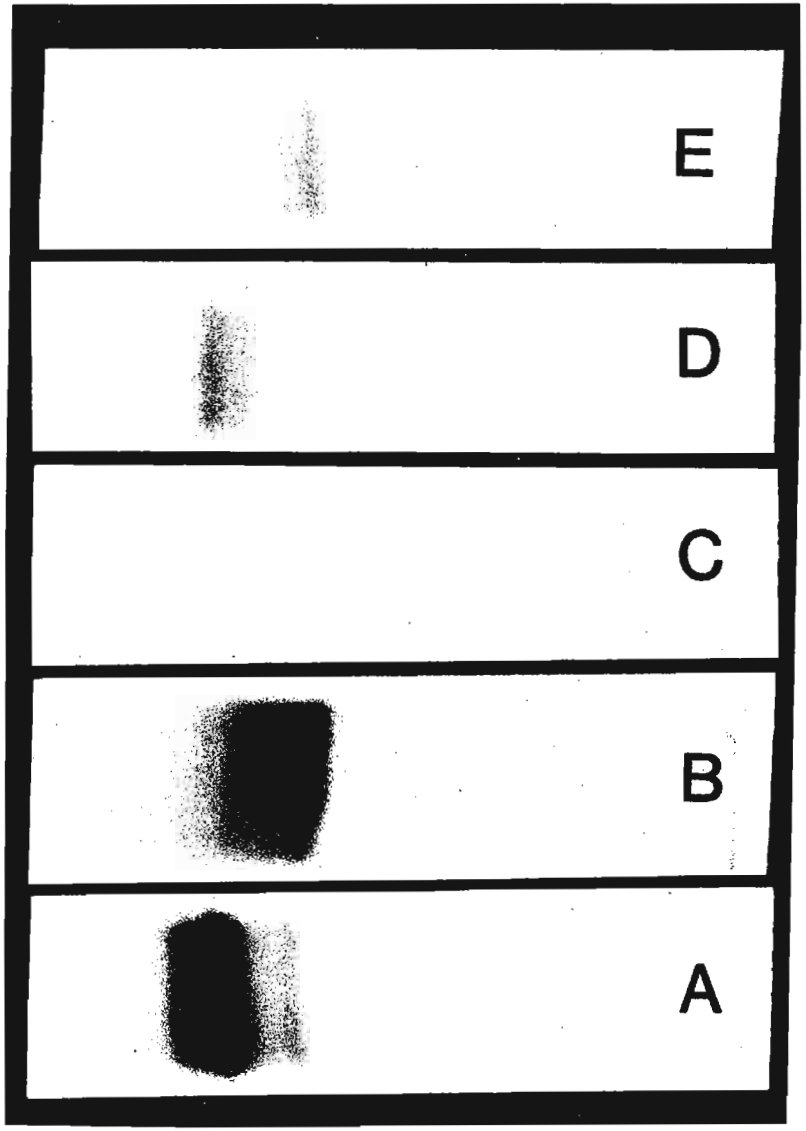


Fig. 2. Zymograms of prominent patterns of each developmental stage in D.melanogaster. A-Larva, B-Early Pupa, C-Middle Pupa, D-Late Pupa, E-Newly Emerged Fly.

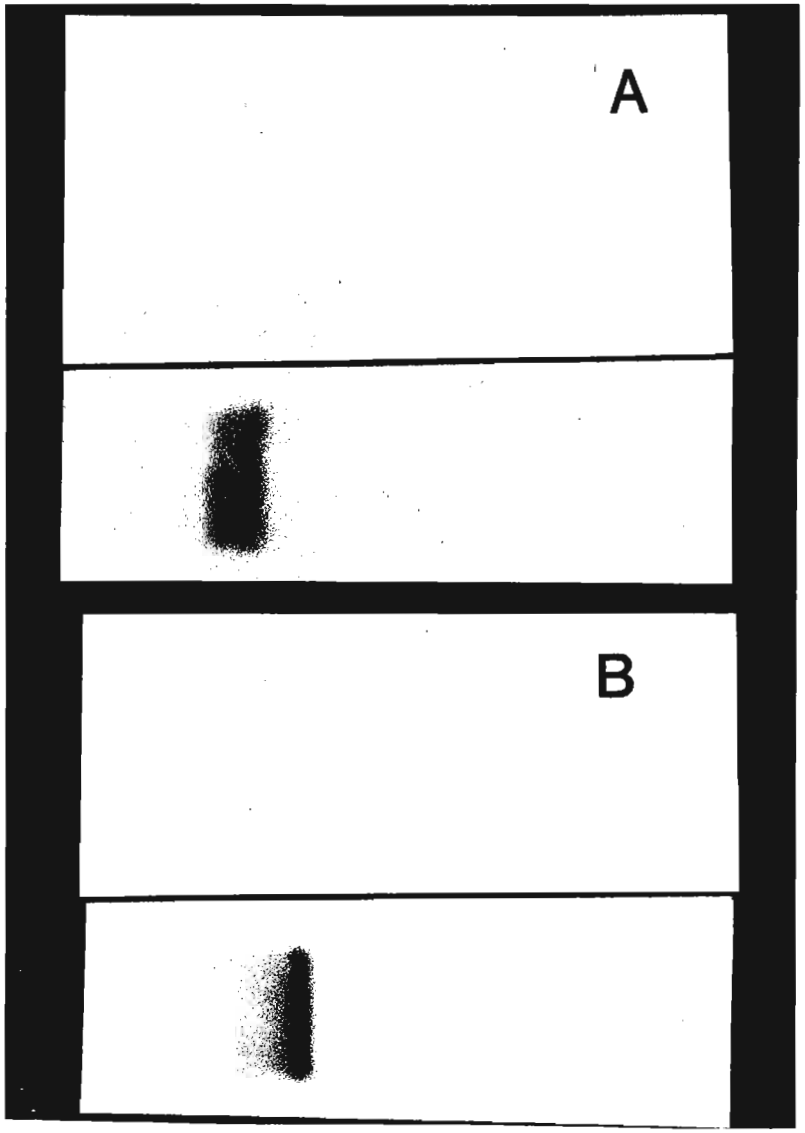


Fig. 3. Comparison between Zymogram and X-IEP patterns. A-Larva, B-Early Pupa.

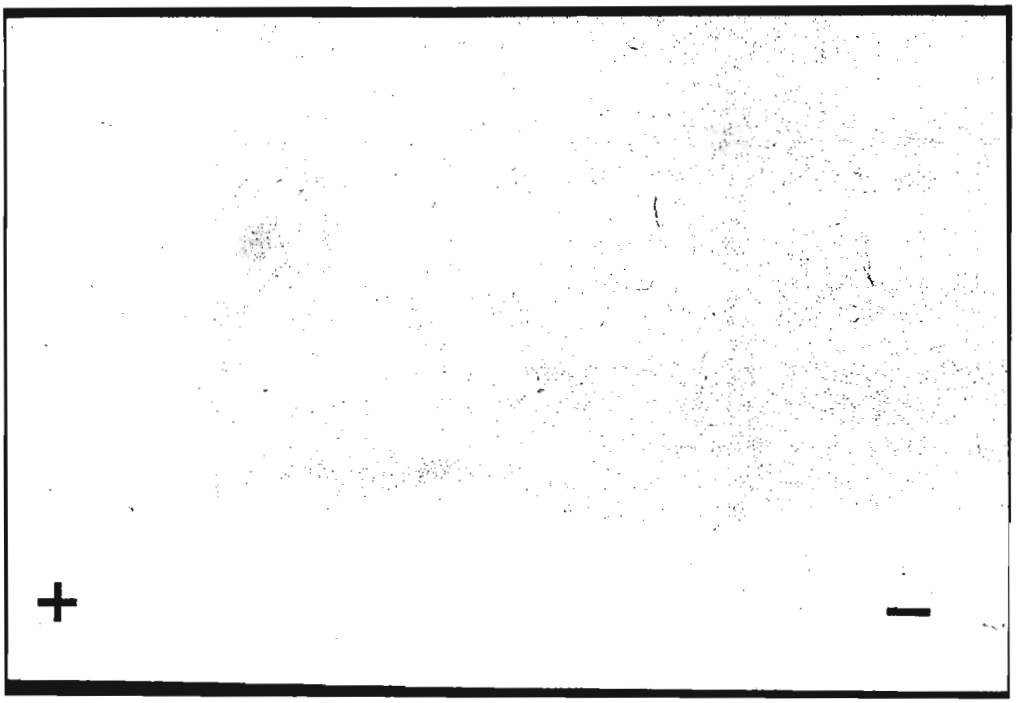


Fig. 4. X-IEP protein pattern of Early Pupa

Yet, though these authors found an activity peak in adult flies at 3 weeks of age, electrophoresis studies yielded insignificant activity in the adult stages. Again, females stained with more frequency than males. The color formation was diffuse and lacked distinct banding patterns. Furthermore, the staining regions tended to shift either anodically or cathodically. This shift did not demonstrate predictability with age, sex, or individual polymorphism. Thus far, it is not certain whether the random adult patterns are due to enzyme processing, gel interaction, substrate preference, protein carriers, membrane-binding, or activity below detectable limits.

Crossed immunoelectrophoresis (X-IEP) confirmed the presence of peroxidase isozymes. In the first dimension, the isozymes were separated electrophoretically, as in the zymograms. The agarose slides were then turned 90 degrees, and the separated proteins were further electrophoresed into a section of gel containing rabbit antibodies to Drosophila proteins from all developmental stages to produce loops of precipitate (Emmett & Crowle 1982). As shown in Figure 3a, both zymogram and X-IEP analyses of a single larva indicated the presence of isozymes 3 and 4, while an early pupa (Figure 3b) demonstrated 3 peaks for isozymes 1, 2 and 3. Since all peaks in the same patterns were the same height and dimensions, the isozyme proteins were approximately of the same concentration. Therefore, the difference in intensities in the zymograms, probably represented differences in enzyme activity. Fusion of the isozyme precipitates indicated that these isozymes were different forms of the same protein, as determined by their immunological identity. An X-IEP for the early pupa in Figure 3b did not detect peroxidase peaks upon staining for protein (Figure 4), but did reveal numerous other proteins against which to compare the peroxidase.

Studies of peroxidase alterations and aging in adult Drosophila must await further developments (i.e., enzyme purification). Nevertheless, zymogram and X-IEP analyses of the 4 detected isozymes offers a unique opportunity to examine the early developmental stages of this organism.

References: Armstrong, D., R.Rinehart, L.Dixon & E.Reigh 1978, Age 1:8-12; Dickinson, W.J. & D.T.Sullivan 1975, Gene-Enzyme Systems in Drosophila: 27-28,98-106; Emmett, M. & A.J. Crowle 1982, J.Immuno.Methods 50:R65-R83; Graham, R.C. & M.J.Karnovsky 1966,J.Histochem,Cytochem. 14:291-302; Herzog, V. & H.D.Fahimi 1973, Anal.Biochem. 55:554-562; Lubinsky, S. & G.C. Bewley 1979, Genetics 91:723-742; Poole, J. & L.Dixon, Masters Thesis: Peroxidase isozymes in D.melanogaster: Enzymatic properties, activity, and proposed functions during development and aging, 1983, DIS 60; Samis, H.V., M.B.Baird & H.R.Massie 1972, J.Insect Physiol. 18:991-1000; Smith,J. & A.Shrift 1979, Comp.Biochem.Physiol. 63B:39-44.

Marengo, N.P. C.W. Post College of Long Island University, Greenvale, New York USNA. Ether-induced failure of the prepupal muscles to shorten the pupal cuticle of D.melanogaster.

Fraenkel & Rudall (1940) have ascribed the shortening of the puparium of Sarcophaga to two principle agents: (1) muscular contractions of the persistent larval muscles and (2) the changing of the orientation of the chitin crystallites from a random arrangement in the unshortened larval cuticle to an arrangement parallel to the transverse axis of the cuticle. They state that this change in orientation explains the degree of contraction in length shown by the cuticle.

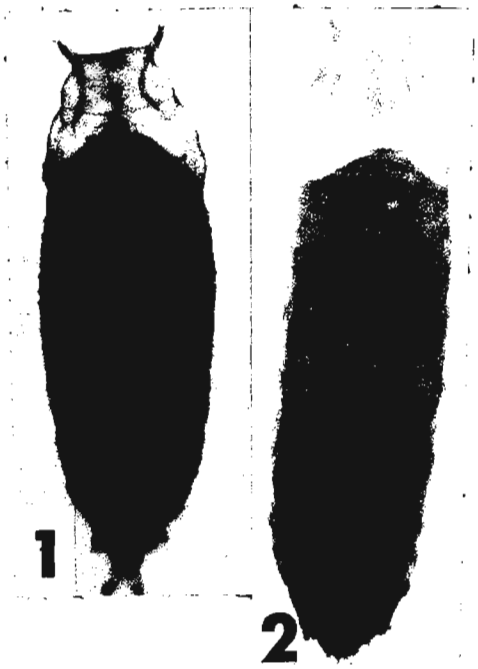


Fig. 1. Control pupa showing normal puparial contour and pupal imago within. X31.

Fig. 2. Pupa which had been etherized during the shortening process. Note normal pupal imago within elongated puparium. X31.

Since Drosophila melanogaster has essentially the same stages in metamorphosis as Sarcophaga (Robertson 1936), this study was devised to establish in D.melanogaster, whether shortening could be prevented by muscular contractions during the shortening process. The results were cleancut and striking, and an account follows.

Wild type Oregon R D.melanogaster were raised on tomato-paste agar at 25°C. When 3rd instar larvae crawled to bottle sides and began movements recognized as just preceding pupation, they were isolated and in petrie dishes and were exposed to ethyl ether for several minutes until the puparium showed signs of hardening. At this time the ether was removed, and the normal process of pupation followed, with the normal-appearing pupa clearly visible within an abnormally elongated puparium (Fig. 2). The control pupa (Fig. 1) pupated normally in the absence of ether. Apparently the anesthetic had no effect on the biochemical changes in the cuticle accompanying the hardening of the puparium, for normal pupation occurred even in the elongated puparium (Fig. 2). Apparently, the removal of the one of the agents described by Fraenkel & Rudall, namely the muscular contractions was enough to prevent normal shortening.

Whether the crystallites of the elongated puparium are arranged parallel to the transverse axis of the puparium can only be determined by X-ray diffraction as used by Fraenkel & Rudall.

References: Fraenkel, G. & K.M.Rudall 1940, Proc. Roy.Soc. B 129:1-34; Robertson, C.W. 1936, J.Morph. 59:351-359.

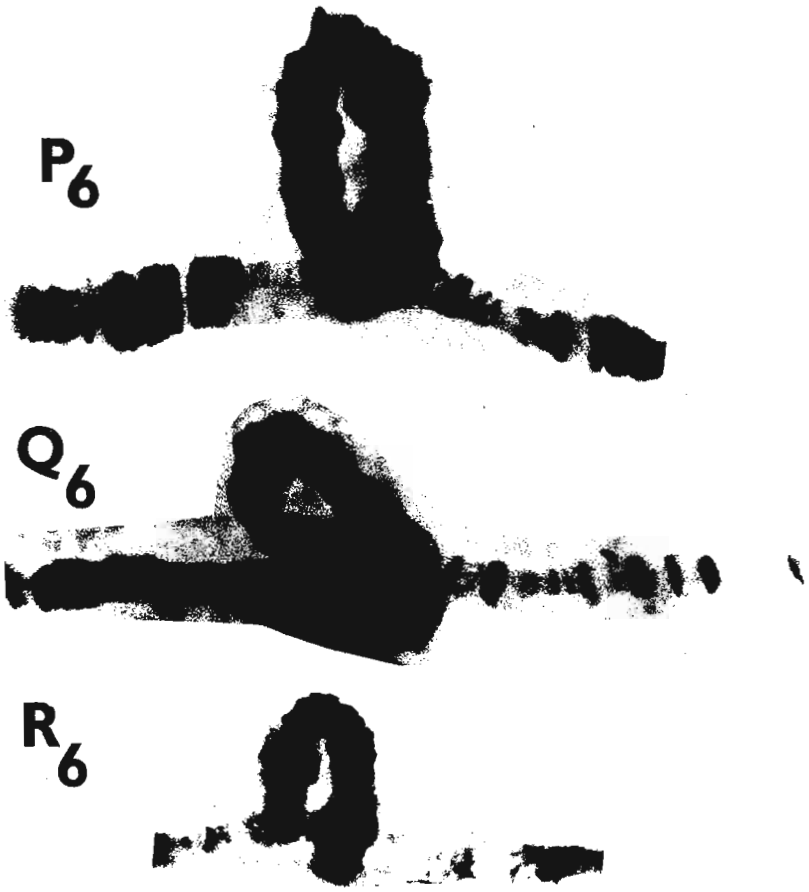
Mather, W.B. and A.K.Pope. University of Queensland, Brisbane, Australia. Inversions from Chiang Mai, Thailand 2nd Report.

In November 1982 thirty-one isolines of D.s. albostrigata and five isolines of D.albomicans were established from Chiang Mai, Thailand.

Inversions in these species were last reported on from Chiang Mai in July 1982 (Mather & Pope DIS 59:82).

(a) D.s.albostrigata. Six simple inversions were detected. All inversions except one had previously been detected in East and Southeast Asia but of these E was new to Chiang Mai. A photograph of the new inversion (P6) is presented and breakpoints assigned (in relation to the standard photographic map)(Thongmeearkom 1977, DIS 52:154). Heterozygosity frequency of all inversions detected is given in Table 1.

(b) D.albomicans. Eight simple and one complex inversion were detected. Seven of the nine inversions had previously been detected in East and Southeast Asia but of these R5, A5 and W5 were new to Chiang Mai. Photographs of the new inversions (Q6 and R6) are presented and breakpoints assigned (in relation to the standard photographic map)(Mather & Thongmeearkom



Photographic Note: The free ends of the chromosomes are to the right.

Table 1.

Inversion	Chromosome	Breakpoints	Het. Freq.%
A5	IIL		45
E	IIL		23
C5	IIR		3
B5	III		10
C1	III		3
P6	III	15.8-21.8	3

Table 2.

Inversion	Chromosome	Simple	Complex	Breakpoints
R5	I	X		
A5	IIL	X		
E'	IIL	X		
S5	IIL	X		
C1	III	X		
W5	III	X		
E6	III		X	
Q6	III	X		42.0-47.2
R6	IIL	X		12.4-18.0

1980, DIS 55:101)(see Table 2).

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, Brisbane, Australia. Inversions from Phuket, Thailand: 4th Report.

In January 1983 twenty isolines of D.s.albostrigata, thirteen isolines of D.albomicans and two isolines of D.kohkoa were established from Phuket, Thailand.

Inversions in these species were last reported on from Phuket in July 1982 (Mather & Pope, DIS 59:83).

(a) D.s.albostrigata. Six simple andone complex inversion were detected. All inversions had previously been detected from East and Southeast Asia but E and K5 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. All inversions had previously been detected from East and Southeast Asia but E', D5, C5 and B6 were new to Phuket. The heterozygosity frequency of all inversions detected is given in Table 2.

(c) D.kohkoa. Three simple inversions were detected (X_4 on chromosome I and Y and U_4 on chromosome III). These had previously been detected from East and Southeast Asia but Y was new to Phuket.

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

Table 1.

Inversion	Chromosome	Simple	Complex	Het. Freq.%
A5	IIL	X		30
D5	IIL		X	55
E	IIL	X		30
C5	IIR	X		75
C1	III	X		60
K5	III	X		5
N5	III	X		65

Table 2.

Inversion	Chromosome	Simple	Complex	Het. Freq.%
E'	IIL	X		54
D5	IIL		X	8
C5	IIR	X		8
C1	III	X		69
T4	III	X		8
E6	III		X	54
L3	III	X		23
B6	III	X		8
N5	III	X		46

Mather, W.B. and A.K.Pope. University of Queensland, Brisbane, Australia. Inversions from the River Kwai, Thailand: 7th Report.

In July 1983, 66 isolines of D.s.albostrigata, 17 isolines of D.albomicans and 1 isoline of D.kohkoa were established from the River Kwai region of Thailand. The inversions from the region were last reported on from a collection made in July 1981 (Mather & Balwin, DIS 58:10).
(a) D.s.albostrigata. Eight simple and one complex inversions 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 inversions were detected. All of the inversions had previously been found at the River Kwai (Table 2).
(c) The one isoline of D.kohkoa established had the complex inversion G6 which had previously been recorded from the region.

Table 1. D.s.albostrigata.

Inversion	Chromosome	Simple	Complex	Het. Freq.%
A5	IIL	X		56
E	IIL	X		5
I2	IIL	X		2
D5	IIL		X	8
C5	IIR	X		33
C1	III	X		6
B5	III	X		2
P5	III	X		3
W2	III	X		3

Table 2. D.albomincans.

Inversion	Chromosome	Simple	Complex
R5	I	X	
S5	IIL	X	
C1	III	X	
E'	IIL	X	
L3	III	X	
E6	III		X

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

McInnis, D.O. and G.C.Bewley. Tropical Fruit and Vegetable Research Laboratory, Honolulu, Hawaii USNA. Laboratory directional selection for dispersal rate and α-GPDH enzyme activity in Drosophila melanogaster.

Quantitative genetic variation for various behavioral traits has been uncovered through directional selection experiments in Drosophila, including phototaxis (Hirsch & Boudreau 1958), and spontaneous locomotion (Grant & Mettler 1969). The effect of major loci, however, has only recently been explored. A major gene-enzyme system involved in flight muscle metabolism in Drosophila is α-glycerophosphate dehydrogenase (α-GPDH, E.C. 1.1.1.8). This enzyme is coupled to a mitochondrial oxidase in the α-glycerolphosphate cycle and provides a mechanism for the rapid and constant energy source required for sustained flight. In this study, (1) directional selection was applied to select for high and low dispersal rates in the laboratory and (2) the relationship between dispersal rate and α-GPDH activity level was investigated.

During 1977, studies were initiated to shed some light upon the important question of how observed variation in realized fly dispersion in the laboratory may be partitioned into genetic and environmental variation. These tests involved wild-raised (1st generation) then laboratory-reared flies (2nd generation onward) of D.melanogaster. Directional selection was applied over successive generations upon tested fly populations with the goal of creating a high line of fast-moving flies and a low line of slow-moving flies. Flies were maintained on standard corn meal molasses medium at 25°C except for the relatively brief periods of the experimental runs. The latter were conducted at 2½-3 week intervals with the adult flies in the same generation. The parents of each generation were removed after a week of mating and egg laying. After eclosion, new generation ♀♀ and ♂♂ (10-14 days old) were allowed to mate prior to the actual experimental run involving selection of fast and slow moving flies. Unfortunately, the control lines for the cage and field work were accidentally lost. However, the divergence between simultaneously run lines was controlled.

Heritability under a system of mass selection (i.e., selected flies mated together en masse) was measured by the regression of the response (R) on selection pressure (S). If all females had mated before selection, and no mating occurred after selection, then $h^2=2$ R/S. If some remating and sperm displacement occurred after selection, then $R/S<h^2<2$ R/S. Estimating this range is appropriate for this work since flies were allowed to mate prior to selection.

Population cage experiments. The following design was established for selecting fast and slow movers in the laboratory. The 'experimental field' was a standard plexiglass population cage (46½ cm long X 14-3/4cm wide X 10½cm high) containing 16 openings for vials (3½ cm diameter X 10cm height) in the floor of the cage. The inside chamber was partitioned into 4 equally sized compartments separated by tightly fitted cardboard. The compartments

Table 1. Directional selection for dispersal rate and α-GPDH activity in a population cage with D.melanogaster (1977).

	Generation number						
	1	2	3	4	5	6	7
Selected line	Compartment position (Avg ± SE; sample size = 200)						
HIGH	(2.52±0.15)	2.78±0.12	2.85±0.11	2.85±0.10	2.85±0.11	2.67±0.14	3.35±0.08
LOW		2.31±0.11	1.98±0.12	2.40±0.14	1.72±0.10	1.62±0.09	1.32±0.07
HIGH-LOW DIVERGENCE	0	0.47	0.87	0.45	1.13	1.05	2.03
CUMULATIVE SELECTION	0	3.00	5.53	7.66	10.21	12.08	14.03
Selected line	α-GPDH activity (units/mg live weight; Avg ± SE)						
HIGH	0.050±.003	0.051±.002	0.050±.002	0.054±.003	0.050±.002	0.054±.002	0.050±.002
LOW	0.058±.003	0.055±.002	0.044±.002	0.048±.003	0.044±.003	0.046±.002	0.040±.002
HIGH-LOW DIVERGENCE	-0.008	-0.004	+0.006	+0.006	+0.006	+0.008	+0.010

were arranged in a horizontal line (i.e., 1-2-3-4) at right angles to light entering the lab from the outside. Flies could travel from one compartment to another by walking through one of 8 circular holes (6mm diameter) punched, equally spaced, around the periphery of the dividers. To provide food and moisture, 4 vials of banana-agar medium were placed in the compartment (#4) most distal to the compartment (#1) wherein the flies were released. Twelve empty vials sealed the remaining holes in compartments 1, 2, and 3.

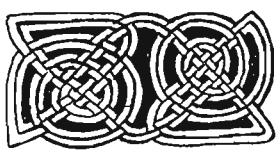
The basic experimental procedure was as follows. One hundred each of locally wild caught ♀♀ and ♂♂ Drosophila melanogaster provided the basis for the first run. These flies were anesthetized briefly with CO_2 gas and while still immobile, placed inside compartment #1 of the experimental cage. The lid was sealed and the flies allowed to revive and move about for approximately 3 to 4 hr after which time the experiment was abruptly stopped. This was accomplished by rapidly CO_2 gassing the flies and aspirating those in each compartment into separate fresh food vials. Flies collected in compartment #4 became the HIGH line parents for the next generation, while flies taken in compartment #1 (the original release site) became the parents for the next LOW line offspring. Such HIGH and LOW lines were maintained in distinct sets of vials for 7 generations and run in separate cages at the time of each selection cycle. Samples were taken for an α-GPDH enzyme activity assay following each experimental run.

α-GPDH enzyme assay. The extraction of α-GPDH for routine assays was performed by the homogenization of adult samples with Dounce glass tissue grinders at a concentration of 20 mg live weight per ml of homogenization buffer which consisted of 0.1M sodium phosphate (pH 7.1) containing 10 nM EDTA and 0.5 nM DTT. Samples were centrifuged for 30 min at 17,000 g at 0°C. The resultant clear supernatant fluid was used as the enzyme source. Each sample consisted of ca. 30 adult flies (evenly divided by sex) and 2 such samples were run and averaged for each tested line. Enzyme activities were monitored in the reverse direction, GP----DHAP, using a Beckman model 25-spectrophotometer with the sample compartment maintained at 20°C. For this reaction, 0.2 ml of properly diluted enzyme was added to a mixture of 0.1M glycine-NaOH (pH 10.0), 4.5 nM NAD^+, and 15 mM GP. Final volumes were 2.5 ml in all assays. A unit of activity is defined as 1 μmole of NAD^+ reduced per minute based upon a molar extinction coefficient for NADH of 6.22×10^3.

Results. The results of directional selection for 6 generations in the population cage are shown in Table 1. The cage data are recorded in terms of the average compartment position attained by HIGH and LOW line flies each generation. The divergence between the lines (HIGH minus LOW) and the cumulative selection pressure are also shown. Movement date in generation 1 are for the original native population prior to selection of HIGH and LOW lines. The 6 generations of selection in the population cage (gens. 1-7) produced an average realized heritability per generation of 0.11 (11%) with a standard error (of the line slope) of 0.027. Divergence and cumulative selection were significantly correlated ($r=0.890$, $p<.01$) and both a sign test for direction (+ or -) of divergence ($p<.05$) and a t-test ($df=6:4.09$, $p<.01$) indicated significant non-zero heritability for dispersal rate averaged over 6 generations. There was no apparent change in the α-GPDH activity of the HIGH line, while the activity of the LOW line declined, largely between gens. 2 and 3. The dispersal rate of the LOW line flies, but not of the HIGH line flies, was significantly correlated with α-GPDH activity ($r=0.850$, $.01<p<.05$).

In conclusion, the evidence indicates that a genetic component exists for small-scale laboratory dispersal ability in D.melanogaster. The available evidence in this study indicates that there is no direct relationship between α-GPDH enzyme activity and fly dispersal rate in the laboratory.

References: Grant, B. & L.E.Mettler 1969, Genetics 62:625; Hirsch, J. & J.C.Boudreau 1958, J.Comp.Physiol.Psychol. 51:647.



McInnis, D.O. and H.E.Schaffer. Tropical Fruit and Vegetable Research Laboratory, Honolulu, Hawaii USNA. Directional selection for field dispersal rate in Drosophila melanogaster.

Directional selection experiments in Drosophila have been used to modify various behavioral traits, at least since Hirsch & Boudreau (1958) obtained responses rapidly for both positive and negative phototaxis. Two other forms of locomotion in D.melanogaster were studied by Connolly (1966) and Grant & Mettler (1969). Connolly selected high and low activity lines of flies which moved atop a closed grid, while Grant & Mettler selected for 'escape' behavior in an I-maze. Both of these studies achieved significant differences between the high and low lines. Ewing (1963) demonstrated with D. melanogaster that attempts to select for one behavioral trait (spontaneous activity) could result instead in changes in other behavioral traits. In this study, we attempted to select for fast and slow movement in a field situation and to estimate the heritability of any resulting difference between fast and slow moving lines.

During 1977, experiments were inititated in search of a genetic component for dispersal ability in Drosophila. These experiments involved wild-raised (1st generation) then laboratory-reared flies (2nd generation onward) of D.melanogaster. Directional selection was applied over successive generations upon tested fly populations with the goal of creating a high line of fast-moving flies and a low line of slow-moving flies. Flies were maintained on standard corn meal molasses medium at 25°C except for the relatively brief periods of the experimental runs. The latter was conducted at 2½-3 week intervals with the adult flies in the same generation. The parents of each generation were removed after a week of mating and egg laying. After eclosion, new generation ♀♀ and ♂♂ (10-14 days old) were allowed to mate prior to the actual experimental run involving selection of fast and slow moving flies. Unfortunately, the control lines for the cage and field work were accidentally lost. However, the divergences between simultaneously run lines were controlled.

Heritability under a system of mass selection (i.e., selected flies mixed together (en masse) was measured by the regression of the response (R) on selection pressure (S)). If all females had mated before selection, and no mating occurred after selection, then $h^2=2$ R/S. If some remating and sperm displacement occured after selection, then R/S<h^2<2 R/S. Estimating this range is appropriate for this work since flies were allowed to mate prior to selection.

The experimental procedure for the open field was as follows. Several hundred locally collected wild ♀♀ and ♂♂ D.melanogaster provided the first release population. Later, however, experiental flies had to be marked so as to distinguish them from native D.melanogaster attracted to fermenting bait. Marking was accomplished by lightly dusting flies with a micronized fluorescent dust (Helecon Pigments, U.S. Radium Corp.). One thousand marked flies per line per generation (except only ca. 500 in gen. 1) were released at a central site during the later afternoon at a time when sufficient sunlight would induce scattering of flies to the shade and coolness of perimeter trees and shrubs. Just prior to the release, 8 traps were set evenly spaced around the periphery of the field, each one 50 meters away from the

Table 1. Directional selection for dispersal rate in an open field with D.melanogaster (1977).

	Generation number						
	1	2	3	4	5	6	7
Selected line	Average distance moved (±SE) (meters)						
HIGH		35.33±2.09 (n=118)	22.49±1.69 (n=181)	39.15±1.07 (n=330)	34.55±1.02 (n=439)	39.43±0.75 (n=342)	33.95±1.03 (n=432)
	(32.77±2.24) (n=112)						
LOW		34.27±2.21 (n=110)	19.26±1.65 (n=164)	37.41±1.29 (n=248)	27.02±1.13 (n=386)	31.58±0.98 (n=390)	27.1±0.98 (n=504)
HIGH-LOW DIVERGENCE	0	1.06	3.23	1.74	7.53	7.85	6.81
CUMULATIVE SELECTION	0	40.00	78.94	115.71	153.97	186.44	218.59

release point. The traps consisted of 2-gallon wax paper buckets containing some fermenting banana to attract Drosophila. One-half hr after the release of flies 4 traps were again evenly spaced on a circle roughly 10 meters from the center. Two additional traps were placed at the center. These 6 latter traps were kept sealed outside the experimental field until needed. For 2 hr after the release, flies were collected from all 14 traps by sweeping with a net over the bait. Flies unable to fly (at the central traps only) were aspirated into a vial and kept separate from the 'flying' flies. These walking flies invariably turned out to be marked and were not included in the LOW line flies selected, since the act of handling these flies may have injured them, impairing flight ability. At the conclusion of the experimental run, all flies were returned to the laboratory where marked and unmarked flies were sorted out under ultraviolet light. Marked flies collected at the periphery became the parents of the next HIGH line generation, while viable marked flies trapped at the 2 center traps produced the following LOW line generation. A total of 7 such releases were conducted in an open grassy field on the campus of N.C.State University.

Results. The results of directional selection for 6 generations in the field are shown in Table 1. The divergence between the lines (HIGH minus LOW) and the cumulative selection pressure are also shown. Movement data in generation 1 are for the original native population prior to selection of HIGH and LOW lines. As tabulated, only between 11 and 50% of the 2,000 HIGH and LOW flies released each generation were recaptured. Six generations of selection in the field resulted again in significant divergence between HIGH and LOW selected lines (sign test, $p<.05$; t-test ($df=6:4.94$, $p<.01$)). Heritability over the 6 generations for the controlled divergence was estimated to average 0.04 (4%) with a standard error of 0.0081. The estimates of heritability for both cage and field work should be doubled if all mating took place prior to selection. Therefore, a heritability range of 4-8% is obtained.

A significant trend was associated with selection progress in the field in that the percentage of released flies recaptured increased from ca. 11% in the first 2 generations to above 40% in both HIGH and LOW lines by generation 7 (sign test, $p<0.001$).

In conclusion, the evidence indicates that a genetic component, albeit small, exists for field dispersal ability in D.melanogaster. The presence of such heritable variation in natural populations provides some flexibility to adapt should there be selection in favor of either fast or slow moving flies.

References: Connolly,K. 1966, Anim.Behav. 14:444; Ewing, A.W. 1963, Anim.Behav. 11:2; Grant, B. & W.E.Mettler 1969, Genetics 62:625; Hirsch, J. & J.C.Boudreau 1958, J.Comp. Physiol.Psycho. 51:647.

Miglani, G.S. and A.Thapar. Punjab Agricultural University, Ludhiana, India.

Modification of recombination frequency by ethyl methanesulphonate and chloroquine phosphate in female D.melanogaster.

Effect of ethyl methanesulphonate (EMS) and chloroquine phosphate (CHQ) was studied on the frequency of recombination in female germ cells of D.melanogaster by dividing the 96 hr larval period (at 25°C) into three equal parts. Using LD_{50} as a criterion, optimum doses of EMS and CHQ were determined. For the 1st, 2nd and 3rd parts of D.melanogaster larvae, the LD_{50} values for EMS, respectively, were 0.90, 0.75 and 0.75%; the corresponding values for CHQ were 0.185, 0.165 and 0.180%. These concentrations of EMS and CHQ were used in the present experiments. Thirty-five to forty females of stock dumpy black cinnabar (dp b cn: 2nd chromosome markers) were mated with wild type (Oregon-K) males for 1-2 days. Inseminated females were starved for 2-3 hr and then allowed to lay eggs for 2 hr. The resultant eggs were transferred on to the food medium with or without EMS or CHQ. The F_1 larvae were thus reared in the 1st, 2nd or 3rd part of larval life on food mixed with respective optimum dose of EMS or CHQ in ratio 9:1. A two-day old F_1 female was mated with 3-4 dp b cn males. The difference in frequency of recombination obtained in the treated and untreated testcross populations was tested using z-test.

Genetic positions of the second chromosome markers in standard genetic map are: dp - 13.0; b - 48.5; cn - 57.5. In the present studies, the percentages of recombination in untreated F_1 females of D.melanogaster in regions dp-b and b-cn were very close to the values of standard genetic map (Table 1).

Decrease in the recombination frequency was observed in both the regions (dp-b and b-cn) studied when treatment with EMS or CHQ was given in the 1st, 2nd or 3rd part of larval life

Table 1. % recombination in regions 1 (dp-b) and 2 (b-cn) in EMS- and CHQ-treated F_1 females of D.melanogaster.

Chemical/ Larval period treated	No. F_1 females treated	Population size	% recombination in region 1	2
Control	20	3723	35.4	9.0
EMS/I	24	3596	29.4[c]	6.4[c]
II	18	2761	26.6[c]	5.1[c]
III	34	1794	32.2[a]	9.0
CHQ/I	24	3206	30.0[c]	5.3[c]
II	22	5370	26.4[c]	4.1[c]
III	23	2302	30.8[c]	6.7[b]

$\bar{P}$ values: [a]0.05; [b]0.01; [c]0.001

(Table 1) (except in one case in region b-cn where the decrease was non-significant when EMS was given in the 3rd part.

Throughout the entire larval life the ovaries contain only oogonia, no oocytes. The oogonia present in the 1st, 2nd and 3rd part of larval life of D.melanogaster responded differently with regard to reduction with EMS and CHQ in the frequency of recombination in regions dp-b and b-cn; the oogonia present in the 2nd part of larval life appear to be most sensitive for reduction in recombination frequency with EMS and CHQ for both the regions studied.

Mittler, S. and S.Wimbiscus. Northern Illinois University, DeKalb, Illinois USNA.
Black pepper (Piper nigrum) is not mutagenic to D.melanogaster.

Black pepper is a widely used spice. Recently there has been some evidence that pepper is carcinogenic (Concon et al. 1979); however, Rockwell & Row (1979) found black pepper to be non-mutagenic with respect to Salmonella/microsome assay. Whole peppercorns showing no evidence of mold or insect infestation were ground into a fine powder. To make sure that the pepper was ingested by the Drosophila, various concentrations of the pepper were mixed with the Drosophila media: 1.9g Carolina Biolgoical Supply Co. instant Drosophila media 4-24; 0.1g Brewer's yeast and 9ml of water on which OR/y^+B^S were reared from egg to adult. Offspring survived to adulthood only on concentration of less than .005g of pepper/10ml of food. The loss of X or Y chromosomes was determined by rearing OR/y^+YB^S on media containing .002 or .003g of pepper/10ml of food. Adult male offspring which emerged were allowed to feed for an additional two days on the media to insure that mature spermatozoa were also exposed to the pepper and then mated to y^2w^{sp} females for two, three day broods. The data presented in Table 1 indicated that the feeding of .002 and .003 pepper/10ml of food did not increase nondisjunction, the loss of the X or Y chromosome, the loss of B^S or y^+ in any of the broods compared to the controls. A 2 x 2 contingency table with Yates' correction factor was used in the analysis.

To determine whether feeding on black pepper during the entire larval life would induce recessive sex-linked lethals, Oregon R males were also permitted to feed for two additional days as adults and then mated to M-5 females for broods 0-3 and 3-6 days.

Table 1. Nondisjunction and loss of X and Y chromosomes, loss of B^S and y^+ in offspring of OR/y^+YB^S fed black pepper.

Pepper Conc/10g	Brood in days	Total Gametes	XXY	loss of X or Y	loss of B^S	loss of y^+
Control	0-3	11100	2(.018%)	9(.081%)	0	0
	3-6	12629	4(.032%)	19(.15%)	2(.016%)	1(.0079%)
.002g	0-3	21745	2(.0092%)	12(.055%)	3(.014%)	1(.0046%)
	3-6	23727	7(.0295%)	35(.1475%)	2(.008%)	0
.003g	0-3	23045	9(.039%)	17(.074%)	2(.0087%)	2(.0087%)
	3-6	25125	2(.008%)	28(.111%)	3(.012%)	2(.008%)

Table 2. Incidence of sex-linked recessive lethals.

	Brood	Number of chromosomes	Recessive lethals
Control	0-3 days	8783	4 (0.04%)
	3-6 days	8478	4 (0.05%)
.003g of Black Pepper per 10ml of food	0-3 days	1508	0
	3-6 days	1321	0

Black pepper when fed to Drosophila melanogaster did not increase loss of chromosomes or increase the incidence of sex-linked recessive lethals.

References: Concon, J.J., T.W. Swerczek & D.S.Newburg 1979, Nutrition and Cancer 1(3):22; Rockwell, P. & I.Raw 1979, Nutrition and Cancer 1(4):10.

Montague, J.R. Barry University, Miami Shores, Florida USNA. Spatial and temporal dispersions of mushrooms and mycophagous Drosophila in a central New York woods.

Recent investigations of mycophagous drosophilid species focused on ecology and life-history traits (Kimura et al. 1978; Charlesworth & Shorrocks 1980), genetic variation and niche breadth (Jaenike & Selander 1979; Lacy 1982), and the evolution and diversity of host-mushroom preferences (Jaenike 1978; Kimura 1980). Jaenike & Selander (1979) suggested that mushrooms are highly unpredictable oviposition substrates, and a number of mycophagous drosophilid species have evolved "generalist" oviposition preferences (Lacy 1982).

This note reports spatial and temporal dispersions of mushrooms, and fluctuations in drosophilid population density during the summer months of 1980. These data should provide some insight into the availablity and abundance of suitable breeding substrates, as well as insight into the population dynamics of mycophagous drosophilds.

The field site was mixed Beech-Maple woods adjacent to a swampy area in Fayetteville, New York (Figure 1). An approximate 100,000 m^2 area was searched during five periods (May 18-20, May 28-30, June 9-15, July 7-8, and August 13-19). Five types of mushrooms were collected and removed from the area: Polyporous squamosus (a large, fleshy polypore); an unidentified polypore-like species; Tricholomopsis platyphylla (a singly-occurring agaric); a group of unidentified, singly-occurring "agaric-like" spp.; and clusters of "Coprinus-

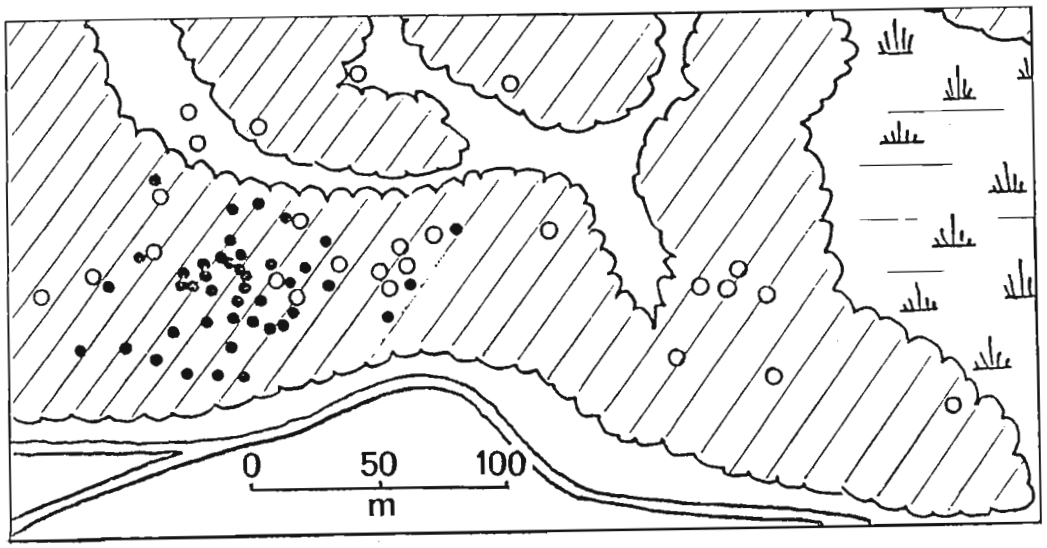


Figure 1. Field site in Fayetteville, New York. Open circles mark locations of natural mushroom substrates during Summer 1980; the solid circles mark the locations of artifical baits used in the mark-recapture census in Summer 1980.

like" (inky caps and shaggy manes) spp. Although the exact taxonomic status of most of the mushrooms could not be determined, each attracted mycophagous drosophilids, and were simply classified by general appearance.

The mean distance between mushrooms was estimated for each collecting period by a method described by Endler (1979): A= area of smallest circle enclosing all mushrooms; N = number of mushrooms, $\sqrt{A/N}$ = mean distance between mushrooms. The July 7-8 collection contained only two mushrooms, so the mean distance was simply the linear distance between them.

The field site was examined on April 20, April 28, and May 8 for drosophilids. The mycophagous drosophilids first appeared on May 15.

The population density of drosophilids was estimated from mark-recapture data from six collection periods (May 20-23, June 19, June 26-28, July 8-11, August 7-8, and August 26). Prior to each period, adults were collected from rotted commercial mushrooms and dusted with micro-fluorescent dusts (Helecon Pigments, U.S.Radium Corp.). The rotted mushrooms were then removed. The marked drosophilids were released at dusk from the center of an area containing 41 baits (Figure 1). Each bait consisted of a small square cloth wrapped around several rotted commercial mushrooms and soaked in rotted mushroom juices. The mean distance between baits was 21 meters (after Endler 1979). Flies were continuously aspirated from the baits every morning (7AM-10AM) until no marked adults were found, or until rain interrupted the collection schedule (June 20, August 27). Temperatures were recorded at 9AM on re-capture days. The September and October populations were not assessed.

Two density estimates described by Begon (1979) were calculated. Jackson's Positive Method (JPM) was used when collections over successive days were available. A simple Peterson Estimate (PE) was

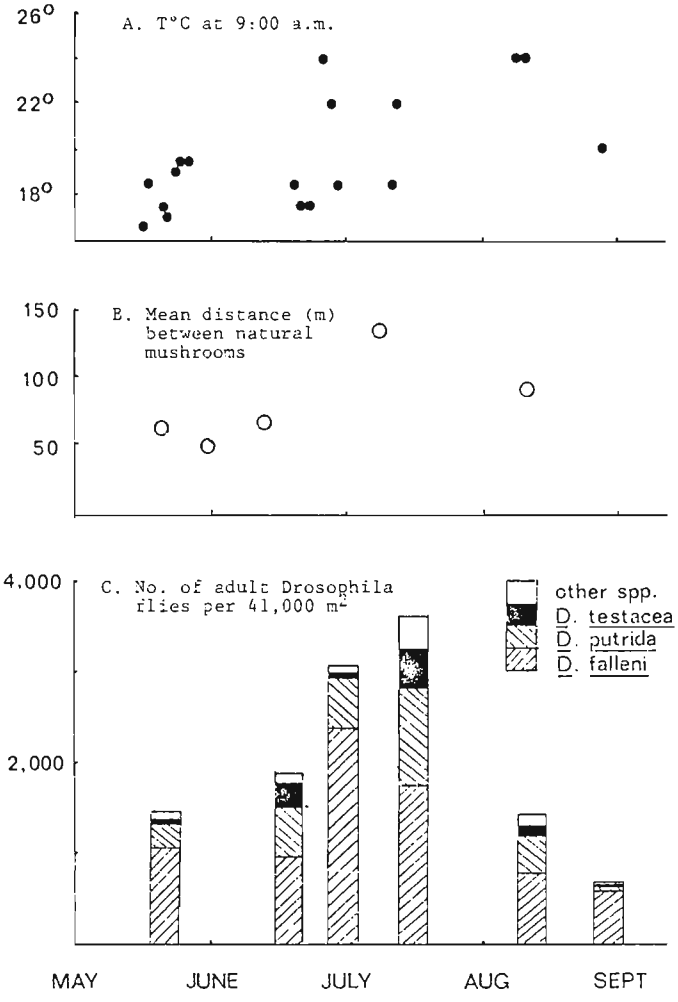


Figure 2. A: seasonal variation in temperature at 9 AM on re-capture days. B: mean distance in meters between natural mushroom substrates. C: estimates of the number of mycophagous drosophilid adults within the area of artificial baits (Figure 1).

Table 1. Temporal dispersions and diversity of natural mushroom substrates (these do not include mushrooms that were too dry to attract mycophagous drosophilids).

Date	"polypore-like" mushrooms	Singly-occurring "agaric-like" mushrooms	Clusters of "Coprinus-like" mushrooms
May 18-20	11	0	0
May 28-30	16	0	2
June 9-15	18	5	3
July 7-8	0	1	1
Aug 13-18	1	3	3

when only a single day's catch was available. Both methods are based upon the proportions of recaptured marks in the total catch (Begon 1979).

The spatial and temporal dispersions of natural mushrooms during 1980 are shown in Table 1 and Figure 1. The mean distances between mushrooms are shown in Figure 2b. During May and June, the "polypore-like" mushrooms increased in density. These large mushrooms became dry and unattractive to drosophilids during the warmer days of July and August (Figure 2a). The predictability of breeding opportunities is difficult to assess since these data were collected within a single season. Statistical comparison between years would be necessary to determine if the observed variation in mushroom densities is significant or merely due to sampling error. Pilot surveys completed during the summer months of 1979 suggested a similar pattern: high polypore density in May and June followed by a decline in total density in July.

The total catch of drosophilids is shown in Table 2, and estimates of the total drosophilid density are shown in Figure 2c. D.fallini (from the quinaria sub-group of the New World hirtodrosophila radiation), and D.putrida and D.testacea (from the testacea sub-group of the New World hirtodrosophila radiation) are among the most common mycophagous drosophilid species in the Eastern U.S. deciduous forests (Collier 1978; Jaenike 1978). Little is known about the full range of oviposition preferences of these species. Kimura (1980) and Lacy (1982) suggested that D.putrida and D.testacea may oviposit in deciduous sap exudates, decaying fruits and vegetation as well as in the fruiting bodies of mushrooms.

The highest drosophilid density was observed during mid July, which coincided with the lowest density of mushrooms (Figure 2b,c). The decline in drosophilid density during August is possibly the result of increased mortality in late July and August. An alternative explanation is that the decline in mushroom density in July resulted in a significant decrease in breeding opportunities. Further investigations of Autumn populations need to be completed.

In summary, observed dispersions of mushrooms and the Drosophila that breed in them suggest seasonal variation in species diversity of the mushrooms, as well as seasonal fluctuations in Drosophila density.

Acknowledgements: I thank my PhD advisor, William T. Starmer (Syracuse University) for his suggestions and patience. John Jaenike and Robert Lacy contributed taxonomic information and useful suggestions.

References: Begon, M. 1979, Investigating animal abundance, University Park Press, Baltimore; Charlesworth,P. & B.Shorrocks 1980, Ecol.Entomol. 5:315-326; Collier, G.E. 1978, DIS 53:168-169; Endler, J.A. 1979, Genetics 93:263-284; Jaenike, J. 1978, Ecology 59:1286-1288;

Table 2. Total catch of Mycophagous Drosophila, Fayetteville, New York, Summer 1980.

Date		D.falleni	D.putrida	D.testacea	Others	Total	#marks recaptured / #released	Population Estimate(a)
5/20-23	Total	1386	408	75	100	1969	57/200 = 0.29	JPM
	♀♀/total	0.34	0.37	0.04	0.04	0.32		
6/19	Total	129	93	41	8	271	236/850 = 0.28	PE
	♀♀/total	0.30	0.52	0.24	0.13	0.36		
6/26-28	Total	407	117	8	3	535	65/420 = 0.15	JPM
	♀♀/total	0.42	0.38	0.25	0.00	0.40		
7/8-11	Total	208	148	41	38	435	13/270 = 0.05	JPM
	♀♀/total	0.28	0.39	0.17	0.24	0.30		
8/7-8	Total	81	50	6	10	147	13/175 = 0.07	JPM
	♀♀/total	0.37	0.38	0.50	0.30	0.37		
8/25	Total	57	6	2	6	71	7/70 = 0.10	PE
	♀♀/total	0.35	0.50	0.50	0.50	0.40		

(a) PE = Petersen Estimate, JPM = Jackson's Positive Method

Jaenike,J. & R.K.Selander 1979, Evolution 33:741-748; Kimura, M.T. 1980, Evolution 34:1009-1018; Kimura,M.T., K.Beppu, N.Ichijo & M.J.Toda 1978, Bionomics of Drosophilidae (Diptera) in Hokkaido. II drosophila testacea, Kontya, Tokyo 46(4):585-595; Lacy, R.C. 1982, Evolution 36:1265-1275.

Moya, A., A.Barbera and J.Dopazo. Universidad de Valencia, Espana. Simulation of the larval competition process.

The present work is an attempt to bring light on the relevance of Wallace's (1981) "biological space unit". The validation procedure was the simulation of the larval competition process, where the medium is divided into K preexisting biological space units. The following assumptions were made: (i) This simulation has no replacement. (ii) Once one larva occupies a biological space unit it will remain in it. This occupation will be at random, taking into account the relative frequency of genotypes before the random number is generation. (iii) Once the larva is inside a unit an intrinsic probability of survival exists, which is obtained from a normal distribution for each genotype. The mean value and standard deviation used for each genotype are obtained from experimental data on Drosophila. (iv) The process ends when the units are exhausted. Then the survivors of each genotype are counted, and the relative viability for each is calculated.

This simulation is a first attempt to find density- and frequency-dependent selection using Wallace's concept. For this reason several values of biological space units (density-dependent selection) and different genetic compositions (frequency-dependent selection) were essayed.

The programming used was PASCAL. The abbreviations used for the parameters were the following:

- N : number of total larvae
- K : number of biological space units
- N_1 : number of larvae of genotype 1
- N_2 : number of larvae of genotype 2
- m_1 : intrinsic viability of larvae of genotype 1
- m_2 : intrinsic viability of larvae of genotype 2
- s_1 : standard deviation of m_1
- s_2 : standard deviation of m_2
- V_1 : viability larva-to-adult of genotype 1
- V_2 : viability larva-to-adult of genotype 2

The results showed that no frequency-dependent selection existed in this kind of simulation (at least when these assumptions). On the contrary, positive density-dependent selection was generated, according to the available biological space unities. Table 1 shows the mean value of viability according to genetic composition for each density. As can be seen, no differences appear between the viabilities of genotypes 1 and 2 due to the similarity of m_1 and m_2. Results not shown here indicate that neither do reductions in the values of s_1 and s_2 statistically permit differences to be on between the viabilities for the different frequencies of the same genotype and density. The same occurs when the mean values of the frequencies are taken and the viabilities of genotypes 1 and 2 are compared. Great differences between m_1 and m_2 will permit differences between V_1 and V_2 to be found and only when other additional parameters are taken into account will it be possible to detect some kind of frequency-dependent selection. More simulations are needed.

Table 1. Results of the simulations: mean viabilities with standard errors. $m_1 = 0.755$, $s_1 = 0.378$, $m_2 = 0.760$, $s_2 = 0.380$

N	n*	K	V_1	V_2
20	19	4	0.19630 ± 0.04638	0.15945 ± 0.02600
		8	0.32514 ± 0.05515	0.25372 ± 0.05427
		12	0.35762 ± 0.02643	0.38887 ± 0.03730
		16	0.50645 ± 0.04924	0.56807 ± 0.04152
		19	0.62756 ± 0.02937	0.63428 ± 0.03918
30	14	6	0.11469 ± 0.01595	0.11158 ± 0.03164
		12	0.26000 ± 0.02799	0.24654 ± 0.02940

Table 1 (contin.)

N	n*	K	V_1	V_2
		18	0.39002 ± 0.03610	0.42584 ± 0.02954
		24	0.47445 ± 0.03206	0.59281 ± 0.03643
		29	0.61254 ± 0.03290	0.59318 ± 0.04483
40	19	8	0.10161 ± 0.01282	0.12610 ± 0.01833
		16	0.26831 ± 0.02801	0.28024 ± 0.02342
		24	0.40296 ± 0.02798	0.33257 ± 0.03474
		32	0.47619 ± 0.03331	0.49050 ± 0.03612
		39	0.59710 ± 0.02394	0.64023 ± 0.02412
50	16	10	0.14573 ± 0.01596	0.11992 ± 0.01388
		20	0.22489 ± 0.02269	0.23789 ± 0.01761
		30	0.38186 ± 0.02159	0.34837 ± 0.03453
		40	0.47197 ± 0.03047	0.51342 ± 0.02742
		49	0.62752 ± 0.02343	0.57838 ± 0.03744
60	19	12	0.13127 ± 0.01544	0.14540 ± 0.01876
		24	0.24562 ± 0.02005	0.25008 ± 0.01861
		36	0.35685 ± 0.03174	0.38115 ± 0.01816
		48	0.50787 ± 0.01634	0.53117 ± 0.03397
		59	0.59681 ± 0.01882	0.59052 ± 0.01538
70	17	14	0.12178 ± 0.01341	0.10846 ± 0.01586
		28	0.25899 ± 0.01385	0.21884 ± 0.02080
		42	0.35761 ± 0.02267	0.40287 ± 0.02263
		56	0.48298 ± 0.03544	0.52257 ± 0.02168
		69	0.66098 ± 0.02802	0.62936 ± 0.02067

* = number of genetic compositions for each pair of N, K.

References: Wallace, B. 1981, Basic Population Genetics, Columbia University Press, New York.

Nahmias, J. and G.C.Bewley. North Carolina State University, Raleigh, North Carolina USNA. Catalase-specific CRM in flies euploid and aneuploid for the cytogenetic region 75D-78A.

Generation of segmental aneuploids spanning the entire genome of Drosophila melanogaster has demonstrated that polytene chromosome region 75D-78A is the only segment in the genome exhibiting a dosage sensitive response to catalase activity with a hyperploid to euploid ratio of 1.54 (1). This result has suggested that this region is the site for the catalase structural gene, Cat^+. Analysis of catalase turnover rates using the irreversible inhibitor 3-amino-1,2,4-triazole has attributed this dosage effect to a 1.4 fold increase in the rate of enzyme synthesis while the rate of enzyme degradation remains constant (1). In the present study, we report that this dosage effect is also reflected by an analogous increase in the number of enzyme molecules as evidenced by quantitating levels of catalase-specific cross reacting material (CRM) using antiserum from rabbits injected with purified catalase antigen (2).

Segmental aneuploids were generated by crosses between stocks L131 and R153 which carry (Y;3) translocations with autosomal breakpoints at 75D and 78A respectively (1). Male progeny euploid and hyperploid for region75D-78A were homogenized at a concentration of one fly per 10 µl of 10 mM sodium phosphate buffer (pH 7.0) containing 0.1% Triton X-100 and 5 µl were applied to each well of a 1% agarose gel containing monospecific antibodies against catalase (2). Gels were stained for catalase activity and the area underneath each rocket was estimated. The ratio of 3-dose to 2-dose flies obtained was 1.52 (Fig. 1). This result demonstrates that the 50% increase in activity observed in 3-dose vs. 2-dose flies is not attributed to structural modifications of the enzyme molecules but rather to differential rates of enzyme accumulation to the steady state.

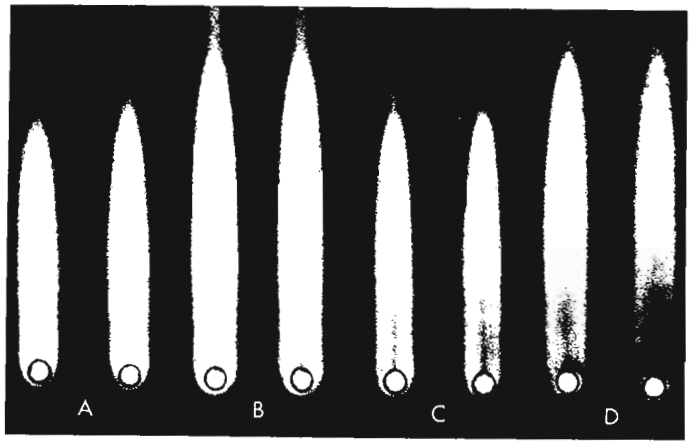


Figure 1. Rocket immunoelectrophoresis of male whole fly extracts bearing 2-doses (A and C) and 3-doses (B and D) of the cytogenetic region 75D-78A. Mean rocket areas ± 1 standard deviation are 258.8 ± 13.9 mm^2 for euploid and 392.9 ± 17.2 mm^2 for hyperploid flies giving a ratio of 1.52.

The data on enzyme levels, CRM levels, and enzyme turnover rate constants all corroborate the notion that the cytogenetic region 75D-78A contains the structural gene for catalase.

Nomenclature Note: There has been some confusion in the literature concerning the gene symbol for catalase and choline acetyltransferase. We have conferred with Jeff Hall on this matter and have mutually agreed that the gene symbol for catalase will remain Cat^+ and that for choline acetyltransferase will be changed to Cha.

References: (1) Lubinsky & Bewley 1979, Genetics 91:723; (2) Nahmias & Bewley 198 , Comp.Biochem. and Physiol. 77B:355.

Najera, C. Universidad de Valencia, Espana.
The maintenance of variability in artificial populations. I. Heterozygotes frequency.

The problem of variability and its maintenance is basic in population genetics.

Considering variability from a selective point of view, one of the several explanatory mechanisms for its maintenance is heterosis (Dobzhansky 1952,1970).

In a previous work the behavior of four eye colour mutants from a cellar was tested against their wild allele from the same cellar, in artificial populations, comparing two culture mediums, one supplemented with alcohol at 10% and the other without alcohol (Najera & Mensua 1983).

Table 1. X^2: Level of significance.

			Without Alcohol		With Alcohol	
2/58A (sepia)	first replica	1° count	1.252	ns	7.429	0.01
		2° count	4.301	0.05	4.657	0.05
		3° count	1.672	ns	8.813	0.005
	second replica	1° count	1.584	ns	18.780	0.001
		2° count	0.111	ns	1.304	ns
		3° count	0.000009	ns	0.092	ns
1/51.3 (Safranin)	first replica	1° count	10.395	0.001	8.562	0.005
		2° count	7.736	0.005	4.149	0.050
		3° count	6.215	0.010	25.169	0.001
	second replica	1° count	10.674	0.001	4.518	0.050
		2° count	3.618	0.050	6.603	0.010
		3° count	5.492	0.025	9.087	0.001
2/54A (cardinal)	first replica	1° count	20.823	0.001	53.760	0.001
		2° count	28.192	0.001	55.370	0.001
		3° count	47.453	0.001	80.606	0.001

Table 1 (contin.)

			Without Alcohol		With Alcohol	
	second replica	1° count	28.021	0.001	28.211	0.001
		2° count	41.056	0.001	34.788	0.001
		3° count	57.591	0.001	74.871	0.001
2/74B (cd+cn+?)	first replica	1° count	2.516	ns	21.973	0.001
		2° count	8.592	0.005	16.213	0.001
		3° count	0.886	ns	21.744	0.001
	second replica	1° count	11.704	0.001	33.394	0.001
		2° count	16.585	0.001	8.575	0.005
		3° count	6.437	0.010	6.128	0.010

Table 2. Gene frequencies.

		Without Alcohol	With Alcohol	t	
2/58A se	1° count	0.453±0.014 0.372±0.012	0.432±0.015 0.410±0.014	0.20	ns
	2° count	0.335±0.014 0.344±0.015	0.380±0.016 0.337±0.016	0.87	ns
	3° count	0.344±0.014 0.321±0.014	0.298±0.011 0.275±0.012	2.86	ns
1/51.3 sf	1° count	0.229±0.013 0.233±0.011	0.285±0.014 0.308±0.017	5.74	0.05
	2° count	0.226±0.015 0.241±0.015	0.280±0.014 0.280±0.015	6.35	0.05
	3° count	0.239±0.014 0.222±0.013	0.259±0.011 0.291±0.011	5.76	0.05
2/54A cd	1° count	0.271±0.013 0.219±0.014	0.299±0.015 0.237±0.015	0.57	ns
	2° count	0.216±0.014 0.225±0.016	0.203±0.014 0.244±0.018	0.14	ns
	3° count	0.221±0.015 0.217±0.013	0.254±0.012 0.236±0.012	2.83	ns
2/74B	1° count	0.153±0.014 0.158±0.011	0.179±0.013 0.187±0.014	6.11	0.05
	2° count	0.120±0.013 0.134±0.014	0.128±0.014 0.161±0.016	1.01	ns
	3° count	0.090±0.012 0.106±0.013	0.131±0.012 0.098±0.011	0.92	ns

The four mutants (2/58A-sepia; 1/51.3-safranin; 2/54A-cardinal and 2/74B-strain segregating cardinal and cinnabar mutants), attained different gene frequencies at equilibrium: 0.32, 0.27, 0.15 and 0.08 approximately.

In order to test whether this equilibrium frequency is due to an excess of heterozygotes, the heterozygote frequency of the populations was studied at 18, 36 and 84 weeks from starting, coinciding with three of the counts.

One hundred wild phenotype male were taken from each of the sixteen populations and were crossed with mutant virgin females, and the heterozygote frequency was verified by means of the Cotterman (1954) formula. By means of this formula, the gene frequency of the mutant and its variance was calculated.

Periodically, in every case except in the 2/58A mutant(se), there was an excess of heterozygotes over the number expected.

Table 1 shows the level of significance of the excess of heterozygotes observed compared to the number expected.

It can be seen that in the 2/58A mutant (sepia) there is not a significant excess of the heterozygotes in the non alcohol medium whereas in the alcohol medium there is.

In the 1/51.3 mutant (safranin), there is always a significant excess of heterozygotes, the same as in the 2/54A mutant (cardinal), although in this latter the significance is higher and more homogenous.

In the 2/74B mutant in non alcohol food there are some cases of non significance but in alcohol food there is always a high degree of signficance.

On comparing the gene frequencies obtained by this method (Table 2) in the two media, it was observed that in the 2/58A mutant (se) there is no significant difference between the two media, and the same was found in 2/54A (cd) populations. In the 2/74B mutant there is only a case of significance at the first count. In the 1/51.3 mutant (sf) the frequency is always higher in the alcohol medium, at a 0.05 level of significance, which confirms the findings for the artificial populations (Najera & Mensua 1983).

It can be concluded that in these populations there is a higher frequency of heterozygotes than could be expected. For this reason one can consider a gene heterosis effect in the maintenance of these mutations which affect the eye colour.

References: Cotterman, C.W. 1954, Statistics and Mathematics in Biology, Iowa State College Press, Ames, Iowa; Dobzhansky, Th. 1952, Heterosis, J.Gowen (ed), Iowa State College Press, Ames, Iowa; Dobzhansky, Th. 1970, Genetics of the evolutionary process, Columbia University Press, New York; Najera, C. & J.L.Mensua 1983, DIS 59:94-95.

Najera, C. and R.deFrutos. Universidad de Valencia, Espana. The maintenance of variability in artificial populations. II. Frequency of inversions.

The existing knowledge of chromosome polymorphism due to the presence of inversions (Chigusa, Mettler & Mukai 1969), together with seemingly permanent linkage disequilibrium between these inversions and some isozyme genes (Mukai, Mettler & Chigusa 1971) gives occasion for numerous investigations of chromosome variants in D.melanogaster populations.

The environmental conditions which determine differences in species distribution, could also determine changes in the frequency of inversions. For example, studies of the distribution of the ecological niches of D.melanogaster and D.simulans show that the first species, more tolerant to ethanol, is distributed both inside and outside cellars but the second is found only outside cellars (McKenzie & Parson 1972,1974); in the same way D.melanogaster is polymorphic for chromosome arrangements and D.simulans is monomorphic.

A study of the inversions frequencies was made in the artificial populations described in the previous work as well as in the five strains which gave rise to these populations, to verify if the strong heterosis present could be explained by the maintenance of inversions in heterozygosis.

The inversions were analyzed through crosses with the "rucuca" strain, homozygous for standard-sequence chromosomes.

One male was crossed with two rucuca virgin females. From the offspring of the cross seven third instar larvae were collected and the giant salivary glands extracted.

A chromosomic line was considered non-carrier of inversions if in none of seven preparations observed, inversion handles appeared.

Ten crosses per population and per strain were made.

The probability of observing the two male chromosomes was $1-(1/2)^7 = 0.99$.

The method used was the conventional: stain in orcein-lactic-acetic (80-20) and squash.

The cytological nomenclature followed that of Lindsley & Grell (1968) and the breakpoints of the inversions were identified by reference to the standard map of Bridges (1935).

The inversions found and their frequency are shown in Table 1.

The mutations were not found within any of the inversions found in the strains.

Table 1. Types and frequencies of inversions in strains and populations.

STRAINS	NUMBER OF CHROMOSOMES ANALYZED	INVERSIONS	FREQUENCY % 2°	3°
2/63(wild)	20	In(2R)NS	5	
2/58A(sepia)	20	-		
1/51.3(safranin)	20	In(3R)87C-93D*		30
2/54A(cardinal)	20	-		
2/74B(cd+cn+?)	20	In(2L)t	100	

POPULATIONS	NUMBER OF CHROMOSOMES ANALYZED	TYPES AND FREQUENCIES OF INVERSIONS WITH ALCOHOL	W/O ALCHOHOL
2/63/2/58A	40	In(3R)87C-93D*--5%	
2/63/1/51.3	40		In(2R)NS--5%
2/63/2/54A	40	In(3R)P--5%	In(3R)P--5%
2/63/2/74B	40	In(2R)NS--10%	In(2R)NS--15%

* = new chromosomal inversion.

As far as the inversions found in the populations are concerned not one included the mutations studied although some are on the same chromosome. Only the cardinal mutation was just at the beginning of the inversion (3R)P, which could cause a linkage disequilibrium. Nevertheless the small inversion frequencies in these populations make this impossible in practice.

It can therefore be concluded, in general, that the strong heterosis present in these artificial populations cannot be explained by the maintenance of inversions in heterozygosis.

References: Bridges, C.B. 1935, J.Heredity 26:60-64; Chigusa,S.I., L.E.Mettler & T. Mukai 1969, Genetics 61:s10; Lindsley,D.L. & E.H.Grell 1968, Carn.Inst.Wash.Publ. 627; McKenzie,J.A. & P.A.Parsons 1972, Oecologia 10:373-388; McKenzie,J.A. & P.A.Parsons 1974, Genetics 77:385-394; Mukai,T., L.E.Mettler & S.I.Chigusa 1971, P.N.A.S. 68:1065-1069.

Novitski, E. University of Oregon, Eugene USNA. Search for a tetraploid male.

The obvious usefulness of a tetraploid line in melanogaster has led a number of us to try to put together a tetraploid male. The existence of the entire compounds for both autosomes has made this project more hopeful. In addition, we have a compound X stock with a completely functional Y chromosome at the centromere region, this point being indisputable since the compound is a tandem metacentric which generates simple rings fertile in the male without a free Y chromosome.

A triploid line was constructed with the compound X and Basc, C(1)TM, XYS.YLX,y/Basc. Such females were mated to males with two second chromosomes attached together, C(2)EN, as well as two thirds joined together, C(3)EN. It would be anticipated that some of the gametes from the female would be diploid and would carry also the Y in the compound X, and that some of the gametes of the male would have two sets of the large autosomes, and a Y chromosome. The resulting zygote from the combination of the two would be 2X2Y;4A. The small fourth chromosomes were uncontrolled, except that the triploid stock was fresh and probably carried three fourth chromosomes, in some individuals at least.

366 triploid females of the above constitution were mated to an excess of C2;C3 males. The diploid progeny included females: 148 B/+, 38 y; males: 76 w^a B, 51 + and 6 y (the latter coming from crossing over within the TM). There were 27 B/+ and 6 y intersexes, 31 B/+/+ triploids and 40 non-B triploids. A few unusual products of crossing over or non-disjunction appeared: 2 w^a B females and 2 B males.

Of particular interest of course were the possible tetraploids. These included 4 y males and 3 B males with the large wing cell size characteristic of polyploids. There also appeared one female with a highly suppressed B phenotype and unusually large wing cells which might have been 4A in autosomal composition. All these individuals proved to be sterile, and it appears likely that the males were in fact male-like interesexes (a not too common occurrence) and that the female was 3X;4A.

From these results it would appear that if such higher level polyploids are viable and fertile in melanogaster, they are not easily produced by way of these entire compounds, although it can be surmised from the types of progeny described above that the 3N females and the C(2);C(3) males both produce the required diploid gametes.

Oguma, Y., S.Akai and H.Kurokawa. University of Tsukuba, Sakura-mura, Japan. Mating behavior in D.auraria complex.

We attempted to clarify what consists of components in mating behavior and how it genetically relates to species discrimination among the four siblings of D.auraria complex. A serial studies by means of observation for successive mating behavior were conducted by using a videocorder with a small observation chamber (ϕ15mm) under a light condition (1500 lux). The principal results obtained are as follows:

1. It was preliminarily found that there were a little but critical differences in sexual maturation between the four species. D.triauraria matured somewhat faster, conversely, D.biauraria did slower than the others. The male flies of the four species, in any case, all matured sexually by 4 days after eclosion at room temperature of 25°C. We accordingly conducted thereafter the video-observation by using flies of both sexes of 4-days through 6-days old.

2. None of fly of the all four species has mated under which set 30 minutes observation period in a dark condition. The flies tested, however, came to mate when they were shifted from a dark to a light condition with only 3 lux illumination. This implies a small amount of illumination is substantially permissible for beginning of mating in these flies.

3. Another experiment using a larger observation chamber (50x50x4mm) in which 10♀♀ and 15♂♂ were placed together revealed that the male flies behaved to show orientation to the females by means of their "sight". Furthermore, we could examine a critical distance which they could notice females was only 20mm. This fact indicates the more interesting subject of "sight" is characteristically used for partner recognition in this group.

4. After successive observations we could recognize 13 different but consecutive components belonging to mating behavior in this complex. All or almost all of male flies of D.auraria and D.quadraria similarly did not represent "wing vibration" while those of D.biauraria and D.triauraria did it. At the stage of attempted copulation, males of all four species consistently showed "wing display" and simultaneously the females spread both wings, following copulation. Just before copulation, males of D.triauraria postured at right rear of females, those of D.auraria and D.biauraria postured at a diagonal rear of females, and those of D.quadraria behaved both ways mentioned above. A tapping of females by males was intensely observed in D.triauraria.

Pascual, L. and R.deFrutos. Universidad de Valencia, Espana. Heat shock puffs in Drosophila subobscura polytene chromosomes.

It is well known that heat shock causes a response in larvae or early prepupae gene activity of Drosophila. Thus, a characteristic puffing pattern was described in the salivary gland chromosomes from several Drosophila species (Ritossa 1962; Berendes & Holt 1964; Ashburner 1970; etc.).

D.subobscura larvae, cultured at 19°C and synchronized for "prepupa 0h." stage (moment of eversion of the anterior spiracles), showed 93 active loci after heat shock (37°C during 10, 20, 30, 45 or 60 min).

Four different groups of chromosome regions reacting to the heat shock could be distinguished:

GROUP I: Puffs "induced" by heat shock and not normally observed at 19°C in this strain: 14AB, 27A, 31C/D, 54C/D, 60C/D, 89A and 94A. The loci 14AB, 54C/D and 60C/D are small and variable in their response (see Figure 1).

GROUP II: Puff which became highly active after the heat treatment when they were not seen to be active in normal development at this stage: 15DE and 18C (see Figure 1).

Figure 1. Principal heat shock puffs in Drosophila subobscura.

GROUP III: Puffs active during normal development at this stage which tended to maintain or to increase their activity. A total of 38 loci belong to this group. The loci 5D, 16B, 40D-41A, 63BC, 65B, 74A, 85AB, 86A and 98C could be distinguished as significantly increasing their activity after the shock.

GROUP IV: Puffs active during normal development at this stage which regressed markedly in their activity after the heat shock. A total of 20 loci belong to this group.

In addition to the puffs in these four groups several puffs were found which showed little or no activity either in the control individuals or in the shocked ones.

Finally, it must be mentioned that the length of time under heat shock does not seem to have a decisive effect on puff formation at the times investigated. The greatest development of puffs tends to appear between 20 and 45 min.

References: Ashburner, M. 1970, Chromosoma 31:356-376; Berendes, H.D. & Th.K.H.Holt 1964, Genen en Phaenen 9:1-7; Ritossa, F.M. 1962, Experientia 18:571-573.

Pechan, P.A. and M.L.Tracey. Florida International University, Miami, USNA.

Passive anti H-Y immunization of Drosophila melanogaster females reduces progeny sex ratio.

Tissue grafts among members of highly inbred populations are accepted, in general, as readily as autografts. Eichwald & Silmser (1955) detected a weak rejection reaction of male skin grafts by female C57BL/6 mice; all other skin grafts were readily accepted. They hypothesized that the observed male to female rejection was governed by a male specific transplantation antigen, H-Y antigen. Serological identification of H-Y antigen was first demonstrated by Goldberg et al. (1971) who used serum from male grafted female mice to kill sperm in the presence of complement. Subsequent in vitro studies provide further support for the hypotheses of H-Y antigen male specificity and an early developmental role in sex determination: (1) in anti H-Y antibody cytotoxicity assays, male eight cell stage embryos are lysed; female embryos are not lysed (Krco & Goldberg 1976; Epstein et al. 1980; Ohno 1979). (2) When cultured testicular cells are lyso-stripped of H-Y antigen, they organize ovarian follicle-like aggregates (Ohno et al. 1978). Similarly, the addition of H-Y antigen to cultured ovarian cells induces the formation of testicular-like tubules (Zenzes et al. 1978). (3) XX bovine gonad primordia undergo testicular conversion in whole organ cultures which contain H-Y antigen (Ohno et al. 1979). Moreover, surveys of both vertebrates and invertebrates report the detection of H-Y antigen in heterogametic individuals (Wachtel 1983).

These reports suggest that similar effects should be detectable in vivo. For example, female mice producing H-Y antibodies should produce fewer male progeny than mothers who are not producing H-Y antibodies. Comparison of sex ratios between litters from H-Y antibody producing C57BL/6 mothers (40% male progeny) and non H-Y producing mothers (52% male progeny) supports this hypothesis ($t=2.35$; $P<0.05$; Pechan unpubl.). Given the ubiquity of H-Y antigen, we decided to attempt a similar in vivo test using passively immunized D.melanogaster females.

Three to four day old virgin females were injected with 0.5 µl of mouse monoclonal H-Y antibody, mouse monoclonal dinitrophenol antibody, 1/64 dilution of mouse monoclonal H-Y antibody (the antibody is not detectable at this dilution), polyclonal H-X serum from females previously injected with female spleen cells, and female mouse serum. Uninjected females were also used. Two males were added immediately after injection and the first 24 h egg

Table 1. Progeny Sex Ratios.

Immunization	Number	Sex Ratio	Number	Sex Ratio
1. H-Y antibody	797	0.44 <.001	1524	0.48 ns
2. H-X	491	0.52 ns	1309	0.46 <.01
3. DNP antibody	226	0.52 ns	309	0.51 ns
4. 1/64 H-Y dilution	381	0.50 ns	334	0.49 ns
5. female serum	301	0.51 ns	252	0.47 ns
6. uninjected	2344	0.48 ns	2707	0.50 ns
pooled 2-6	3743	0.49 ns	4911	0.49 ns

collection or brood was collected 24 h later, that is 24-48 h post-injection. A second brood was collected 48-72 h post-injection. The progeny sex ratios (#males/total) were computed by counting all males and females in broods one and two between days eight and sixteen after egg laying. Differences in the sex ratio between broods as well as among treatments were tested for significance, because we did not expect the passive immunization effect to last more than 24 h due to protein degradation.

Among treatments within the first brood, only the H-Y immunized mothers produced fewer sons than expected. All other sex ratios were in agreement with a 0.50 expectation (Table 1). In the second brood the progeny sex ratio of mothers immunized with serum from mice injected with female spleen cells (H-X) differed significantly from 0.50. All others were in agreement with the 0.50 expectation. Sex ratios from females of increasing age have been shown to increase with maternal age (Lauge 1980); however, the differences are not significant over a 48 h period, and we did not observe an effect in our controls. The decreased production of sons by H-Y immunized females is consistent with the mouse data and supports the hypothesis of an early male differentiation role for H-Y antigen. The low second brood sex ratio among progeny of H-X immunized mothers is problematical.

References: Eichwald, E.J. & C.R.Silmser 1955, Transp.Bull. 2:148-149; Epstein,C.J., S. Smith & B.Travis 1980, Tissue Antigens 15:63-67; Goldberg,E.H., E.A.Boyse, D.Bennett, M. Scheid & E.A.Carswell 1971, Nat. 232:478-480; Krco,C.J. & E.H.Goldberg 1976, Sci. 193:1134-1135; Lauge, G. 1980, in M.Ashburner & T.R.F.Wright, Genetics and Biology of Drosophila 2d: 33-106; Ohno, S. 1979, Major Sex-Determining Genes; Ohno,S., Y.Nagai & S.Ciccarese 1978, Cytogenet.Cell Genet. 20:351; Ohno, S., Y.Nagai, S.Ciccarese 1978, Cytogenet.Cell Genet. 20:351; Ohno,S., Y.Nagai, S.Ciccerese & H.Iwata 1979, Rec.Progr.Horm.Res. 35:449-476; Wachtel,S. 1983, H-Y Antigen and the Biology of Sex Determination; Zenzes,M.T., U.Wolf & M.Engel 1978, Hum.Genet. 44:333-338.

Pelliccia, J.G. and D.G.Couper. Bates College, Lewiston, Maine USNA. Intragenic complementation at the Adh locus.

Intragenic complementation is a process where, in a multiple subunit protein, two or more non-functional subunits produced by null activity alleles, interact to produce an active enzyme. We are interested in determining some of the properties of enzymes produced by this process as compared to their wild type counterparts.

A large number of null activity mutations of the alcohol dehydrogenase (Adh) gene have been isolated and the properties of their respective protein products have been studied (Sofer & Hatkoff 1972; O'Donnell et al. 1975). Heterozygotes for certain pairs of CRM positive Adh null activity mutations show levels of enzyme activity ranging from 1% of normal up to almost 23% (W.Sofer, unpubl. data). All such animals have either the Adhn11 or n18 mutation as one member of their complementing pair of alleles. ADH enzyme is active only as a dimer so we assume that the heterodimer is the active form in these hybrid animals.

One such combination of complementing alleles results from crossing an Adhn6 cn vg male with a b Adhn11 cn vg female with the resulting F1 having approximately 13% of the enzyme activity found in the b AdhF cn vg strain from which these mutant strains were derived (F indicates the 'FAST' electrophoretic variant). The results were similar when the reciprocal cross was done.

Parallelling the decreased enzymatic activity of the hybrid adults was their decreased survival on ethanol supplemented media. Whereas AdhF flies have an LD50 at 6½% ethanol under our conditions of testing, (25 four day old males placed in a plastic shell vial with Carolina instant media reconstituted with an ethanol solution of known concentration and covered with parafilm for 24 hr), the hybrid flies had an LD50 of 1%. Homozygous n6 or n11 males showed 100% mortality when fed media supplemented with 1% ethanol. Thus, the enzyme activity levels predict the in vivo susceptibility to environmental alcohol.

Adults of the b AdhF cn vg strain show a pattern of accumulating enzyme activity as they age. Enzyme specific activity (units of enzyme per mg soluble protein) rises to a maximum between days 4 and 5 and remains constant thereafter. As shown in Figure 1, the specific activity of the hybrid flies peaks at day 2 and then remains constant. Thus, not only is a lower level of activity maintained, but that level is reached earlier in the developmental profile of the adult. Pelliccia & Sofer (1982) showed that both the n6 and n11 strains produced inactive ADH at rates similar to wild type but maintained steady state levels lower

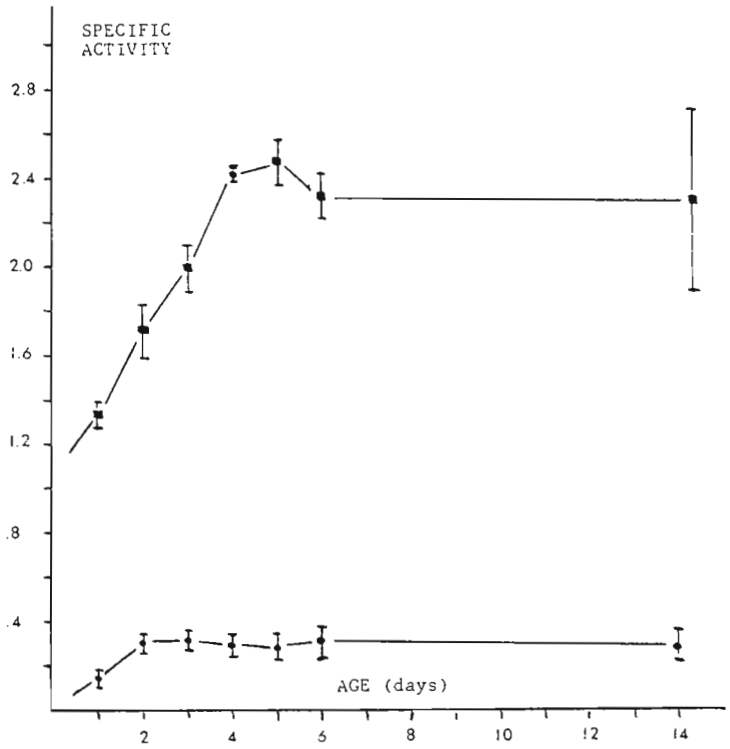


Figure 1. Developmental profile for alcohol dehydrogenase specific activity in AdhF (squares) and hybrid (circles) adults. Each point represents the mean ± standard deviation for at least 3 separate determinations.

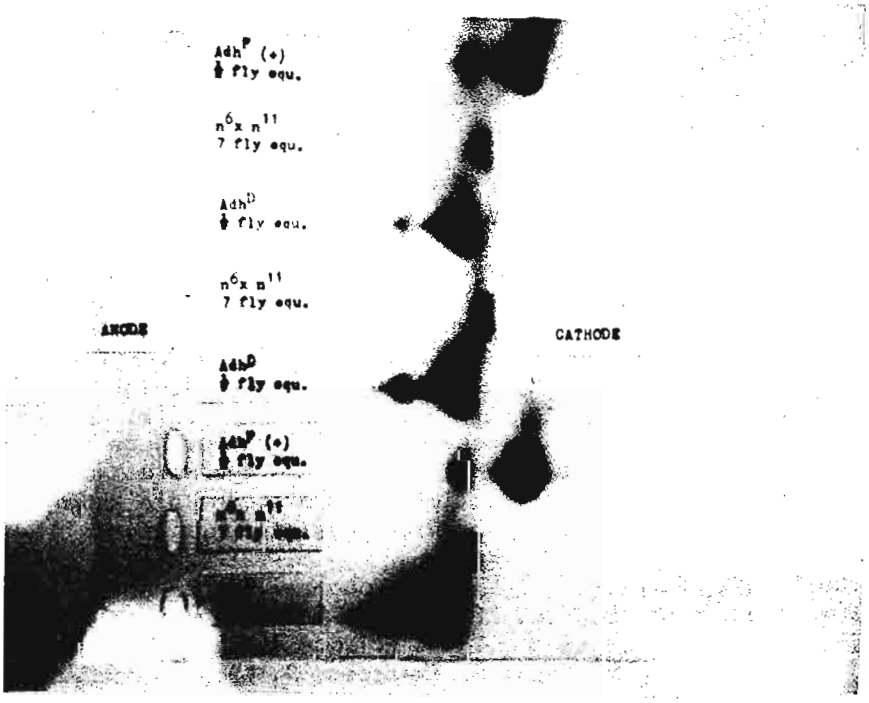


Figure 2. Agar gel electrophoresis of 20,000 x g supernatants from bulk fly homogenates. The bands were visualized with ADH activity stain. 1 'fly equivalent' is that amount of bulk supernatant containing protein equivalent to that from a single fly.

than normal due to an increased rate of protein degradation. The shorter time necessary for the hybrid enzyme to reach its steady state level of activity could be explained if it too showed a similar rate of synthesis and an increased rate of degradation when compared to wild type.

A more direct test of enzyme stability was performed by using the technique of heat denaturation on extracts partially purified from AdhF and hybrid flies by salt fractionation and hydroxylapatite chromatography. This procedure produces a preparation of approximately 35 fold greater purity than a crude homogenate. Equal volumes of the extracts were prepared which contained 100 enzyme units of activity. After 2 minutes at 42°C, the hybrid enzyme had completely lost its catalytic activity while the F enzyme still retained 15% of its initial activity. In fact, after 4 minutes at the elevated temperature, the F enzyme still retained 12% of its activity. At this temperature, the hybrid enzyme lost activity more rapidly than the wild type, but the F extract appeared to contain a small percentage of ADH molecules more heat resistant than average. Schwartz et al. determined that the ADH 1 isozyme is more heat stable and catalytically less active than the ADH 3 or ADH 5 isozymes due to the binding of a small NAD+ carbonyl adduct (Schwartz et al. 1979). Thus, our data suggests that although the hybrid protein denatures more quickly than the F enzyme, the prolonged stability of a small portion of the F extract may be due to an increased proportion of adduct when compared to the hybrid.

As a first step in resolving this issue, we used agar gel electrophoresis to separate the isozymes produced by AdhF, hybrid, and AdhD flies. The AdhD allele was obtained by Grell (1965) from AdhF flies by EMS mutagenesis and produces a protein which migrates more slowly toward the cathode. This is reasonable as the amino acid change in ADH D has been found to be from a glycine in ADH F to a glutamic acid (Schwartz & Jornvall 1976). This change accounts for an overall charge difference of 2 in the dimeric ADH molecule. Adh n11 protein also differs from ADH F by a single amino acid as it has an aspartic acid instead of a glycine at position #14 (Thatcher 1980). Thus, if we assume that the n6 protein does not have a charge difference from F, then the hybrid enzyme would have an intermediate mobility between that of ADH F and ADH D. Figure 2 demonstrates that this is, indeed, true. Since the n6 and n11 homodimers would therefore differ from the heterodimer in mobility, it should prove possible to quantitate the amounts of the three different species produced by a heterozygous fly.

Note the presence of the different isozymic forms for the F and D proteins. In certain overstained gels, these forms were also seen for the hybrid, indicating that this protein also has the capacity to bind the NAD+ carbonyl adduct. However, we were not able to estimate whether the hybrid flies maintained similar levels of the three isozymic forms as the F strain.

We again used the partially purified extracts described above to determine the kinetic parameters for the substrates ethanol and 2-butanol. The data is summarized in Table 1. ADH F protein shows a higher Vmax for the secondary alcohol as compared to the primary. The hybrid enzyme shows the same pattern. What is interesting is that whereas, for the F enzyme, Vmax for ethanol is about 30% that of Vmax for 2 butanol; the difference is markedly greater for the hybrid as here Vmax for ethanol is only 2.1% as large as it is for 2 butanol. In addition, the hybrid protein binds ethanol more tightly than the F enzyme while this is reversed for 2-butanol. Thus, the interaction of the two different subunits which restores a functional catalytic site seems to produce one with altered substrate binding properties. We are testing other substrates to determine if other differences exist.

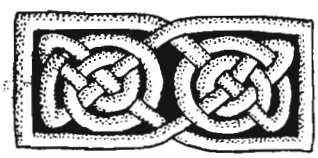
Table 1. Kinetic parameters for F and hybrid ADH Vmax is expressed in enzyme units. The units for Km are mM.

	Hybrid		F	
Substrate	Km	Vmax	Km	Vmax
Ethanol	65	0.4	73	8.5
2 Butanol	48	19	29	28

Finally we used the technique of antibody competition to measure the number of cross reactive molecules in extracts from AdhF and hybrid flies. 100 enzyme units of the hybrid extract contained approximately 12% more cross reacing molecules than a similar F extract. We suspect that this is due not only to the presence of the inactive homodimers (which should be present at low levels) but also to the reduced catalytic efficiency of the hybrid enzyme which would therefore require more molecules to produce 100 enzyme units. Again, as these molecules are electrophoretically separable, it should be possible to determine the relative contributions of these two ideas. Complete purification of the heterodimer enzyme would also allow a direct comparison of catalytic activity per molecule.

In conclusion, intragenic complementation at the Adh locus of the fly can produce a functional enzyme which has several interesting properties when compared to the wild type protein. Since the amino acid differences in certain null activity ADH proteins are known (Thatcher 1980) information correlating the change in sequence with physical parameters of the enzyme should be possible.

References: Sofer, W. & M.Hatkoff 1972, Genetics 72:545-549; O'Donnell,J. et al. 1975, Genetics 79:73-83; Pelliccia,J.G. & W.Sofer, Biochem. Gen. 20:297-313; Schwartz, M. et al. 1979, Arch.Biochem.Biophys. 194:365-378; Grell,E.H. et al. 1965, Science 149:80-81; Schwartz,M. & H.Jornvall 1976, Eur.J.Biochem. 68:159-168; Thatcher, D. 1980, Biochem. J. 187:875-886.



Pla, C., J.B.Toral and A.Fontdevila. Universidad Autonoma, Bellaterra (Barcelona), Espana. Genetic analysis of five morphological mutants recovered from a natural population of Drosophila buzzatti.

On October 1981, more than 300 adults of Drosophila buzzatti were collected at Calablanca, a country farm located at the outskirts of Sitges, about 45 Km south of Barcelona (Spain). This collection was performed at a row of Opuntia ficus-indica stands either using banana traps or aspirating the adults directly from the cactus rots. Thirty inseminated females of this collection were placed individually in vials and each offspring was investigated for its hidden morphological variability following the method of Spencer (1947). Accordingly, eight sib-pairs were established from each isofemale F_1 progeny. The analyses of their F_2 offspring unveiled the presence of five recessive eye color mutants, although test crosses showed that two of them were alleles of the same locus. Chromosomal assignment of the four independent mutants was performed by conventional linkage analyses using the offspring of crosses with marked strains. The tester strains used were homozygous for the following allozyme markers: M12 (Estβ, chromosome 2); S (ADH, chromosome 3) and M16 (PGM, chromosome 4) (Pla et al. 1984). Results of this analysis and considerations of reported data on chromosomal and mutant homologies (Stone 1955; Linsley & Grell 1968; Zouros 1976) substantiate the following tentative names, symbols and chromosome locations for the analyzed mutants: vermilion - v (X); mahogany - ma (2); scarlet - st (4) and brown -bw (5).

These are the first morphological mutants ever described in D.buzzatti. The low frequency of mutants in the studied sample (only four mutants out of thirty isofemale lines) may be explained in terms of the dynamics of natural populations of D.buzzatti and other cactiphilic species. Spencer (1940, 1941, 1944) has found obvious differences in frequency of occurrence of visible mutations between two natural populations of D.immigrans and D.hydei. The different degree of genetic variability for each of both populations of these species has been interpreted in terms of the reduction of crossbreeding as a consequence of a sharp population reduction during winter periods, which produces bottlenecks every year. In our particular case, the population of Calablanca is maintained by the rotting fruits and pads of not more than a few dozens of O.ficus-indica pads. The abundance of these natural substrates is seasonal, being high when there is an adequate combination of temperature and humidity. These optimal conditions occur only at few occasions, as we know by our collecting experience during several years. Consequently, population size experiments dramatic bottlenecks followed by expansions, which results in a low effective population size. This increases the frequency of homozygous and the effect of homoselection, producing a low equilibrium mutation-selection for morphological characters.

References: Linsley,D.L. & E.H.Grell 1968, Carn.Inst.Wash.Publ. 627; Pla,C., J.B.Toral, H.Naveira & A.Fontdevila 1984, submitted to Experientia; Spencer, W.P. 1940, Ohio J.Sci. 40:345-361; ____ 1941, Ohio J.Sci. 41:190-200; ____ 1944, Genetics 29:520-536; ____ 1947, Avd.in Genet. 1:359-402; Stone,W.S. 1955, Symp.Quant.Biol. 20:256-270; Zouros,E. 1976, Genetics 83:169-179.

Poole, J.H. and L.K.Dixon. University of Colorado at Denver, Colorado USNA. Drosophila peroxidases: I. Three major isozymes observed.

The response of peroxidase (PO) activity to pH was measured in homogenates of Drosophila melanogaster, and the correlations among variant forms of PO was determined.

Preparation of extract. A water soluble extract of Drosophila homogenate was prepared as follows. Flies were killed by placing in the freezer in pre-chilled bottles (-15°C) for 9 minutes (this was found to be the minimum time sufficient to kill all flies). A sample of 200-500 imagoes of mixed age and sex was weighed, and homogenized in distilled water (0.100 ml/mg tissue) with a motorized teflon-glass rotary tissue grinder (20 pulses per extraction at 10 second intervals). Use of pre-chilled (2°C) water for the extraction was sufficient to prevent frictional heating of the homogenate above 35°C. Homogenate was centrifuged at 2400 rpm for 20 minutes. All assays were performed on the supernatant solution.

A series of stock buffers was prepared covering pH 4.0-11.0 in increments of 0.1 pH unit, using phosphate-tris-borate (30 mM each) in the range pH 5.5-11.0, and phosphate-tris-phthalate (30 mM each) in the range pH 4.0-6.0. Then 0.20 ml Drosophila extract was mixed with 0.60 ml of each buffer and the resultant pH measured. These samples were adjusted to each

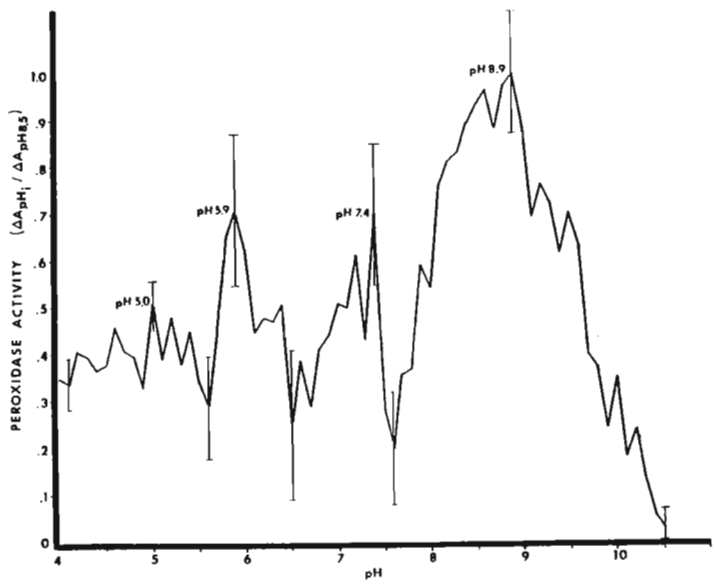


Fig. 1. Peroxidase activity vs. pH.

pH increment (±0.03) by blending samples of adjacent pH. In this manner, a series of samples was obtained for a given extract, spanning the desired pH range.

Assay method. To measure PO activity, the rate of oxidation of p-phenylenediamine (PDA) by hydrogen peroxide was photmetrically determined (Aurand et al. 1956; Armstrong et al. 1978). In all assays, 100 ul PDA was mixed with 100 ul H_2O_2 (final concentration of each substrate = 20 mM) at 20°C in a 1 cm light-path cuvet, and the reaction was initiated by the addition of 600 ul buffered Drosophila extract (total volume = 0.8 ml). Photon absorbance at 485 nm was measured for 10-20 minutes, and zeroed throughout the reaction against a blank cuvet. (The contents of the blank were the same as for the sample cuvet, but with plain buffer rather than extract.) The rate of change in A_{485} was used as a measure of the reaction velocity (i.e., formation of the reaction product, diaminophenazine). PO activity was determined from the mean slope in the linear portion of the reaction curve.

pH optima. A total of 580 measurements of PO activity were obtained between pH 4.0 and 10.5, at 0.1 pH increments (7-14 independent determinations per point). Raw activity scores were transformed to percent of the activity at pH 8.5, prior to calculation of the mean relative activity at each pH increment. This analysis resolved the presence of four activity optima, at pH 5.0, 5.9, 7.4 and 8.9 (Fig. 1). For each of these peaks the level of PO activity was significantly greater than that of the flanking minima (one-tail t-test), with $p<.02$, .02, .01 and .001 respectively. The three largest peaks displayed activities 3-5 times greater than the adjacent minima. These peaks will be referred to as acid-PO (pH 5.9), neutral-PO (pH 7.4) and alkaline-PO (pH 8.9). The fourth, small peak (pH 5.0) was not given further attention in this initial analysis.

Armstrong et al. (1978) also reported a pH optimum for Drosophila PO at pH 7.4. The other peaks reported here were not observed, perhaps due to the smaller number of measurements taken over wider pH increments in that study.

Evidence of isozymal status. Distinct pH optima generally correspond to variant forms of an enzyme (see IUPAC-IUB, 1976, for discussion). The question may be raised whether the three major peaks observed in this study correspond to distinct isozymes of PO. Another possibility is that only a single polypeptide is synthesized, which is subsequently modified by conjugation, cleavage or conformational changes (such alternate forms are not properly considered isozymes). If the latter alternative were correct, one would expect to find consistent correlations among the activities of each enzymatic form.

As a preliminary test of these alternatives, the activities of acid-PO, neutral-PO and alkaline-PO were measured in males of 12 inbred strains and a random-bred population of D. melanogaster (3-week-old imagoes). For each strain, four independent assays were performed at each pH, with 10 flies per assay. Across all strains, the following product-moment correlations were obtained among the activities of the PO variants (12 df):

$r_{\text{acid-PO neutral-PO}} = 0.35$ (N.S.)

$r_{\text{neutral-PO alkaline-PO}} = 0.52$ (N.S.)

$r_{\text{acid-PO alkaline-PO}} = 0.30$ (N.S.)

The absence of significant correlation (at the .05 level) strongly indicates that these PO variants are in fact independent isozymes. The distinct kinetic properties of the PO's (see the following paper in this series) provide further verification of the isozymal status of these variants.

It is of interest to note that the only electrophoretic analysis of insect peroxidases reported to date (Coles 1966) isolated three major PO isozymes in locusts. The three Drosophila PO's observed in the present study may well be homologous to the three locust isozymes. Electrophoretic analyses of the Drosophila peroxidases are currently in progress in our lab.

References: Armstrong,D., R.Rinehart, L.Dixon & D.Reigh 1978, Age 1:8-12; Aurand,L.W., W.M.Roberts & J.T.Cardwell 1956, J.Dairy Sci. 39:568-573; Coles,G.C. 1966, J.Insect Physiol.. 12:679-691; IUPAC-IUB Commission on Biochemical Nomenclature 1976, Handbook of Biochemistry and Molecular Biology, 3rd ed., V2, G.D.Fosman ed. CRC Press, Cleveland Ohio 1976 pp 84-84.

Poole, J.H. and L.K.Dixon. University of Colorado at Denver, Colorado USNA. Drosophila peroxidases: II. Isozyme kinetics, and optimum conditions for assays utilizing p-phenylenediamine.

The purpose of this study was to obtain initial estimates of the kinetic properties of Drosophila peroxidase (PO) isozymes, and to develop a sensitive photometric assay for each PO, for use on Drosophila tissue homogenates. A series of reactions was carried out to measure the effect of substrate concentration on reaction velocity, and to identify optimum assay conditions.

PROCEDURES: Flies were killed and extract prepared as described in the previous study in this series (see previous note). Subsequent to finding three major pH optima for PO activity (previous study), all extractions were carried out in tris-phosphate buffer (33.3 mM each) at pH 5.90, 7.50 or 9.05 (±0.01), 0.100 ml buffer/mg tissue. This yielded homogenates with the target pH of 5.9, 7.4 or 8.9 (±0.05).

PO activity was determined from the rate of oxidation of p-phenylenediamine (PDA) by hydrogen peroxide, measured as the change in absorbance at 485 nm (see previous note for general procedure). For each of the extracts (pH 5.9, 7.4, 8.9) a series of reactions was carried out with final concentration of H_2O_2 fixed at 20 mM, and the final concentration of PDA varied between 0.156 mM and 80 mM. This permitted estimation of the K_m for the PDA substrate. Following this determination, a series of reactions was carried out with the concentration of PDA fixed at 40 mM (i.e., greater than 75% enzyme saturation) and H_2O_2 concentration varied between 0.156 and 80 mM. This allowed estimation of the K_m for the H_2O_2 substrate.

KINETIC PROPERTIES OF THE PEROXIDASE ISOZYMES: Each of the three PO's produced linear Eadie-Hofstee plots (i.e., Michaelian kinetics) at all concentrations of PDA, and at concentrations of H_2O_2 below 5 mM. Catalatic activity was apparent as bubbling in the cuvet above 5 mM H_2O_2 --consistent with the observation of Angermueller and Fahimi (1981) that catalase activity predominates above 10 mM H_2O_2. The following estimates of K_m were obtained for each isozyme, based on the Eadie-Hofstee plots. For the H_2O_2 substrate, acid-PO had the highest apparent K_m=31±16.3 uM. Alkaline-PO's apparent K_m was 110±65 uM. For the PDA substrate, apparent K_m= 9±4.2 mM for acid-PO, 3.0±1.10 mM for neutral-PO, and 5.5±2.15 mM

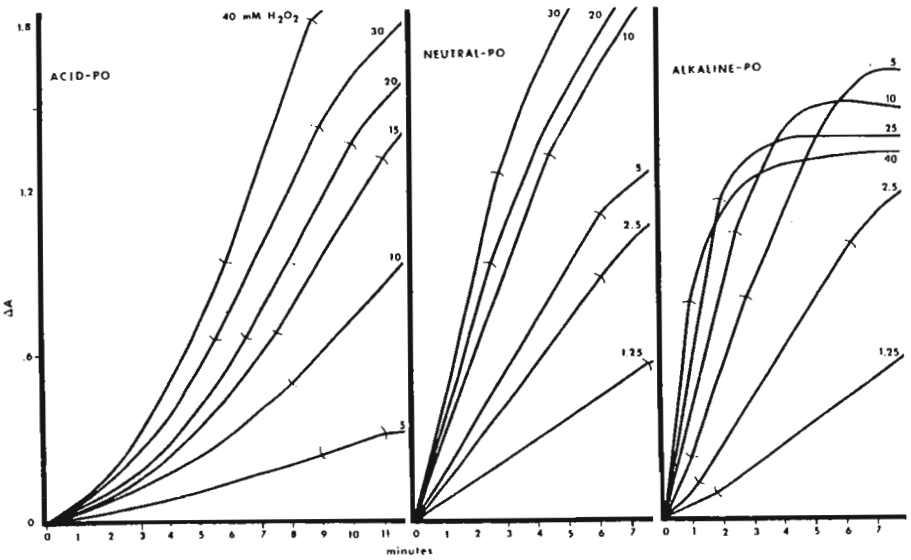


Fig. 1. Time course of the peroxidase reactions at various substrate concentrations. In all reactions the concentration of PDA was twice the concentration indicated for H_2O_2. Hysteretic lags were evident for acid-PO and alkaline-PO activity. Parentheses enclose the period of maximum linear velocity.

for alkaline-PO. It may be of interest to note that values of K_m are often indicative of the substrate concentrations normally encountered by an enzyme under physiological conditions (Hochachka & Somero 1973).

Two of the isozymes, acid-PO and alkaline-PO, displayed hysteretic kinetics (i.e, a lag phase prior to maximum linear reaction rate--Fig. 1). Alkaline-PO typically had a lag of 0-3 min before maximum velocity was attained. The lag for acid-PO was longer; an incubation period of 2-8 min was observed. For alkaline-PO the rate of transition to maximum velocity was found to be directly proportional to the concentration of PDA (r=0.991) and independent of changes in H_2O_2 concentration (Fig. 2). F r acid-PO the opposite was observed, with H_2O_2 concentration directly determining the rate of transition to maximum velocity (r=0.998). Frieden (1979) has pointed out that such hysteretic lags allow an enzyme to control substrate flux through metabolic pathways by damping the response to brief fluctuations in substrate concentration. This behavior may be important where a relatively constant level of enzyme activity is critical for homeostasis. In contrast, no hysteresis was observed for neutral-PO, which attained maximum linear velocity at the moment of substrate addition (Fig. 1).

OPTIMUM ASSAY CONDITIONS: In general a 1:2 ratio of H_2O_2 to PDA concentration was found to give best results. Raising the concentration of H_2O_2 above this ratio tended to produce bubbling in the cuvet, a catalatic reaction, which interfered with photon absorbance measurement. Lower levels of H_2O_2 reduced the rate and duration of linear reaction. Thus the 1:2 ratio produced the most rapid and sustained linear reaction.

A series of reactions was then carried out at the 1:2 ratio of substrates (ranging from 80 mM PDA + 40 mM H_2O_2 to 5.0 mM PDA + 2.5 mM H_2O_2). In this manner, the substrate conditions producing the most sustained linear reactions were obtained (Table 1).

STABILITY OF PEROXIDASE ACTIVITY IN HOMOGENATE: The peroxidase activity in the extract supernatant was found to change gradually over an eight hour period at 20°C. The acid-PO activity increased linearly at a rate of 5.7% ± 1.50% per hour. Neutral-PO activity decreased

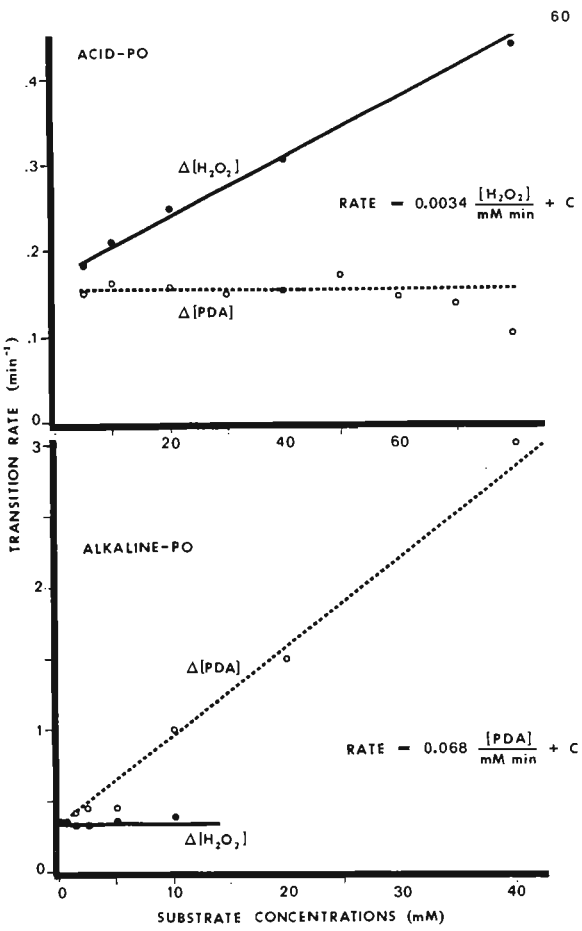


Table 1. Assay conditions for Drosophila peroxidase isozymes.

Volume	Initial Concentration	Final Concentration
ACID-PO ASSAY (pH 5.9)		
0.1 ml PDA	160.0 mM	20.0 mM
0.1 ml H_2O_2	80.0 mM	10.0 mM
0.6 ml extract	33.3 mM tris-phos	25.0 mM
0.8 ml total		
NEUTRAL-PO ASSAY (pH 7.4)		
0.1 ml PDS	20.0 mM	2.50 mM
0.1 ml H_2O_2	10.0 mM	1.25 mM
0.6 ml extract	33.3 mM tris-phos	25.00 mM
0.8 ml total		
ALKALINE-PO ASSAY (pH 8.9)		
0.1 ml PDA	40.0 mM	5.00 mM
0.1 ml H_2O_2	20.0 mM	2.50 mM
0.6 ml extract	33.3 mM tris-phos	25.00 mM
0.8 ml total		

For each assay, blanks were prepared as above, with plain buffer (pH 5.9, 7.4, 8.9) rather than extract.

Fig. 2. Effect of substrate concentration on the rate of hysteretic transition. The transition rate of acid-PO was dependent on H_2O_2 concentration, while that of alkaline-PO was dependent on PDA concentration.

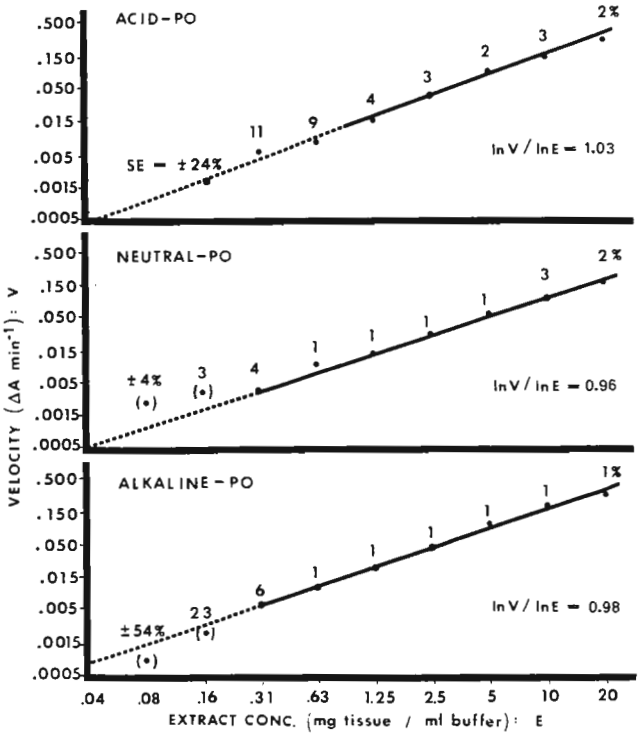


Fig. 3. Linearity and reproducibility of assays. Reaction rate vs extract concentration (by serial dilution) is graphed on a log-log plot. Relative standard error is indicated for each point (2 df). Solid lines indicate velocities that can be considered within the range of reliability for each assay (based on both linearity and reproducibility).

linearly at 2.7% ± 0.61% per hour. Alkaline-PO showed no significant change over time (0.0% ± 0.52% per hour). Consequently a standard period, post homogenization, was used for all assays.

STANDARD ASSAYS: Based on the above observations, the following protocol was established for assays of each isozyme's activity. After freezing, sexing and weighing, 5-10 organisms (or body segments) are placed in 1.00 ml buffer (33.3 mM tris-phosphate at pH 5.90, 7.50 or 9.05 (±0.01), prechilled to 3°C). The samples are homogenized with a motorized teflon glass tissue grinder, centrifuged (2400 rpm, 20 min) and the supernatant decanted for assay. Enzyme assays are then targeted to a one-hour window, set at 5.0 h (±0.5 h) after homogenization of samples.

See Table 1 for reagent proportions in each assay. Addition of extract initiates the reaction (time-zero), and A_{485} is recorded (±0.005) at intervals of 1 min (±5 sec), for 10 minutes.

The reaction velocity is calculated from the most linear portion of the curve over a minimum of three minutes (i.e., four readings). For acid-PO, these readings are usually at t=7 to 10 min: for neutral-PO and alkaline-PO at t=2 to 9 min.

SENSITIVITY AND REPRODUCIBILITY OF ASSAYS: Figure 3 summarizes findings on the usable range for each isozyme's assay, under the reaction conditions described above. Linearity of the assays, in response to various concentrations of PO is shown. Reproducibility is indicated by the value of the standard error at each level of activity (2 df).

The acid-PO assay was found to be linearly proportional to enzyme concentration at all levels of activity tested. On a log-plot plot, the slope is very close to unity (the index of direct proportionality), with a value of 1.03 (r=.997). The relative standard error is less than 5% for enzyme activities obtained with extract concentrations as low as 1.25 mg tissue/ml. This is equivalent to one fly per assay, under the above assay protocol. Reproducibility is marginal (SE>10%) at lower concentrations, equivalent to fractions of a fly per assay.

The neutral-PO and alkaline-PO assays were found to be three to five times as sensitive as the acid-PO assay. Log-log slopes of 0.96 and 0.98 (r=.999), as well as relative standard errors of less than 5%, were obtained for extract concentrations as low as 0.31 mg tissue/ml--or approximately one-third of a fly per assay, under the above assay protocol.

SCALING OF ASSAYS: The IUPAC-IUB Commission of Biochemical Nomenclature (1973) recommends use of katal units in all reports of enzyme activity (1 kat= the activity converting one mole substrate to product per second). It is recommended that use of the former "enzyme unit" (1 U = micromoles per minute) be discontinued.

In order to convert ΔA to katal units, the change in molar absorptivity (ε) at 485 nm, upon oxidation of PDA, was determined at each of the three assay pH's (based on absorbance measurements on standard solutions of PDA and oxidized PDA). At pH 5.9, $\Delta\varepsilon$=2.686±0.024. At pH 7.4, $\Delta\varepsilon$=2.137±0.0075. At pH 8.9, $\Delta\varepsilon$=1.98±0.078.

Based on the molar absorptivities, the extraction and assay volumes, the substrate concentrations used in these assays, and the values of K_m for each isozyme, the following

conversion factors may be readily calculated: acid-PO = 1.37×10^4; neutral-PO = 1.20×10^4; alkaline-PO = 1.23×10^4. Multiplying raw data of the assays (ΔA_{485} min^{-1}) by the above conversion factors gives the V_{max} PO activity of the 1 ml extract, in picokatals (pkat).

CONCLUSIONS: This assay procedure utilizes total soluble extracts of Drosophila tissue, without prior concentration or isolation of the PO isozymes. Thus, the measurements of enzyme activity cannot be assumed to reflect enzyme concentration alone, but may also be influenced by natural inhibitors and activators of peroxidases, present within the tissue homogenates. The method has the advantage of speed and yields reasonably reproducible results. We are currently analyzing the PO isozymes of Drosophila for developmental changes in activity, tissue distribution and functional significance.

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Poole, J.H. and L.K.Dixon. University of Colorado at Denver, Colorado USNA. Drosophila peroxidases: III. Developmental profile of isozymes.

In this report, we present our findings on the expression of peroxidase (PO) isozyme activity over the lifespan of Drosophila melanogaster.

METHODS: Wildtype stocks were maintained on cornmeal-molasses-agar-yeast medium in half-pint bottles, with a diurnal cycle of 12 hr in the light at 30°C and 12 hr in the dark at 20°C. Imagoes were collected at eclosure and transferred weekly to fresh medium, thereby maintaining distinct age-cohorts. For assay, third instar larvae were collected while wandering outside the medium prior to pre-pupal immobilization. Pupae were collected after completion of body segmentation and wing-bud/leg eversion but prior to initiation of eye pigmentation. Imagoes were collected for assay at eclosure ("week 0") and at weekly intervals thereafter. For each assay 6-10 organisms of a given age and sex were frozen, weighed, homogenized in buffer, centrifuged and photomerically assayed with PDA/H_2O_2 as previously described (see Report II). In addition, protein content of the supernatant was assayed by UV absorbance at 260 and 280 nm (Layne 1957). Three independent extractions and assays were performed for each PO isozyme, at each age and sex (except for the last two weeks of life, when sufficient numbers of organisms survived to perform only 2 independent determinations). Larvae and pupae were not sexed for this series of assays. PO activity is reported in picokatals (picomoles H_2O_2 reduced per second) at V_{max}, per mg body mass (see previous note for conversion factors).

PO ISOZYME ACTIVITIES DURING DEVELOPMENT: The activity of each PO isozyme across the lifespan of wildtype D. melanogaster is shown in Figure 1. Each data point represents the mean and standard error of the mean for PO activity at a given age.

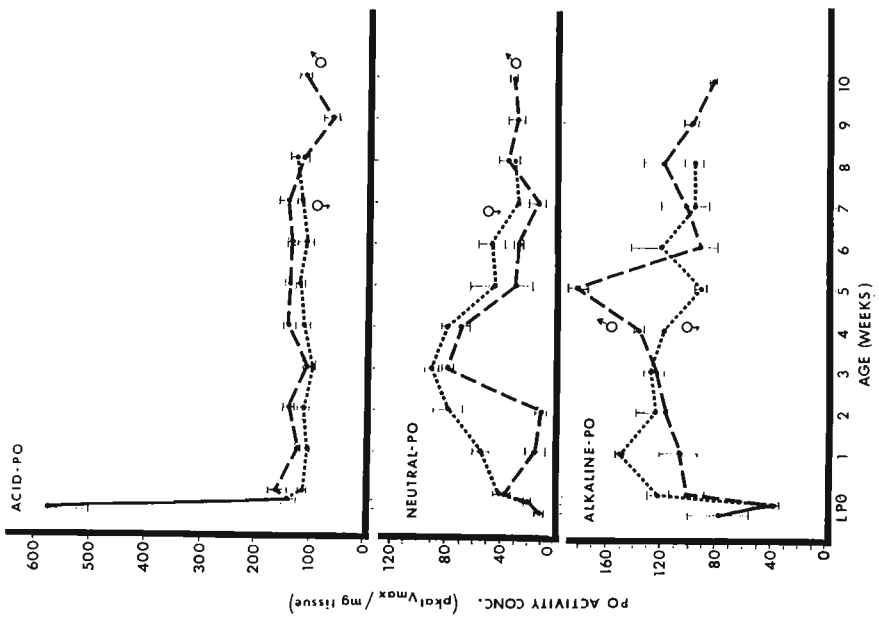


Fig. 1. Activity concentration of peroxidase isozymes vs age (wildtype females and males).

The three isozymes were quite distinct from one another in their developmental expression. Acid-PO showed maximum activity in the pre-pupating larvae. The activity of this isozyme dropped about 80% in pupae and remained low in both sexes for the remaining lifespan. In contrast, neutral-PO activity was quite low in the pre-adult stages, peaked at age 3 weeks in both sexes and then declined by about 60% with age. Alkaline-PO activity was lowest in the pupae. This isozyme appeared to peak briefly (age 1 week in females, age 5 weeks in males), but otherwise remained at relatively constant levels during adult life.

Specific activity (PO activity per mg protein) was also determined for each isozyme. This index of enzyme activity places two of the peaks at slightly later ages than were indicated by activity per mg body mass (neutral-PO in males at age 4 weeks, alkaline-PO in females at age 3 weeks). Otherwise the indices are in general agreement and portray similar developmental trends in the isozyme activity levels.

Armstrong et al. (1978) also measured the PO activity at pH 7.4 (corresponding to neutral-PO) across the lifespan of D.melanogaster. The developmental profile in that study was quite similar to that of neutral-PO in the present study. Armstrong et al. found peak neutral-PO activity at two weeks of age in female imagoes and at three weeks of age in male imagoes. The isozymes activity also declined precipitously with age, as in the present study. It appears that the developmental profile of at least this isozyme is quite reproducible.

We are currently analyzing the functional properties of each PO isozyme and the possible significance of these patterns of isozyme expression during Drosophila development and aging.

References: Armstrong,D., R.Rinehart, L.Dixon, D.Reigh 1978, Age 1:8-12; Layne,E. 1957, Methods in Enzymology, V3, S.P.Colowick & N.O.Kaplan (eds), Academic Press, New York, pp. 451-454.

Preston, C.R. and W.R.Engels. University of Wisconsin, Madison USNA. Movement of P elements within a P strain.

Wild strains of Drosophila melanogaster carry a family of movable genetic elements known as P elements. These elements are known to transpose at a high rate when crossed into laboratory, (M), strains having the condition known as M cytotype (Engels 1983). Though P element activity within a P strain (flies with P cytotype and P elements) is much reduced, the observations reported here demonstrate that transposition and excision still occur at significant and measurable frequencies.

By in situ hybridization of polytene chromosomes, we have seen changes in P element locations in branches of a P strain after the lines had been maintained separately for many generations. These changes presumably represent transpositional activity of P elements within P strains.

Π_2, a wild caught P strain from a Madison population was maintained as described previously (Engels & Preston 1979) through twelve generations of full-sib matings and five subsequent bottle stock generations. At that time, a single pair mating produced the adults that were branched into ten single female lines (lines a-j, see Figure). These branches were kept at room temperature (21°C) by single female matings for three generations and small mass (vial) generations thereafter. At approximately the same time, two more pairs were branched and subsequently maintained at a higher (28°C) and a lower (18°C) temperature in quarter-pint bottle populations. Π_2 20c is yet another line that was branched from the $\Pi_2 c_1$ line at generation 52 and maintained independently for twenty generations as a bottle stock.

After 77-80 generations for the room temperature and 28° lines and 47 generations for the 18° line, larvae were selected for in situ hybridization to P element sequences. The results (see Figure) show the variation of P element positions on the X chromosome within these lines. The number of larvae analyzed per branch line is shown to the right of each chromosome. In cases where more than one larva was examined, there was a possibility of detecting polymorphism (circled points) within the line. We looked at more slides of the "hot" and "cold" lines expecting more variability because of the larger population size. This proved to be especially true in the 28° line where each of the twelve P element sites was missing in at least one case.

Because P element DNA probes were unavailable when the Π_2 strain was isolated in 1977, P element sites of the original line at that time are unknown. However, because these branches all originated from a highly inbred stock, the observed differences must represent

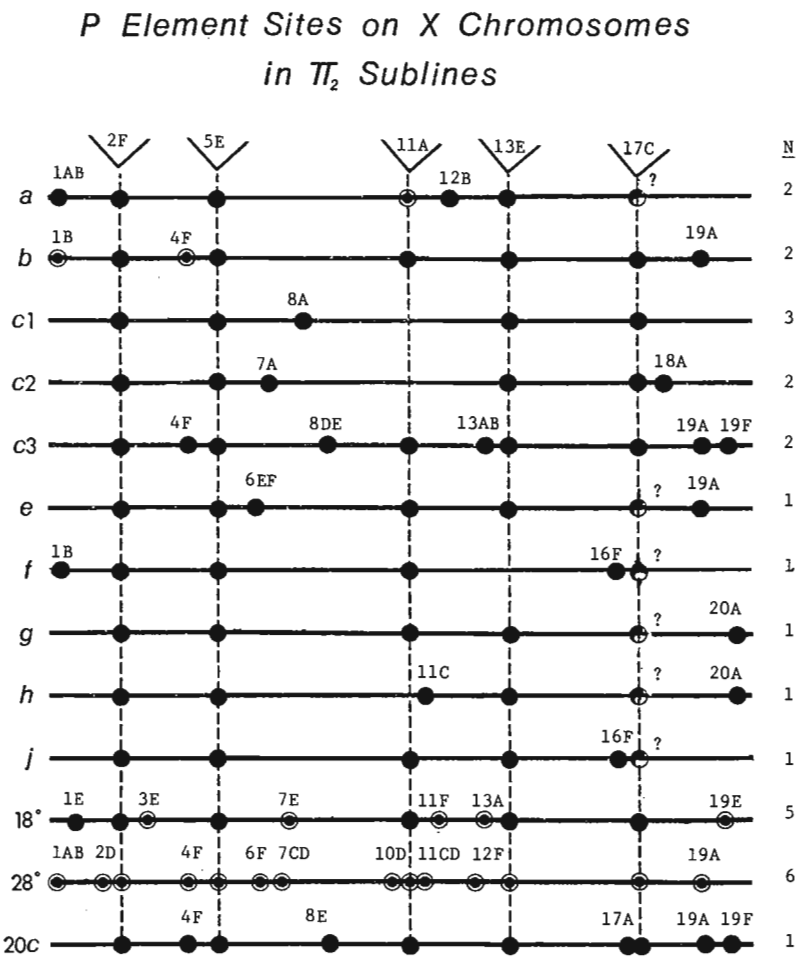


Figure 1. Horizontal lines depict X chromosomes of the Π_2 branch lines, and dots mark the positions of in situ hybridization. The probe in most cases was a 1:1 mixture of the HindIII fragment and the HindIII-SalI fragment internal to the complete P element (O'Hare & Rubin 1983). The rest of the slides were labeled with the pΠ25.1 probe which contains a complete P factor plus flanking sequences from position 17C. For those cases, the presence of a P element at 17C is uncertain, as indicated by the question marks in the Figure. Circled points represent polymorphic sites.

transpositions of P elements within P strain conditions. The vertical dashed lines show points of frequent (7 or more) sites which most likely represent the original Π_2 line. Several other sites, 1B, 4F and 19A, occur in several branches and may represent polymorphic sites in the original Π_2 stock prior to branching despite the close inbreeding.

To estimate the transposition and excision rates, we assume the original stock had X chromosomal sites at positions 2F, 5E, 11A, 13E and 17C, and that all changes indicate single, selectively neutral, events. If polymorphic sites are weighted by their observed frequencies, then the estimated rate of gain of sites is 0.29±0.005 per X chromosome per generation. The standard error is based on the assumption that these events have a Poisson distribution. We cannot estimate the rate of transposition on a per element basis since some of the donor sites might have been on the autosomes which were not monitored. Our estimate of the excision rate per element per generation (excluding the 17C element in cases where only the pΠ25.1 probe was used) is 0.0015±0.0006. These Π_2 stocks are apparently not in equilibrium since they are gaining sites more rapidly than they lose sites. We conclude that P transposition and excision occur at appreciable frequencies in the P cytotype. However, these rates are only approximately one thirtieth of rates previously estimated for the M cytotype (Engels & Preston 1981; Bingham et al. 1982).

References: Bingham,P.M., M.G.Kidwell & G.M.Rubin 1982, Cell 29:995-1004; Engels,W.R. 1983, Ann.Rev.Genet. 17:315-344; Engels,E.R. & C.R.Preston 1979, Genetics 92:161-175; Engels,W.R. & C.R.Preston 1981, Cell 26:421-428; O'Hare, K. & G.M.Rubin 1983, Cell 34.25-35.

Ramachandra, N.B. and H.A.Ranganath.
University of Mysore, Manasa Gangotri, India. Preliminary studies on the differences in the nutritional requirements in Drosophila.

For Drosophila, a satisfactory standard culture medium must be nutritious, inexpensive, have a high moisture content and a firm texture, and be resistant to mould and bacterial contamination. Most standard media contain sugar source, a grain base, agar, a mould inhibitor and yeast (Ashburner & Thompson,Jr 1978). Baumberger (1919) and Hassett (1948) have demonstrated that sugar was a dietary requirement and the role of different sugars on the biology of Drosophila.

The present project was undertaken to understand, the ecological differences, if any, under laboratory conditions among ecologically/phylogenetically closely placed forms of Drosophila.

D.melanogaster and D.ananassae are cosmopolitan species. They are sympatric in the domestic habitats. Morphologically they are different and taxonomically, D.melanogaster belongs to melanogaster subgroup while D.ananassae comes under ananassae subgroup. D.n.nasuta and D.n.albomicana are morphologically identical and they are cross fertile. These chromosomal races are allopatric in their distribution. Both belong to the nasuta subgroup of Drosophila.

Utilising these strains of Drosophila, preliminary studies have been made to record the relative preference of these forms to different sources of sugar, namely, glucose, fructose and sucrose. Flies were maintained on wehat cream agar media containing either glucose or fructose or sucrose or without any one of them. The relative preference of different strains to different sources of sugar as assessed by their 'overall population size' for over ten months is given in the Table:

	Wheat cream agar media with:			
Strain	Fructose	Glucose	Sucrose	No sugar
D.melanogaster	++	++	++	+
D.ananassae	++	+	+	-
D.n.nasuta	+++	++	+++	+
D.n.albomicana	++	+++	++	-

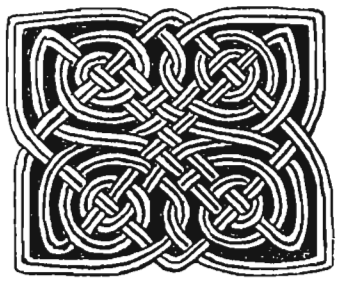
Relative prefrence: +++ > ++ > + > - .

D.ananassae and D.n.albomicana failed to maintain their population in a media which was devoid of sugars, while D.n.nasuta and D.melanogaster have managed to survive in sugarless media even after ten months. It appears that D.ananassae prefers the media with fructose than with glucose or sucrose, while D. melanogaster is found to have no such discrimination and it survives equally well on all the three types of media. For D.n.nasuta, media with fructose or sucrose are found to be more suitable, while D.n.albomicana maintains a better population size in the media with glucose than in others.

Thus, these preliminary experiments conducted for over a period of ten months do indicate the existence of 'subtle' differences between ecologically closed placed (D.melanogaster and D.ananassae) and between phylogenetically closed linked (D.n.nasuta and D.n.albomicana) forms of Drosophila in their preference to the media with different types of sugars. Further experiments are in progress to quantify these differences.

Acknowledgements: Authors are grateful to Prof. N.B.Krishnamurthy, Head of the Department of Zoology, for his help and encouragement; to the University Grants Commission and the Indian National Science Academy for financial assistance.

References: Ashburner,M. & J.N.Thompson,Jr 1978, The laboratory culture of Drosophila, IN The Genetics and Biology of Drosophila, V2a:2-109 (Ashburner & Wright-eds), Academic Press, London; Baumberger,J.P. 1919, J.Exp.Zool. 28:1-81; Hassett,C.C. 1948, Biol.Bull. Woods Hole 95:114-123.



Reiling, H., W.-E.Kalisch and T.Whitmore.
Ruhr-Universität Bochum, FR Germany.

Computerized EM Chromosome maps.

We have made use of the combination of three techniques which together enable us to plot computerized, semi-quantitative chromosome maps of the EM band-interband pattern from polytene chromosomes in Drosophila. These being: (1) the surface spread polytene (SSP) chromosome preparation technique, (2) a digitization technique for the cytological data of EM photos of SSP chromosomes and (3) a computer program for plotting chromosome maps.

The usability of the SSP chromosome preparation technique has already been proven for LM, TEM and SEM analyses of the band-interband patterns in Drosophila and Chironomus (Kalisch 1982a,b; Kalisch & Hägele 1981,1982; Kalisch & Jacob 1983; Kalisch & Whitmore 1983). We could show that in EM micrographs of selected SSP chromosomes all the polytene structures (i.e., individual bands and their right-handed interbands) can be depicted individually. Recently we have found a way to digitize the cytological data of the EM band-interband pattern of the SSP chromosomes from Drosophila by using a set of 'graphic elements' for the characterization of an individual polytene structure (in preparation; details will be given on request by the authors). These graphic elements are symbolized by variables: A = band diameter; B = band type; C = band thickness; D = interband length; E = outlines of interbands and puffs; R = band radius (for curved bands); W = sectioning of the chromosome map; V$ = reference numbers (for details see Table 1). The values of variables A-V$ come in part from measurements of EM micrographs of SSP chromosomes (variables A, C and D) and from standardized computer-internal and plotter-internal values (variables B, R, W, and V$). A computer had to be used to compile and to store the large amount of data.

In Table 1 we present our standard computer program, especially written for Drosophila chromosomes, by which the digitized data can be recorded, stored and plotted as a chromosome map. The program is written in EXTENDED BASIC for a SHARP PC-1500 pocket computer, which is coupled with a plotter (SHARP CE-150) and extended to ca. 11.5 Kbytes RAM with a memory module (SHARP CE-155). For the serial storage of data we used a taperecorder (SHARP CE-152). The program offers the choice of three options (in the following the first program line number concerned is given in brackets): (1) digitized data of an individual polytene structure can be recorded [50-110], (2) recorded data can be corrected [120], plotted as a chromosome map [140-895] or stored on tape [135-139]. (3) Data stored on tape can be corrected again [2000-2130] and/or plotted as chromosome map [140-895]. Data (A-V$ of each polytene structure are stored together with a code number [50] for recalling an individual data from the tape [3000]. We use the (extended) reference numbers of previously published chromosome maps as code numbers. For the plotting of chromosome maps one can select from a menu of four different possibilities: (1) only the band-interband pattern is plotted [140-895], (2) the pattern is plotted together with a (C/D) scale, which visualizes the individually measured values (in 0.2 mm units) of band thicknesses (variable C) and interband lengths (variable D) [1300-1490], (3) the pattern is plotted together with a sectioning of the chromosome (divisions, subdivisions and/or individual bands) and the reference numbers [1200-1290], (4) the pattern is plotted together with the C/D scale, the sectioning and the reference numbers [140] as to be seen in Fig. 1. For special inputs of the menu see [28-33]. Due to the limitations and specifications of the equipment used the minimum plotting unit is 0.2 mm. By the latter one, the chromosome outlines (band diameters, puff sizes) have to be calculated relative to the maximum value of 160 units.

Fig. 1 shows, as an example, the computerized EM chromosome map of region 83A-85C in D.hydei. The map (shown in its original plotting size; i.e., 3.2 mm of the map equals 1 µm of an X3,200 EM micrograph) is based on data from several EM micrographs of SSP chromosomes of salivary glands from late 3rd instar larvae. Compared with the hand-drawn LM chromosome map so far existing (Berendes 1963), the computerized EM map shows a 44% (50:72) increase in the number of bands. Besides the semi-quanitativeness of the A, C and D values the computer maps have the distinct advantage that the actual pattern can be plotted according to subsequent cytological analyses. A flow diagram of the program listed in Table 1 will be sent on request by the authors.

Research was supported by the Deutsche Forschungsgemeinschaft, Ka 309/6.

References: Berendes,H.D. 1963, Chromosoma(Berl.) 14:195-206; Kalisch,W.-E. 1982a, DIS 58:85-87; ____ 1982b, Genetica 60:21-24; Kalisch,W.-E. & K.Hägele 1981, J.Cell Biol. 31:91-138; ____ 1982, In: Advances in Genetics, Development and Evolution of Drosophila, Plenum Publ. Co., New York, p.1-10; Kalisch,W.-E. & H.J.Jacob 1983, Cytobios 36:39-43; Kalisch,W.-E. & T.Whitmore 1983, Cytobios 37:37-43.

Table 1. Standard computer program for digitized cytological data from EM micrographs of SSP chromosomes in Drosophila by which data can be recorded, stored and plotted as chromosome maps. The term 'field' (line numbers 404, 504 and 604) stands for 'decondensed chromosome band'.

```
 5:REM    PROGRAM FOR SHARP PC-1500
 6:REM    + PLOTTER CE-150
 7:REM
10:REM ****************************
11:REM                            *
12:REM    "ROU/COU/TCP" =         *
13:REM    PROGRAM FOR RECORDING/  *
14:REM    CORRECTING/PLOTTING     *
15:REM    VARIABLES FOR CHROMO-   *
16:REM    SOME BANDS BY TAPE      *
17:REM                            *
18:REM ****************************
19:REM
20:REM ****************************
21:REM                            *
22:REM    INPUTS FOR VARIABLES    *
23:REM                            *
24:REM ****************************
25:REM
27:DIM E(30)
28:WAIT 150:PRINT "ROU=RECORDING OF
   VARIABLES":PRINT "MENUE = ROU+S
   +C/D"
29:PRINT "COU=CORRECTING OF VARIABL
   ES":PRINT "MENUE = COU"
30:PRINT "TCP=TAPE CONTROLLED PLOTT
   .":PRINT "MENUE = TCP+S+C/D"
31:INPUT "INPUT ROU/COU/TCP = ";Q$
32:IF Q$="COU"GOTO 2000
33:IF (Q$="TCP")OR (Q$="TCP+S")OR (
   Q$="TCP+S+C/D")GOTO 3000
37:FOR I=1TO 30
38:E(I)=0
39:NEXT I
41:C=0,B=0,C=0,D=0,R=0
42:U$=" D",M$="D",N$="D"
43:F=0,I=0,J=0,K=0,M=0,S=0
44:T=0,W=0,X=0,Y=0,Z=0,L=0
45:PRINT "NEW BAND"
50:INPUT "PROGRAM LINE, INPUT T = ";
   T
55:INPUT "BAND DIAMETER, INPUT A = "
   ;A
57:IF A)160GOTO 1510
60:INPUT "BAND TYPE, INPUT B = ";B
65:IF B<106OTO 1520
66:IF B>69GOTO 1530
70:INPUT "BAND THICKNESS, INPUT C =
   ";C
75:INPUT "INTERBAND, INPUT D = ";D
80:IF M$="N"GOTO 90
83:IF B)=28GOTO 85
84:GOTO 100
85:IF B)=50GOTO 90
86:IF B)=40GOTO 100
90:INPUT "RADIUS, INPUT R = ";R
93:IF R=0GOTO 100
95:IF R<=A/2GOTO 1540
100:INPUT "DIVISION BORDER, INPUT W =
   ";W
103:IF M$="N"GOTO 110
104:IF W=9GOTO 113
105:IF W)36GOTO 1550
106:IF W)1GOTO 113
110:INPUT "DIVISION NO., INPUT U$ = "
   ;U$
113:IF D<0GOSUB 1600
115:WAIT 250:PRINT T;A;B;C;D;R;W;U$
116:IF D)=0GOTO 119
117:PRINT T;A;B;C;D;E(1);E(-D/2+0.5)
119:M$="Y"
120:INPUT "CORRECT VARIABLES ? Y/N=
   ";M$
121:IF M$="N"WAIT 150:PRINT "CHANGE
   DATA":GOTO 50
122:M$="Y"
123:INPUT "PLOTTING OF BAND? Y/N = "
   ;M$
124:IF M$="N"GOTO 135
125:GOSUB 140
126:N$="Y"
129:INPUT "CORRECT GRAPHING ? Y/N =
   ";N$
130:IF N$="N"PRINT "CHANGE DATA":
   GOTO 50
135:PRINT #1,T,A,B,C,D,R,W,U$
136:IF D)=0GOTO 139
138:PRINT #1,B,D,E(*)
139:GOTO 32
140:IF (Q$="COU")OR (Q$="ROU+S")OR (
   Q$="ROU+S+C/D")GOSUB 1200
142:IF (Q$="TCP+S")OR (Q$="TCP+S+C/D
   ")GOSUB 1200
144:IF C=0GOTO 700
145:IF B)=60GOTO 600
146:IF B)=50GOTO 500
147:IF B)=40GOTO 400
148:IF B)=30GOTO 300
149:IF B)=20GOTO 200
150:REM
152:REM ****************************
153:REM                            *
154:REM          BAND              *
155:REM                            *
156:REM ****************************
157:REM
160:Z=0
165:GRAPH
170:LINE (J10-INT (A/2),-I)-(J10+INT
   (A/2),-I),B-10
180:Z=Z+I
185:IF Z<CTHEN 165
190:IF (Q$="TCP+C/D")OR (Q$="TCP+S+C
   /D")GOSUB 1300
191:IF (Q$="ROU+C/D")OR (Q$="ROU+S+C
   /D")OR (Q$="COU")GOSUB 1300
195:GOTO 700
200:REM
202:REM ****************************
203:REM                            *
204:REM         CONVEX BAND        *
205:REM                            *
206:REM ****************************
207:REM
210:S=B-20,J=10,Z=0,I=0
213:IF S)0GOTO 220
215:I=2
220:K=INT (SQR (R*R-A/2*A/2))
230:GRAPH
235:GLCURSOR (J10,-K):SQR6N
240:LINE -(INT (-A/2),K),9
245:FOR I=R-INT (A/2)TO R+INT (A/2)
    STEP J
250:X=INT (R-I)
255:Y=INT (SQR (R*R-X*X))
260:LINE -(-X,Y-Z),B-20
265:NEXT I
270:Z=Z+I
275:IF Z<CGOTO 240
280:GLCURSOR (0,K-C):SQR6N
290:IF (Q$="TCP+C/D")OR (Q$="TCP+S+C
   /D")GOSUB 1300
291:IF (Q$="ROU+C/D")OR (Q$="ROU+S+C
   /D")OR (Q$="COU")GOSUB 1300
295:GOTO 700
300:REM
302:REM ****************************
303:REM                            *
304:REM         CONCAVE BAND       *
305:REM                            *
306:REM ****************************
307:REM
310:S=B-30,J=10,Z=0
313:IF S)0GOTO 320
315:I=2
320:K=INT (SQR (R*R-A/2*A/2))
330:GRAPH
335:GLCURSOR (J10,K):SQR6N
340:LINE -(INT (-A/2),-K),9
345:FOR I=R-INT (A/2)TO R+INT (A/2)
    STEP J
350:X=INT (R-I)
355:Y=INT (SQR (R*R-X*X))
360:LINE -(-X,-(Y+Z)),B-30
365:NEXT I
370:Z=Z+I
375:IF Z<CGOTO 340
380:GLCURSOR (0,-(K+C)):SQR6N
390:IF (Q$="TCP+C/D")OR (Q$="TCP+S+C
   /D")GOSUB 1300
391:IF (Q$="ROU+C/D")OR (Q$="ROU+S+C
   /D")OR (Q$="COU")GOSUB 1300
395:GOTO 700
400:REM
402:REM ****************************
403:REM                            *
404:REM           FIELD            *
405:REM                            *
406:REM ****************************
407:REM
410:Z=0
415:GRAPH
420:LINE (J1)-INT (A/2),-I)-(J1)+INT
   (A/2),-I),B-40
425:Z=Z+I
430:IF Z)=CTHEN 485
435:LINE (0,-2)-(0,-2),9
440:Z=Z+I
445:IF Z)=CGOTO 485
450:GRAPH
455:LINE (J09-INT (A/2),-I)-(J09+INT
   (A/2),-I),B-40
460:Z=Z+I
465:IF Z)=CGOTO 485
470:LINE (0,-2)-(0,-2),9
475:Z=Z+I
480:IF Z<CGOTO 415
485:IF (Q$="TCP+C/D")OR (Q$="TCP+S+C
   /D")GOSUB 1300
486:IF (Q$="ROU+C/D")OR (Q$="ROU+S+C
   /D")OR (Q$="COU")GOSUB 1300
490:GOTO 700
500:REM
502:REM ****************************
503:REM                            *
504:REM         CONVEX FIELD       *
505:REM                            *
506:REM ****************************
507:REM
510:I=10,Z=0,S=B-50
513:IF S>0GOTO 515
514:I=2
515:K=INT (SQR (R*R-A/2*A/2))
520:GRAPH
522:GLCURSOR (J10,-K):SQR6N
524:LINE -(-INT (A/2)-I,K),9
526:FOR I=R-INT (A/2)-ITO R+INT (A/2
    )-JSTEP J
528:X=INT (R-I)
530:Y=INT (SQR (R*R-X*X))
532:LINE -(-X,Y-Z),B-50
534:NEXT I
537:Z=Z+I
540:IF Z)=CGOTO 585
543:LINE -(0,K-2-Z),9
545:Z=Z+I
548:IF Z)=CGOTO 585
550:LINE -(-INT (A/2)+I,K),9
553:FOR I=R-INT (A/2)+ITO R+INT (A/2
    )+JSTEP J
555:X=INT (R-I)
558:Y=INT (SQR (R*R-X*X))
560:LINE -(-X,Y-Z),B-50
565:NEXT I
568:Z=Z+I
570:IF Z)=CGOTO 585
575:LINE -(0,K-2-Z),9
578:Z=Z+I
580:IF Z<CGOTO 524
585:GLCURSOR (0,K-C):SQR6N
590:IF (Q$="TCP+C/D")OR (Q$="TCP+S+C
   /D")GOSUB 1300
591:IF (Q$="ROU+C/D")OR (Q$="ROU+S+C
   /D")OR (Q$="COU")GOSUB 1300
595:GOTO 700
600:REM
602:REM ****************************
603:REM                            *
604:REM         CONCAVE FIELD      *
605:REM                            *
606:REM ****************************
607:REM
610:I=10,Z=0,S=B-60
613:IF S)0GOTO 615
614:I=2
615:K=INT (SQR (R*R-A/2*A/2))
620:GRAPH
622:GLCURSOR (J10,K):SQR6N
624:LINE -(-INT (A/2)-I,-K),9
626:FOR I=R-INT (A/2)-ITO R+INT (A/2
    )-JSTEP J
628:X=INT (R-I)
630:Y=INT (SQR (R*R-X*X))
632:LINE -(-X,-(Y+Z)),B-60
634:NEXT I
637:Z=Z+I
640:IF Z)=CGOTO 685
643:LINE -(0,-(K+2+Z)),9
645:Z=Z+I
648:IF Z)=CGOTO 685
650:LINE -(-INT (A/2)+I,-K),9
653:FOR I=R-INT (A/2)+ITO R+INT (A/2
    )+JSTEP J
655:X=INT (R-I)
658:Y=INT (SQR (R*R-X*X))
660:LINE -(-X,-(Y+Z)),B-60
665:NEXT I
668:Z=Z+I
670:IF Z)=CGOTO 685
675:LINE -(0,-(K+2+Z)),9
678:Z=Z+I
680:IF Z<CGOTO 624
685:GLCURSOR (0,-(K+C)):SQR6N
690:IF (Q$="TCP+C/D")OR (Q$="TCP+S+C
   /D")GOSUB 1300
691:IF (Q$="ROU+C/D")OR (Q$="ROU+S+C
   /D")OR (Q$="COU")GOSUB 1300
695:GOTO 700
700:REM
702:REM ****************************
703:REM                            *
704:REM          INTERBAND         *
705:REM                            *
706:REM ****************************
707:REM
710:IF D<0RETURN
720:IF D<0GOTO 800
730:GRAPH
740:LINE (0,0)-(0,-D),9
750:TEXT
760:IF (Q$="TCP+C/D")OR (Q$="TCP+S+C
   /D")GOSUB 1400
761:IF (Q$="ROU+C/D")OR (Q$="ROU+S+C
   /D")OR (Q$="COU")GOSUB 1400
770:RETURN
800:REM
802:REM ****************************
803:REM                            *
804:REM           PUFF             *
805:REM                            *
806:REM ****************************
807:REM
810:Z=0,I=0
820:FOR I=1TO (-D/2)+0.5
825:GRAPH
830:LINE (0,-2)-(0,-2),9
835:LINE (J09-INT (E(I)/2),-I)-(J1)-
    INT (E(I)/2),-I),0
840:LINE (J09+INT (E(I)/2),-I)-(J1)+
    INT (E(I)/2),-I),0
845:LINE (0,-1)-(0,-1),9
850:Z=Z+I
855:IF Z)=ABS (D)GOTO 880
860:LINE (0,-2)-(0,-2),9
865:Z=Z+I
870:IF Z)=ABS (D)GOTO 880
875:NEXT I
880:TEXT
885:GOTO 750
1200:REM
1202:REM ****************************
1203:REM                            *
1204:REM          LABELING          *
1205:REM                            *
1206:REM ****************************
1207:REM
1210:IF W=0RETURN
1211:GRAPH
1215:IF W=0GOTO 1225
1216:IF W=1GOTO 1235
1217:IF W)1GOTO 1245
1225:LINE (0,I)-(100-INT (A/2),I),0
1230:IF W<36GOTO 1270
1235:LINE (0,I)-(100-INT (A/2),I),1
1240:IF W<36GOTO 1270
1245:LINE (12,I)-(100-INT (A/2),I),
     3
1250:TEXT
1260:RETURN
1270:IF U$="NO"GOTO 1290
1273:CSIZE 1
1275:LINE (0,0)-(0,-3),9
1277:ROTATE 1
1280:LPRINT U$
```

Table 1 (contin.)

```
1283:ROTATE 0
1285:LINE (0,0)-(0,0),9
1287:'EXT
1290:RETURN
1300:REM
1302:REM ****************************
1303:REM                            *
1304:REM      C/D SCALE             *
1305:REM                            *
1306:REM ****************************
1307:REM
1310:Z=0
1315:CRAPH
1320:LINE (0,0)-(0,C),9
1325:CRAPH
1330:GLCURSOR (200,-1):SORGN
1335:LINE -(20,0),0
1340:Z=Z+1
1345:'F Z>=CRETURN
1350:GLCURSOR (0,-1):SORGN
1355:LINE -(0,0),9
1360:Z=Z+1
1365:'F Z>=CRETURN
1370:GLCURSOR (0,-1):SORGN
1375:LINE -(10,0),0
1380:Z=Z+1
1385:'F Z>=CRETURN
1390:GOTO 1350
1400:Z=0
1410:CRAPH
1420:LINE (0,0)-(0,ABS (D)),9
1425:CRAPH
1430:GLCURSOR (135,-1):SORGN
1435:LINE -(15,0),0
1440:Z=Z+1
1445:'F Z>=ABS (D)RETURN
1447:GLCURSOR (5,0):SORGN
1450:GLCURSOR (0,-1):SORGN
1455:LINE -(0,0),9
1460:Z=Z+1
1465:'F Z>=ABS (D)RETURN
1470:GLCURSOR (0,-1):SORGN
1475:LINE -(10,0),0
1480:Z=Z+1
1485:'F Z>=ABS (D)RETURN
1490:GOTO 1450
1500:REM
1502:REM ****************************
1503:REM                            *
1504:REM   ORDERS FOR CORRECTING    *
1505:REM   MISTAKES IN INPUTS       *
1506:REM                            *
1507:REM ****************************
1508:REM
1510:BEEP ON
1511:BEEP 10,20,1000
1515:PRINT "CHANGE A : SMALLER A"
1516:GOTO 55
1520:BEEP ON
1521:BEEP 10,20,1000
1525:PRINT "CHANGE B : LARGER B"
1526:GOTO 60
1530:BEEP ON
1531:BEEP 10,20,1000
1535:PRINT "CHANGE B : SMALLER B"
1536:GOTO 60
1540:BEEP ON
1541:BEEP 10,20,1000
1545:PRINT "CHANGE R : LARGER R
1546:GOTO 30
1550:BEEP ON
1551:BEEP 10,20,1000
1555:PRINT "CHANGE W : SMALLER W"
1556:GOTO 100
1560:BEEP ON
1561:BEEP 10,20,1000
1565:PRINT "CHANGE E(*): SMALLER E(*
     )
1566:GOTO 1618
1570:BEEP ON
1571:BEEP 10,20,1000
1575:PRINT "END OF PROGRAM"
1576:END
1580:BEEP ON
1581:BEEP 10,20,1000
1585:PRINT "CHANGE INF. PREVIOUS BA
     ND"
1586:GOTO 2015
1600:REM
1602:REM ****************************
1603:REM                            *
1604:REM       INPUTS FOR PUFFS     *
1605:REM                            *
1606:REM ****************************
1607:REM
1608:REM
1610:WAIT 60:Z=0,1=0
1615:'F -D/2>)INT (-D/2)GOTO 1630
1620:FOR I=1TO -D/2
1630:'NPUT "A FOR PUFF-OUTLINES = "
     :E(I)
1635:'F E(I)>160GOTO 1560
1640:Z=Z+2
1650:PRINT "Z = ";Z/2;"REST = "; -D/
     2-Z/2
1660:NEXT I
1670:RETURN
1690:FOR I=1TO -D/2+0.5
1700:'NPUT "A FOR PUFF-OUTLINES = "
     :E(I)
1705:'F E(I)>150GOTO 1560
1710:Z=Z+2
1720:PRINT "Z = ";Z/2;"REST = "; -D/
     2+0.5-Z/2
1730:NEXT I
1740:RETURN
2000:REM
2002:REM ****************************
2003:REM                            *
2004:REM    INPUTS FOR FINDING      *
2005:REM    THE RELEVANT BAND       *
2006:REM                            *
2007:REM ****************************
2008:REM
2010:PRINT "SEARCHING PREVIOUS BAND
     "
2015:'NPUT "PROGRAM LINE,INPUT I =
     ";F
2020:'NPUT #I,A,B,C,D,R,W,U$
2025:'F D>=0GOTO 2040
2030:'NPUT #I,B,D,E(*)
2040:'F F>I GOTO 2020
2050:WAIT 250:PRINT I;A;B;C;D;R;W;U
     $
2060:'F D>=0GOTO 2070
2065:PRINT I;B;D;E(I);E(-D/2+0.5)
2070:'NPUT "CORRECT VARIABLES ? Y/N
     = ";M$
2075:'F M$="N"GOTO 3100
2080:M$="0"
2100:REM
2102:REM ****************************
2103:REM                            *
2104:REM    INPUTS FOR              *
2105:REM    CORRECTING VARIABLES    *
2106:REM                            *
2107:REM ****************************
2108:REM
2110:A=0,B=0,C=0,D=0,R=0,W=0
2111:E=0,U$=" D ",Q$="0",I=0
2112:FOR J=1TO 30
2113:E(J)=0
2114:NEXT J
2120:PRINT "INPUTS FOR FOLLOWING BA
     ND"
2130:GOTO 50
3000:REM
3002:REM ****************************
3003:REM                            *
3004:REM    INPUTS FOR TAPE         *
3005:REM    CONTROLLED PLOTTING     *
3006:REM                            *
3007:REM ****************************
3008:REM
3010:I=0
3030:'NPUT "FIRST BAND = ";F
3040:'NPUT "LAST BAND = ";L
3050:'F F>1GOTO 3070
3060:I=I+1
3070:'F I>=L+1GOTO 1570
3080:'NPUT #I,A,B,C,D,R,W,U$
3085:'F D>=0GOTO 3100
3090:'NPUT #I,B,D,E(*)
3100:'F F>I GOTO 3060
3110:GOSUB 148
3120:GOTO 3060
```

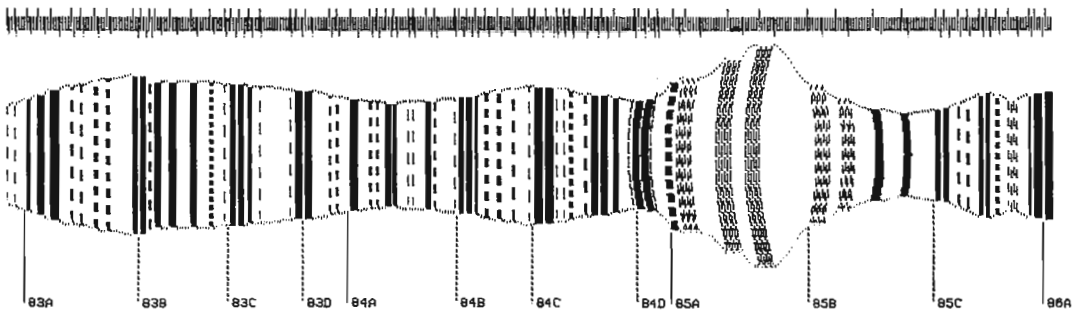


Figure 1. Computerized EM chromosome map of region 83A-85C in D.hydei. The map is based on measurements of cytological data (band diameters, band thicknesses and interband lengths) from several EM micrographs of SSP chromosomes.

Roca, A. and J.Rubio. Universidad de Oviedo, Espana. Breakage of polytene chromosomes of D.virilis under genetic control.

Some individuals from a stock of D.virilis kept in our laboratory since 1975, were found to experience abundant and variable breakage of their salivary gland polytene chromosomes when treated in the usual preparatory way for observation, that is dissection in aceto-lactic acid, followed by staining the glands in a drop of 2% aceto-lactic orcein on a slide for 30 min, and squashing the glands covered by a cover-slide (by observation under a stereomicroscope we can control, in the squash, the degree of extension of the chromosomal arms).

The breakage takes place even at normal squashing pressure apparently as a result of the squashing since a direct relationship between the pressure applied and the amount of chromosome fragmentation is observed. The chromocenter region is always the first to break up; then, the oligotenic points of the chromosomes (α-heterochromatin) are broken with increasing pressure, indicating a higher frailty than the rest of the chromosome. A typical view after a minimal pressure upon the stained glands would be to have all 5 chromosomes disjointed from each other at the chromocentric region and their arms broken at the following points: chromosome 1 at point P-Q; chromosome 2 at Mc and chromosome 4 at F-G (notation as in HSU maps, Patterson & Stone 1952); the chromosomes 3 and 5 are usually found without breakage when squashed lightly. When the squashing pressure increases to normal (as necessary for a good extension of the chromosomes), more breakages occur at various sites, so that eventually many chromosome fragments of variable size appear and the breakage points seem to be random (Figure 1).

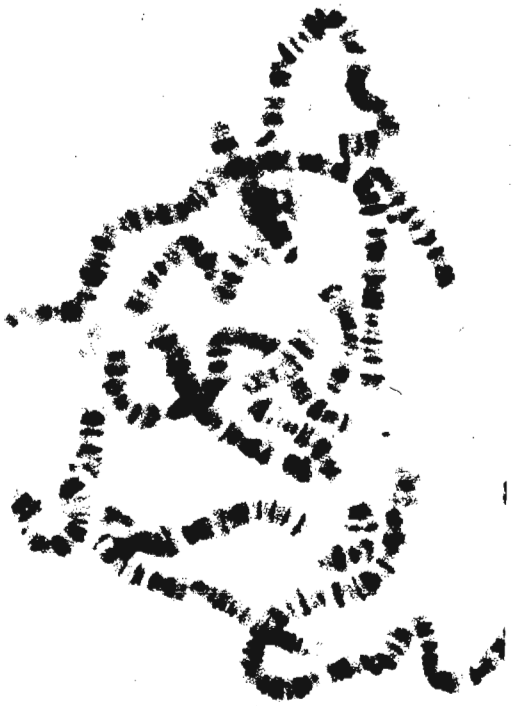


Figure 1. Frail chromosomes.

Taking off components from the preparatory solution (orcein or acetic or lactic acid) does not improve the stability of the polytene chromosomes to squashing pressure, nor change the pattern of breakage. It was not possible to make preparations without aceitc and lactic acids (only in distilled water) nor in saline solution because no nuclear membrane disruption is obtained under these conditions, probably due to the lack of elasticity that the acetic acid (Lefevre 1976) and the lactic acid (unpubl.) confer to the membrane and the chromosomes. The use of detergenets did not show any results, because they do not break the nuclear membranes.

All these results move us to consider that although the squash is the main cause of chromosomal breakage, the preparatory solutions can not be considered as related to the breakage phenomenon.

A true-breeding stock for such polytene chromosome frailty was established, where the adults are otherwise phenotypically normal and without any signs of their viability or fertility being affected. Reciprocal crosses to a normal stock of D.virilis from different origins yield progenies segregating as expected for a recessive sex-linked allele with 100% penetrance (Table 1).

We called "fra" to the allele responsible for the chromosome frailty.

Closer examination of the banding in heterozygous individuals under the optic microscope does not reveal any difference between the two homologous endocopy fasces. Even though it is indubitable that the structure of the polytene chromosomes of individuals with chromosome frailty is different than that of the normal individuals in the species. How the chromosome fine structure might differ remains to be seen.

Table 1. Progenies of the reciprocal crosses.

PARENTS:	♀♀ frail chrom.;	♂♂ normal chrom.
1st GENERATION:	♀♀ all normal;	♂♂ all frail
2nd GENERATION:	♀♀ normal (22);	♀♀ frail (27)
	♂♂ normal (14);	♂♂ frail (17)
PARENTS:	♀♀ normal chrom.;	♂♂ frail chrom.
1st GENERATION:	♀♀ all normal;	♂♂ all normal
2nd GENERATION:	♀♀ normal (45);	♀♀ frail (0)
	♂♂ normal (15);	♂♂ frail (19)

References: Lefevre,Jr, G. 1976, Genetics and Biology of Drosophila, V1a, Academic Press Inc., London; Patterson,J.T. & W.S.Stone 1952, Evolution in the genus Drosophila, Macmillan Co., New York.

Sampsell, B.[1] and B.Latham.[2] 1-Roswell Park Memorial Institute, Buffalo, New York USNA. 2-Chicago State University, Illinois USNA. Survival of ADH thermostability variants on naturally-occurring alcohols.

Wild Drosophila melanogaster probably feed and breed on a variety of fermenting fruits and other substances derived from human activities. Examination of the alcohol content of several rotting fruits showed that ethanol was the most common alcohol present with concentrations ranging up to 4%, while 1-propanol and 2-propanol occurred in concentrations of 1% or usually less (McKechnie & Morgan 1982). Ethanol concentrations as high as 12% were observed in certain portions of a pile of grape residues outside a winery (McKenzie & McKechnie 1979). We have measured the ability of flies with various Adh genotypes to survive on several alcohols presented singly and in combination over a range of concentrations.

Alcohol tolerance was measured using a modification of the method of Starmer et al. (1977). Newly-emerging adults were collected from uncrowded breeding vials and transferred to fresh food vials to age for 7 days. The flies were then lightly etherized and groups of 20 males or females selected. They were returned to an empty vial for 4 hours to recover from the ether. They were then transferred to a 35 ml food vial, empty except for a 1-inch square of filter paper on which 0.1 ml of an alcohol solution of the indicated concentration (v/v) had been absorbed. From this point on in the experiment, this alcohol solution served as the sole source of nourishment and moisture. It should also be noted that flies were exposed to the alcohol by inhalation as well as ingestion. The vials were covered with parafilm, and observations were made at regular intervals to count the number of dead flies. After more than 50% of the flies in a given vial had succumbed, the number of hours to 50% mortality was determined by interpolation between observed points. Each point in the graphs represents the average of 6 vials (3 of males and 3 of females). Females generally lived somewhat longer than males; a fact that is probably the result of their larger body size and hence greater store of food. Three strains of flies, each homozygous for one of the three common alleles, were tested. The strains were Sf 1 (Adh-Fr), Ore (Adh-Fm), and SSS (Adh-Sm). Information about these strains may be found in Sampsell & Steward (1983). All tests were conducted at room temperature.

As previously reported, certain alcohols can serve as nutrients for the flies prolonging their life beyond that possible on plain water. In the case of ethanol, survival peaks occurred at 4-8%; however, survival time decreased sharply at concentrations above this level. On 1-propanol, the survival peak was seen at much lower concentrations of only 0.5 to 1%. Either this alcohol offers less nutritive benefit than ethanol or higher concentrations are more toxic. Survival on a combination of the two alcohols suggested that the latter explanation is probably correct. When ethanol and 1-propanol were both present in a 10:1 ratio, survival times peaked in the 2-4% ethanol range. 2-propanol was apparently even more toxic, since on this alcohol alone, flies did not survive longer than on plain water. Presented in combination with ethanol, 2-propanol's toxic effect was again apparent from the fact that peak survival occurred at around 4% ethanol. Finally when all three alcohols were present (with ethanol concentrations 10X that of either of the other two alcohols) a peak in hours to 50% mortality occurred at 2% for all three strains. The most interesting thing about these findings is the fact that the alcohol concentrations at which longest survival times were observed are very similar to those observed in natural food sources.

Comparisons among the strains for differential survival times were hampered by the small sample sizes, but several conclusions can be drawn. Based on measurements of ADH activity levels in which Ore > Sf 1 > SSS, we might have expected survival times to parallel these differences. In fact, SSS (Adh-Sm) flies generally had shorter survival times when there were any significant differences among the strains. Sf 1 (Adh-Fr) survival times, however, were generally equal to or longer than those of Ore (Adh-Fm). The Adh-Fr allele occurs in most natural populations, but only at low frequencies (1-16%). Flies with this allele, however, do not seem to have impaired alcohol tolerance compared to individuals with

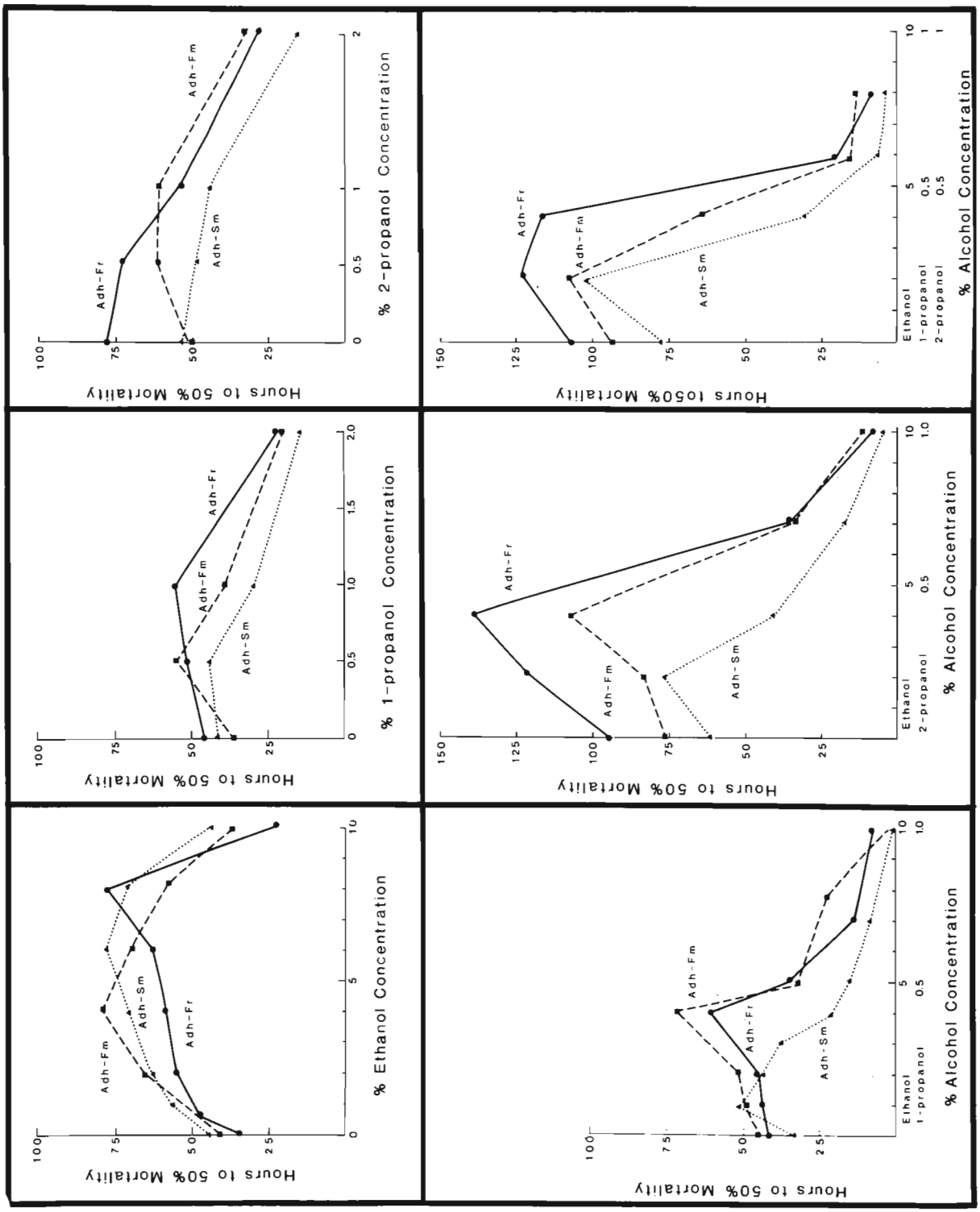


Figure 1. Survival of flies with different Adh genotypes on various combinations of naturally-occurring alcohols.

either of the more common forms, at least under the conditions tested. Explanation of the frequency of this ubiquitous, but uncommon, allele awaits further experimentation. (Supported by NIH Grant RR-08043-10)

References: McKechnie,S.W. & P.Morgan 1982, Aust.J.Biol.Sci. 35:85-93; McKenzie,J.A. & S.W.McKechnie 1979, Oecologia 40:299-309; Sampsell,B. & E.Steward 1983, Biochem.Genet. 21: 1071-1088; Starmer,W.T., W.B.Heed & E.S.Rockwood-Sluss 1977, Proc.Natl.Acad.Sci.USA 74:387-391.

San Miguel Salán, E. Universidad de León, España. Developmental time in four populations of formaldehyde-treated Drosophila melanogaster.

Formaldehyde is a toxic, contaminant, and mutagenic environmental agent (Auerbach 1967, 1971; Perera 1982). Several samples of eggs from four populations of D.melanogaster underwent toxification by the larval feeding method. The populations were caught on different sites from Asturias (Spain) with clear geographic and ecological diversities. They were maintained in the laboratory by mass culturing. Two types of culture media were used: control medium (basic yeast and sucrose), and treated medium (0.2% v/v formaldehyde in control medium). Ten ml of culture medium were poured into each vial (10 x 2.5cm ø).

Four day old virgins of both sexes from each population were put in plastic cylindrical cages (3.60 x 7.80cm ø) where females laid eggs for 24 hr on control medium. The eggs were transferred into vials (50 per vial). The experiment was performed at 21±1°C.

The developmental time was determined from egg-to-adult (in days). Results presented in Table 1 indicate the mean values of each sample for males and females together, and also the standard errors (X^{+}-S.E.). In parentheses are given the number of vials. Naranco, Felguera and Urbana population samples that were treated show a vial amount less than their respective control samples. This is due to 100% lethality found in some vials, which were excluded from the analysis.

The main effect of the treatment is the extreme increase in developmental time, as derived from the differences between the mean values of treated and control samples.

On the other hand (see Table 2), there are significant differences (at 5% level) between control samples and also for treated samples, as shown by the SNK test (Sokal 1979). Populations appear in increasing order of developmental time. Underlined populations show no significant differences at 5% level. Ms, mean square; Df, degrees freedom.

Table 1.

	Populations			
Samples	TEVERGA	NARANCO	FELGUERA	URBANA
Control	11.79±0.11 (10)	13.65±0.07 (20)	13.83±0.06 (20)	14.25±0.09 (20)
Treated	27.90±0.69 (10)	30.20±0.44 (16)	31.80±0.40 (11)	31.54±0.40 (12)

Table 2. Results by the SNK test.

Samples	Populations	Ms	Df
Control	TEVERGA < NARANCO FELGUERA < URBANA	0.10	66
Treated	TEVERGA < NARANCO URBANA FELGUERA	2.85	46

According to this, the estimation of the relative decrease in development (by the differences between mean values of treated and control samples with reference to this latter $[(\bar{X}_t-\bar{X}_c)/\bar{X}_c]$ was used to determine which populations were the most sensitive to the toxic. The relative decrease for the Teverga, Felguera, Naranco and Urbana populations were 1.37, 1.30, 1.21 and 1.21, respectively, showing that Teverga population was the most sensitive.

A remarkable aspect of this work is the phenotypic variability of the analyzed trait. The coefficient of variation gave an average value of 2.5% for the four control samples, and for the treated samples this coefficient was 5.55%. If the wide differences in developmental time by effect of the contaminant are basically determined by genotypic differences, the possibility exists of quick responses to selection. Further studies are under way to elucidate this point.

References: Auerbach 1967, Science 158:1141-1147; Auerbach & Kilbey 1971, Ann.Rev. Genet. 5:187; Perera 1982, Science 216:1285-1291; Sokal 1979, Biometria, H.Blume, Madrid.

San Miguel Salán, E. Universidad de León, España. Male sterility in four populations of formaldehyde-treated Drosophila melanogaster.

Mutagenic properties of formaldehyde have been known for several years (Slizynska 1957; Auerbach & Kilbey 1971). Its relevance as environmental toxic has been emphasized recently (Perera & Petito 1982). On the other hand, their effects on the partial components of fitness are not yet well understood. In this communication I wish to report the effects of this toxic on the male sterility of D.melanogaster.

Samples of four populations of D.melanogaster were treated with the toxin by the larval feeding method. Populations, experimental treatment, etc., have been described in the previous Research Note (this issue).

Males developed in control medium, and the survivors at 0.2% (v/v) formaldehyde-treated populations were mated invididually with two virgin females of the Cyl/Pm stock, into vials (10 x 2.5cm) containing 10 ml of control medium. The individuals from each vial were kept together for 11 days at 21°C, after which they were removed, and the vials without any emerging adults were scored.

Table 1 indicates the total number of males analyzed (t), and the number of sterile males (s) with their respective percentages (%s). The results suggest that all populations were sensitive to the toxic, at least variably, as shown by the higher sterility percentage in relation to the control populations. More extensive experiments are under way to establish more definitive conclusions.

Table 1.

	Populations							
	TEVERGA		FELGUERA		URBANA		NARANCO	
Samples	s/t	%s	s/t	%s	s/t	%s	s/t	%s
Control	0/50	0.00	1/80	1.25	1/80	1.25	2/80	2.50
Treated	3/37	8.10	5/44	11.36	8/49	16.32	11/80	13.75

References: Auerbach & Kilbey 1971, Ann.Rev.Genet 5:168-187; Perera & Petito 1982, Science 216:1285-1291; San Miguel 1984, DIS 60(in press); Slizynska 1957, Proc.Roy.Soc. Edinburgh 666:284-304.

Sanchez, J.A. and G.Blanco. Universidad de Oviedo, Asturias, Espana. The relationship between variance in rate of development and Adh genotypes in Drosophila melanogaster.

Lerner (1954) has argued that more heterozygous individuals should be characterized by increased developmental stability. Recent reports show evidence for this hypothesis. The more heterozygous populations of different species (lizard, Soule 1979; freshwater bivalves, Kat 1982; killfish, Mitton 1978; monarch butterfly, Eanes 1978; etc.) have lower amounts of fluctuating asymetry and variance for morphological trait. Mitton (1978) retorted that the results are surprising because on the basis of genetic variation of a single locus, a population can be subdivided into two groups that differ in their levels of morphometric variation.

The present paper aims to examine the relationship in D.melanogaster between heterozygosity at an enzyme locus (ADH) and variance of a quantitative trait directly related with the fitness (rate of development).

In this work, the progeny of individuals heterozygous for the Adh locus were classified according to their genotype, sex and rate of development. Two experiments were performed. In experiment A heterozygous flies were obtained from crosses between female F/F and male S/S; and in experiment B from reciprocal crosses. As there are no significant differences in rate of development between the sexes, we combined the data from the sexes in our analysis. Within each genotype (F/F, S/S or F/F) we estimated the phenotypic variation of the character (rate of development) using the variance and the coefficient of variation. The null hypothesis tested here is that individuals heterozygous have the same level of variation as individuals homozygous.

The results show that heterozygous have lower variance and coefficient of variation than both types of homozygotes (Table 1); and in three of the four comparisons these differences are statistically significant (Table 2). Both types of homozygotes have the same levels of variation (Table 1) and there are no significant differences between them (Table 2).

Table 1. Variance and coefficient of variation for the three genotypes in the two experiments performed. σ^2 = variance; n = sample size; c.b.= coefficient of variation.

genotype	F/S	F/F	S/S	Pool of homozygous (F/F + S/S)	
σ^2	1.9185	2.1582	2.2415	2.2063	Experiment A
c.v.	0.0853	0.0914	0.0917	0.0918	
n	817	432	344	776	
σ^2	2.3840	3.0184	3.2675	3.1365	Experiment B
c.v.	0.0928	0.1033	0.1064	0.1048	
n	582	285	255	540	

Table 2. F values in the analysis of variance.

Comparisons	F/S vs F/F	F/S vs S/S	F/S vs Pool homo. (F/F+S/S)	F/F vs S/S
Experiment A	1.1249 n.s.	1.1683 *	1.15 *	1.0385 n.s.
Experiment B	1.2761 * *	1.3705 * *	1.3156 * *	1.0825 n.s.

n.s.=not significant; * = P < 5%; * * = P < 1%.

The differences in level of variation between heterozygous and both types of homozygous and between heterozygous and homozygous combined (F/F+S/S) (Tables 1 and 2), are consistent with observations compiled by Lerner and the other authors previously cited, and reject the null hypothesis tested here.

No significant difference in variance between F/F and S/S homozygous type were found, thus indicating that the level of variation of the homozygous individuals is independent of the particular allele for which these individuals are homozygote.

References: Eanes,W.F. 1978, Nature 276:263-264; Kat,P.W. 1982, Am.Nat. 119:824-832; Lerner,I.M. 1954, In: Genetic Homeostasis (Oliver & Boyel, Edinburgh); Mitton,J.B. 1978, Nature 273:661-662; Soule,M. 1979, Evolution 33:396-401.

Sato, T. Kansas State University, Manhattan, Kansas USNA. A new homoeotic mutation affecting antennae and legs.

We have isolated a new homeotic mutation which arose spontaneously in the stock of $T(1;3)bxd^{111}/TM1$ (Lewis 1981); for reasons described below, the variant is denoted Bristle on arista of Manhattan (symbolized Ba^m). Preliminary experiments showed it to be a second chromosomal, recessive variant. Male and female homozygotes are viable and fertile, and they show partial transformation of antennae to legs, as well as deletion of some leg structures. Tarsal tissue sometimes including claws develops in place of arista and part of the third segment of antenna (Fig. 1). The third antennal segment usually resembles a patchwork of incompletely differentiated leg cuticle, whereas the first and second segments of antenna are unaffected.

For legs, the region affected is restricted to the distal part and seems to be common in all legs (Fig. 2). Abnormal bristle patterns including reversed polarity appear around segmental boundary between tibia and basitarsus in less extreme cases. This segmental boundary is often very incomplete and accompanied by extrusion of supernumerary cuticles (Fig. 2G and H). In more extreme cases, deletion of distal tibia and whole basitarsus is revealed by missing or reduction of numbers of bristles typical to these parts, e.g., transverse rows on prothoracic leg, apical bristle on mesothoracic leg and preapical bristles on all legs for tibia as well as transverse rows on pro- and metathoracic legs for basitarsus (Fig. 2D, E, F). Frequently in these cases, the number of tarsal segments is also reduced to two or three and

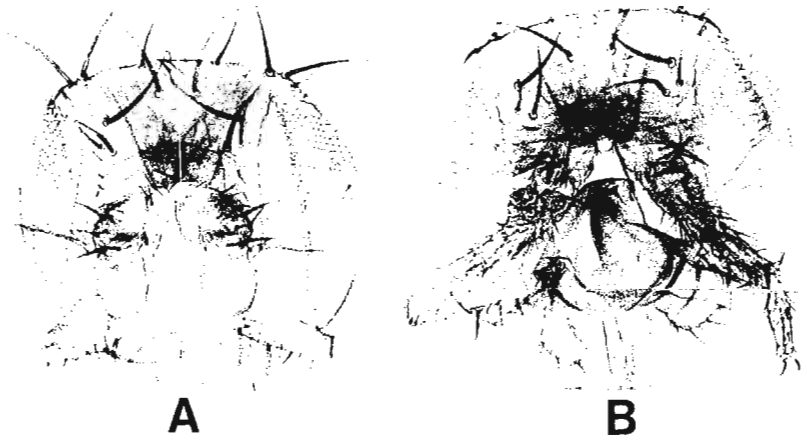


Fig. 1. Transformation of arista to tarsi. Female flies were fixed in three parts 70% ethanol and one part glycerol, and internal tissues were dissolved by heating in 10% KOH solution. After washing in water and then in n-propanol, cuticles of head were dissected and mounted in Euparal.

A, Wild-type (Canton S).
B, Ba^m homozygote.

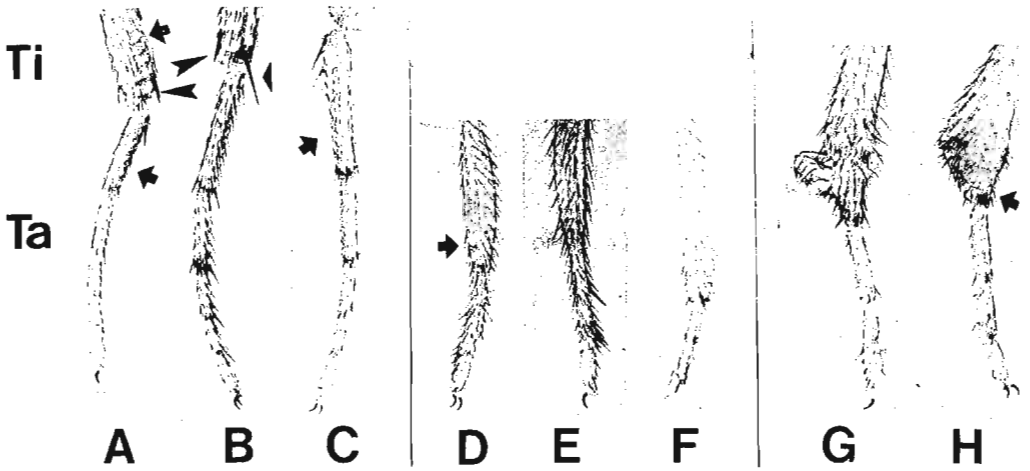


Fig. 2. Deletion in legs. Cuticles of pro-(A,D,G), meso- (B,E), and metathoracic (C,F,H) legs of female flies were prepared and mounted as described in Fig. 1. Distal portion of tibia (Ti) and tarsi (Ta) are shown in this figure. Note the characteristics of each leg; transverse rows (⬆), preapical (➤) and apical (◗) bristles. A-C, Wild-type (Canton S). D-H, Ba^m homozygote.

tarsal segmental boundaries are very obscure. In the most extreme cases, claws are missing. Thus, all tarsal segments can be affected by this mutation. However, proximal segments (trochanter, coxa and femur) and proximal tibia are unaffected. Although distal portion of legs are variably affected, a few transverse rows consistently remain on the distal tibia of the prothoracic leg in most cases, if not all, suggesting that there might be a defined limit of domain of action of this mutation near the distal tip of tibia. Expressivity is variable and significantly higher in females than in males. Penetrance is also dependent on the genetic background, but more than 90% under an optimum condition. The arista phenotype is more penetrant than leg phenotype, and the methathoracic legs show higher penetrance than other legs.

This new variant was mapped by balancing 818 2nd chromosomes transmitted by Ba^m/ix bw sp females. The genetic constitution of each chromosome was determined by subsequent outcrosses to flies bearing Sp L^{rm} Ba^m or ix bw sp chromosome. Ba^m was mapped to the distal right arm of the 2nd chromosome, 0.6 map unit (mu) to right of sp, based on four bw^+ sp^+ Ba^+ and one bw sp Ba^m recombinants. The observed map distances between ix and bw (37 mu) and bw and sp (4.4 mu) approximated standard values. We scored antennal and leg phenotypes independently, and there was no segregation between these phenotypes among 818 chromosomes examined. Therefore, Ba^m seems not to represent multiple mutations. In addition, Ba^m chromosome does not show any aberrancy in banding pattern of polytene chromosome. In trans heterozygotes, Ba^m is complemented by 3 distal deficiencies: Df(2R)Px (60B8-10;60D1-2), $Df(2R)Px^2$ (60C5-6;60D9-10), and $Df(2R)M\text{-}c^{33a}$ (60E2-3;60E11-12).

Sunkel (1983) has described dominant mutations at the Bristle on arista locus with phenotypes very similar to the variant described here. They are lethal when homozygous. He has mapped this locus 0.8 mu distally to sp, and within polytene chromosome region 60E1;2-4. Sunkel (1983) also reported that Ba^m/Ba^1 heterozygotes die as pharate adults with display of extreme malformations of antennae and legs. It appears, therefore, that Ba^m is a leaky mutation of that locus.

References: Lewis,E.B. 1981, v.23:189-208 in Developmental Biology Using Purified Genes (Brown & Fox eds), ICN-UCLA Symposia on Molecular and Cellular Biology, Academic Press, New York; Sunkel,C. 1983, Genetic and developmental analysis of the homeotic mutation Brista in Drosophila melanogaster, PhD thesis, University of Sussex.

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Drosophila of Black Hawk County, Iowa.

Drosophila have been collected from lowland forest, upland forest and sand prairie communities near Cedar Falls, Iowa. The lowland forest, dissected by a stream, consists primarily of box elder, black cherry, hackberry, black walnut, green ash and cork elm. The upland forest, situated on a river bluff dissected by numerous ravines which periodically contain water, consists of oak, hickory, basswood and maple. The rural sand prairie is a virgin mixed grass prairie traversed by a moist swale fringed by big bluestem and Indian grass with elevated xeric areas dominated by little bluestem.

The collection data are shown in Tables 1 (Lowland forest), 2 (Upland forest) and 3 (Sand prairie). All 1982 collections were made by netting flies attracted to buckets containing banana bait. In 1983 all collections were secured in traps baited with bananas (Heim 1978). For three of the species (D.affinis, D.algonquin and D. athabasca) the males can be readily distinguished but distinguishing the females is very difficult. If we only collected males of one of the three species, the females were assumed to be conspecifics; otherwise females were not separated as to species.

The species compositions in our collections from the three communities are compared in Table 4. D.affinis is the most abundant species in all three communities while D.falleni and D.putrida are common in all three. D.robusta is common in two and present in all three. D.tripunctata, although common in the lowland, is apparently absent from the other two communities. This species is most abundant in late summer and fall and may have been excluded from upland forest collections since that community was sampled much earlier in the year. We will sample the upland forest more extensively in the future and thus

Table 2. Upland forest community.

1982:	May		June		July	
Species	♂	♀	♂	♀	♂	♀
D.affinis	10*		103*		112	70
D.falleni	0	0	18*		76	68
D.robusta	0	0	0	0	43	65
D.putrida	0	0	28*		12*	
D.melanogaster	3*		0	0	0	0
Total collections	1		2		2	

* = not sexed.

Table 3. Sand prairie community. Note that a collection during April 1983 yielded no flies. (* = not sexed)

	1982		1983:									
	June		May		June		July		Aug.		Sep.	
Species	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
D.affinis	56	21	0	2	3	7	2	12	2	14	0	0
D.algonquin	0	0	0		0	0	2		0	0	5	6
D.falleni	5*											
	21	0	0	0	0	0	0	1	0	0	0	0
D.putrida	16*		0	0	0	0	1	2	0	0	1	2
D.quinaria	0	0	0	3	0	0	0	0	0	0	0	0
D.buskii	0	0	1	0	0	0	0	0	0	0	0	0
D.robusta	0	0	0	0	0	0	0	0	0	0	2	0
Total collections	1		3		2		2		1		2	

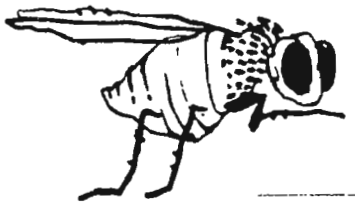

Table 1. Lowland forest community.

Species	1982												1983											
	May		June		July		Aug.		Sept.		Oct.		Nov.		Apr.		May		June		July		Aug.		Sept.		Nov.	
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
D. affinis	619*		564	91	348	194	0	0	5		1		2		0	0	28		332		320	591	14	23	40		6	7
D. algonquin	0	0	0	0	0	0	0	0	0	8	0	7	2	8	0	0	5	17	31	680	0	0	0	0	3	34	0	0
D. athabasca	0	0	0	0	0	0	3	2	15		2		3		0	0	0	0	0	0	0	0	0	0	0	0	0	0
D. falleni	87*		249*		100* 12	9	1	0	5	17	12	12	0	3	0	0	0	1	1	4	7	20	0	1	2	5	0	0
D. tripunctata	0	0	20*		3* 31	13	9	11	62	43	41	17	19	11	0	0	0	0	2	2	9	13	6	1	146	56	12	5
D. robusta	1*		8* 4	21	45	80	1	0	0	2	1	7	0	1	6	5	3	2	2	1	9	9	4	3	24	36	3	6
D. putrida	0	0	13*		29*		0	1	0	0	9	7	1	5	0	0	9	8	7	10	19	15	0	1	35	27	0	0
D. quinaria	0	0	0	0	0	0	0	0	0	0	2	6	0	0	0	0	0	1	1	0	2	0	0	0	2	6	0	0
D. buskii	0	0	0	0	0	0	0	0	0	0	0	1	0	1	2	0	0	0	6	15	1	1	0	0	0	0	3	0
D melanogaster	0	0	0	0	0	0	0	0	0	0	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
D. immigrans	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	3	2	2	0
D. duncani	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	8	0	2	0	0	0	0	0	0
D. testeca	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	4	5	0	0	1	0	0	0
D. victoria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	3	1	0	0	0	0	1	0	0
Total collections	5		3		4		1		5		4		1		1		3		2		2		1		2		1	

NOTE: A collection during October, 1983, yielded no flies.
* = not sexed

Table 4. Species % composition by community.

Species	Lowland forest	Upland forest	Sand Prairie
D.affinis	56.3	48.5	56.1
D.algonquin	0.7	0.0	7.0
D.athabasca	0.4	0.0	0.0
Undetermined aff.-alg.-ath.♀♀	13.3	0.0	7.5
D.falleni	9.7	26.6	14.4
D.tripunctata	9.4	0.0	0.0
D.robusta	5.0	17.8	1.1
D.putrida	3.5	6.6	11.8
D.buskii	0.5	0.0	0.5
D.quinaria	0.4	0.0	1.6
D.duncani	0.2	0.0	0.0
D.testeca	0.2	0.0	0.0
D.immigrans	0.1	0.0	0.0
D.victoria	0.1	0.0	0.0
D.melanogaster	0.1	0.5	0.0
Total collected	5657	608	187

allow a more accurate comparison of the species compositions of the two forest communities.

Few members of the species commonly associated with humans (D.buskii, D.immigrans and D.melanogaster) were collected. We conclude that our samples represent natural Drosophila populations and not human-associated ones.

We thank E. Pilkington for his help with the early collections and L. Throckmorton, M. Wheeler and A.C. Haman for help in keying out our specimens.

Reference: Heim,W.G. 1978, DIS 53:216.

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Partial inhibition of the effect of the mutant red malpighian tubules (red) by other eye colour mutations of Drosophila melanogaster.

A study of eye pigments and related metabolites in adult flies (9 days after emergence) of ten strains of double mutants of D.melanogaster has been carried out. All the strains carry two eye colour mutations, one of them being "red."

The separation of eye pigments and related metabolites in these strains was carried out by means of two-dimensional thin-layer chromatography (TLC) on cellulose plates, using as extraction solvent methanol-acetic acid-water (4:1:5 by vol) and as elution solvent isopropanol-2%-ammonium acetate (1:1, v/v) for first dimension (3 hr) and 3% aqueous ammonium chloride for the second one (50 min). Quantification was made by measuring the fluorescence directly on the plate with a fluorescence spectrophotometer (PERKIN ELMER MPF 44B). The results of the ten double mutants strains are compared with the patterns of the single mutants (Ferre et al. 1983) in Table 1.

Although the effect of mutant "red" consists in a strong reduction of pteridines (except biopterin), some double mutants such as cn red, rb red, and cm red and to a lesser extent ltd red, cl red and v red, present a significantly higher quantity of drosopterins, PDA and sepiapterin and a lower quantity of biopterin compared to the mutation red alone. This reduction may be interpreted as a partial inhibition of the effect of red mutant. Especially interesting is the case of cn red strain, since the gene cn^+ is known to be the structural gene of the enzyme Kynurenine hydroxylase and thus to affect only ommochrome synthesis.

The malpighian tubules of the wild type presents a light yellow colour produced by the accumulated 3-OH-kynurenine; however, the malpighian tubules of the mutant "red" are red-coloured (Oster 1954), due to the conversion of the accumulated 3-OH-kynurenine into ommochromes, of which a small quantity is xanthomatin and a larger quantity is ommin (Wessing & Bonse 1966). In addition this mutant, in the eyes, accumulates 31% of brown pigment (H_2-xanthomatin) and around 5% of drosopterins compared to the wild type (Ferre et al. 1983). Transport defects in malpighian tubules are the basis for the anomaly in some eye colour mutants of D.melanogaster that have reduced amounts of both pteridines and ommochrome (Sullivan et al. 1975; Howells et al. 1977; Sullivan et al. 1979). This fact strongly suggests that the gene red^+ is acting on the transport of pigments precursors, being unable to transport precursors of pteridines and ommochromes efficiently. For reasons not known it seems that certain mutations as cn, rb, cm, ltd, cl and v produce a partial inhibition of

Table I. Percentages of eye-pigments and related metabolites (Or-R has arbitrarily received the values of 100). NDP(Neodrosopterin), DP(Drosopterin),IDP(Isodrosopterin),ADP(Aurodrosopterin),PDA (2-amino-4-oxo-6-acetyl-7,8-dihydro-3H,9H-pyrimido[4,5-b][1-4]diazepine),SP(Sepiapterin), Spot 7(unidentified pteridine), AHP(Pterin), BP(Biopterin), IXP(Isoxanthopterin),XTC (Xanthurenic acid) and H_2-XTM (Dihydroxanthomatin). ND (not detected).

strain	NDP	DP	IDP	ADP	PDA	SP	Spot 7	AHP	BP	IXP	XTC	H_2-XTM
red	6±2	4±2	4±2	9±2	79±4	43±10	66±10	24±5	301±30	51±5	20±7	31±1
cn red	70±14	36±5	39±7	80±5	175±33	84±14	69±18	52±2	174±46	30±2	ND	--
rb red	41±10	16±2	9±1	32±5	145±26	128±30	101±9	22±2	323±42	26±3	37±4	--
cm red	49±10	25±2	21±1	59±3	255±4	161±7	98±9	35±1	232±15	24±1	ND	--
v red	12±1	6±2	7±2	35±2	169±27	47±5	61±8	29±3	122±3	23±2	ND	--
ltd red	13±2	9±1	9±1	40±6	160±14	89±12	76±5	23±4	153±3	17±2	ND	--
cl red	23±2	21±1	19±4	20±4	ND	1750±750	507±99	492±34	340±110	73±4	ND	--
pr red	7±2	5±0	2±1	7±2	20±2	19±5	30±5	22±1	128±15	38±2	16±3	--
pn red	5±2	3±0	2±1	4±2	TRACE	37±10	46±6	16±0	162±23	50±3	ND	--
dke red	4±1	5±1	6±1	12±1	104±1	14±4	71±2	21±1	215±20	22±1	24±5	--
cho red	TRACE	TRACE	TRACE	TRACE	33±4	39±1	61±3	25±2	308±28	35±1	ND	--
cn	91±4	86±8	89±2	91±4	84±16	101±14	80±7	95±13	82±6	63±5	ND	4±0
rb	34±2	25±3	36±3	55±3	94±12	162±7	50±5	37±4	149±14	29±2	7±2	38±1
cm	22±4	19±2	21±2	51±6	202±21	199±15	66±4	34±6	182±30	20±6	6±3	43±1
v	93±2	80±4	79±2	93±4	108±11	146±9	100±9	102±6	103±6	107±4	ND	3±0
ltd	32±1	26±3	25±4	58±2	203±8	140±23	114±6	26±7	165±31	30±8	ND	8±1
cl	19±1	54±1	47±1	8±3	ND	1442±46	474±35	526±40	261±19	117±5	59±3	160±2
pr	45±3	33±9	48±11	33±3	35±4	64±6	58±11	39±10	105±17	109±10	22±7	122±0
pn	22±4	22±2	32±4	18±2	87±17	161±14	95±6	34±2	140±5	88±5	43±6	81±2
dke	20±3	51±13	55±17	86±9	109±15	128±34	61±4	38±4	119±7	66±10	108±15	153±1
cho	4±1	4±1	4±1	11±2	47±3	108±9	125±2	47±11	268±43	58±9	TRACE	66±1

this effect, causing these flies to recover the ability to transport and accumulate this substances in the eyes.

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Additional evidence for cis-acting regulation of ADH activity.

Variation in levels of alcohol dehydrogenase (ADH) activity have been observed for many strains of Drosophila melanogaster (McDonald & Ayala 1978; Maroni et al. 1982; Sampsell & Steward 1983). Both genetic and epigenetic factors may contribute to this variation. Mutations at the structural locus have been shown to yield proteins with different specific activities, while changes in putative regulatory regions have been proposed to explain differences in quantity of ADH produced by strains with the same Adh genotype. Evidence for both closely-linked, cis-acting elements as well as unlinked, trans-acting regions has been reported. Here we offer additional evidence for the presence of cis-acting genes which appear to affect the quantity of slow ADH subunits produced in flies heterozygous for Adh-Fr and Adh-Sm.

The three strains (CS4, CS20, and CS19) were derived from wild flies collected in Chicago, Illinois. By the standard breeding schemes involving the Cy/Pm, D/Sb marker stock, each strain was made homozygous for a different wild second and third chromosome. All three strains are homozygous for Adh-Sm, and so far as can be determined from cellulose acetate electrophoresis, thermostability tests, and activity ratios (Sampsell 1977; Sampsell & Steward 1983) they code for identical ADH-Sm proteins. The three strains do show consistent and significant differences in ADH activity (for data in Table 1, $F_{strain} = 131$, $p<0.001$).

To test whether these activity differences were the consequence of cis-acting regulatory elements, we crossed each of the Adh-Sm strains to a variety of strains homozygous for an Adh-fast allele. Extracts from the heterozygous progeny were subjected to disc-gel electrophoresis according to the methods of Cooper (1977). The gels were stained for ADH using the standard nitro-blue tetrazolium stain and were scanned densitometrically to determine the proportion of ADH activity contributed by each of the dimers. Areas under the two peaks representing the NAD-bound and unbound forms of each dimer were combined. The relative band intensities (area of the scanned peaks) will be a function of the relative number of slow and fast subunits synthesized, the relative dimerizing ability of fast and slow subunits, and the relative specific activities of the various dimers. By making comparisons among heterozygotes with the same ADH subunits, the latter two factors (which are unknown) can be ignored. From the proportions of the three kinds of dimers the relative quantities of fast and slow subunits were determined (given as F/S ratio in Table 2). A one-way ANOVA of these ratios indicated significant differences among the progeny of the three different slow strains. For the crosses to Ric110, $F=6.56$, $p=0.02$; while for the crosses to Si44, $F=14.88$, $p=0.001$.

The relative number of slow subunits produced in the various heterozygotes reflected the different activity levels of the parental slow strains; CS4 consistently had the highest ADH activity and had the lowest F/S ratio among the heterozygous progeny indicating a higher production of slow subunits. CS19 which had the lowest ADH activity, had the highest F/S ratio

Table 1. ADH activity levels in three Adh-Sm strains. Averages of two groups of male flies aged 5-7 days post-eclosion. Methods are those of Sims and Sampsell (1982).

	ADH activity (ΔOD_{340}/min/mg)	
Strain	on 2% isopropanol	on 10% ethanol
CS4	19.4	12.9
CS20	17.3	9.5
CS19	15.3	7.3

Table 2. Proportion of ADH dimers in Adh-Fr/Sm hybrids. Four separate groups of flies were tested for each hybrid cross. Averages are given below.

		Relative amounts of			F/S
Strain	Activity	FF	FS	SS	subunits
CS /Ric110	22.0	0.29	0.46	0.26	1.06
CS20/Ric110	22.2	0.34	0.46	0.20	1.33
CS19/Ric110	17.6	0.35	0.46	0.19	1.38
CS4/Si44	25.6	0.32	0.45	0.23	1.18
CS20/Si44	24.5	0.34	0.46	0.20	1.31
CS19/Si44	22.5	0.37	0.47	0.17	1.53

suggesting a lower production of slow subunits. CS20 was intermediate in parental ADH activity and in F/S ratio. CS4 and CS20 have been crossed to three Adh-Fm homozygous strains, and among the heterozygous progeny the F/S ratio was inversely proportional to the Adh-Sm activity in the parental strain (data not shown). Although we have not yet tested ADH levels directly in the CS strains by immunodiffusion techniques, these results are consistent with the observations on other strains in which differences in ADH activity among strains with the same Adh genotype were shown to be associated with differences in the quantity of ADH (Maroni et al. 1982; Sampsell & Steward 1983).

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References: Cooper,T.G. 1977, The Tools of Biochemistry, Wiley & Sons, New York; Maroni, G. et al. 1982, Genetics 101:431-446; McDonald,J.F. & F.J.Ayala 1978, Genetics 89: 371-388; Sampsell,B. 1977, Biochem.Genet. 15:971-987; Sampsell,B. & S.Sims 1982, Nature 296:853-855; Sampsell,B. & E.Steward 1983, Biochem.Genet. 21:1071-1088.

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Contaminating microorganisms interfere with Southern Blot analysis of Drosophila melanogaster DNA.

During our studies of restriction enzyme polymorphism of the small heat shock genes at the 67B locus in Drosophila we discovered that one of the restriction enzyme fragments which we were observing was actually hybridizing to pBR322, the vector that our heat shock probe was cloned in. In several isofemale lines of Drosophila melanogaster we found one band of about 4.7 kb which showed up when probed with pBR 322 (Fig. 1). We believe our finding may serve as a useful warning to other investigators who are using whole flies as a source of genomic DNA for study of restriction enzyme polymorphism. We would also like to report on the properties of this contaminating fragment which were observed incidentally to our studies of the small heat shock genes.

Our first impression upon discovering this additional fragment was that this was merely a simple case of accidental contamination of some of our genomic DNA stocks by trace quantities of plasmid. If this was true then independent DNA extracts of these lines of flies should be free of the additional fragment. In independent DNA extracts we found that the additional fragment was present in precisely the same lines in which it was found in the first place, and absent in lines in which it was not previously found. This observation could have two possible causes: either there are pBR322 homologous sequences in the Drosophila genome or this fragment was derived from microorganisms which are associated with flies

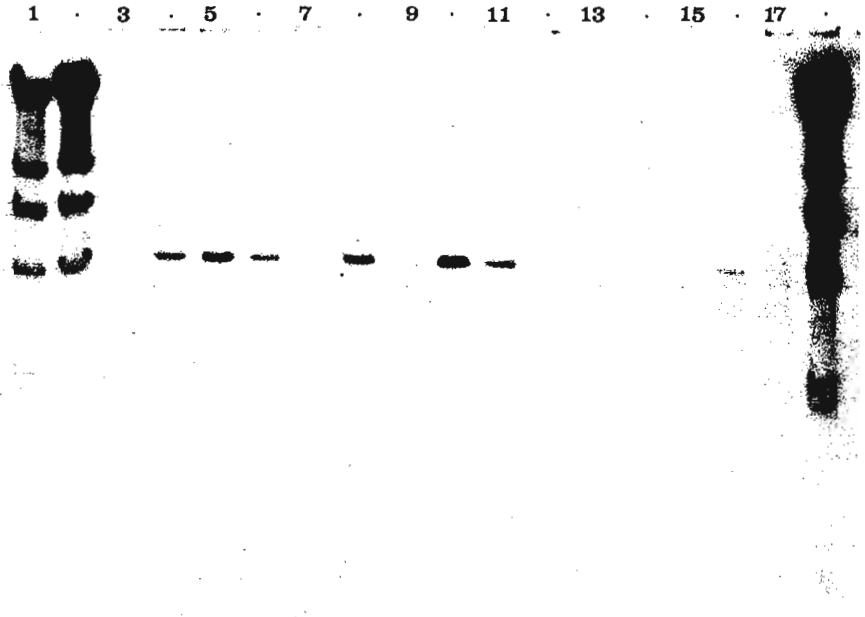

Fig. 1. A Southern Blot of fly DNA from 15 different strains of D.melanogaster. Lane 1, 2 and 18 contain DNA mol. weight markers. Lane 3-9 represent strains from Hamilton, Ontario and lane 10-17 from Brownsville, Texas. The fly DNA is cut with EcoR I and probed with the plasmid (pBR 322) DNA. Migration is from top to bottom. Lane 4, 5, 6, 8, 10, 11 16 show the contaminating 4.7 kb DNA fragment which hybridizes with pBR 322 DNA.

cultured under standard non-axenic conditions. We decided to do a few simple experiments in order to distinguish between these two possibilities.

We attempted to determine whether or not the additional fragment was a part of the Drosophila genome in two different ways. First we tried producing axenic cultures of flies which were known to carry the additional fragment to see if it could be removed. The axenic cultures were produced by surface sterilizing eggs according to the procedure of W.G.Starmer & D.G.Gilbert (DIS 58:170-171). Sterility tests were performed on fly cultures by incubating a fly in bacterial mutrient broth plus 0.2% glucose for several days at 37°C. One line of flies was obtained, which showed no microbial contamination when tested by the above method, and remained sterile throughout the time it was maintained. DNA extracted from these flies did not show the additional fragment when analysed in a Southern blot. We were therefore able to cure the flies of this contaminant by sterilization.

Our second approach was to try to transfer this contaminant from flies which carried it to flies which did not carry it. This was done by taking five males from a contaminated stock, grinding them up in a sterile isotonic buffer and spreading this mixture onto the surface of fresh medium in a clean bottle. Uncontaminated flies were then introduced into the bottle. These flies were collected after one week and their DNA was extracted. Southern Blot analysis of this DNA showed the contaminant band to be present. We were thus able to transfer the contaminant to previously uncontaminated flies.

As we continued our work on restriction enzyme polymorphisms using more enzymes, we identified the contaminating fragment by probing each blot twice, once with just pBR322 and once with pBR322 with the heat shock gene insert. During this process we were also observing digestion patterns of the contaminatn. After using six different restriction enzymes (Bam HI, Eco RI, Pst I, Sal I, Xba I, and Hind III) we found that the contaminating band was the same size in all of the ten lines of flies which carried it. There are three possible explanations for this observation. One is that the fragment is not being cut by any of the six enzymes. The second is that it is a circular fragment with a unique site for all of the enzymes. The third is that it has a unique site for some of the enzymes, is not being cut by other enzymes and that we are unable to distinguish between linear and circular forms of this molecule. After doing a few double digests in an attempt to map some of the heat shock gene restriction fragments, we obtained some results which also helped us distinguish between these three possibilities. We digested one line of flies which carried the contaminant with the following enzyme combinations: Bam HI plus EcoR I, Bam HI plus Pst I and Bam HI plus Xba I. We found no change in size of the contamination fragment following these digestions. This allows us to conclude that the fragment is not being cut by four of the six enzymes we used. The lack of change in mobility of this fragment after cutting it with six different restriction enzymes is therefore not likely to be due to linearizatio of a circular molecule.

The possibility of the contaminating fragment being a circular molecule combined with the fact that it is homologous to pBR322 caused us to speculate that perhaps it is a naturally occurring plasmid of E.coli. At present,however, all that we can conclude from our observations is that since it can be removed by surface sterilization of eggs, the contaminating fragment must be derived from an extracellular microorganism which is associated with some of our fly stocks.

Smith, M.R., G.K.Chambers, L.D.Brooks, F.M.Cohan* and S.C.Cohan.* Museum of Comparative Zoology, Harvard University, Cambridge, Massachusetts USNA. *University of California, Davis USNA. How many Adh clines on the west coast of North America?

Conversations with casual collectors of Adh allele frequencies in the western regions of North America have led us to a growing suspicion that there is no exact western counterpart of the Adh latitudinal cline observed for east coast populations (see Oakesho.t et al. 1982, who also report similar clines in Australasia and Europe/Asia). Recent data we have collected as part of a large geographical survey of genetic variation in D.melanogaster reveal that our suspicions may indeed be well founded.

Field collections were made over a three-month period in 1982 (except for VIN and SEA [Table 1], which were made in 1981). Locations of collections are shown in Figure 1. Isofemale lines were established from wild caught individual females and shipped, usually within 1-2 generations, to the MCZ. Mass cultures were re-established from the separate lines and Adh allele frequencies were determined by cellulose acetate electrophoresis on F1 progeny by the method of Wilks et al. (1980). The data are shown in Table 1.

Table 1. Adh allele frequencies in population samples.

Population	#Iso lines	°Latitude°N	Adh^F	Adh^{Fr}	Adh^S	Sample size (alleles)
LAK	17	32.9	0.44	0.00	0.56	84
WES	21	36.3	0.57	0.00	0.43	28
FNO	23	36.7	0.68	0.00	0.32	196
WAT	26	36.9	0.55	0.00	0.45	28
VIN	55	38.3	0.73	0.01	0.26	196
CAM	28	38.7	0.65	0.01	0.34	196
HAM	37	39.7	0.72	0.00	0.28	196
MED	47	42.4	0.50	0.00	0.50	194
ALO	27	45.6	0.51	0.04	0.45	196
CAR	28	47.6	0.54	0.01	0.45	140
SEA	>50	47.6	0.64	0.05	0.31	42
POC	57	49.3	0.78	0.01	0.21	196

LAK Lakeside,CA; WES Westside Field Station, Five Points, CA; WAT Watson,CA; VIN Vineburg,CA; CAM Camino,CA: HAM Hamilton City,CA; MED Medford,OR; ALO Aloha,OR; CAR Carnation,WA; SEA Seattle,WA; POC Port Coquitlam,B.C.

Table 2. Regression analysis of allele frequency data.

Analysis	r^2	slope	probability
Adh^F vs Latitude (all data)	0.0571	+0.0047	0.455
Adh^F vs Latitude (California)	0.7730	+0.0415	0.009
Adh^F vs Latitude (Oregon & Washington)	0.611	+0.0349	0.118
Adh^{Fr} vs Latitude (all data)	0.383	+0.0019	0.032

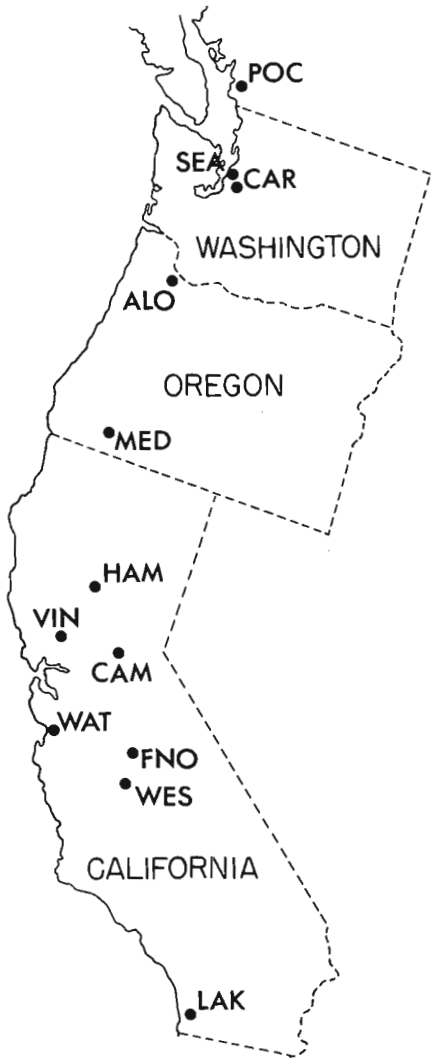

Fig. 1. Location of collections.

It is clear that there is a general trend for Adh^F to increase in frequency as one travels north, but the data do not reveal a uniform pattern of variation. Accordingly, unweighted linear regression analysis of Adh allele frequency on latitude was performed (SAS --General Linear Model). Results are shown in Table 2. The statistical analysis confirmed our initial observation: the regression of Adh^F frequency on latitude does have a positive slope but is non-significant. The data may be better explained by two clines, one in California ($p=0.01$) and one in Oregon, Washington and British Columbia ($p=0.12$). However, we are reluctant to draw firm conclusions from such small selected subsets of the data.

Interestingly, the frequency of Adh^{Fr} is significantly related to latitude ($p=0.03$), but this may be an artifact resulting from its absence over most of California. At this stage we are content to note that Adh^{Fr} is widespread but at low frequency. This distribution resembles that found in Australia by Wilks et al. (1980) and that reported for other U.S. populations by Sampsell (1977).

We conclude that the distribution of Adh alleles on the west coast is complex and it is probable that many more populations will have to be sampled before we can decide between competing descriptions in terms of zero, one or two Adh clines.

We are grateful to T.Prout, J.Coyne, A.Beckenbach, and S.Tuljapurkar who contributed collections.

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Soutullo, D.[1] and E.Costas.[2] 1-Universidad de Santiago, Espana. 2-Instituto Espanol de Oceanografia, La Coruna, Espana. A method to find the genetic origin of a mutational unstability phenomenon in Drosophila melanogaster.

The present method allows us to tell whether a mutational unstability phenomenon is due to the action of mendelian genes or to mutator polygeneic systems, or even whether it is caused by the presence of transponsable genetic elements.

The procedure consists of substituting every chromosome of the mutator strain--which might eventually be responsible for the unstability--for stable chromosomes marked with dominant markers while making sure that no crossing-over will take place between the different chromosomes (due to the presence of inversions in the marked chromosomes). Previously the 'dot' of the unstable strain has been substituted by means of balancers (it is not described in the Figure), and then it is tested whether the unstability remains. In this way we are able to avoid the taking into account of the IV chromosome in order to establish the possible conclusions of the analysis.

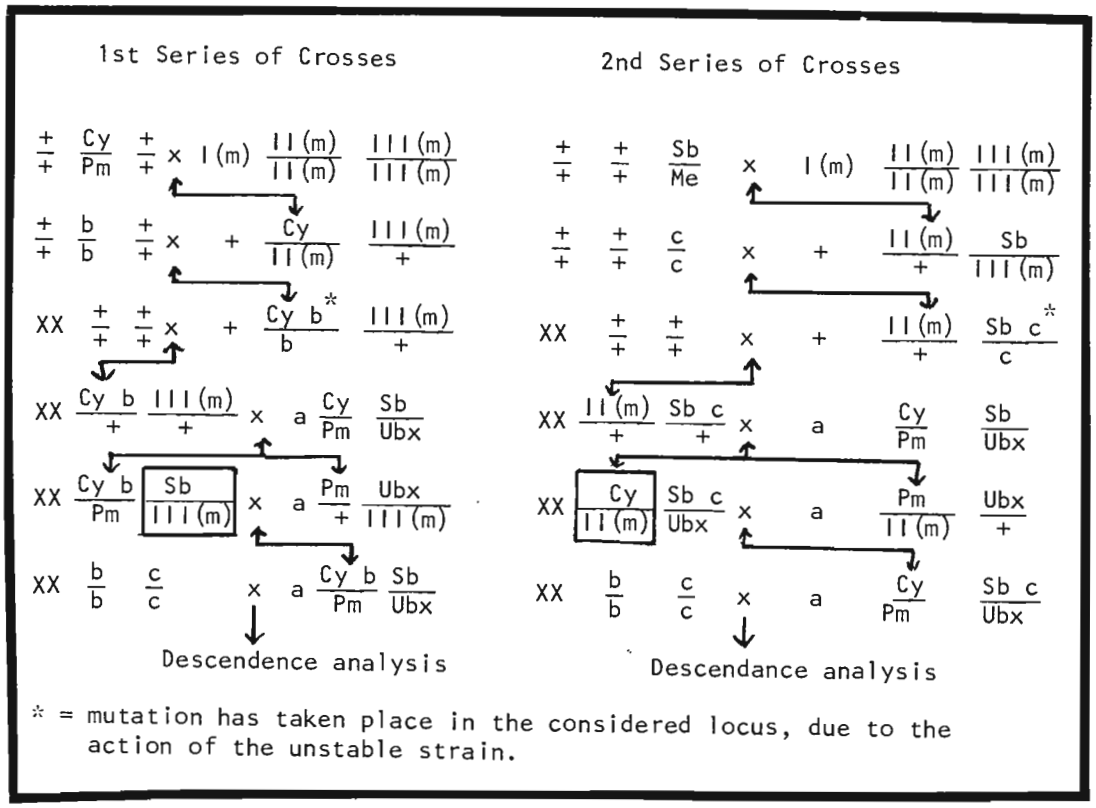
Two series of crosses were performed, one in which the unstability appears initially associated with the II chromosome and another one in which it appears associated with the III chromosome, testing at the end of each series if the unstability remained. This test is performed by checking the sensitive to mutagenic action loci in every major chromosome by means of adequate recessive markers (see Figure).

The persistence of unstability would indicate that its origin is due to the presence of any type of transponsable genetic element, except if a double crossing-over between an unstable chromosome and its stable marked homologous one would have taken place. From the comparison between both series of crosses we observed the hypothetical double crossing-over to have taken place--if it existed--between homologous III chromosomes in the first case, whereas the double crossing-over would have taken place between homologous chromosomes in the second case (noted by squares in the Figure).

From the analysis of results we can get the following conclusions:

Case 1: Presence of mutation at the end of both series of crosses: The unstability would be due to transponsable genetic elements.

Case 2: Presence of mutation at the end of the first series of crosses and absence in the second series: The unstability would be caused by genes of the III chromosome in the unstable strain.



Case 3: Presence of mutation at the end of the second series of crosses and absence on the first one: The unstability would be due to genes of the II chromosome in the unstable strain. Case 4: Absence of mutation at the end of the both series of crosses: The unstability would not be caused by transponsable genetic elements, without being able to specificate which genes of the unstable strain are responsible for it.

NOTES: (1) The chromosomes coming from the unstable strain are indicated by the notation (m). (2) 'a', 'b' and 'c' indicate the recessive markers used in order to detect the presence of mutation.

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Stark, W.S., K.Srivastava, S.D.Carlson* and M.B.Garment.* University of Missouri, Columbia, USNA. *University of Wisconsin, Madison USNA. Characteristics of none, a mutant with no ocelli and narrow eyes.

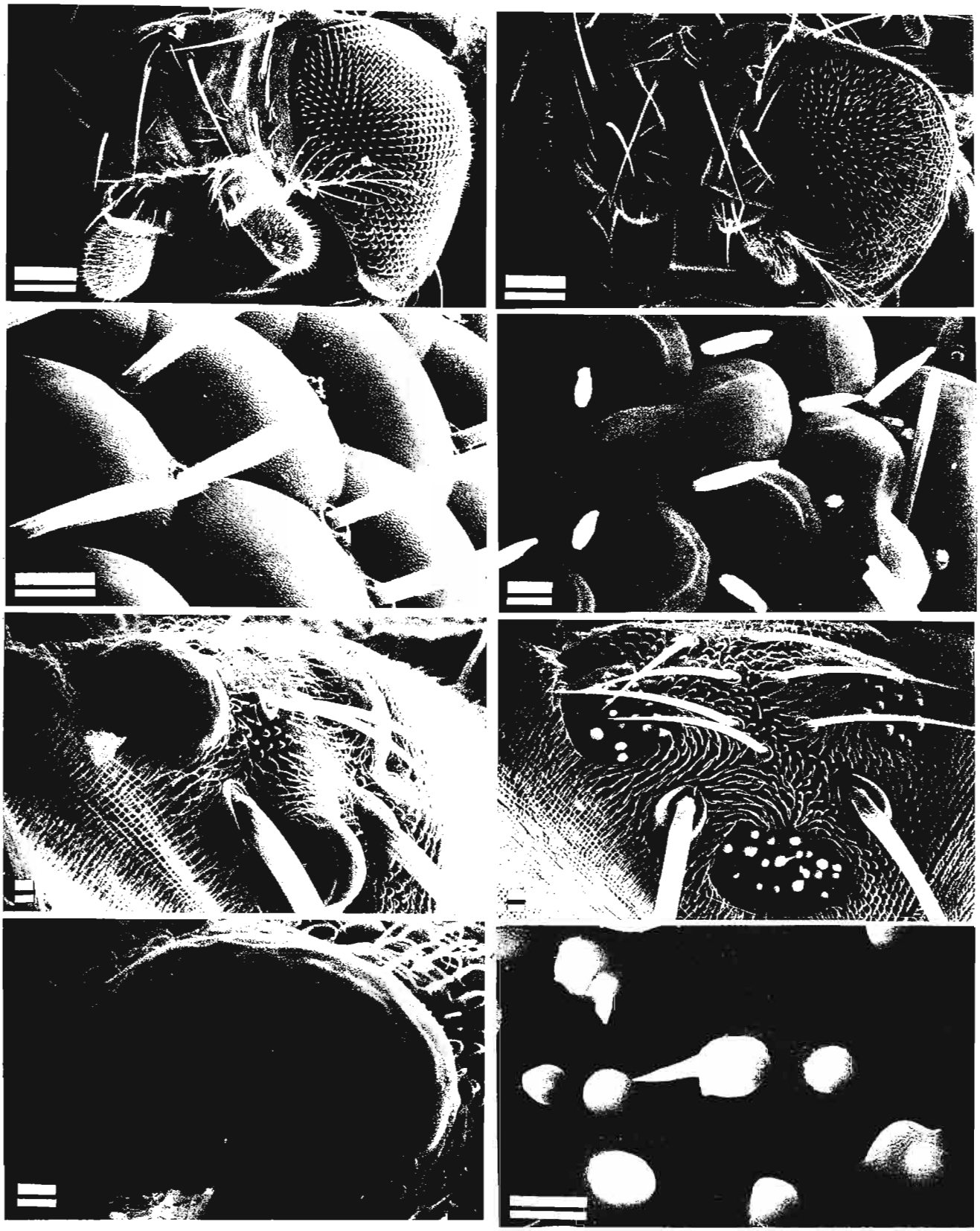
Drosophila mutants with abnormalities of the visual system have been widely used for studies of the development and function of the visual system. Some years ago, one of us (WSS) obtained a mutant from Allen Shearn at The Johns Hopkins Univeristy. Shearn had names this mutant "no ocelli, narrow eyes (none)." In an effort to process this mutant for further electrophysiological studies of receptor function and microspectrophotometric studies of the photopigment, this mutant was made white eyed with w using standard Mendelian genetics. Now only the w;none stock is retained. The initially disappointing finding was that the compound eye had no electroretinogram (ERG), and thus, if there were receptor cells, they did not work (c.f. Stark et al. 1976). Further, the eye showed no deep pseudopupil or optical density changes, thus revealing no photopigment (c.f. Stark & Johnson).

Based on these physiological findings we sought ultrastructural evidence to explain this dysfunction. We processed flies for histology, transmission electron microscopy and scanning electron microscopy. The accompanying plate shows the external featuresof the head from scanning electron micrographs. (Calibration bars show 100 microns, top row, 5 microns, remainingrows.) The external morphology of the compound eye is in some disarray (right) when compared with a control fly (left). On higher magnification (second row) the fusion of corneal facets and displacement of corneal hairs can be observed; yet the mutant does have the characteristic corneal nipples, the fine granularity which functions as an impedance matching device and an anti-reflective coating. On close examination of the ocellar area (third and fourth rows) the normal fly's ocellar lenslets and the remnants of the mutant's lenselts can be observed.

We have preliminary observations of the compound eye from the High Voltage Electron Microscope (HVEM), an NIH Biotechnology Resource in Madison, WI (c.f. Stark & Carlson 1983). In spite of the external corneal disarray, Semper cells are present as are the pseudocones which the former secrete. A corneal lenslet with its underlying pseudocone make up the distal dioptric (optical) apparatus for one ommatidium. Proximally, the compound eye's peripheral retina is separated from the first synaptic neuropile (lamina ganglionaris) by a basement membrane. Between these dioptric and basement membrane areas there is a complete absence of photoreceptor cells. Most of the volume of the peripheral retina is occupied by pigmented glia based on cell morphology, electron density and types of organelles. In a survey of this metaplasia we found no recognizable specializations such as rhabdomeres (the microvillar organelles which house the visual pigment molecules). Also, beneath the basement membrane, glial elements and interneurons exist. Yet we have observed no organization such as the normal fly's optic cartridges which are formed by terminals of receptor axons onto discrete clusters of lamina monopolar interneurons. In conclusion, the finding of an all glial cell mass in the peripheral retina readily explains the lack of an ERG and the deep pseudopupil.

The morphological features of none's compound eye are not unlike those of Glued mutants (Harte & Kankel 1982). Possibly none is an allele of Glued. Unfortunately, the micrographs of Glued do not show the ocellar area.

We hope to further characterize the specific cellular and developmental deficits in this mutant. It would be particularly useful to section the ocelli of none flies and to compare these structures to those of normal flies. To our knowledge, ocellar ultrastructure in



Drosophila has only been presented in thesis form (Schmidt 1975) though ocellar ultrastructure has been published for the fleshfly (Toh et al. 1971).

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Toda, M.J. Hokkaido University, Sapporo, Japan. The northernmost subarctic Drosophilidae.

The northernmost areas of the Holarctic region are the most interesting for consideration of biogeographical relationships between the two continents, Eurasia and North America, for it is there that the two continents were sometimes connected in the past and are the closest even at the present time, through Beringia and several Arctic islands. In a recent monograph on drosophilid biogeography (Ashburner et al. eds. 1981), Wheeler (1981) and Bächli & Rocha Pite (1981) reviewed Nearctic and Palaearctic drosophilids, respectively, but did not specify the northernmost fauna in the two regions.

The strong cohesion of drosophilid distribution to woodland areas has been confirmed not only latitudinally (Basden 1956; Wheeler & Throckmorton 1960) but also altitudinally (Burla 1951; Basden & Harnden 1956; Bächli 1977), except for some specimens sporadically collected far beyond the forest boundary. It can be, therefore, concluded that the northernmost drosophilid fauna as a biogeographical entity is virtually confined to the subarctic forest zone, never deeply entering the real tundra.

Basden (1956) listed a total of 23 arctic species by choosing arbitrarily the Arctic Circle as the southern limit of the area, though this is obviously artificial and biologically meaningless as recognized by himself. Since then, considerable information on northern drosophilid fauna has been brought from several subarctic localities, Alaska (Wheeler & Throckmorton 1960), northern Finland (Lumme et al. 1979), and Mackenzie Delta, N.W.T., Canada (Takada & Toda 1981). By reviewing these reports, the northernmost subarctic drosophilid fauna are listed below. The chorological types are classified into four: Palaearctic (P), Nearctic (N), Holarctic (H) and Cosmopolitan (C); and are given before the specific number.

P 1 Cacoxenus(Paracacoxenus)argyreator Frey
P 2 Stegana (Stegana) furta (Linne)
P 3 St. (Steganina) stroblii Mik
H 4 St. (Stn.) coleoptrata (Scopoli)
P 5 Amiota (Amiota) alboguttata (Wahlberg)
N 6 A. (A.) quadrata Takada et Toda
N 7 A. (A.) sp.Wheeler & Throckmorton 1960
P 8 Chymomyza fuscimana (Zetterstedt)
N 9 Ch. aldrichii Sturtevant
N 10 Ch. coxata Wheeler
N 11 Ch. tetonensis Wheeler
N 12 Ch. wirthi Wheeler
H 13 Ch. caudatula Oldenberg
H 14 Ch. costata (Zetterstedt)
P 15 Scaptomyza (Scaptomyza) flava (Fallen)
P 16 Sc. (Sc.) griseola (Zetterstedt)
N 17 Sc. (Sc.) nigrita Wheeler
H 18 Sc. (Sc.) graminum (Fallen)
H 19 Sc. (Sc.) montana Wheeler
H 20 Sc. (Sc.) teinoptera Hackman
P 21 Sc. (Sc.) sp. (= Finnish Sc. ?montana Basden 1956)
N 22 Sc. (Hemiscaptomyza) terminalis (Loew)
H 23 Sc. (Hsc.) trochanterata Collin
H 24 Sc. (Hsc.) unipunctum (Zetterstedt)
C 25 Sc. (Parascaptomyza) pallida (Zetterstedt)
P 26 Sc. sp. Lumme et al. 1979
N 27 Sc. sp. Wheeler & Throckmorton 1960
P 28 Drosophila (Sophophora) alpina Burla
P 29 D. (So.) bifasciata Pomini
P 30 D. (So.) eskoi Lakovaara et Lankinen
P 31 D. (So.) obscura Fallen
P 32 D. (So.) subsilvestris Hardy et Kaneshiro
N 33 D. (So.) athabasca Sturtevant et Dobzhansky
N 34 D. (So.)populi Wheeler et Throckmorton
C 35 D. (So.) melanogaster Meigen
P 36 D. (Lordiphosa) fenestrarum Fallen
C 37 D. (Dorsilopha) busckii Coquilett
P 38 D. (Hirtodrosophila) lundstroemi Duda
P 39 D. (H.) subarctica Hackman
C 40 D. (Drosophila) funebris (Fabricius)

P 41 D. (D.) ezoana Takada et Okada
P 42 D. (D.) littoralis Meigen
P 43 D. (D.) lummei Hackman
N 44 D. (D.) borealis Patterson
H 45 D. (D.) montana Patterson et Wheeler
C 46 D. (D.) immigrans Sturtevant
H 47 D. (D.) testacea von Roser*
P 48 D. (D.) phalerata Meigen
N 49 D. (D.) rellima Wheeler
H 50 D. (D.) transversa Fallen
N 51 D. (D.) melanderi Sturtevant

* Takada & Toda (1981) reported D.putrida from MacKenzie Delta, but that was a misidentification of D.testacea.

The northernmost subarctic drosophilid fauna is characterized by the relative richness in species number of the following taxa: Chymomyza, Scaptomyza, the obscura group (Nos. 28-33) and the virilis group (Nos. 41-45). It is notew thy that the southernmost antarctic drosophilid fauna is monopolized by Scaptomyza (Brncic & Dobzhansky 1957). The relative percentages of the four chorological elements, calculated by excluding unidentified species, are as follows: Palaearctic (19 spp., 40.4%), Nearctic (12 spp., 25.5%), Holarctic (11 spp., 23.4%) and Cosmopolitan (5 spp., 10.6%). The relatively high percentage of Holarctic elements suggests that the intercontinental faunae exchange, possibly across Beringia, repeatedly occurred until relatively recent times in the northernmost subarctic region.

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Tolchkov, E.B. and V.A.Gvozdev. Institute of Molecular Genetics, USSR Academy of Sciences, Moscow USSR.

The structure of two rearrangements resulting in the Pgd gene position effect in Drosophila melanogaster.

The study shows that the previously described rearrangements T(1;4)pn2 and Tp(1)pn3 (Ilyina et al. 1980) are pericentric inversions, designated In(1LR)pn2a and In(1LR)pn2b, respectively, with very similar genetic structurs.

Analysis of recombination in pn2a/y cv v f car females has shown the genetic map of the rearrangement to differ from that of the normal X chromosome. The pn2a rearrangement is characterized by the following order of the markers: cv-v-f-car-y (cf. y-cv-v-f-car in the normal chromosome). The distances between the y gene and the markers nearest to it, car and f, in the rearrangements are in good agreement with the reported (Schalet & Lefevre 1976) distances between these markers and the centromere. The easiest way to explain these results is to assume that the distal section of the X chromosome carrying the y^+ gene is transferred to the centromeric region of the X chromosome and not to the 4th chromosome, as formerly believed. The genetic maps of the rearrangements pn2a and pn2b do not differ. Analysis of the polytene chromosome shows the distal end of the rearranged chromosome to break off in the 2DE region. The telomere of the rearranged chromosome consists of heterochromatic material, as attested by its metachromasy (bluish staining with azure-eozine, as opposed to the violet staining of the bulk of the chromosomes) and the presence of highly repetitive sequences, probably satellite DNA, revealed by in situ hybridization with total labelled DNA in a set-up where the hybridization of highly repetitive DNA is selectively favoured. THe 1A-2DE region is associated with the chromocenter through the 2DE segment. The metaphase chromosomes show an enlarged XR the size of the 4th chromosome, which probably corresponds to the 1A-2DE fragment. Comparative analysis of the data on the recombination and structure of the polytene and metaphase chromosomes suggests that the rearrangements are pericentric inversions of the X chromosome (Figure 1).

The euchromatic break point of the inversions lies in the 2D-F region, whose fine genetic structure has been studied earlier (Gvozdev et al. 1973; Gvozdev et al. 1975). Genetic analysis has demonstrated that in both inversions the genes corresponding to

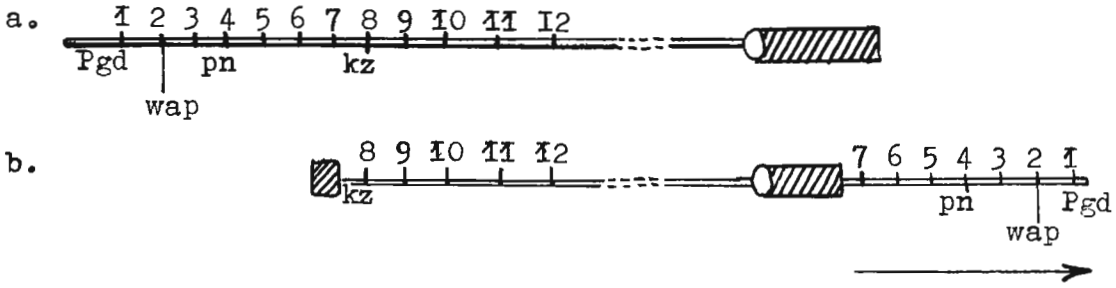

Figure 1. Structure of the normal X chromosome (a) and the rearrangements pn2a and pn2b (b). Numbers designate complementation groups for the region which has been studied earlier through saturation by lethals (Gvozdev et al. 1975). The shaded rectangle denotes the heterochromatin right arm of the X chromosome. The arrow shows the direction in which gene inactiviation proceeds.

complementation groups 1 (Pgd), 2 (wap), 3,4 (pn), 7 (groups 5 and 6 were not analyzed, as the corresponding lethals had been lost) of the 2D-F region were localized in the right arm, and complementation groups 8 to 12 in the left arm (Figure 1).

In the In(1LR)pn2a and In(1LR)pn2b inversions the 2D-F region is divided into two units, each finding itself close to the XR heterochromatin (Figure 1), which is known to cause the position effect for euchromatic genes that have been moved to it. Indeed, one observes a strong position-effect inactivation of the genes in the right arm of the inversions, while the left-arm genes are not inactiviated.

The inactivation of the Pgd gene in the pn2a rearrangement is sharply enhanced by the removal of the Y chromosome. In the females the Pgd gene activity in In(1LR)pn2a is about 50% of the normal level, as assessed by the 6PGD activity in crude extracts. The Pgd gene activity in XO males amounts to 25% of the normal level. The heterochromatic Y chromosome, which is known to suppress position-effect inactivation, normalizes the Pdg gene is observed in the pn2b rearrangement: in males it comes to only 15% of the normal level. The inactivation of the pn gene is also stronger in the pn2b rearrangement.

The inactivation of genes corresponding to complementation groups 2,3,7 sharply reduces the viability of females that carry the inversions in a heterozygote with lethals for those groups. Their viability does not exceed 3% of the normal value. The inactivation is stronger in the case of the pn2b inversion.

The above results show, within the accuracy of the methods used, that the two rearrangements have the same structure but differ considerably in the intensity of the position effect. The causes of the difference in inactivation intensity are not clear. This difference might be due to autosomal modifiers. However, the difference in the position effect intensity persists when the 1A-2DE region, associated with heterochromatin, is transferred to another genotypic environment. This result suggest that the factors responsible for the difference are linked to the centromeric regions of the rearrangement.

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Trusis, S.N. and A.J.Hilliker. University of Guelph, Ontario, Canada. Analysis of the distribution and homzygous viability of translocation breakpoints with respect to linkage group conservation in D.melanogaster.

Several lines of cytological evidence (Comings 1980; Evans & Filion 1982) argue that, in particular eukaryotic species, at least some portions of the interphase chromosomes (often centromeres and telomeres) are nonrandomly arranged relative to each other. Recently, it has been shown that, at least in one embryonic cell-type in Drosophila, each of the five major chromosome arms is found to occupy a relatively exclusive subvolume or chromosomal domain of the nucleus (Hilliker, manuscript submitted). Each chromosomaldomain appears to be a physically discrete unit consisting of an extensively folded chromosome arm. It is possible that this arrangement is of functional significance.

Indeed, chromosome linkage groups as defined by Drosophila chromosome arms may have remained largely intact during the evolution of the higher Diptera. Sturtevant & Novitski (1941) noted that, chromosome arms were maintained as distinct linkage groups within the (super) genus Drosophila. That is to say, the same genes, as defined by either homologies in polytene chromosome patterns or parallel mutations, were always found associated on the same chromosome arm. In comparing over 14 different species of Drosophila, these chromosome arms maintained their identities, though variability in the linear ordering of the genes within each arm was extensive. A recent study (Foster et al. 1981) shows that linkage group conservation may be a property of the higher Diptera per se. In assaying for biochemically and/or morphologically similar mutations in Lucilia cuprina, and in comparing their linkage relationships to Musca domestica and Drosophila melanogaster, it was discovered that the major linkage groups were conserved. The only major difference found between these Dipterans, which from an evolutionary viewpoint had diverged millions of years prior, was that the linkage groups, as denoted by chromosome arms in D.melanogaster, were actually metacentric chromosomes in L.cuprina and M.domestica. Based on these observations, we proposed that this linkage group conservation might be due to the functional arrangement of the interphase chromosomes.

As a means of testing this hypothesis, a series of reciprocal translocations were synthesized. These translocations altered the established chromosome associations, resulting in the creation of new linkage groups. By assaying for recessive lethality of these induced rearrangements, it might be possible to deduce whether the new type of linkage group created had disrupted any important linkage associations, and thus whether the integrity of the chromosomal region disrupted (not simply that in the immediate vicinity of the breakpoints) was of any functional significance.

Oregon-R males of Drosophila melanogaster, aged 4-5 days, were treated with approximately 2000 rads of gamma irradiation to recover reciprocal translocations between chromosomes 2 and 3. These translocations were analyzed cytologically to determine their polytene chromosome breakpoints, and were subsequently tested for homozygous viability or lethality. Table 1 summarizes these results.

As shown in Table 1, about 53% of 47 analyzed translocations are lethal when homozygous. Earlier studies of induced autosomal 2-3 translocations, similarly obtained by exposing sperm to 2000 rads of irradiation, report slightly higher levels of homozygous lethality. Ytterborn (1970) reported that 66% of 35 synthesized translocations were lethal in the homozygous condition, while Sobels (1972) found that of 84 translocations, 62.2% were homozygous lethal. It is important to note here that, in both cases, no attempt was made to determine the actual rearrangement breakpoints. Since these earlier studies failed to consider the breakpoints or the complexity of the rearrangements, it is possible that the reduced lethality levels we observed could be explained by the fact that the data set presented in Table 1 did not include the majority of complex rearrangements which we obtained. (Upon diagnosis, these stocks were placed aside and never precisely analyzed.)

Analysis of the results presented in Table 1 entailed correlating the homozygous viability or lethality with the actual rearrangement breakpoints. It should be noted though that in analysing the viability of homozygous translocations, the recessive lethality may be attributed to two factors. First, one or both of the actual breakpoints may fall within a vital gene locus. This being the case, the observed lethality would then be a direct result of the breakpoints themselves. On the other hand, it is possible that a proportion of the homozygous lethality may be due to the disruption of a region of the chromosome which requires a linear integrity. Another possibility exists in that new linkage associations may

Table 1. Induced autosomal II-III translocations listed according to polytene chromosome breakpoints and homozygous viability or lethality.

Translocation no.	Chromsome II breakpoint	Chromosome III breakpoint	Viability of translocation homozygote	Status of stock	New stock ID # (if available)
T(2;3)-1	43F1-2	87D4-13	viable	available	T(2;3)-1
T(2:3)-2	2Rh	100F2-5	lethal	lost	
T(2;3)-3	60C2-7	76A2-3	lethal	available	T(2;3)-3
	42A6-19	83D5-E1			
		In(3R)84F12-16;98C3-D1			
T(2;3)-4	50A10-15	90C8-D1	viable	lost	
T(2;3)-5	2Rh	62E3-8	lethal	lost	
T(2;3)-6	60D6-9	94A1-3	lethal	lost	
		In(3R)87D3-10;96F9-11			
T(2;3)-7	57F	70C2-12	lethal	lost	
	het	90C			
T(2;3)-10	44C5-D1	84D3-8	viable	available	T(2;3)-10
T(2;3)-11	36C2-E1	3h	viable	lost	
T(2;3)-13	2Rh	83D	viable	available	T(2;3)-13
T(2;3)-14	53D3-E1	79E2-5	viable	available	T(2;3)-14
T(2;3)-15	47B	92D3-9	viable	available	T(2;3)-15
T(2;3)-16	56D2-E1	67C2-4	viable	lost	
T(2;3)-19	43B1-C1	87D3-E1	viable	available	T(2;3)-19
T(2;3)-20	51D2-7	96E5-9	viable	available	T(2;3)-18
T(2;3)-22	het	het	lethal	lost	
T(2;3)-23	2h	72E2-F1	viable	lost	
T(2;3)-25	42B1-4	82C2-D1	lethal	available	T(2;3)-5
T(2;3)-26	57A2-4	65F2-66A1	lethal	lost	
T(2;3)-27	2Rh	62D6-E2	viable	available	T(2;3)-17
T(2;3)-28	2rh	88C10-E1	viable	lost	
T(2;3)-29	24D1-2	3h	viable	available	T(2;3)-9
T(2;3)-31	38D	69F2-70A1	viable	lost	
T(2;3)-32	21E2-F1	83C2-D1	viable	available	T(2;3)-12
T(2;3)-35	2Rh	98D1-2	lethal	available	T(2;3)-8
T(2;3)-36	33A1-B12	71F2-72A1	viable	lost	
T(2;3)-38	50C11-20	3Lh	viable	lost	
T(2;3)-40	60F1-2	3L(and Y^S?)	lethal	lost	
T(2;3)-42	38A2-C1	89A1-3	viable	lost	
		3Lh			
T(2;3)-43	59C5-D1	3Lh	lethal	available	T(2;3)-16
T(2;3)-44	het	het	lethal	available	T(2;3)-11
T(2;3)-46	38DE	78B	viable	available	T(2;3)-2
T(2;3)-47	24D2-E1	78C	lethal	available	T(2;3)-7
T(2;3)-48	22A1-2	3Rh	lethal	lost	
T(2;3)-49	35B2-B9	3h	viable	lost	
T(2;3)-51	56C3-D1	82E7-8	lethal	lost	
T(2;3)-52	2Lh	99C3-D1	lethal	lost	
T(2;3)-54	36C	3h	viable	lost	
T(2;3)-55	23A	98BC	lethal	lost	
T(2;3)-57	51F	61C7-9	lethal	lost	
T(2;3)-61	23E1-2	64E1-2 (and Y)	lethal	lost	
T(2;3)-63	2h	99D2-4	lethal	lost	
	42A	3h			
	In43F-42A	99D			
T(2;3)-64	24BC	87B4-5	viable	available	T(2;3)-4
T(2;3)-65	59D	64A1-B2	lethal	lost	
T(2;3)-66	21D2-E1	3Lh	viable	lost	
T(2;3)-67	2Rh	99A8-B1	lethal	lost	
T(2;3)-68	21B2-8	82F8-83A1	lethal	available	T(2;3)-6

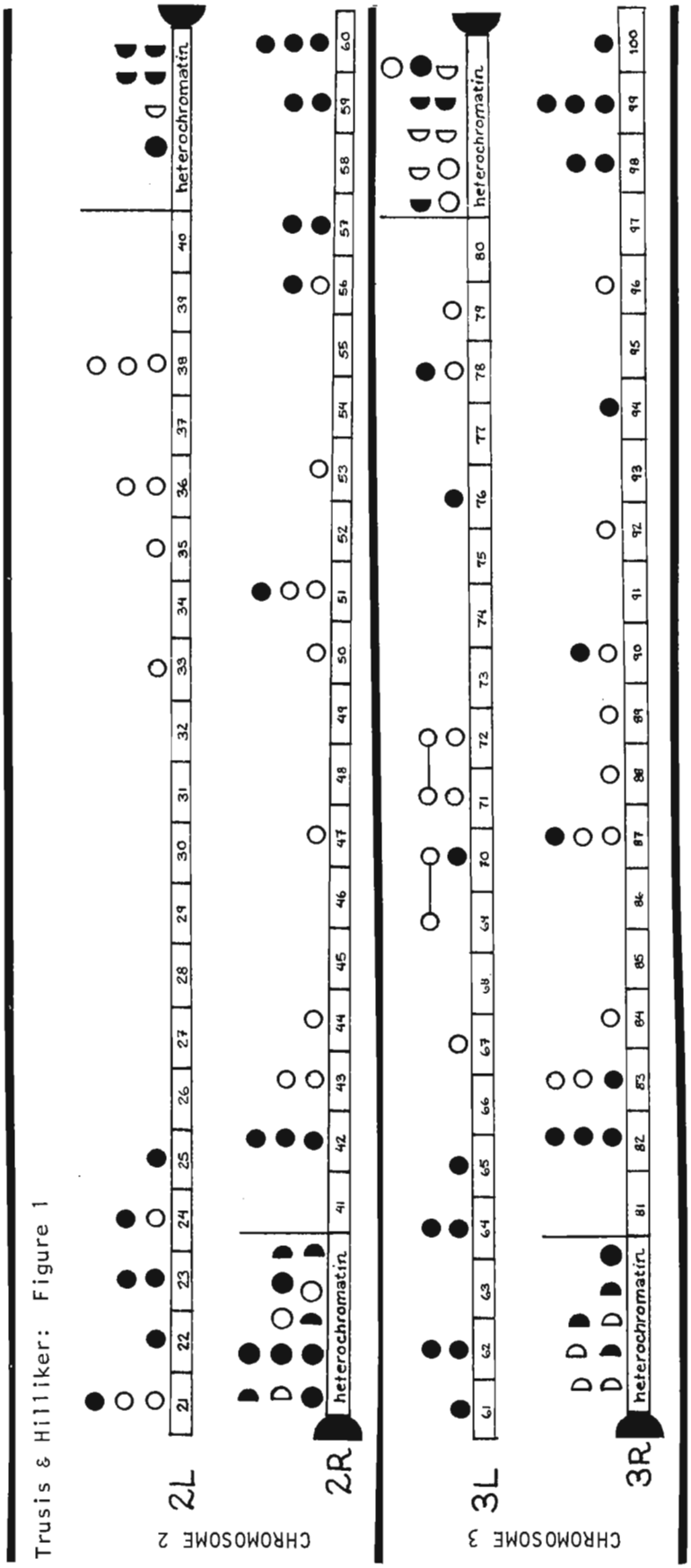

Fig. 1. The distribution of translocation breakpoints with respect to polytene chromosome section indicating homozygous viability (o) or lethality (●). Breakpoints which span more than one polytene section are indicated by o—o. Half circles (◖ or ◗) represent heterochromatic breakpoints which could not be assigned to specific chromosome arms, and are diagrammed as such in both the left and right arm figures. Note also that heterochromatic symbols are arbitrarily placed and do not reflect the relative position of the breaks within heterochromatin.

bring together "new" sets of genes which result in lethal combinations. However, we expect that these latter effects would be dominant lethal and therefore not recoverable.

It was of course necessary to analyse the viability of these translocations in the homozygous condition. Lethal effects would not be evident in the heterozygotes as each translocation heterozygote would still possess one normal homologue for each translocated chromosome, oriented in its appropriate domain. Further, by virtue of somatic pairing, these undisturbed homologues might tend to bring the translocation elements per se into proper alignment with respect to their original linkage groups during interphase.

Figure 1 illustrates the distribution of homozygous lethal and viable breakpoints with respect to each chromosome arm (or domain). Despite the fact that many more translocations are needed for a complete analysis of the genome, Figure 1 documents that the linear integrity of the chromosomal domains can be disrupted quite dramatically. Although this may be simply due to sampling, it appears that the more proximal and distal breakpoints are associated with much higher proportions of homozygous lethality relative to those

Table 2. Inter se complementation crosses tested for viability of trans heterozygotes

Translocation numbers tested

♂	1	3	4	5	6	10	13	14	15	16	19	20	22	23	25	26	28	29	31	32	35	36	42	43	44	46	47	48	49	52	54	55	57	61	64	65	68	♀
1		C					C					C																							C	C		
3				C				C	C			C		C									C			C			C			C						
4		C		C	C		C	C	C						C															C								
5							C	C				C								C			C							C		C	C					
6	NC		C				C																C						C	C								
10	C	C			C		C	C	C			S			C			C				C				C			C					C	C		
13			C	C																										S						C		
14	C	C		C		C								C				C					C			C								C				
15		C					C	C							C				C				C		C		C						C						
16			C																										C									
19		C	C	C								C									C												C		C			
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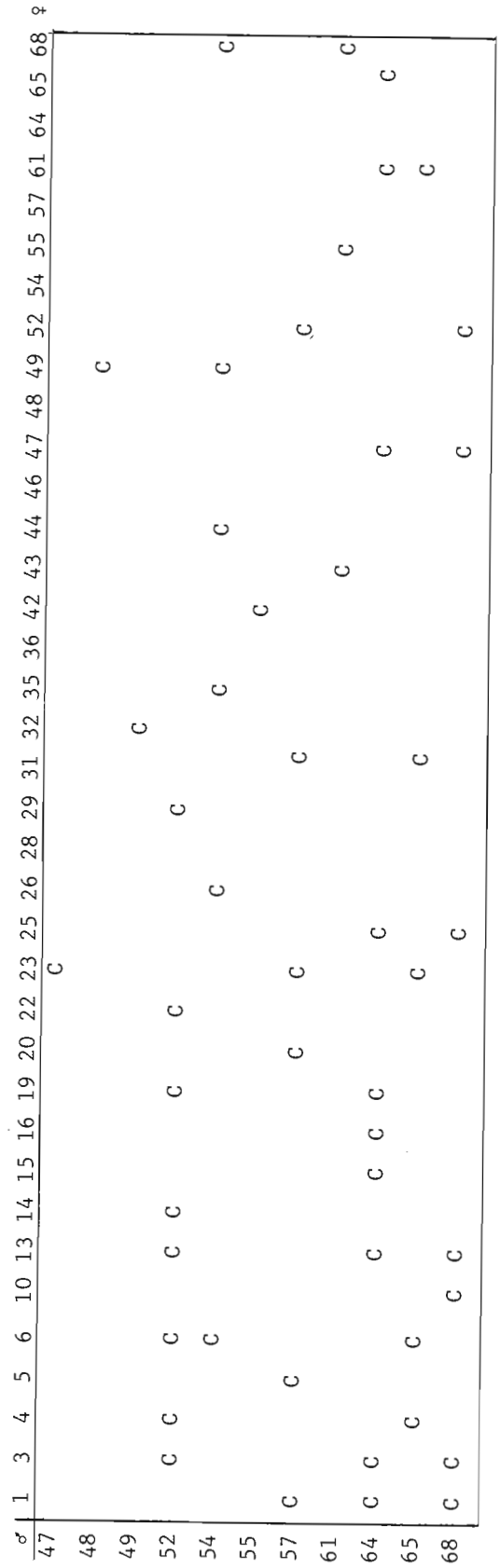
28							C								C														C						C			
29		C	C					C							C								C				C											
31		C		C										C	C												C											
32																							C				C		C	C	C						
35																										C				C								
36		C	C							C						C							C										C					
42	C		C*		C*	S		C			S							C																				
43	C																																				C	
44		C							C						C												C											
46				C						C	C	C			C	C																		C					

Table 2. Translocation numbers tested (contin.)



to those associated with internal or medial segments.

These findings are contrary to what one might expect, taking into consideration our previous hypothesis regarding linkage group conservation. If maintaining linkage groups is indeed of functional significance, then one might expect that breaks occurring at the telomeres and centromeres would be associated with lower levels of lethality for each respective chromosomal arm since the linkage group would still be largely intact. Similarly, one would expect medial breakpoints to be associated with recessive lethality as they constitute major disruptions of the linkage gorups. Perhaps further data will reveal that the linear integrity of some large sub-regions of the telomeres and possibly the centromeres is important; although the linear integrity need be present in one (normal) chromosome of the translocation heterozygote.

As mentioned earlier, a problem arises in analyzing homozygous lethal translocations, since it is not known whether the lethality is a result of the new linkage associations or merely a function of one or both breakpoints disrupting a vital gene locus. If it happens that the creation of the new linkage groups (i.e., the destruction of the old linkage groups) results in a lethal combination, then two translocations bearing similar yet not identical breakpoints within the disrupted region should also prove to be lethal. To assay this possibility, inter se complementation crosses were carried out between the various analyzed translocations. Stocks of the translocations were balanced and maintained as heterozygotes over TM3. Complementation analysis was subsequently performed by crossing two heterozygous translocation stocks and assaying for surviving trans-heterozygotes. F1 progeny showing none of the dominant phenotypic markers associated with TM3 were presumed to be viable trans-heterozygotes and were therefore said to complement. Table 2 summarizes the results of the initial complementation screen. Unfortunately, the majority of these translocation stocks were lost, thereby limiting the size of the data set presently available.

Despite this limitation, the available data indicates that almost all inter se combinations are viable. (The majority of complementations involved translocations with dissimilar breaks, and might be considered as control crosses.) Although these control crosses do certainly represent the majority of data shown in Table 2, several inter se combinations between related translocations also complemented. Of these combinations, it is interesting to note that the cross involving

a T(2;3)-1, having a break in polytene section 42A6-10, and a T(2;3)-3 ♀, having a break in 43F-1, showed complementation. In referring back to Figure 1, it is evident that this proximal region of 2R euchromatin is associated with a high proportion of homozygous lethal translocations.

Examples of other inter se combinations having similar breakpoints which also complemented include: T(2;3)-5♂ x t(2;3)-13♀ , 2Rh/2Rh ; T(2;3)-10♂ x T(2;3)-3♀, [84D3-8/83D5-E1]; T(2;3)-14♂ x T(2;3)-46♀, [79E2-5/78B]; T(2;3)-42♂ x T(2;3)-1♀, [89A1-3/87D4-13]; T(2;3)-13♂ x T(2;3)-3♀, [83E/83D5-E1]; T(2;3)-57♂ x T(2;3)-20♀, [51F/51D2-7]; T(2;3)-57♂ x T(2;3)-5♀, [61C7-9/62E3-8]; T(2;3)-19♂ x T(2;3)-3♀, [43B1-C1/42A6-19].

Table 2, however, also lists several exceptional combinations which were noted. Four different inter se combinations appeared to be sterile. In each of these combinations, T(2;3)-13♂ x T(2;3)-52♀, T(2;3)-42♂ x T(2;3)-10♀, T(2;3)-42♂ x T(2;3)-19♀, and T(2;3)-10♂ x T(2;3)-20♀, eggs were laid in a dispersed pattern indicating that fertilization had occurred, yet none had appeared to have hatched. It is also interesting to note that one of the reciprocal crosses T(2;3)-13♀ x T(2;3)-52♂ was not sterile and produced progeny according to the expected frequencies. This result was rechecked by repeating each of the reciprocal crosses and again yielded the same results. Two additional inter se combinations involving the crosses T(2;3)-42♂ x T(2;3)-50♀, and T(2;3)-42♂ x T(2;3)-3♀ proved to complement; however, all of the trans-heterozygotes scored (approximately 40 or more in each case) were males. Meiotic segregation per se does not appear to be involved, as each translocation heterozygote male (T(2;3)/bw;ve st e) segregated euploid products (i.e., T(2;3) or t;ttt) in equal proportions. It is possible however that segregation may have been unusual in conjunction with TM3 and/or in translocation heterozygote females. Unfortunately, we can offer no explanation for these results.

The only cross which failed to show complementation was T(2;3)-6♂ x T(2;3)-1♀. In analyzing the significance of this result, it should be noted that only 23 F1 progeny were recovered. Also, upon examining the breakpoints of these translocations, we find that T(2;3)-6 is a complex rearrangement, including an inversion which bears a breakpoint in the same polytene section as T(2;3)-1. It is therefore possible that this lethality, or failure to complement, may be due to common breakpoints within one gene, rather than the type of chromosome configuration produced by this inter se combination. Unfortunately, these exceptions cannot be further analysed since all crosses involve at least one, if not both of the translocations which are no longer available as stocks.

Research is presently ongoing to obtain and analyse additional translocations. This may allow us to identify any important regions of association. Despite the fact that much additional information is still required, a picture is starting to emerge. The results presented here document that many major linkage associations need not be maintained. Some observations, however, suggest that certain regions involving the proximal and distal extremities of each linkage group may require a linear integrity.

A.J.Hilliker was responsible for cytological determination of all polytene chromosome translocation breakpoints. Synthesis and subsequent genetic analysis of the translocations was done by S.N.Trusis.

References: Comings,D.E. 1980, Human Genetics 53:131-143; Evan,K.J. & W.G.Filion 1982, Can.J.Genet.Cytol. 24:583-591; Foster,G.F., M.J.Whitten, C.Konovalov, J.T.A.Arnold & G.Maffi 1981, Genet.Res. 37:55-69; Sobels,F.H. 1972, DIS 48:117; Sturtevant,A.H. & E.Novitski 1941, Genetics 26:517-541; Ytterborn,L. 1970, DIS 45:158.

Ukil, M., K.Chatterjee and A.S.Mukherjee. University of Calcutta, India. Cytophotometric analysis of in situ binding of non-histone protein to the chromatin in Drosophila melanogaster.

The role on non-histone chromosomal protein in the control of gene activity has been reported earlier (Paul & Gilmour 1968; Spelsberg & Hnilica 1969). Since hyperactivity of the X chromosome in Drosophila male is a consequence of relatively higher net transcription of the X chromosome, it is conceivable that the non-histone protein may have a role in mediating the hyperactivation of the X linked genes. Conversely, it may be predicted that non-histone protein may bind differently with X chromosomal DNA sequences in male and female and to substantiate this presumption we carried out the cytophotometric analysis.

Table 1.

Experiment	No. of observed nuclei	Integrated absorbance ratio 433/547 (Mean ± SE) Proximal segment of X chromosome	Distal segment of X chromosome	Autosome
1. CONTROL				
Male	10	1.78 ± 0.18*	1.97 ± 0.19**	1.32 ± 0.11
Female	10	1.33 ± 0.11	1.32 ± 0.07	1.24 ± 0.13
2. ONLY BUFFER				
Male	15	1.13 ± 0.33	1.16 ± 0.05	1.08 ± 0.06
Female	15	1.05 ± 0.03	1.03 ± 0.05	1.15 ± 0.04
3. BUFFER + NHP				
Male	20	1.75 ± 0.03***	1.73 ± 0.03***	1.23 ± 0.03
Female	20	1.30 ± 0.03	1.31 ± 0.04	1.25 ± 0.04

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

First, the non-histone chromosomal proteins (NHP) were isolated separately from male and female Drosophila melanogaster by following standard methods (Elgin & Hood 1973; Phillips & Forest 1973; Chiu et al. 1976) with certain modifications. Formaldehyde-fixed slides were prepared from third instar larval salivary glands, and grouped as follows: (1) treated with 2M NaCl, 1M urea in phosphate buffer (pH 7.6) for 90 min and subsequently with non-histone proteins (Conc. 1 mg/ml); (2) treated with only 2M NaCl, 1M urea in 50 mM phosphate buffer (pH 7.6) for 90 min; (3) control slides without any treatment.

The treated and control slides were stained with Schiff's reagent for 90 min and counter-stained with Napthol-Yellow S for 60 min. The slides were scanned cytophotometrically by using interference band filters at 547 nm for DNA measurements and at 433 nm for NHP measurements. Two area-one wave length method was used for cytophotometric measurements and the values were transformed into AE (integrated absorbance). Results are shown as ratios of AE for 433/547 nm.

Data reveal that the ratio at 433:547 nm is always greater in the male than in the female. Results also reveal that the extraction buffer (2M NaCl, 1M urea in 50 mM phosphate buffer, pH 7.6) unequivocally removes the non-histone proteins from both X chromosome as well as autosomes, although the extraction is non-random. Interestingly, the relatively higher proportion of NHP binding to X chromosome of male is observed in control as well as NHP bound chromatin. Results clearly show that the single X chromosome in the male has a higher binding affinity than that in the female. These data corroborate the proposition that DNA-NHP-Histone organization of the chromatin of X chromosome is mainly responsible for evoking a hyper-template activity of the X chromosome in male Drosophila (Mukherjee 1982).

References: Paul,J. & R.S.Gilmour 1968: In Differentiation and Immunity, 135; Spelsberg, T.C. & L.S.Hnilica 1969, Biochem.Biophysics 195:629; Elgin & Hood 1973, Biochem. 12:4984-4991; Phillips,J. & H.Forest 1973, J.Biol.Chem. 248:265-269; Jen-Fu-Chiu et al. 1976, Method in Cell Biol. 16:283-286; Mukherjee, A.S. 1982, Current Science 51(5):205-212.

VanDelden, W. and A.Kamping. University of Groningen, Haren, Netherlands.

Selection for increased alcohol tolerance in Drosophila melanogaster in relation with the Adh locus.

It has repeatedly been shown that the alcohol dehydrogenase (Adh) locus in Drosophila melanogaster is of great importance in the detoxification of ethanol and other alcohols (review in VanDelden 1982). Survival of the Adh genotypes on alcohols is positively correlated with in vitro ADH activity.

Experiments were designed to study the effects of long term exposure of D.melanogaster strains, either homozygous for the Adh^S or the Adh^F allele to particular alcohols. For this purpose Adh^S and Adh^F strains, originally derived from the Groningen population, were kept for many generations on ethanol supplemented food (18 vol.%), on propanol supplemented (3.5%) on hexanol supplemented food (0.525%) and on regular food without alcohol. The procedure for founding and maintaining these strains was described in Van Delden and Kamping (1983).

The aim of the experiment was to determine whether the different strains kept on various alcohols had developed resistance to propanol. At the time of the test the strains had been exposed to their particular alcohols for many generations: 70 generations for the ethanol strains SSE and FFE; 90 generations for the propanol strains SSP and FFP; 90 generations for the hexanol strains SSH and FFH; while the control strains SSC and FFC had been kept for 140 generations. Tolerance to propanol was determined in mortality tests on adult flies (5 to 9 days old; sexes separated). Mortality was measured on control food and on four different propanol concentrations (3.5, 4.5, 5.5 and 6.5 vol.%). There were 10 replicates, each with 10 flies, per sex and per concentration. Mortality was determined after three days of exposure to propanol.

The results are given in Table 1. It appears that both the SSP and FFP strains (previously exposed to propanol) are considerably more resistant to propanol than the controls SSC and FFC. Especially FFP females are highly resistant to propanol: an increase in LD50 exceeding 400% is found. But also in the ethanol and hexanol strains a significant increase in resistance to propanol is observed. It was previously found that these strains had become resistant to their "own" alcohol. Apparently the genetic changes in the ethanol and hexanol strains, both SS and FF, also provide higher tolerance to an alcohol which the strains never experienced. This suggest a general mechanism in the development of tolerance to alcohol stress.

Table 1. Median lethal doses (LD50) of propanol for adult survival of control, ethanol, propanol and hexanol strains (95% confidence limits given in parentheses).

Strains	Females	Males
SSC	6.2 (5.9-6.5)	4.5 (4.2-4.7)
FFC	5.9 (5.7-6.2)	5.5 (5.3-5.8)
SSE	12.0 (10.2-13.7)	8.1 (7.4-9.0)
FFE	6.6 (6.2-7.0)	6.7 (6.3-7.2)
SSP	11.2 (9.7-12.9)	6.3 (6.0-6.6)
FFP	33.3 (26.0-42.6)	8.3 (7.7-8.9)
SSH	15.4 (13.2-17.8)	7.6 (7.1-8.2)
FFH	8.5 (7.7-9.4)	9.8 (8.4-11.5)

Concerning the role of ADH in the adaptation to propanol it is striking that the absolute height of in vitro ADH activity cannot be the main factor responsible for increased tolerance to propanol as both the SSE, SSP and SSH strains are considerably more resistant to propanol than the FFC strain, though the latter has a much higher in vitro ADH activity. These results are in agreement with earlier obtained results (Van Delden & Kamping 1983).

References: VanDelden, W. 1982, Evol.Biol. 15:187-222; VanDelden,W. & A.Kamping 1983, Ent.exp.&appl. 33:97-102.

Vargo, M. and J.Hirsch. University of Illinois, Urbana-Champaign, Illinois USNA.

Bidirectional selection for central excitation.

A food-deprived, water-satiated fly shows an increased frequency of the proboscis extension reflex (PER) to a water stimulus which follows sucrose stimulation of the labellum but not to a water stimulus which precedes sucrose stimulation. This sucrose induced responsiveness is termed the central excitatory state (CES) with previous studies performed with the blow fly Phormia regina (Dethier et al. 1965,1968). Bidirectional selection experiments for high and low expression of CES in Phormia (McGuire 1981; Tully & Hirsch 1982a) and hybrid analyses of the selected lines (Tully & Hirsch 1982b) have found two segregating alleles of one major gene correlate of CES. Further behavioral experiments revealed the existence of additional components of the proboscis extension reflex (PER; Tully & Hirsch 1983) and that CES was positively correlated with excitatory conditioning of PER in Phormia (Tully et al. 1982).

CES has since been demonstrated in Drosophila melanogaster (Vargo & Hirsch 1982a,b). Furthermore, from other studies there is reason to believe that CES is involved with the summation of courtship stimuli in female D.melanogaster (Bennet-Clark et al. 1973). It would now be valuable to have selected lines of D.melanogaster with extreme expression of CES so that more detailed studies with other behaviors can be performed. The importance of these lines lies in the ability to use CES as a controlled variable in studies of other behavioral constructs which may have CES as a correlated trait.

Two foundation populations of Drosophila melanogaster were used; the Berlin wild type strain obtained from Marburg, Germany in 1975 and an outbred population called Austin, produced by the interbreeding of 12 Austin inbred lines obtained from Birmingham, England. All stocks were kept on a 16/8 hr L/D cycle at 25°C and 50% RH and maintained on Instant Drosophila medium (Formula 4-24, Carolina Biological Supply Co., Burlington, NC).

The basic test procedure was the same as that used in Vargo & Hirsch (1982a,b). Each animal received in a single trial (a) a 5-sec stimulation of the tarsi, and labellum if the proboscis was extended, with distilled water (pretest), (b) a 5-sec stimulation of the tarsi and labellum with .25 M sucrose immediately following pretest, (c) a 45-sec inter-stimulus interval (ISI) immediately following sucrose stimulation in which no stimuli were administered, and (d) a 5-sec presentation of distilled water (posttest) again applied to the tarsi first and labellum if extended. Animals were allowed to imbibe water on pretest and posttest to control for thirst and labellar contact with sucrose was required for a response to be recorded. All animals were approximately 48 hr old at the time of testing and food-deprived by placing them for 36 hr in a vial containing water-soaked cotton. Before a test session all subjects were given water 15 min ad lib to ensure water satiation.

The CES test consisted of 8 trials with a 6-min inter-trial interval (ITI). Proboscis extension was scored all or none (Position 3 or better on the Dethier et al. scale, 1965), therefore the range of scores for pretest, sucrose, and posttest was 0 to 8, with posttest being the measure of CES in an animal.

Animals were stimulated automatically using the apparatus described in Vargo et al. (1983) and Holliday et al. (1983). In the automatic method, the solutions were contained on Whatman #3 filter paper strips which were placed on the surface of a kymograph drum. The animals were positioned along the side of the kymograph. With the drum turning, as the strips approached the fly, it extended its tarsi and walked over the strips, thereby being stimulated. Animals were discarded from analysis if (a) sucrose score was less than 6 or (b) the animals did not participate (not walking over either the pretest, sucrose, or posttest strips) 3 or more trials.

Animals selected to breed the high line were required to score 6 or more on posttest and 2 or less on pretest, whereas to qualify for the low CES line, flies had to score 2 or less on both pretest and postttest. In each generation 4 pairs were mated. A control line was also maintained in which 4 pairs of untested flies were bred each generation. In each generation, equal numbers of males and females were tested. Approximately 30 flies were tested each generation with N ranging from 10 to 50. Four bidirectional selection experiments were performed, three with Berlin and one with Austin. From the four experiments only one high line (termed HE) and one low line (termed LE) were obtained (Figure 1). HE was founded out of Berlin while LE was founded out of Austin. It is interesting to note that from the other selection experiments not presented, Berlin never produced a low line and Austin never produced a high line. Given the above result, the most appropriate control line with which to compare HE and LE would be a hybrid between the Berlin and Austin foundation populations. Such a population was mated. The Berlin, Austin, and hybrid of the two were all tested for

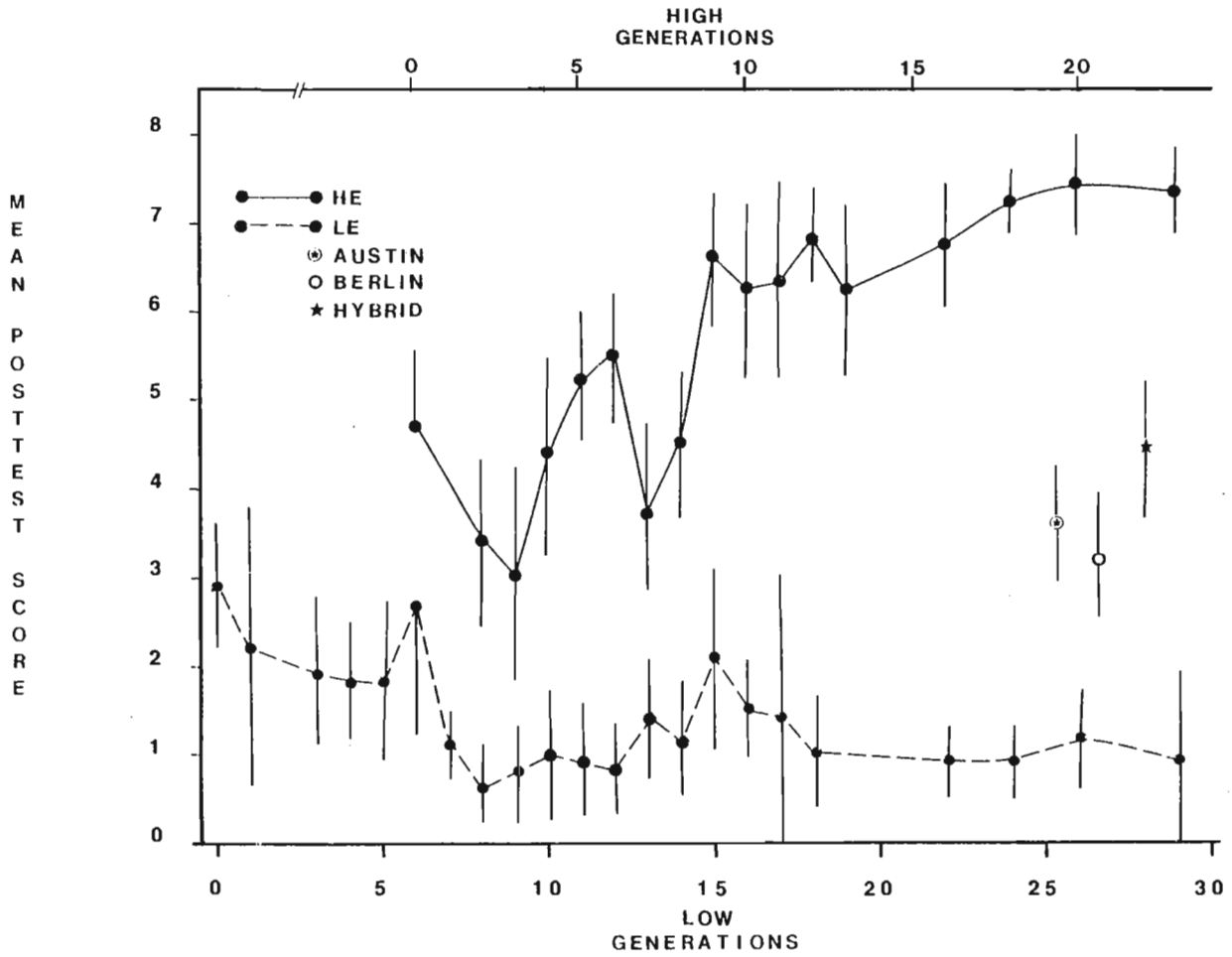


Figure 1. Selection data for the high and low CES lines. The high line was first tested at the 7th generation of the Austin attempt. Plots are presented in this manner to indicate which generations were tested concurrently. Data points for unselected populations of Austin, Berlin, and the Austin X Berlin population are likewise shown to demonstrate the effect of selection and displaced to later generations just for clarity. Data points are shown with their 95% confidence intervals.

CES in the 19th generation of selection in LE and are shown in Figure 1.

Our results are not unique in the study of this phenomenon. Kemler (1974), while studying classical conditioning in D.melanogaster, also assayed CES in the subjects (Oregon-R, maintained at the University of Nebraska at Lincoln). Kemler likewise artifically selected for high and low CES and stated that "selection is particularly effective in the direction of reduced arousability" (p.70). We interpret this statement to indicate that Kemler had success in developing a low line but not a high line, a result similar to ours with Austin.

In summary, high and low CES lines have been established, albeit from different foundation populations, which can now be analysed genetically and used to assay the effect CES may have on other behaviors.

Acknowledgement: This work was supported by Grants from the National Science Foundation (BNS-83-00353), the National Institute of Mental Health for training in research on Institutional Racism (MH15173), and a Biomedical Research Support Grant (NIH RR 7030) from the National Institute of Health.

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Villarroel, H. and P.Zamorano. Academia Superior de Ciencias Pedagogicas, Valparaiso, Chile. Drosophila species which inhabit the National Park "La Campana".

The particular geographic configuration which presents Chile, both externally and internally (Brncic 1970), has permitted the development of a flora and fauna fundamentally endemic (Reiche 1907; Fuenzalida 1950). The Drosophilidae family constitutes a good example of this phenomenon.

The purpose of this work is to carry out a preliminary search of the Drosophila species which live in the National Park "La Campana" Valparaiso. This site is considered as one of the most interesting ecological areas in Central Chile (Rundel & Weisser 1975).

The collections were made during the period of October 1982 and March 1983. The capture was done by means of the usual trapping method with fermented banana bait.

Table 1. Total number of flies and their corresponding percentages.

Species	No. of Flies	Percentages
D.amplipennis	192	9.68
D.araucana	569	28.69
D.busckii	1	0.05
D.immigrans	428	21.58
D.pavani	27	1.36
D.repleta	66	3.33
D.subobscura	565	28.50
Total	1983	100.00

Of the 33 species described for Chile by Brncic (1957a, 1962a), 9 Drosophila species (Table 1) were collected in the National Park, which have been grouped according to Brncic (1970) in: (a) widespread species: D.busckii, D.immigrans, D.melanogaster, D.repleta & D.simulans; (b) endemic and ecologically restricted species: D.amplipennis; (c) endemic and ecologically versatile species: D.araucana and D.pavani.

We must add that on this occasion samples of D.subobscura were also collected, which correspond to a colonizing species for Chile (Brncic & Budnik 1980).

Finally we desire to point out that the place chosen for our study presents very interesting biological characteristics, such as the presence of one set of typical Drosophila species, which is found in relation to specific habitats. This event will permit us to carry out important studies on the biology of populations of these organisms.

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Whitmore, T. and W.-E.Kalisch. Ruhr-Universität Bochum, FR Germany. Hoechst 33258 staining of surface spread polytene chromosomes in D.hydei.

The bibenzimidole derivative Hoechst 33258 has been used extensively in the past as a DNA-specific fluorochrome in cytofluorometric investigations of metaphase chromosomes (see for example, Holmquist 1975; Latt & Wohlleb 1975; Wheeler & Altenberg 1977; Singh & Gupta 1982). Its use with polytene chromosomes has been, however, rather limited (Holmquist 1975; Lakhotia & Mishra 1980; Martin & Sedat 1982). We found that it can also be used, similar to

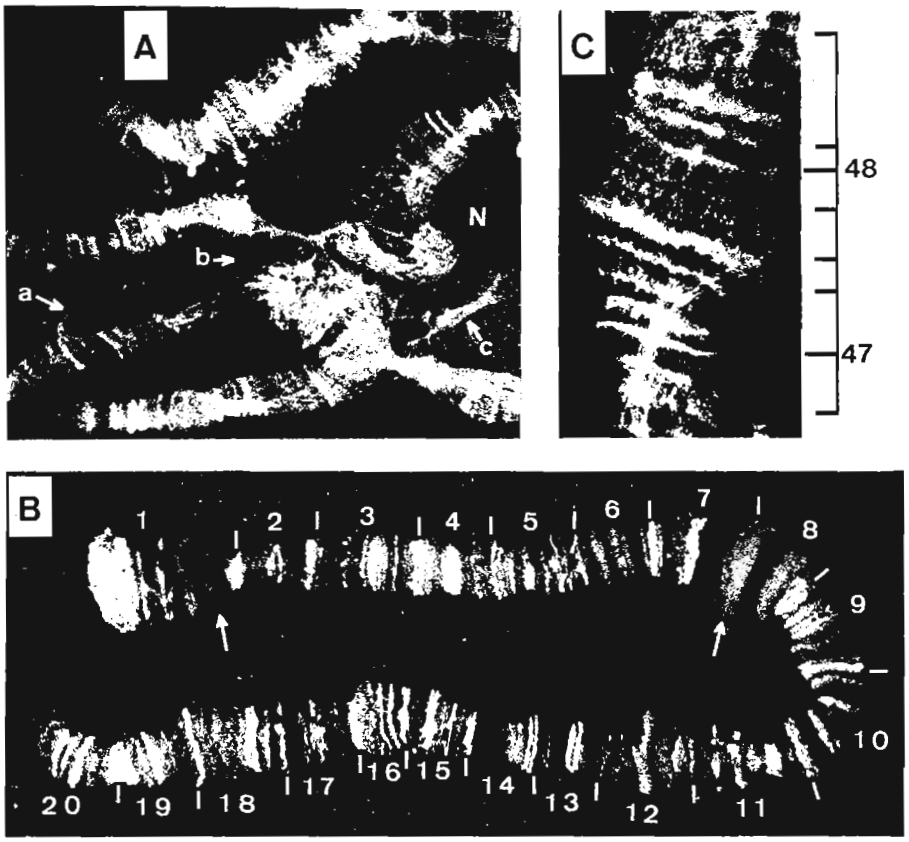


Figure 1. (A) D.hydei, chromoccenter with nucleolus (N), arrows indicate (a) intercalary DNA "ectopic pairing", (b) proximal connections between chromosomes and (c) intranucleolar DNA (nucleolar chromatin thread). x 750
(B) D.hydei, X-chromosome, arrows point to two examples of puffed regions by 1d and 7d/8a. x 550
(C) D.hydei, Chromosome 2, region 46D-48C, showing the band-interbadn pattern of a highly spread chromosome. x 650

corophosphine O (Nash & Plaut 1965; Barr & Plaut 1966), as a simple tool to help demonstrate with salivary gland polytene chromosomes intranucleolar DNA (nucleolar chromatin threads), intercalary chromoregions "ectopic pairing" (Fig. 1A), puffed regions (Fig. 1B) as well as band-interband patterns (Fig. 1C).

Surface spread polytene (SSP) chromosomes were prepared basically as described by Kalisch & Whitmore (1983) with the exception that the crhomosomes were picked up on subbed slides instead of EM grids (for further details on surface spreading of polytene chromosomes, see also: Kalisch & Hägele 1981, 1982; Kalisch 1982a,b; Kalisch & Jacob 1983). The staining with Hoechst 33258 was done with a modification of the method of Lakhotia & Kumar McIllvaine buffer (0.1 M, pH 4.0), rinsed with rinsed with distilled water and mounted in the same McIllvaine buffer, then sealed with rubber cement and stored approx. 24-48 hr in the dark before viewing which helps against photofading. We used a rather high concentration of Hoechst 33258 to ensure sufficient fluorescence of even extremely small chromatin finbers seen for example in the interband regions or those connecting the proximal ends of the chromosomes (Fig. 1A).

Photos were taken using a Zeiss photo microscope coupled with a Zeiss III RS fluorescence attachment and 25/40 Plan-NEOFLUAR oil/water immersion objections on either Agfapan 25 ASA or Kodak Tri-X 400 ASA film.

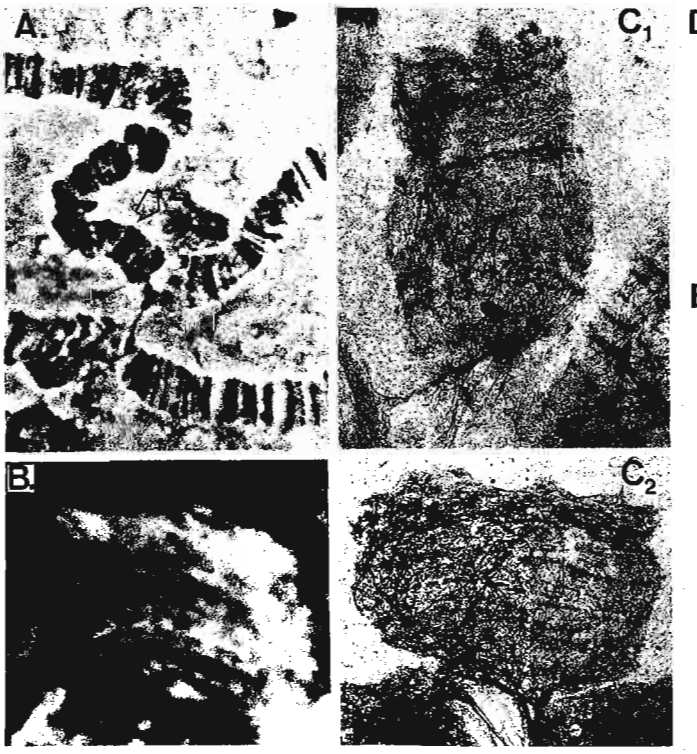
References: Barr,H.J. & W.Plaut 1966, J.Cell Biol. 31:C17-C22; Holmquist,G. 1975, Chromosoma (Berl.) 49:333-356; Kalisch,W.-E. 1982a, Genetica 60:21-24; ____ 1982b, DIS 58: 85-87; Kalisch,W.-E. & K.Hägele 1982, IN S.Lakovaara (ed): Advances in Genetics, Development and Evolution of Drosophila, p1-10, Plenum Publ.Corp, New York; Kalisch,W.-E. & H.J.Jacob 1983, Cytobios 36:39-43; Kalisch,W.-E. & T.Whitmore 1983, Cytobios 37:37-43; Lakkotia,S.C. & M.Kumar 1979, Cytobios 21:79-89; Lakkotia,S.C. & A.Mishra 1980, Chromosoma (Berl.) 81:137-150; Latt,S.A. & J.C.Wohlleb 1975, Chromosoma (Berl.) 52:297-316; Mortin,L.I. & J.W.Sedat 1982, J.Cell Sci 57:73-133; Nash,D. & W.Plaut 1965, J.Cell Biol 27:682-686; Singh,B.K. & J.P.Gupta 1982, Chromosoma (Berl.) 87:503-506; Wheeler,L.L. & L.C.Altenburg 1977, Chromosoma (Berl.) 62:351-360.

Whitmore, T., W.-E.Kalisch and H.Reiling.
Ruhr-Universität Bochum, FR Germany.

An EM map of chromosome 6 of D.hydei.

Among the various species probably the most difficult polytene chromosomes to investigate are the so called "dot" or small rod shaped chromosomes. In contrast to the relatively well defined banding pattern of the longer chromosomes these extremely short chromosomes are characterized by their faint and unclear bands. To complicate the problem further, they are usually attached to or embedded in the chromocenter along with the proximal ends of the other chromosomes. Together, this makes good photo-cytological maps of these chromosomes extremely difficult and rather lacking in detail (see for example the photo-maps by Ananiev & Barsky 1982; Parkash & Rajput 1983).

We found recently working with other polytene chromosomes that a combination of the surface spread polytene (SSP) chromosome technique (Kalisch & Hägele 1981, 1982; Kalisch 1982a,b; Kalisch & Whitmore 1983) and fluorochrome staining with Hoechst 33258 led to an improved resolution over routine squash preparations (in prep., see also Whitmore & Kalisch 1984, this issue). We have attempted, therefore, to provide a more detailed photomap of the dot chromosome of D.hydei using fluorescence light microscopy (Fig. 1 a,b) in addition to transmission electron microscopy (Fig. 1c1-2). We have used in both cases SSP chromosome preparations.



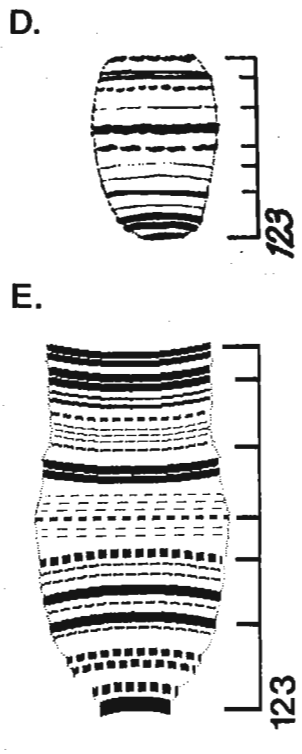


Fig. 1. D.hydei, salivary gland chromosome 6. (A) Squash preparation, orcein stained, indicates chromosome 6. x 1,450 (B) Fluorescence microscope photo of chrom. 6 complexed with DNA specific fluorochrome Hoechst 33258. x 3,200. (C1-2) Two EM micrographs from SSP chrom. preparations showing typical forms of chrom. 6. x 3,200 (D) Chrom. map based on squash preparations (from Berendes 1963). (E) Computerized map based on 6 electron micrographs (width shown is 2:1 reduction due to the limitation of plotter used).

As previously described by Berendes (1963)chromosome 6 of D.hydei is partly heterochromatic and consists of 17(15) concentric bands (Fig. 1d). Its shape is variable, but usually takes on a top- or rod-like configuration. Based on the data obtained from an analysis of 6 EM micrographs we have tentatively included in our computerized map (Fig. 1e) 30 bands (for details on the computerization of polytene chromosomes (see Reiling, Kalisch & Whitmore this issue). We have not included those bands seen only once or twice and which would bring the total number of bands 34.

References: Ananiev,E.V. & V.E.Barsky 1982, Chromosoma (Berl.) 87:239-345; Berendes, H.D. 1963, Chromosoma (Berl.) 14:195-206; Kalisch,W.-E. 1982a Genetica 60:21-24; Kalisch, W.-E. 1982b DIS 58:85-87; Kalisch,W.-E. & K.Hägele 1982, In: S.Lakovaara (ed.): Advances in Genetics, Development and Evolution of Drosophila,p1-10, Plenum Publ. Corp, New York; Kalisch,W.-E. & T.Whitmore 1983, Cytobios 37:37-43; Parkash,R. & P.S.Rajput 1983, DIS 59:96-97.

TECHNICAL NOTES

Achary, P.M.R. and P.K.Dutta. University of Calcutta, India. Apparatus for mutagenizing Drosophila.

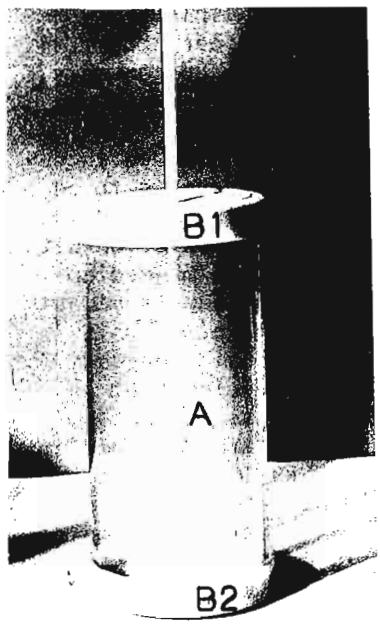


Fig. A Drosophila mutagenizing apparatus.

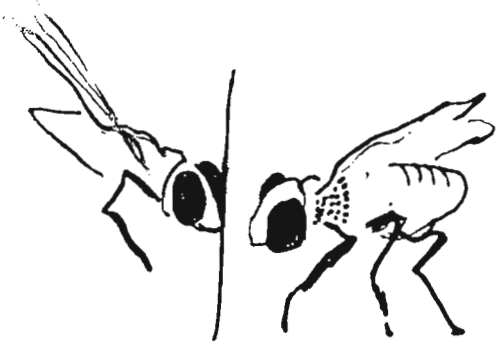
Methods for mutagenization of Drosophila by feeding have been proposed by Lewis & Bacher (1968) and Sharma (1974). We have developed a method in our laboratory which proved to be suitable for mutagenization of Drosophila by feeding.

The apparatus consists of a plexiglass barrel (A) of height 12 cm, diameter 9 cm and two plastic petridishes of diameter 9.2 cm. The plexiglass barrel is fitted on one side with a specially designed etherizer made of a plastic petridish (B1), which consists of small pores on the upper surface and a circular uniform cotton pad pasted to the inner surface. In addition, a hole (of size enough for the insertion of a pastuer pipette) is bored, through which a pastuer pipette is introduced. The other end of the plexiglass barrel is fitted with another plastic petridish (B2) containing filter paper (of diameter 11 cm) or a glass filter paper, as being chemically inert (Lee 1976), saturated with the mutagen solution of desired strength. The filter paper can be kept moist by passing the mutagen solution through the pastuer pipette. Flies to be mutagenized are etherized and placed on the filter paper. After one hour of complete recovery from etherization (Lewis & Bacher 1968) the filter paper is saturated with the mutagen solution of desired strength. Mutagenized flies can be etherized directly in the chamber after the desired time of treatment by dropping ether through the small pores onto the cotton pad of the etherizer, care is taken and seen that the etherizer side of the apparatus is placed downward after removal of the pastuer pipette, otherwise anaesthesized flies will get stuck to the filter paper containing mutagen solution.

In employing this method of feeding we have seen that the number of surviving flies is much high, for the fact that very less number of flies get stuck to the filter paper.

Advantages of the apparatus: (1) a large number of flies can be mutagenized at one time, (2) the apparatus can be easily neutralized after the treatment schedule, (3) the filter paper can be wetted with the mutagen solution through the pastuer pipette from time to time to prevent drying off, (4) the apparatus can also be used for time-gap chemical mutagenesis, e.g., flies can be treated with a certain mutagen for a particular time and then the mutagen treatment can be removed by changing the petridish, containing the treatment medium. After the desired time-gap, same or another mutagen treatment can be resumed by changing the petridish, and (5) unlike glass apparatus it is unbreakable.

References: Lewis,E.B. & F.Bacher 1968, DIS 43:193; Sharma,R.P. 1974, DIS 51:143; Lee, W.R. 1976 (see "The Genetics and Biology of Drosophila", Ashburner & Novitski (eds), V1c, Academic Press, New York).



Backhaus, B., E.Sulkowski and F.W.Schlote. Institute of Biology II (Zoology), RWTH Aachen, FR Germany. A semi-synthetic, general-purpose medium for D.melanogaster.

In the course of long-term experiments on the toxicity, accumulation, and metabolism of heavy metals in Drosophila populations we encountered difficulties in comparing and interpreting results from different laboratories. We understand this as a result of the wide variety of general and specialized culture media that are now available. Most of them are introduced without any comments about pH, amount of living yeast, osmotic values, etc.

For our studies, careful control of nutrition is of great importance. Completely defined, synthetic media are of no use for either population experiments or other long-term studies because they are expensive and laborious to prepare (e.g., Hunt 1970) and can only be turned to good account when sterile conditions are guaranteed. Problems with contaminating microorganisms and low viability stocks grown on complex media (e.g., SPENCER 1950) have often been described. They are not sufficiently defined because they have to be inoculated with live yeast. Semi-synthetic or partly defined media so far published have to be seeded with live yeast in order to warrant reproducible growth of Drosophila (PEARL 1926).

For 4 years, we have worked with a semi-synthetic medium which renders inoculation with live yeast superfluous because an excess of dried brewer's yeast (4.5×10^8 cells/ml medium) and yeast extract. The components are easily stocked and less variable than baker's yeast, maize, cornmeal, etc. This highly reproducible medium is capable of producing from two to three times as many offspring as some standard medium (e.g., SPENCER 1950). The medium is attractive for oviposition and has given good results in cultivating both larvae and adults of Drosophila. Its smooth surface makes it easy to count fertile and non-hatched eggs even after 48 hr from oviposition. No living yeast hinders exact counting, which is when using ordinary agar-baker's yeast media.

Table 1. Ingredients for 1 liter of new medium.

Components	Quantities	Manufacturers
Brewer's yeast (dead, dry)	80 g (4.5×10^8 cells/ml)	DIASANA, Radolfzell[1]
Yeast extract	20 g	DIFCO, Detroit[2]
Peptone	20 g	DIFCO, Detroit[2]
Agar	10 g	FLUKA, Buchs[3]
Sucrose	30 g	MERCK, Darmstadt[1]
Glucose	60 g	MERCK, Darmstadt[1]
$MgSO_4 \times 6H_2O$	0.5 g	FLUKA, Buchs[3]
$CaCl_2 \times 2H_2O$	0.5 g	FLUKA, Buchs[3]
Nipagin mixture (added to food as a 10% solution in 96% ethanol)	10 ml	CAESAR&LORETZ, Hilden[1]
Propionic acid	6 ml	RIEDEL-DE HAENAG, Hanover[1]
aqua bidest.	added to a total of 1000 ml	

1=FRG; 2=USA; 3=Switzerland.

The ingredients required for 1 liter of medium are given in Table 1. For some biometrical characters see Table 3.

Preparation. The ingredients are stirred in one at a time except for propionic acid and Nipagin and brought to a boil. The temperature is then allowed to drop below 60°C and the Nipagin mixture is added, stirring constantly. Propionic acid is stirred into the medium just before pouring it into petri dishes or glass vials with a temperature between 40 and 60°C. It should not be poured too early because brewer's yeast and yeast extract sink down when the agar solution is still too hot and fluid.

When down to room tempera, ture, the vials or dishes are stored at about 8°C until ready to use. No (live) yeast is

Table 2. Some chemical characters of the new medium compared with the standard medium by SPENCER (1950).

Parameter	New Medium	Medium by SPENCER
pE	4.75	5.5
water content	80%	90%
osmotic value	875 mOsmol/l	210 mOsmol/l
Ca-content	400-500 µg/ml	±70 µg/ml
Zn-content	± 10 µg/ml	± 5 µg/ml
(live) yeast	not added	added

added. This medium has been used fresh and after several weeks stored in a refrigerator and was shown to be equally effective in either case.

Table 2 presents some chemical properties of the new medium which are important for the accumulation, metabolism, and toxicity of heavy metals in Drosophila. In comparison, the standard medium of SPENCER (1950) is listed.

Some developmental data of D.melanogaster grown on the medium at 23°C and 75% relative humidity are given in Table 3.

Although no living yeast is added, a certain amount of cells can be regularly found in the presence of D.melanogaster grown on the new medium, even when surface-sterilized eggs are deposited on it (Figure 1). This phenomenon has already been mentioned by BEGON (1974). We could show that on our medium these amounts of yeast are of no nutritional importance for the development of Drosophila: Experiments on the effects of Cd-accumulation, the results of which are described elsewhere (in prep.), showed 5 µg Cd/ml medium to be highly toxic to baker's yeast, but developmental time and survival rates of Drosophila were not effected by this concentration. On standard medium (SPENCER 1950), however, even lower Cd-concentrations provided negative effects not only on yeasts but also on flies. Therefore, live yeast is not a major source of food for developing larvae in our medium. This argument is supported by the fact that we have an excess of brewer's yeast (4.5×10^8 cells/ml medium) compared with live yeast cells.

Conclusions. While this recipe is more expensive than media using baker's yeast, maize, etc., we feel its advantages offset the extra costs: Inoculation with live yeast is rendered superfluous. The medium allows standardization of culture conditions and has shown no contaminations with fungi or bacteria if handled in the described manner. The simple handling of its preparation and the developmental data of D.melanogaster grown on this medium justify its general use.

Table 3. Some cultivation data of D.melanogaster grown on the new medium in an air-conditioned room (23°C, 75% relative humidity).

Parameter	Cultivation Data
Optimal culture conditions	
FLY DENSITY[1] ≦ 25 PAIRS/20 CM^2	MEDIUM:
total egg number	20-50 eggs/female a day
non-hatched eggs	5-10% of total egg number
FLY DENSITY[2] ≦ 50 PAIRS/20 CM^2	MEDIUM:
mean life span of adults	Females: 50% mortality after 30 days Males: no significant mortality before 40 days
EGG DENSITY[3] ≦ 50 EGGS/CM^2	MEDIUM:
mean developmental time from egg to adult	10 days + 8 hr (highly reproducible)
survival rate from egg to adult	80-90%
larvae mortality	± 5%
pupae mortality	± 1%
mean dry weight	Females: 280 µg; Males: 220 µg;
Mass culture conditions[4]	
yield of flies	about 1300 flies/vial=250% of standard medium conditions (e.g. SPENCER 1950)

1= several fly densities from 10 to 25 pairs/20 cm^2 medium with 3-6 days old flies (pre-fed as larvae on standard medium) were created in order to measure the following parameters. Egg numbers and percentage of eggs hatching decline with the age of flies.

2= the medium was renewed every 2 days.

3= 25 pairs of flies aged 3-6 days (pre-fed as larvae on standard medium) were allowed to deposit eggs for 24 hr. The adults were removed and their progeny cultivated for the following parameters to be measured (depth of medium: 1cm).

4= 10 pairs of flies aged 2-4 days were allowed to deposit eggs for 7 days in 1/2 pint bottles containing 35 ml medium. The adults were removed and their total progeny counted.

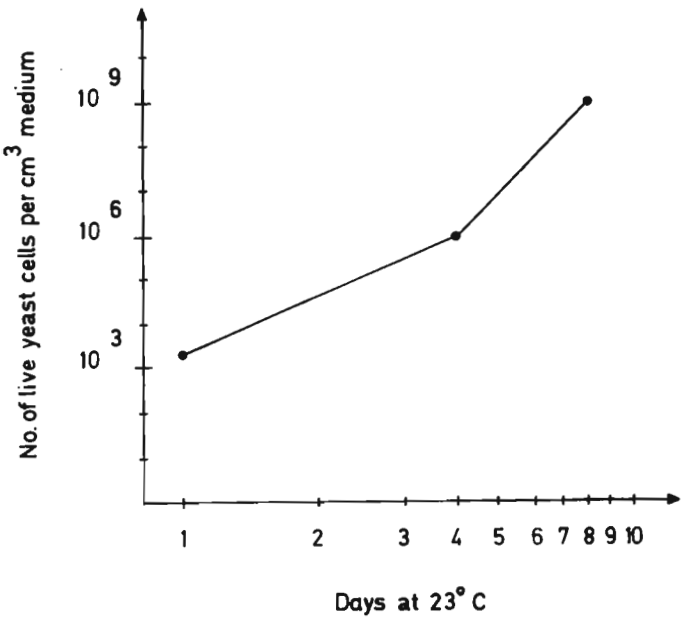


Figure 1. Growth of baker's yeast in the new medium in the presence of D.melanogaster. 25 pairs of flies aged 3-5 days were allowed to lay eggs for 24 hr before day 1. The adults were removed and their progeny cultivated for 8 days. Yeast cells were counted immediately after the removal of adults (day 1), with second instar larvae growing in the medium (day 4), and after all larvae having left the medium for pupation (day 8). From: Köhne, A., A method for determining yeast growth in the medium of D.melanogaster (examination paper, Aachen, 1982, unpubld.).

Acknowledgements: We want to thank M. Dohms for her careful technical assistance and would like to express our appreciation to A.Köhne for her examination-paper utilized in this study.

References: Begon,M. 1974, DIS 51:106; Hunt,V. 1970, DIS 45:179; Pearl, R. et al. 1926, Am.Nat. 60:357-366; Spencer,W.P. 1950, Collection and laboratory culture, IN: Demerec,M. (ed) Biology of Drosophila, Wiley, New York.

Band, H.T. Michigan State University, East Lansing, Michigan USNA. A high protein medium using soybean protein flour.

The removal of Kellog's Concentrate from the market has created problems for Drosophila workers doing research with species requiring a high protein medium. Two such media used this ingredient (Wheeler & Clayton 1965; Band 1981). In our laboratory we used a high protein diet preparation for a year in place of Kellogg's Concentrate, but this and similar products have been withdrawn from the market. Kellogg's NutriGrain Wheat did not adequately maintain fertility in Chymomyza amoena.

We have found soybean flour to be an acceptable substitute for Kellogg's Concentrate and the high protein diet preparations. The product we use is called Vibrant Health Protein Powder from Michigan Vitamin, Ferndale, MI 48220. We have also continued to use Kellogg's NutriGrain Wheat in our medium since it lists vitamins not specifically mentioned in other ingredients. The following recipe is our current high protein medium:

15 gm Gerber's Hi-Pro	500 ml Spartan applesauce
15 gm Kretschmer's Wheat Germ	650 ml distilled water
5 gm Kellogg's NutriGrain Wheat	45 gm Quick Cream of Wheat
5 gm soybean protein flour	3 ml proprionic acid
7 gm Bacto-agar	9 ml 95% ethyl alcohol

To Prepare: Blend the first 4 ingredients in a Waring Blender for several minutes. Add the applesauce and blend 5 min more. Boil 450 ml of water in a large vessel, add agar and stir to dissolve. Add the applesauce mixture; rinse the blender with 100 ml of water and add to the food mixture. Add the remaining 100 ml of water to the cream of wheat and stir it into the food mixture as it begins to boil. Reduce heat and stir until thickened, usually about

5 min. Remove from heat, continue to stir to cool. Add the propionic acid and ethyl alcohol. Pour into food cups or vials. Store in a refrigerator after the food has cooled.

References: Band,H.T. 1981, DIS 56:171; Wheeler,M.R. & F.E.Clayton 1965, DIS 50:98.

Barr,C. and L.Søndergaard. University of Copenhagen, Denmark. An efficient safety etherizer without health risk.

Ether vapour is the most widely used agent for the immobilization of Drosophila. Being an organic solvent, ether has a potential health risk. Exposure to even low concentrations of ether vapour may give headaches, irritation to eyes and nose as well as dizziness. Long-term exposure may lead to permanent brain damage.

In our lab alternatives to ether have been tried (CO_2, triethylamin, and dichlormethan). However, they all show undesired side effects (too easy to overexposure, too short immobilization time, toxicity, etc.). Due to the increased attention of the health authorities to laboratory safety and to prevent ether pollution of the fly room, a closed etherizer which is permanently connected to a ventilation system was constructed. After installation of this etherizer with forced ventilation, it has not been possible to detect ether vapour in the fly lab, and all inconveniences of ether in the laboratory have been removed.

The etherizer consists of a vented ether container and an air lock, all made from aluminum (i.e., no light and hence no peroxide formation). Flies are introduced into the etherizer via the air lock by a special perspex cartridge which matches the vials used in the lab so that the flies are easily transferred from the vial to the cartridge. Due to the air lock ether will not escape from the apparatus not even when the flies are introduced into the etherizer. Before the flies are removed from the etherizer, they are effectively vented to remove ether vapour adherent to the flies. A sealed compartment houses an adjustable electronic timer/alarm system. The timer is adjustable from 1 to 20 sec. and starts automatically when the flies are introduced into the etherizer. At the end of the preset exposure time an acustic and optic alarm is turned on. The flies are vented while in the car-

FIGURE 1. A SAFETY DROSOPHILA ETHERIZER.

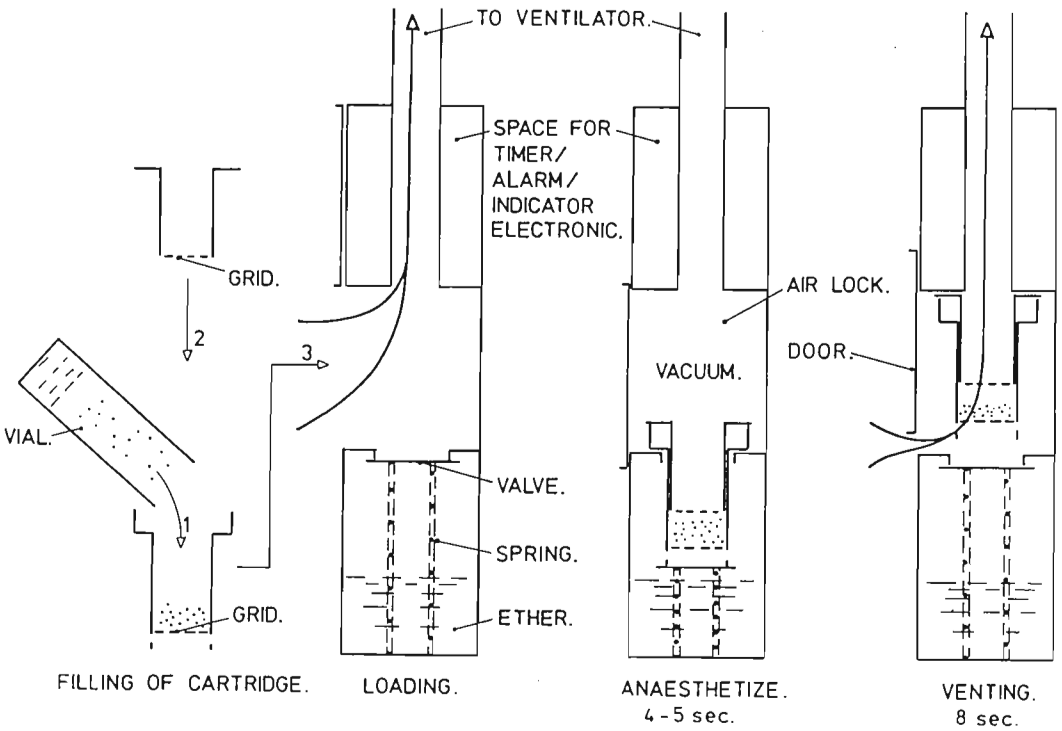
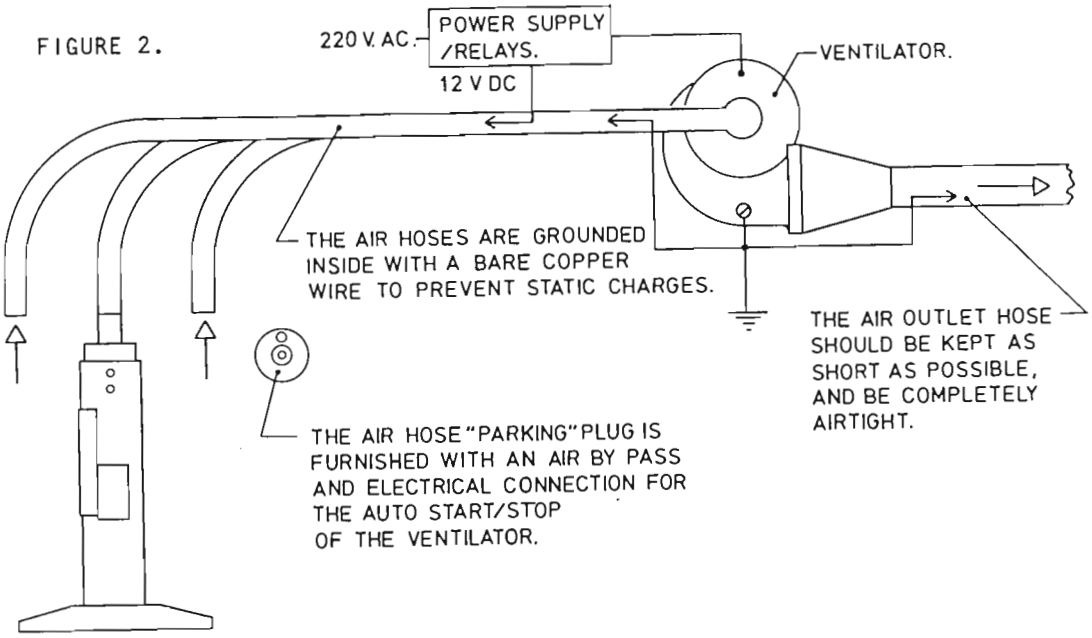


FIGURE 2.



A LAB SYSTEM CONSISTING OF THREE SAFETY ETHERIZERS.

tridge in the etherizer, and after 8 sec an acustic alarm indicates the end of the venting period. If the door of the air lock is left open, an alarm indicates this after 16 sec. Typically, after 5 sec exposure the flies are immobilized for about 3 min.

The vent system automatically switches-on when being connected to the etherizer, and sufficient air bypass prevents accumulation of ether vapour in the system. The whole electrical and air system is grounded to prevent static electricity.

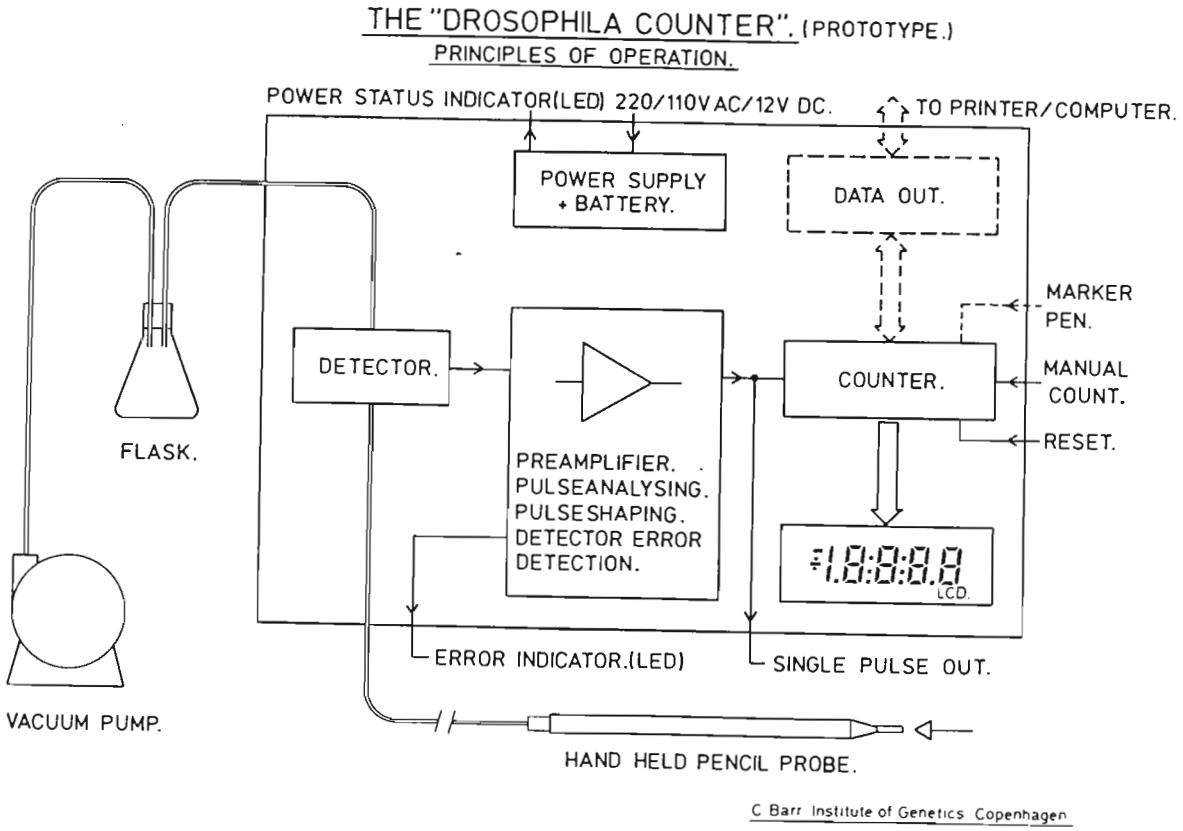
Ether consumption by the new etherizer is very low. The average consumption was 0.3ml/hr over a 50 day period (max. 1 ml/hr during constant use). From this it can be calculated that the maximal ether vapour concentration in the vent system is 1/1400 of the explosion limit of ether. The etherizer can be installed with no expensive rebuilding of the laboratory.

The etherizer can be made by an well-equipped workshop. Request further information from C.Barr, Inst. of Genetics, Univ. of Copenhagen, Øster Farimagsgade 2A, DK-1353 Copenhagen K, Denmark.

Barr, C. and L.Søndergaard. University of Copenhagen, Denmark. A reliable Drosophila counter.

In many Drosophila experiments (mutagenesis, non-disjunction, population experiments, etc.) considerable effort is used in the process of counting enough flies to get statistically reliable results. In order to diminish the counting time an electronic fly counter was designed. Prototypes of the counter have now been in use for more than one year in our lab and in Professor E.B.Lewis's lab at Caltech; millions of flies have been counted without technical problems and the counting time has been reduced by a factor 3-4; 3000 flies are easily counted in half an hour.

Anaesthetized Drosophila are sucked up with a pencil-shaped probe. The air flow is delivered by a small membrane vacuum pump. Flies travel along a plastic hose into the counter where they pass a photodetector. The electrical signals from the photodetector go to an amplifier, delivering TTL pulses to the internal counter display and to an output for external data computing. The instrument is furnished with an internal rechargeable "back-up" battery to prevent loss of counts in the event of a power failure. With fully charged battery and no outside power, the counter will perform and keep counts for one-half hour.



The counter is most conveniently operated while sorting the flies on a large piece of cardboard (A4 size). During the genotyping or sexing of the flies they are separated with a scalpel or a fine artist's brush so that the distance between the flies is at least 2 mm. The flies are alive and undamaged after they have been counted. Operated this way the counter makes the counting and sorting very effortless and more importantly the operator can concentrate on the pertinent part of the experiment, i.e., the genotyping or sexing of flies. In a population cage experiment in our lab one person has easily sorted and counted egg samples containing about 20,000 flies in less than a day. Accuracy: -1.3%10.9% counts.

A control light on the front panel indicates if the detector is contaminated or the light source is worn out. This has not yet happened, but when it does, it can easily be fixed by a person without technical background.

The counter is now under preparation for commercial production and will soon be available. Request information from C. Barr, (see also announcements section of this issue): Inst. of Genetics, Univ. of Copenhagen, Øster Farimagsgade 2A, DK-1353 Copenhagen K, Denmark.

Bourgin-Rosenberg, M. and S.Paumard.
University of Paris VII, France.
The "double subculturing method."

For different reasons, it could be often useful to reduce the subculturing frequency of Drosophila stock cultures.

We have developed a "double subculturing" method which can at any wanted temperature, increase twice the period required between two subculturings.

For the first subculturing, 5 ml of standard medium is poured into scintillation counter vials (Figure 1), inclined, to have the maximum of surface for egg-laying. These vials have two advantages: first they can be capped full ofmedium and stored at -20°C as long as wished; secondly, they are cheap enough to be discarded after utilization.

Twenty pairs of flies of the desired strain are introduced into these vials (screw-caps are replaced by foam plugs) where the females are allowed to lay eggs for three days at 25°C.

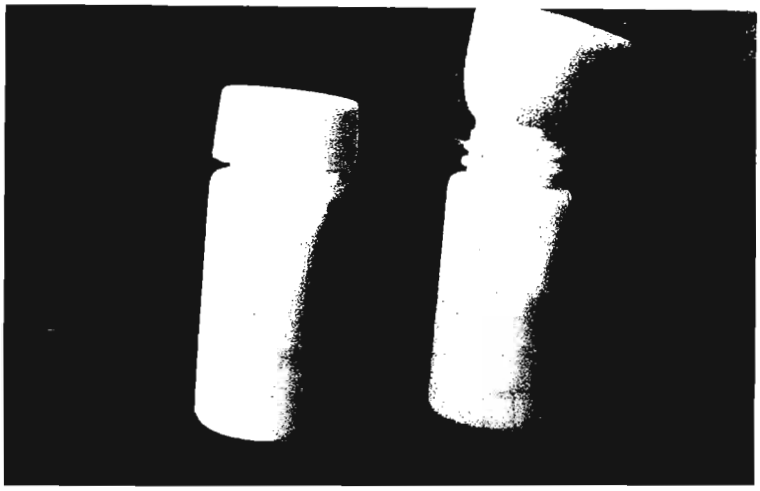


FIGURE 1.

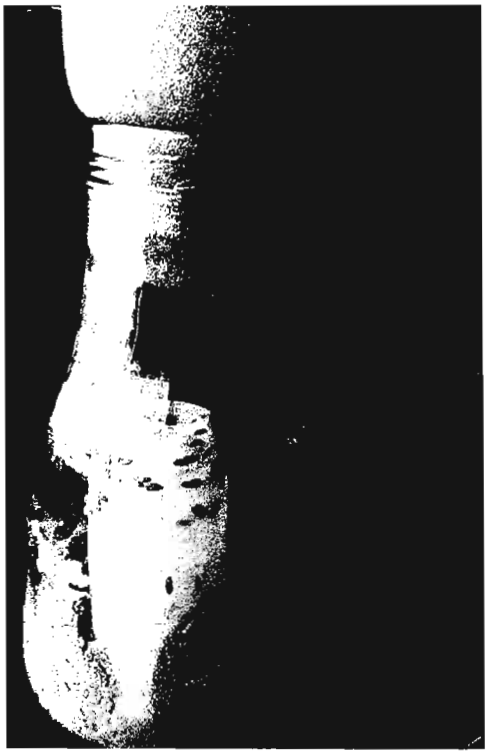


FIGURE 2.

The parents are then removed and each vial is introduced into a bottle containing standard medium: the bottom of the alcohol cleaned vial is lightly sunk into the agar medium. The whole is allowed to develop in an incubator at the desired temperature.

This procedure has the advantage that eggs, larvae and even pupae stay into the vials or on its walls (Figure 2). Therefore, when the flies of the first generation emerge, the medium in the bottle is nearly intact: it is not tilled. Although no subculture has been made, the females of the first generation will lay their eggs on a fresh medium where later larvae will develop, giving rise to the second generation of flies.

Brooks, L.D. Harvard University, Cambridge, Massachusetts. A new multiply marked third chromosome of Drosophila melanogaster.

I created a third chromosome that has a more even distribution of eight recessive markers than rucuca does. The markers and Lindsley & Grell (1968) map positions on chromosome three are:

ve	h	th	cu	sr	e^s	ro	ca
0.2	26.5	43.2	50.0	62.0	70.7	91.1	100.7 .

This chromosome arose as a double recombinant between ve h th cu e^s ro ca (kindly supplied by Dr. R.Grell) and ru h th st cu sr e^s ca (rucuca from Bowling Green, Ohio). It was extracted, starting with one male and crossing with TM3, Sb Ser/Ly st (from Davis, California) females for 5 generations, to establish a stock that is homozygous for the marked third chromosome and has other chromosomes from the TM3 stock. The stock has good viability and fertility. It may be obtained from the Bowling Green stock center.

Reference: Lindsley,D.L. & E.H.Grell 1968, Carn.Inst.Wash.Publ. 627, Genetic Variations of D.melanogaster.

Craymer, L. California Institute of Technology, Pasadena, California USNA.

A pericentric inversion screen.

The following screen was devised in the course of development of techniques for manipulating pericentric inversions (Genetics 99:75-97, 1981). The screen operates to recover pericentric inversions as translocations between free-armed chromosomes. Figure 1 illustrates the basic idea and diagrams a T(2;3)rn, D^3 Sb Ubx/ F(2L); F(2R); F(3L); F(3R) genotype and the euploid gametes which it produces. A male of this constitution produces the four euploid gametic types shown: (a) T(2;3), (b&c) half-translocation plus complementary free arms, and (d) F(2L); F(2R); F(3L); F(3R). A translocation between either the F(2L) or F(3L) and either the F(2R) or F(3R) will cause the (b&c) gametic types to be aneuploid and result in the lethality of zygotes produced by the fertilization of a euploid egg by either type of sperm. Thus T(2L;2R)'s or T(3L;3R)'s will cause D^3 to show pseudolinkage with Sb and Ubx [T(2L;3R)'s and T(3L;2R)'s will also cause this pseudolinkage, but these are easily screened out in later generations as behaving like T(2;3)'s].

By using Sb and Ubx as lethals, the screen can be simplified to the point that one need only look for cultures lacking D^3. The T(2;3)rn, D^3 Sb Ubx/free arms males can be mated to Sb/In(3R)Ubx^{80} (or other rearranged Ubx: the rearrangement prevents crossing over between Sb and Ubx): this prevents the (a&b) gametes from being recovered in surviving progeny, and the (c) gametic type--carrying D^3--will not be recovered if an appropriate translocation has been induced.

Three stocks have been built for this screen: (1) 2^PB238; F(2R)VH2; 3^PJ17; 3^PJ139--the free-armed stock. 2^P238 was derived from T(Y;2)B238 (Lindsley-Sandler) so that the short arm of 2^PB238 is capped with the tip of the X and variegates for y; 3^PJ17 and 3^PJ139 were also derived from Lindsley-Sandler translocations, and 3^PJ139 carries a variegating B^S.

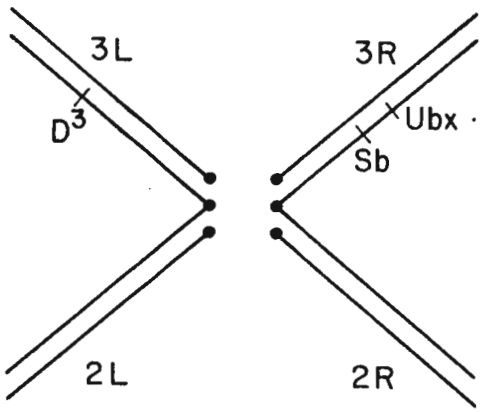
(2) T(2;3)rn, D^3 Sb Ubx/In(3LR)C190

(3) C(1)M4, y^2/shi^{ts}; or If; Sb/In(3R)Ubx^{80}. shi^{ts} is present to automate virgin collection; for collecting virgins, one need only to clear the cultures and put the bottles at 28°C.

The screening crosses are:

P_0 T(2;3)rn, D^3 Sb Ubx/In(3LR)C190 females x 2^PB238; F(2R)VH2; 3^PJ17; 3^PJ139 males (irradiated)

F_1 C(1)M4, y^2; or If; Sb/In(3R)Ubx^{80} females (5 to 10 per culture) x T(2;3)rn, D^3 Sb Ubx/free arms males (1 per culture)



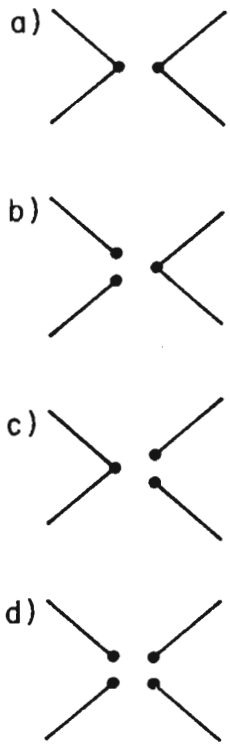


Figure 1. T(2;3)rn, D^3 Sb Ubx/2^PB238; F(2R)VH1; 3^PJ171 3^PJ139 and the four types of euploid gametes normally produced by this genotype:

a) T(2;3)rn, D^3 Sb Ubx

b) 2^PB238; 3^PJ17/2R.3R, Sb Ubx.

c) 2L.3L, D^3/F(2R)VH1; 3^PJ139.

d) 2^PB238; F(2R)VH1; 3^PJ17; 3^PJ139.

F_2 Look for cultures lacking D^3 (this can be done without etherization). From these cultures, test for the presence of a T(2L;3R) or T(3L;2R) by crossing or If; Sb/free arms males to structurally normal females and discard any cultures which show pseudo-linkage of If and Sb. Isolate stocks of the putative In(2LR)'s and In(3LR)'s and check for inversions cytologically.

A small scale test (about 300 F_1 males tested; 4000 r) yielded one In(2LR), five In(3LR)'s, and one translocation between 3L and the short arm of 3^PJ139. These are further described under New Mutants (this DIS). Techniques for freeing the inversions from the free arm complex are detailed in Genetics 99:75-97.

Engeln, H. Institut fur Genetik, Freie Universitat Berlin, FR Germany. Apparatus for measuring temperature preferences in Drosophila.

For measuring temperature preferences of adult Drosophila in short time experiments with many replicates a smaller and more simple apparatus as that one presented by Fogleman (1978) may be sufficient. Our thermal gradient field consists of a sheet of aluminum heated at one end by a heating flex and cooled by circulating water at the other end (Figure 1). Heating is controlled by a rheostat and a contact-thermomemter switching the flex on and off. Continuously circulating cold water is obtained by a small laboratory cooler. Different stable temperature gradients can be adjusted in this way. In Figure 2 temperature profiles are shown along the centre line and along the margins of the aluminum sheet.

Two cages, each consisting of three observation chambers, run parallel with the center line (Fig. 1). These chambers are made from transparent plexiglass without any bottom and put directly on the surface of the aluminum sheet. Each chamber is divided lengthwise into 10 fields by optical marks. To avoid influences of different degrees of relative humidity caused by the temperature gradient moistened filter paper is placed on the upper horizontal surface of the aluminum, so that about 100% relative humidity will be obtained everywhere in the chambers. For immediate anaesthetization of the tested flies carbon dioxide is conducted through pipes and little holes into each observation chamber (Fig. 1). All remaining parts of the aluminum sheet which are exposed to airflow are covered with styrofoam insulation.

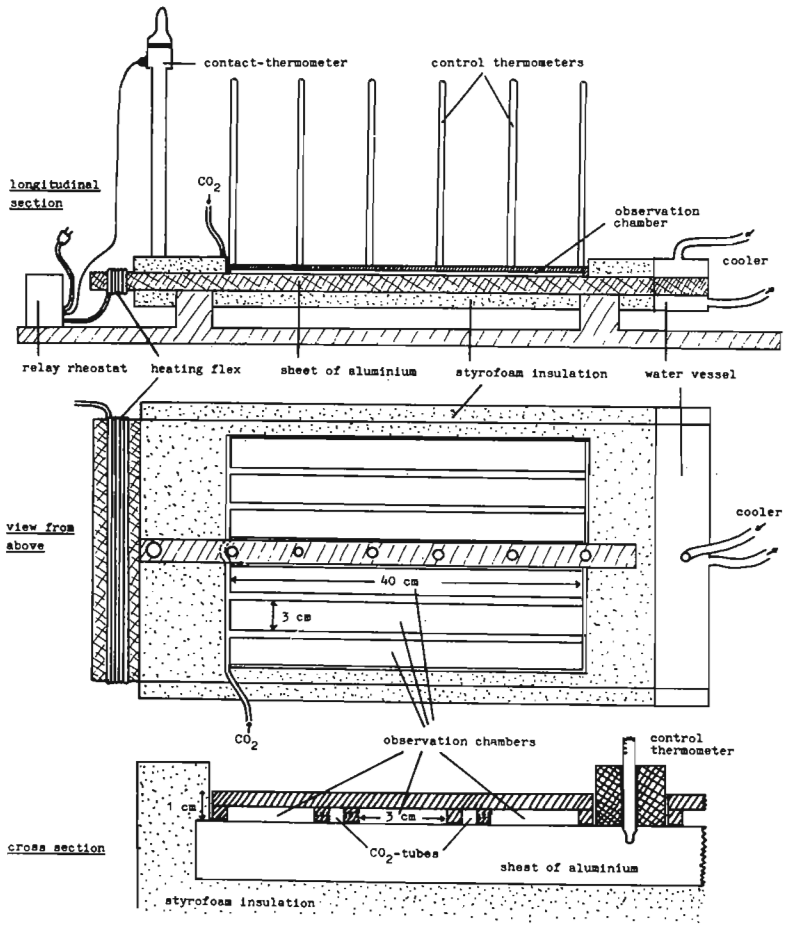


Fig. 1. Apparatus for measuring temperature preferences in Drosophila.

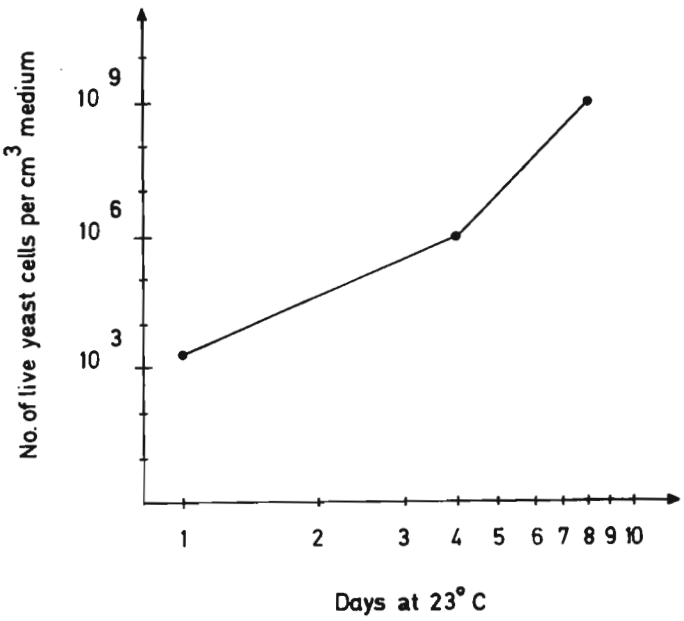


Figure 1. Growth of baker's yeast in the new medium in the presence of D.melanogaster. 25 pairs of flies aged 3-5 days were allowed to lay eggs for 24 hr before day 1. The adults were removed and their progeny cultivated for 8 days. Yeast cells were counted immediately after the removal of adults (day 1), with second instar larvae growing in the medium (day 4), and after all larvae having left the medium for pupation (day 8). From: Köhne, A., A method for determining yeast growth in the medium of D.melanogaster (examination paper, Aachen, 1982, unpubld.).

Acknowledgements: We want to thank M. Dohms for her careful technical assistance and would like to express our appreciation to A.Köhne for her examination-paper utilized in this study.

References: Begon,M. 1974, DIS 51:106; Hunt,V. 1970, DIS 45:179; Pearl, R. et al. 1926, Am.Nat. 60:357-366; Spencer,W.P. 1950, Collection and laboratory culture, IN: Demerec,M. (ed) Biology of Drosophila, Wiley, New York.

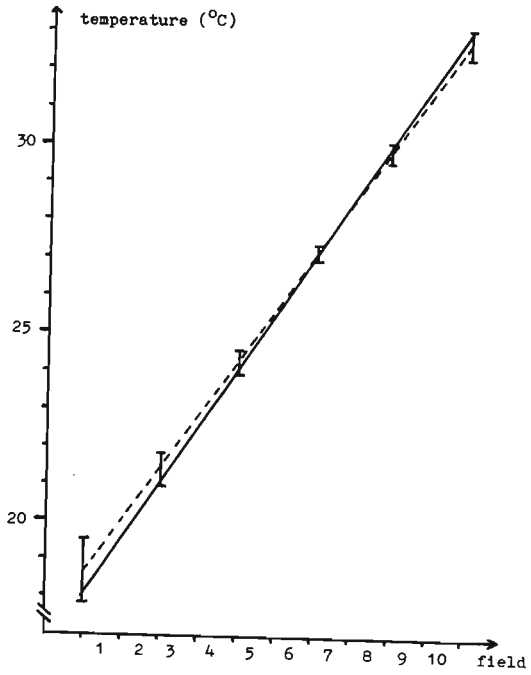
Band, H.T. Michigan State University, East Lansing, Michigan USNA. A high protein medium using soybean protein flour.

The removal of Kellog's Concentrate from the market has created problems for Drosophila workers doing research with species requiring a high protein medium. Two such media used this ingredient (Wheeler & Clayton 1965; Band 1981). In our laboratory we used a high protein diet preparation for a year in place of Kellogg's Concentrate, but this and similar products have been withdrawn from the market. Kellogg's NutriGrain Wheat did not adequately maintain fertility in Chymomyza amoena.

We have found soybean flour to be an acceptable substitute for Kellogg's Concentrate and the high protein diet preparations. The product we use is called Vibrant Health Protein Powder from Michigan Vitamin, Ferndale, MI 48220. We have also continued to use Kellogg's NutriGrain Wheat in our medium since it lists vitamins not specifically mentioned in other ingredients. The following recipe is our current high protein medium:

15 gm Gerber's Hi-Pro	500 ml Spartan applesauce
15 gm Kretschmer's Wheat Germ	650 ml distilled water
5 gm Kellogg's NutriGrain Wheat	45 gm Quick Cream of Wheat
5 gm soybean protein flour	3 ml proprionic acid
7 gm Bacto-agar	9 ml 95% ethyl alcohol

To Prepare: Blend the first 4 ingredients in a Waring Blender for several minutes. Add the applesauce and blend 5 min more. Boil 450 ml of water in a large vessel, add agar and stir to dissolve. Add the applesauce mixture; rinse the blender with 100 ml of water and add to the food mixture. Add the remaining 100 ml of water to the cream of wheat and stir it into the food mixture as it begins to boil. Reduce heat and stir until thickened, usually about



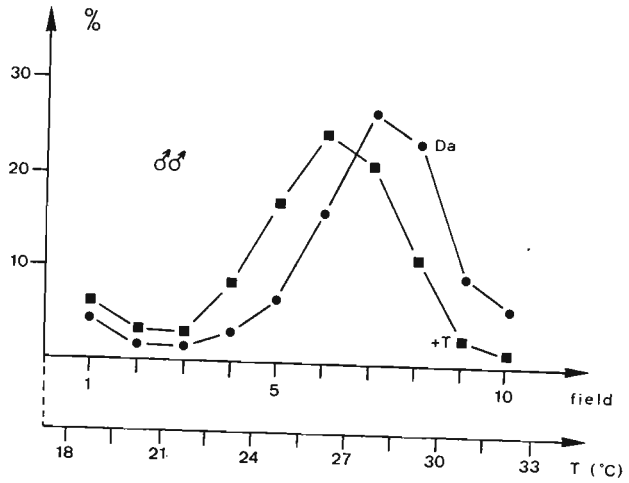


Fig. 3. Distributions of males of two strains of D.melanogaster along the temperature gradient (average of 12 replicates).

Fig. 2. Temperature profiles measured in the center (solid line) and at the margins (dotted line) of the apparatus. The temperature gradient in the center line is very stable and deviations are within the precision of the thermometers (±0.1°C). Vertical bars indicate maximum deviations measured at the margins of the aluminum sheet.

In each replicate about 50 anaesthetized flies are introduced into the center of an observation chamber and are allowed to distribute according to their preferences for about an hour. After this period carbon dioxide flows into the chambers and the flies become immobilized immediately and their number per field is counted. Figure 3 demonstrates the frequency distributions of males of a European and an African population from Berlin (+T) and from Benin (Da) along the temperature gradient. Each distribution is an average of 12 replicates.

Reference: Fogleman,J. 1978, DIS 53:212-213.

Kambysellis, M.P. New York University, New York USNA. A highly efficient method for collection of hemolymph, hemocytes or blood-borne organisms from Drosophila and other small insects.

Manual collection of hemolymph from individual Drosophila is time-consuming, the yield is low, and this method is often frustrating. An alternative method published previously (Kambysellis 1978), although faster and yielding higher volumes, has the disadvantage that unless extreme care is taken, the crop and the gut can be ruptured, and their contents together with cell debris are often obtained along with the hemolymph.

A modified method is presented here for collecting high yields of hemolymph, including the hemocytes, and yet free of other contaminants. The hemolymph is collected by centrifugation in an assembly adapted from the techniques of recombinant DNA work (deBruijn & Lupski 1984). We use two different size Eppendorf centrifuge tubes which precisely fit inside each other, a 0.5 ml tube and a 1.5 ml tube. With a hot needle (gauge #23), we open a small hole in the bottom of the smaller tube, which is then packed with glass wool up to the end of the narrow part of the tube (Fig. 1). The large amount of glass wool provides a cushion for the flies and prevents the squeezing of the flies during centrifugation which often leads to rupture of the crop. It is advisable but not essential to siliconize the glass wool prior to use. The tube with glass wool is then inserted into the larger tube which

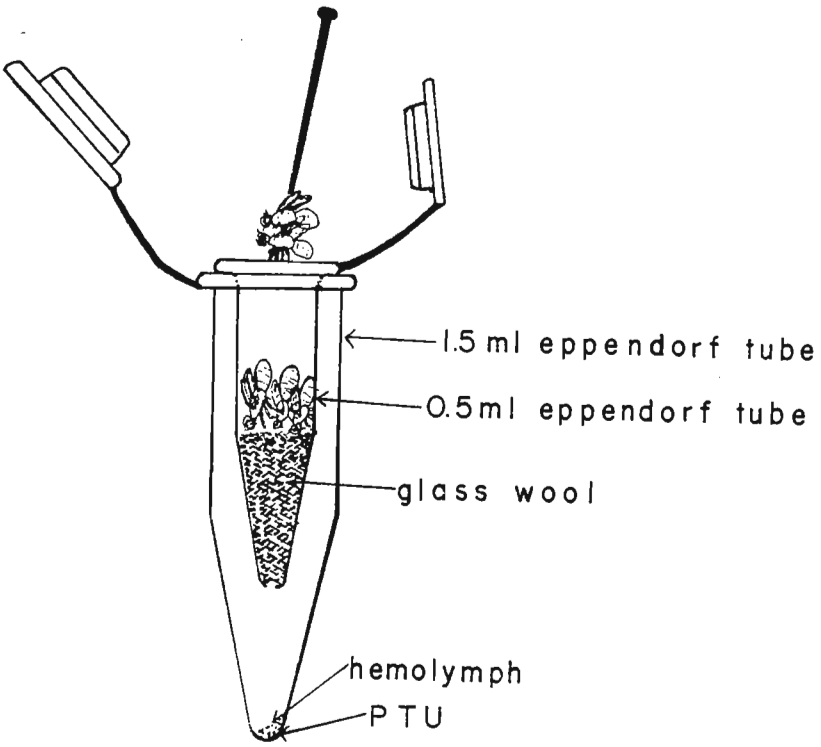


FIGURE 1.

contains 2-3 crystals of phenyl thiourea (PTU) to prevent tyrosinase activity in the hemolymph (Fig. 1). This assembly can now be wrapped in aluminum foil and sterilized if the material which will be collected is to be used for tissue culture work.

When ready to collect the hemolymph, the assembly is placed on ice for 10 min to cool. Under a dissecting microscope, the anaesthetized flies are stabbed in the thorax with #5 forceps or an insect pin, several at a time, and released with another forcep or pin directly into the smaller tube (Fig. 1). It is important in this step not to injure the abdomen of the fly, because the crop or gut will be ruptured and the hemolymph will become contaminated. It is also important to work fast and keep the apparatus cool to prevent protein denaturation in the hemolymph due to the presence of proteolytic enzymes, and also to reduce the chances of sealing of the wound by the hemocytes. We have found that for certain proteins (e.g., vitellogenins and serum proteins), even these precautions are not sufficient to prevent some degradation of the proteins. The degradation products although not detectable on stained SDS-polyacrylamide gels, became apparent in our immunological studies with Western blots, in which picogram quantities of vitellogenins (20-30 pg) can be detected. In such cases, degradation can be prevented by placing a few microliters of a proteinase inhibitor on the glass wool. (We routinely use 5 µl of 1mM phenylmethylsulfonyl fluoride.) If hemolymph protein concentrations are to be determined, then of course the appropriate allowance for the dilution with the inhibitor should be made.

When 20-40 flies are accumulated in the tube (this takes about 5 min), the assembly is placed in a refrigerated centrifuge and centrifuged for 3 min at 3,000 rpm. If a refrigerated centrifuge is not available, you can use a table-top centrifuge, the buckets and adaptors of which have previously been cooled on ice or in the refrigerator for 15-20 min. After centrifugation, the small tube with the flies is removed and discarded if not needed. (We routinely use these flies to dissect out ovaries or extract DNA.) The supernatant hemolymph is then carefully removed from the large vial so that the pellet of hemocytes and any organisms circulating in the hemolymph is not disturbed. The pellet is then resuspended in 100 µl of tissue culture medium or insect Ringer's for washing, followed by recentrifugation for 2 min at 2,000 rpm. The culture medium is then removed and discarded and the cells harvested in the appropriate buffer for a particular experiment. The duration and speed of centrifugation and the number of washings required to free the cells of hemolymph varies for different cell types and the optimal conditions should be determined in pilot experiments.

We have successfully used this technique to collect hemolymph from a variety of Hawaiian Drosophila species (Kambysellis et al. 1984), and to isolate the sex-ratio organisms from several other Drosophila species (Williamson & Kambysellis, unpubl. data). This method has also been adopted in malaria work to isolate Plasmodium knowlesi sporozoites from the salivary glands of decapitated infected mosquitoes (Ozaki et al. 1984).

This work was supported by NIH grant AG 01870 and NSF grant PCM-7913074.

References: deBruijn & J.Lupski 1984, Gene, in press; Kambysellis 1978, DIS 53:218; ____, P.Hatzopoulos & E.M.Craddock 1984, W.Roux's Archiv, submitted; Ozaki,L.S., R.Gwadz & G.N.Godson 1984, J. Parasitology, submitted.

Marenco, M.J., M.Galissie & G.Vaysse.
Université Paul Sabatier, Toulouse, France.

Experimental modifications of the larval nutritive medium in Drosophila melanogaster, and learning process of the imago.

It has been shown that serotonin (5-HT), a chemical mediator synthetized from tryptophan, is present in each larval instar and in the imago of Drosophila melanogaster. In the latter, the 5-HT level in the head is twice that of the whole body. By HPLC, we found about 18 µg of 5-HT for 1 mg of fresh head (18 µg/mg) and about 8 µg/mg in the body.
-- Larvae fed with a synthetic nutritive medium (from Hinton 1951) containing 0.3 g/l of para-chlorophenylalanine (p-CPA): an inhibitor of serotonin synthesis;
-- Larvae fed with a similar medium without an amino acid: the D-L tryptophane.
Both being rehabilitated after the pupal instar, have been observed from a learning point of view.

The tarsal reflex (a proboscis extension in response to a sugar stimulation of the foreleg tarses, refer to Holliday, DIS 59) is normally inhibited, if each extension is followed by a negative reinforcement (a bitter stimulation of quinine on the tarses, in the paradigm of Medioni et al. 1978).

As shown in Figure 1, the treated flies (p-CPA or deprivation group) persist to extend the proboscis in response to a sugar stimulation in spite of the presence of quinine.

Other experiments (habituation of the tarsal reflex itself, locomotor activity, taking-off, sexual behaviour) give arguments to a selective action of 5-HT on this kind of learning.

References: Holliday,M., M.Vargo & J.Hirsch 1983, DIS 59:140; Hinton,T., D.T.Noyes & J.Ellis 1951, Physiol.Zool. 24:335-353; Medioni,J., N.Cadieu & G.Vaysse 1978, C.R.Soc. Biol. 172:961-967.

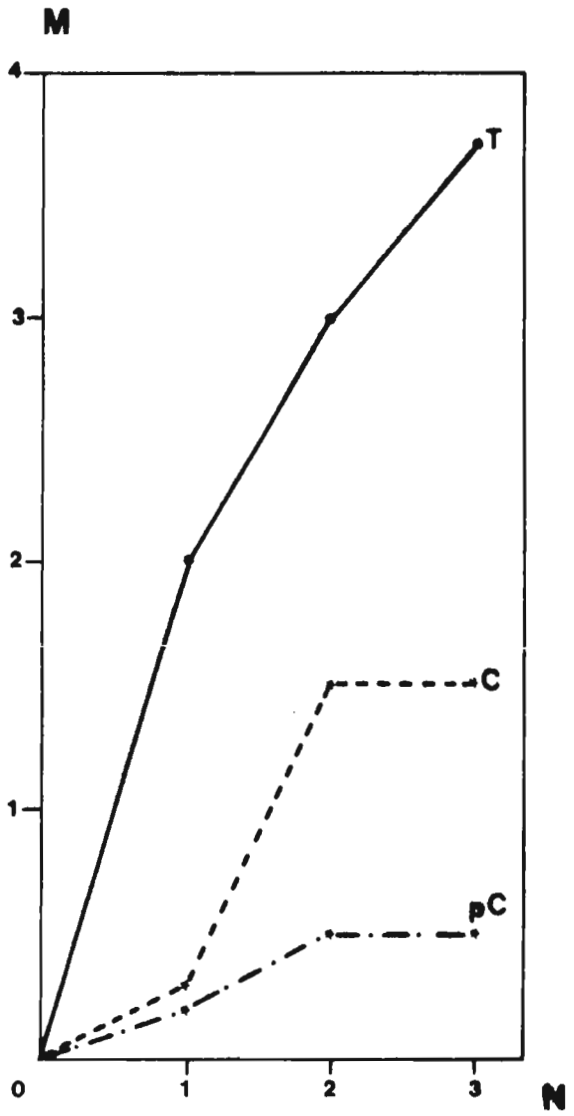


Fig. 1. Mean of eliminations (M) of each of the 3 blocks of 5 tests during the conditioning. T=test group; C=deprived group; pC=group treated with p-CPA.

Miklos, G.L.G. The Australian National University, Canberra, Australia. The isolation of high molecular weight DNA from adult heads of D.melanogaster.

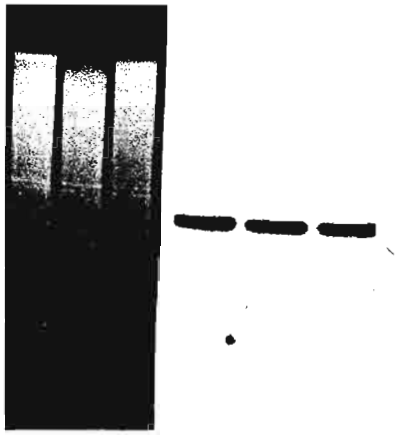
The conventional source of DNA from diploid tissues is early embryos of inbred or homozygous stocks of D.melanogaster. In crosses where chromosomal rearrangements such as deficiencies or duplications are segregating, however, the embryonic pool can contain a mixture of different genotypes and, in general, is an inappropriate starting point for DNA isolations.

Adult heads appear to be an excellent alternative source of DNA since the various genotypes from a given cross can first be identified. Furthermore, the brain contains about half the neural tissue of the adult and is probably near diploid. In brief outline, flies of the appropriate genotype are sorted under the microscope, snap frozen in liquid nitrogen, and stored at -70°C until required. The fractionation of body parts through wire sieves follows the general procedure introduced by Oliver & Phillips (1970). DNA is then extracted and spun to equilibrium in cesium chloride/ethidium bromide gradients. The details of the technique are as follows:

1. Flies of the required genotype are sorted from their sibs, starved for four hours, placed in shell vials and frozen by pouring liquid nitrogen into the vials. When this has evaporated, the vials are stored at -70°C.
2. When required for DNA isolations, liquid nitrogen is again added to a vial containing about one quarter of its volume as adults. As soon as the liquid nitrogen has evaporated, the vial is shaken vigorously half a dozen times. The flies are decapitated by this procedure as well as losing their legs and wings.
3. The shattered debris is quickly poured onto the first of two sieves and quickly moved around with a paint brush. The mesh size of the first sieve (1 mm x 1 mm) allows the heads, legs and wing parts to pass through but retains the bodies. The heads, legs and other small debris are kept cold by immediately pouring more liquid nitrogen into the catchment vessel. The debris is now poured over a second sieve of mesh size (0.5 mm x 0.5 mm) which retains only the heads.
4. The heads are placed in a glass homogeniser, more liquid nitrogen is again added, and upon its evaporation the heads are crushed using a very loose fitting teflon plunger.
5. Approximately 5 mls of ice cold buffer (10 mM TRIS, 20 mM EDTA, pH 8.0) is added and the heads are homogenised at 4°C with half a dozen strokes of a tight fitting teflon homogeniser.
6. The detergent Sarkosyl NL-30 is added to a final concentration of 3% and lysis is allowed to continue for 3 min.
7. 5 mls of the viscous lysate is then added directly to 5 gm of solid cesium chloride in a polyallomer centrifuge tube and allowed to dissolve at room temperature. 0.3 mls of ethidium bromide at 10 mg/ml in TE buffer (10 mM TRIS, 1 mM EDTA, pH 8.0) is added and the solution is overlayed with paraffin oil and spun to equilibrium.
8. Upon completion of the run, the DNA bands (main band and any satellites) are clearly visible with a UV lamp and are withdrawn from the tube by side puncture with a 19 gauge needle.
9. The ethidium bromide is removed by six extractions with isopropanol and the DNA is dialyzed against TE buffer for 16-19 hr.
10. The DNA in TE buffer isethanol precipitated in Eppendorf tubes, resuspended to the desired volume and used as such for restriction digests.

The results illustrated in Figure 1 using this method are from Southern blots of EcoRl genomic digests of Canton S DNA challenged with a radioactive probe made from a cloned Drosophila alcohol dehydrogenase fragment. The same 4.8 kb band is apparent in digests from (a) embryos, (b) ovaries, and (c) male adult heads. No significant DNA amplification or rearrangement events are detectable, as occurs in ovarian follicle cells for the chorion genes (Spradling 1981). We have challenged blots containing DNA from embryos, larval brains, adult ovaries and adult heads with different autosomal and X chromosomal cloned probes and have invariably found that adult heads produce qualitatively and quantitatively similar signals to the above sources.

The DNA from adult heads is of high molecular weight, runs at limiting velocity on agarose gels and cleaves to completion with all restriction endonucleases tested. At least 50 micrograms are available from less than a third of a vial of adults.



a b c a b c

We have found the method to be an easy adjunct to genetic experiments, since instead of discarding the appropriate genotypes at the time of scoring, they are retained at -70°C for use in recombinant DNA experiments. It should also be noted that storage of adults in this way provides an excellent source of undegraded total and polyadenylated RNA from heads as assayed by Northern blots (M.Healy, unpubl.). Since nearly two-thirds of the polysomal RNA complexity of the entire life cycle is present in adult heads (Levy & Manning 1981), adult heads may be a far more useful source of nucleic acids than has previously been imagined.

References: Oliver,D.V. & J.P.Phillips 1970, DIS 45:58; Spradling, A.C. 1981, Cell 27:193-201; Levy, L.S. & J.E. Manning 1981, Developmental Biology 85:141-149.

Rapport, E.[1], J.Kleinjan[2] and R.Dadd[2]. 1-University of Toronto, Ontario, Canada. 2-University of California, Berkeley USNA.

Egg sterilization without dechorionation.

We have adapted a method used for sterilizing mosquito eggs for use with fruit fly eggs. Eggs laid over a 3-5 hr period are loosened with a brush from agar flooded with water. A yeast paste made from sterile killed baker's yeast is used on the agar as an oviposition stimulant. Eggs are collected in a small fine meshed sieve and washed to remove visibly adhering particles. Eggs are then transferred to a sterile 50 ml container with 20 ml of 80% alcohol. Following a suggestion by H.Gordon a vacuum is applied (about 5-10 Hg is adequate) for about a minute. Presumably the alcohol is a wetting agent which is effectively pulled into the interstices of the chorion under vacuum. After 45-60 min in alcohol, using a sterile sieve, the eggs are transferred to a 50 cc sterile snapcap jar with 20 cc aqueous 0.3% Hyamine 10X which has been autoclaved for about 5 min. This compound is available from Sigma as methylbenzethonium chloride. An alcohol sterilized cap is snapped on and the eggs are shaken for 30-40 min. Finally the eggs are placed in sterile water and pipetted into sterile media. In a typical experiment where approximately 10 eggs were transferred to each media vial 85% of the vials remained free of contamination.

Success of this method seems to depend on using only 200-300 eggs per container, using "clean" parents (we do not add live yeast to our cultures) and using "fresh" eggs where there are no hatched larvae. Overnight eggs are less successful.

Rose, V.M. University of Texas, Arlington, Texas USNA.

Method of preparation of Drosophila for scanning electron microscope studies.

Modification of the method of Hodgkin & Bryant (1978) for preparation of Drosophila for SEM studies has resulted in a simple procedure which yields excellent results with minimal distortion of the specimen.

Adult flies were first etherized and submerged in either 70% ethanol or 70% acetone; less damage was observed in the specimens dehydrated with ethanol. In addition, soaking in 70% ethanol for two to three days appeared to reduce damage. The flies were then dehydrated as follows: 70% ethanol for 15 min, 95% for 15 min, and three changes of 100% for 15 min each. Specimens were then placed in sample holders constructed as described by Postek et al. (1980) and slowly critical point fluid. The use of the sample holders necessitates longer diffusion time, but reduces damage due to excessive handling. Whole flies were mounted directly on metal stubs with carbon paint which eliminated charging. Appendages and heads were attached to glass coverslips with double-stick cellophane tape which gives a smooth background; the coverslips were then mounted on metal stubs. Specimens were sputter coated with approximately 200 A of gold-palladium. Specimens were examined with a JEOL 35C scanning electron microscope using an accelerating voltage of 10 kV and a load current of approximately 100 μA. Polaroid 4x5 Land Film Type 55/Positive-Negative produced the best quality prints.

Fig. 1. D.simulans tarsal claw. Acetone dehydration, attached with double-stick tape. Bar = 10 µm.

Fig. 2. D.simulans. Ethanol dehydration, attached with carbon paint.

I thank Dr. H.J. Arnott for the use of his laboratory and equipment, and Mark Grimson for technical assistance.

References: Hodgkin,N.M. & P.J.Bryant 1978, IN The Genetics and Biology of Drosophila, V2c (Ashburner & Wright, eds), Academic Press, New York, p337-358; Postek et al. 1980, Scanning Electron Microscopy, A Students Handbook, Ladd Research Industries, Inc, p.143-144.

Sparrow, J.C. and J.R.Warr. University of York, Heslington, Great Britain.

A new fungicide for Drosophila medium.

Heavy and persistent fungal infections which were resistant to the propdionic acid and Nipagin (Tegosept) which are routinely added as fungicides to the yeast-sucrose-agar medium we use (based on Carpenter 1950) led us to look for a more effective fungicide. Benzimidazole compounds are often used as fungicides in agriculture and horticulture. We now routinely add methyl benzimidazole carbamate as the fungicide for our Drosophila medium. This compound is insoluble in water and only sparingly soluble in ethanol. We make a solution of 200 µg/ml of MBZ in ethanol. (It is effective even though this may not all dissolve unless left for an extended period.) 10 ml of this solution is added to 500 ml of Carpenter's medium after autoclaving. This addition has no discernible effect on fertility, fecundity, development rate or viability of any of our stocks of D.melanogaster or D.hydei. We have not tested for any mutagenic effect and care should be taken if studying non-disjunction as these compounds inhibit microtubular function and effect non-disjunction in fungi. We have now used this fungicide for two years and had no problems with fungal infections during that time. Yeasts appear to grow on this medium. We obtain methyl benzimidazole carbamate from BASF (UK) Ltd., Lady Lane, Hadleight, Ipswich, Suffolk, IP7 6BQ, U.K.

Reference: Carpenter, J.M. 1950, DIS 24:96-97.

Tetzel, H.D. and B.Backhaus. Institute of Biology II (Zoology), RWTH Aachen, FR Germany. An improved electronic counting device for determining large quantities of fruit flies (D.melanogaster).

For counting large numbers of fruit flies (D. melanogaster) we built and electronic counter according to Cuperus et al. (1969,1970). We encountered great difficulties in getting reliable results and hereby want to present some improvements we think to be useful.

A complete circuit diagram of power supply and counting device is given in Fig. 1 and 2, respectively. As to the apparatus originally described by Cuperus et al. (1969,1970), our device differs in the following details:

In the counting head (Fig. 3), 2 light barriers are arranged crosswise in order to increase the accuracy of counting. 2 IR-diodes (3 mWatt) with a maximum energy at 940 nm are used as light sources. The impulses caused by the flies which are sucked through the channel are given to 2 IR-receivers. Their signals are combined in the comparator which works as a summating amplifier (Fig. 2).

Since our device does not have to be shielded from natural sources of light, all 3 channels--the 2 counting channels and the suction pipe--could be fitted in plexiglass. Thus the counting head can be examined from outside which facilitates any repairs or alterations (e.g., cleaning or changing of a scratched glass pipe).

Integrating circuits are used which allow faster counting compared with discrete-component circuits. The threshold frequency in our device is 100,000/s which was tested with a sine wave generator. The minimum distance, d_{min}, between 2 impulses that still guarantees reliable counting can be calculated from the threshold frequency, f, and the air speed,

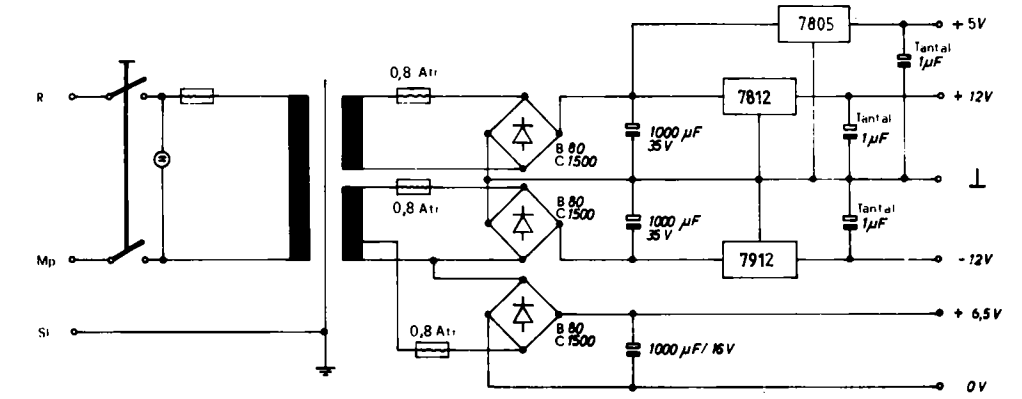


Fig. 1. Power Supply R, Mp, S1 - main connections (220V).

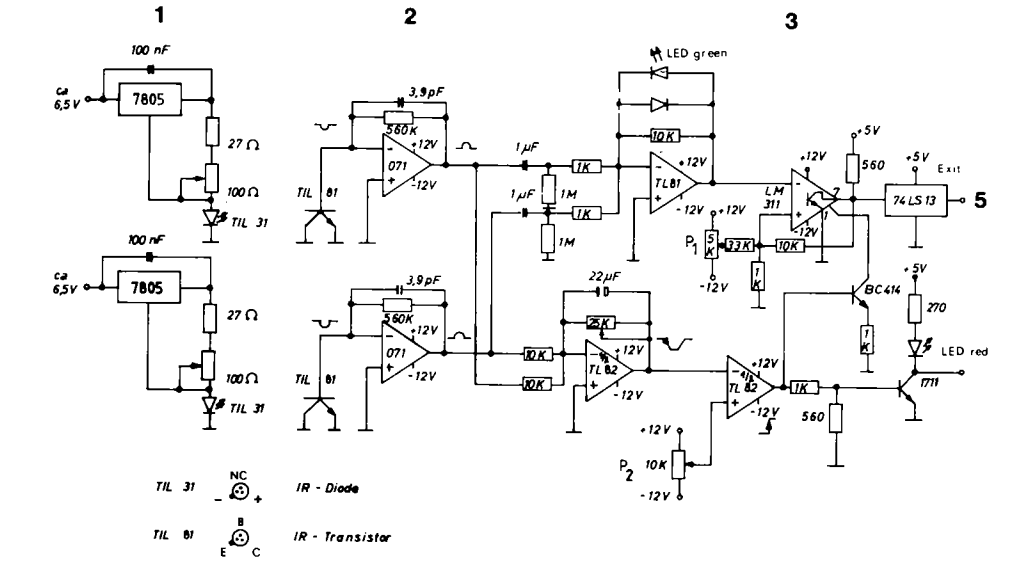


Fig. 2. Complete circuit diagram: (1) constant current supply and light transmitters (IR-diodes). (2) high speed receivers (IR-transistors) and amplifiers. (3) impulse forming device (AC coupled): comparator (working as a summating amplifier) and Schmitt-trigger. (4) transmission control (DC coupled): very low frequency pass combined with an integrator, comparator, and visual indicator. (5) electronic counter. P1,P2 - sensibility for counting of flies and dirt in the suction tube, respectively.

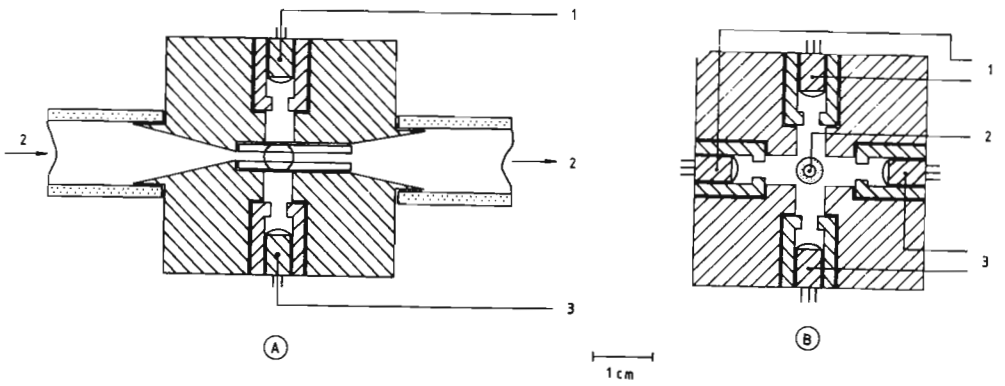


Fig. 3. Counting head:
A Cross-sectional view.
B Longitudinal view.
(1) transmitter.
(2) air flow through suction pipe.
(3) receiver.

v_{air}, according to: $d_{min} = v_{air} \times t_{int}$; with t_{int} = duration of the interval between 2 impulses (for f = 100,000/s and τ = 10 µs we have t_{int} = 5 µs per interval).

The maximum number of flies which theoretically can be counted within a given time, n_{max}, may be determined according to the following equation:

$$n_{max} = \frac{v_{air}}{l_{fl} + d_{min}}$$; where v_{air} represents the air speed at the sensor, l_{fl} the mean fly length and d_{min} the minimum distance between 2 flies being sucked through the channel.

With a suction power of 5001/h and the diameter of the suction tube being 2 mm the mean air speed at the sensor is about 45 m/s. Taking a fly length of 2.5 mm and a minimum distance of 45,000 mm/s x 5 µs = 0.225 mm, theoretically about 15,000 flies may be reliably counted.

With flies as well as with poppy seeds (diameter:<1mm), we found couting deviations of less than 0.5%. Even very small flies--which are found especially in population experiments --were counted with the same accuracy.

Losses of flies being killed during the counting procedure are less than 0.5%.

When the apparatus is used daily, problems with counting accuracy may arise from the dirt being deposited in the suction tube. Therefore, we established a signal that shows optically when the transmission through the suction pipe drops below a critical value. At the same time the counting process is interrupted. This is effected by DC-coupling the impulses caused by the flies and leading them to an extreme low-frequency pass combined with an integrator (Fig. 2).

More detailed information can be obtained on request from the first-named author.

By varying the diameter of the suction tube and if necessary the suction power this counting device may be applied to other biological objects.

References: Cuperus et al. 1969, DIS 44:134-135; Cuperus et al. 1970, DIS 45:176.

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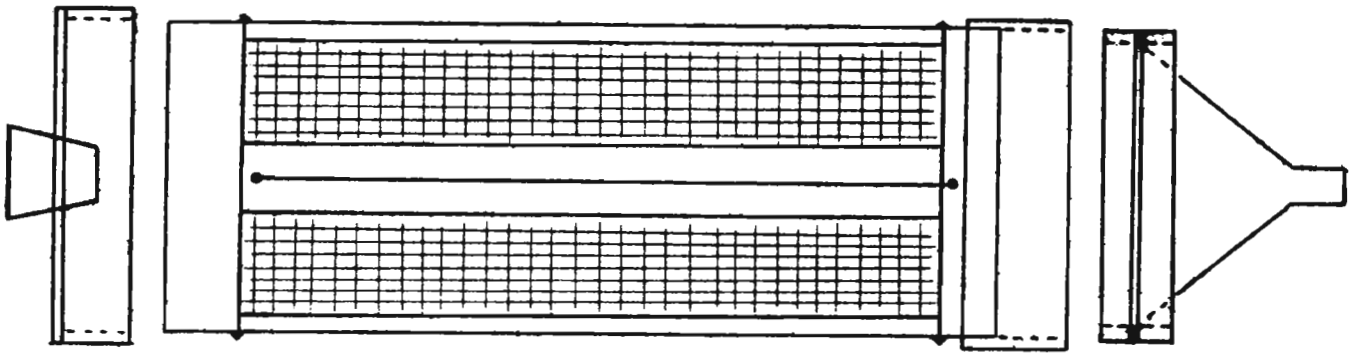
A modular mating chamber for Drosophila.

Although density has been one of many variables studied by those interested in the mating behaviors of Drosophila, it appears that no one has designed a mating chamber in which the density of flies can be altered by incremental changes in the size of the chamber as well as by altering the number of flies in a chamber of constant size. The chamber described here is of modular construction; its size can be varied by combining two, three, or even more of the basic units. The single unit has been used successfully in studying the kinetics of the mating behavior of a sepia strain of D.melanogaster for densities ranging from 5 males plus 10 females to 320 flies of each sex over a half-hour period. Because the results of replicated tests were highly consistent, this mating chamber seems suitable for studying a number of aspects of mating behavior.

The basic unit of the mating chamber is a 12" length of rigid clear plastic tubing $4\frac{1}{2}$" O.D. ($4\frac{1}{4}$" I.D.; i.e., 1/8" wall). Attached to one end is a $1\frac{1}{2}$" sleeve of the same type of



clear plastic tubing cut from a piece of 4-3/4" O.D. (4½" I.D.); this sleeve overlaps the main tube one-half inch. Because of the imprecision in the manufacture of plastic tubing, it may be necessary to cut the sleeve in order for it to fit over the main unit; if so, the gap must be filled by cementing in an appropriately sized piece from a 1½" ring sacrificed just for this purpose.

Vanes of nylon window screening (1¼" strips) are provided as resting surfaces for the flies. These are supported by pairs of crosswires (cut from straightened coat hangers) that are inserted through small holes drilled in the walls of the chamber. One pair is located just behind the sleeve; the other just more than 1" from the opposite end of the tube (so the wires do not interfere with the sleeve of the next module, or with the terminal cap). The wires are fixed in place by brushing clear fingernail polish into and over the holes in the walls of the tube.

The nylon vanes are held taut by attaching one end to 1¼" pajama elastic; the elastic and the nylon strip can then be glued to cloth-mounted picture hooks using Elmer's glue to moisten (and supplement) the glue that is already on the mounting cloth.

Moisture is provided f the flies by a dampened tubular wick that extends (with light weight spiral springs at either end) from the intersecting wires at one end of the chamber to those at the opposite end. The spring tension keeps the wick from sagging against any of the four vanes. The wick is put in place and removed with the help of a small hook shaped from a piece of coat hanger wire.

The chamber is capped at one end by a 1" ring of 4-3/4" O.D. plastic (extended in diameter as needed) to which a circular plate of clear plastic is cemented. A hole bored into the plastic provides a port through which flies may be introduced. The hole should accommodate a 25 mm x 95 mm shell vial; it is closed by a rubber stopper.

A funnel is provided at the opposite end for the removal of flies. A 1" ring of 4½" O.D. plastic (which, of course, can fit into the terminal sleeve of the mating chamber) holds the funnel. A ½" ring of 4½" O.D. plastic from which an appropriately sized segment has been removed is cemented snugly within the 1" ring. A ½ pint Fairgrove funnel (#174, Aluminum Housewares Co., Inc., Maryland Heights, Missouri 63043) with its rim unrolled and flattened fits within the 1"ring and rests on the ½" inner ring. A second ½" ring is then cemented within the 1" ring, on top the flattened rim of the funnel. A rubber stopper fits over the tip of the funnel; nylon curtain material is fastened over the hole in the stopper by means of Divro plastic rubber.

The various components of the mating chamber, when properly assembled, fit together snugly. The pieces are held firmly in place, however, by the use of 3/16" wooden pins that are fashioned from doweling; each pin protrudes about one inch from a "head" consisting of a one-half or three-quarter inch segment of 1" doweling. The large heads make handling of the pins convenient. Holes, 3/16" in diameter, are drilled on opposite sides of each component. In the mating chambers I built, one hole was drilled on either side of a terminal cap; one of these (the black hole) was identified with a Magic-marker pen. This cap then served as the model for drilling holes into each mating chamber. Care was taken to pin the first hole before drilling the second one on the opposite side; for each pair, the Magic-marker was used to identify the black hole. The mating chambers were used in turn as models for drilling holes in other terminal caps and in the sleeves at the ends of the mating chambers; holes in the sleeves served as models for drilling holes in the funnel assembly. In each case, the

pieces were held together as firmly as possible as the new hole was drilled; the first one was then pinned before drilling the second; and the appropriate one in each case was marked with black ink. As a result, the pieces are freely interchangeable. When needed, a fly-tight seal can be made by smearing Elmer's glue on the "male" end of the basic unit (or funnel) and allowing it to stand overnight on brown paper toweling. When the excess paper is trimmed off the following day, a thing paper gasket remains on the plastic wall at the end of the tube. Such gaskets have been sufficient to seal small spaces caused by inaccuracies in the construction of the present chambers.

When flies are to be removed from the mating chamber, CO_2 is led into the unit through the opening in the terminal plate; the tube from the CO_2 tank pierces a cork that fits into the port hole of the terminal plate. The nylon-covered stopper on the funnel allows displaced air to escape without building up pressure within the chamber. *The use of ether in conjunction with mating chambers as large as these would seem ill-advised.*

Because the flies seem to prefer the funnel as a resting place, future tests are planned in which a clear plastic plate, rather than a funnel, will be inserted into the sleeve at that end of the unit.

TEACHING NOTE

Perez-Chiesa, Y. University of Puerto Rico, Rio Piedras PR-USA. Incidence of Drosophila melanogaster flies with melanotic tumors for demonstrating conditionality, penetrance and variable expression.

A sex-linked, temperature-sensitive melanotic tumor mutation in Drosophila melanogaster, tu(1)Sz^{ts} (Rizki & Rizki 1980) is excellent for demonstrating conditionality, penetrance and variable expression with changes in temperature. It also allows for learning the chi-square contingency test and for discussing dosage compensation in Drosophila, as well as other aspects of insect physiology. As reported by Rizki & Rizki (1980) tu(1)Sz^{ts} larvae develop melanotic tumors at 26°C, whereas 18°C inhibits tumor formation. However, penetrance may vary in melanotic tumor strains depending also on genetic background, crowding conditions and food media used (Sparrow 1978).

Experimental Procedure: Students are given two stocks of D.melanogaster: wild type, non-tumor forming strain and tu(1)Sz^{ts}. They set up two cultures of each stock and place them in incubators: one culture of each at 18°C, the others at 26°C. Three days later the parents are removed and their progeny is allowed to continue development at the same temperature at which they started. After eclosion students classify the flies in terms of sex and mutant phenotype: presence of melanotic tumors. The tumors are usually found in the abdomen and less frequently elsewhere. The students are asked to determine whether there are significant differences in the incidence of flies with tumors between the sexes and between the stocks used at each temperature. We have done the experiment at 22°C vs 29°C; there will be tumor formation at 22°C but the incidence of flies with tumors is still significantly different from that of flies grown at 29°C. Cultures can be coded to avoid bias.

References: Rizki,T.M. & R.M.Rizki 1980; Wilhelm Roux's Archives189:197-206; Sparrow, J.C. 1978, IN The Genetics and Biology of Drosophila (Ashburner & Wright eds), V2b, Academic Press, London p277-313.

SUBMITTED STOCK LISTS - D. melanogaster

AMHERST COLLEGE, Webster Center for Biological Sciences, Amherst, Massachusetts USNA 01002

Triploid Stocks

C(1) RM, cm ct^6 sn^3 and FM6 ♀/FM6 ♂
C(1) RM, g^{53d} sd and FM6 ♀/FM6 ♂
C(1) RM, ras^{79f19} m and FM6 ♀/FM6 ♂
C(1) RM, y w and FM6 ♀/FM6 ♂

LAB AM

Chromosome 1

Basc
C(1)DX, y f/cm ct^6 sn^3 oc
C(1)DX, y f/cm ct^6 sn^4 oc ptg
C(1)DX, y f/cm ct^6 sn^{36a}
C(1)DX, y f/ct^6 oc
C(1)DX, y f/ct^n oc
C(1)DX, y f/ec rb^{64f14} cv (see notes)
C(1)DX, y f/oc
C(1)DX, y f/oc ptg
C(1)DX, y f/sn
C(1)DX, y f/sn^2 oc ptg^3
C(1)DX, y f/sn^3 oc
C(1)DX, y f/sn^4 oc ptg^3
C(1)DX, y f/sn^{36a}
C(1)DX, y f/w sn^5
C(1)DX, y f/y^2 sn^3 ras^4 m
C(1)RM, cm ct^6 sn^3 & FM6 ♀/FM6 ♂
C(1)RM, g^{53d} sd & FM6 ♀/FM6 ♂
C(1)RM, ras^{79f19} m & FM6 ♀/FM6 ♂ (see notes)
C(1)RM, y w & FM6 ♀/FM6 ♂
cm
cm ct^6
cm ct^6 sn^2
cm ct^6 sn^3
cm ct^6 sn^4
cm ct^6 sn^{34e}
ec
FM6/y w^{sp}
g
g sd
g^3
g^3 sd
g^{50e}
g^{53d}
g^{53d} sd
g^w
mg^{64b11} f^{36a} (see notes)
ras dy
sd
sn^2
sn^3
sn^3 g^{53d}
sn^4
w
w^a
wy^2
wy^2 g
wy^2 g^2
wy^2 g^3
wy^2 g^4
wy^2 g^{53d}
wy^2 g^{53d} sd
y ct^6 ras^2 f

Chromosome 2

;al cl b c sp^2/SM5
;al cl nub sca^2 sp^2
;al nub lt stw^3 sca^2 sp^2
;b B1 vg bw/In(2L)Cy In(2R)Cy, Cy cn^2 bw^{45a} or ^{45a} sp^2
;B1 L^2/SM5
;cl
;cn bw (not tested for lethals)
In(2L)Cy In(2R)Cy, dp^{1v1}B1 Lt^3 cn^2 L^4 sp^2/ In(2LR) bw^{V1}, ds^{33K} bw^{V1}
;In(2L)t In(2R)Cy, Roi cn^2 bw^{45a} or ^{45a} sp^2/ In(2R)vg^U, vg^U
In(2R)vg^u, S^x Sp vg^U/SM5
;In(2R)vg^U, Sp vg^U lf/SM5
;In(2R)vg^U, vg^U lf/SM5
;lt stw^3
;net^{38j} b^{38j} cn^{38j} bw^{38j}
;Sp BL L^2/SM5
;vg^{51h25}

Chromosome 3

;;bar-3
;;cu gl^3
;;cu ss^{ax}
;;h th st p^p cu sr e^s
;;In(3L)D, D^3/In(3LR)TM3, y^+ ri p^p sep Sb bx^{34e} e^s Ser
;;In(3L)D, D^3/TM1
;;In(3L)D, D^3 Sb/In(3L)P In(3R)P
;;ru h st cu sr e^s ca
;;ru st ss ca
;;se ss $Su(ss)^3$
;;se^{50K}
;;ss bx $Su(ss)^3$
;;ss gl^3
;;ss^{ax}
;;ss^{ax} gl^3

Chromosome 4

;;;ey^2
;;;spa^{pol}

Multiple Chromosomes

;bw;h
;In(2L)Cy In(2R)Cy, Cy cn^2 Sp^2/In(2LR)bw^{V1} ds^{33k} dp b bw^{V1}
;In(3LR)Dcx^f D/In(3R)Mo, Sb sr
;In(2L)Cy In(2R)Cy, Cy cn^2 bw^{45a} or^{45a} sp^2 ; TM3, Sb Ser; T(2;3) bw h, bw h (see notes)
;net or ^{45a} sp^2 ;ru bv
;SM5;TM1;T(2;3)gl^{63d}, gl^{63d} (see notes)
;;TM2;T(2;3)ap^{Xa}. ap^{Xa}
;vg^{51h25};se^{50k}
;vg^{51h25} bw;se^{50k} e
;$vg^{51h25}sp^2$;se^{50k}
;vg^{51h25};se^{50k};spa^{pol}

Notes

rb^{64d14}, $ruby^{64d14}$ 1-7.5. Found by P.T.Ives in one sperm of an Oregon-R ♂ that had been irradiated with 1 kr X-ray. Phenotype like rb.

g^{64b11}, $garnet^{64b11}$. 1-44.4 Found by P.T.Ives in one sperm of an Oregon-R ♂ that had been irradiated with 1 kr X-rays. Phenotype like g.

sca^2 is the allele described as sca^{65131} in DIS 48:16.

T(2:3) gl^{63d29}. Found by P.T.Ives in one sperm of an Oregon-R ♂ that had been irradiated with 1 kr X-rays. It is lethal free and its phenotype is white with yellowish center. A.S.Robinson (Robinson & Curtis 1972, Canadian J. of Genetics and Cytology 14:129-137) puts its breaks at 47B and 91A, the latter being near gl and suggesting that the gl phenotype is a position affect mutation. The T and the mutant have not been observed to separate by crossing over.

T(2:3) bw h found by P.T.Ives after irradiation of bw;h ♂ with 1 kr X-rays. Not studied cytologically and not tested for the amount of crossing over between the T and the markers.

ras^{79f19}, $raspberry^{79f19}$. 1-32.2. Found by P.T.Ives in one sperm from a ♂ that was heterozygous for Massachusetts and Tennessee chromosomes. Phenotype like ras^4. ♀♀ either sterile or produce few developing eggs at 25°C.

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Wild type - Oregon R^+

Mutant Stocks

Chromosome 1

B
Basc (Muller-5)
ct^n otu^1 v^{24}/FM3, In(1)FM3, y^{31d} sc^8 dm B
gt w^a
otu^3 v^{24}/FM3, In(1)FM3, y^{31d} sc^8 dm B
w
y f :=/+ +
y v
y w sn^3
y Df(1)62 gt^8/w^+Y/y f :=/w^+Y
y dnc^{M11} cv v f/FM7, y^{31d} sc^8 w^a sn^{X2} v^{of} g^4 B
y dnc^{M14} cv v f/FM7, y^{31d} sc^8 w^a sn^{X2} v^{of} g^4 B
y sc^4 bb^G sc^8(Y's extra)
In(1)B^{M1}, v B^{M1}
In(1)B^{M2}, v^{rv} B^{M2}
In(1)rst^3, y rst^3 car bb
In(1)sc^{4L} sc^{8R}, y sc^4 sc^8 cv/B^SY/C(1)DX, yf
IN(1)sc^8, y^{31d} sc^8 w^a

Chromosome 2

fes dp^{tr} sp/al^2 Cy lt^3 sp^2
M(2)017/SM5, al^2 Cy lt^v cn^2 sp^2
S fes Alu lt/al^2 Cy cn^2 L^4 sp^2
vg

Chromosome 3

cu
e^s
ecd^{tsl} st ca
1(3)3^{DTS}/TM2, In(3LR)Ubx^{130}, Ubx^{130} e^s
se cu
st ca
Df(3R)GC^9/TM3, In(3LR)TM3, y^+ ri p^p sep bx^{34e} e^s
Df(3R)GC^9/TM6B, h e Tb ca
Df(3R)GC^{14}/TM3
Df(3R)GC^{14}/TM6B
In(3R)$GC^{18L}GC^{23R}$/TM3
Df(3R)e^{Gp4}/TM3
Df(3R)e^{Gp4}/TM6B
Df(3R)e^{H5}/TM6B
In(3R)C cd Df(3R)e^{F4}/ca H e^4 Pr

Chromosomes 1 and 3
gt w^a; e^s
gt w^a; ecd^{tsl} st ca
gt w^a; $1(3)3^{DTS}$/In(3LR)TM2, $In(3LR)Ubx^{130}$, Ubx^{130} e^s
$T(1;3)e^{H2}$, red $e^{variegated}$/In(3R)C Sb e 1(3)4

Chromosomes 2 and 3
vg; se
SM5, $In(2LR)SM5$, al^2 Cy lt^v cn^2 sp^2/+; TM2, $In(3LR)Ubx^{130}$, Ubx^{130} e^s/+
$T(2;3)e^{H1}$: 40-41;93D red e

UNIVERSIDAD DE LOS ANDES, Instituto de Genetica, Bogota, D.E., Colombia.

Markers (mutants):
Vermellon (I°)
White (I°)
Dumpy (II°)
Vestigial (II°)
Cyl//Pm (II°)
Ebony (III°)
Sepia (III°)
Yellow-Forked-Cross-Veinles-Vermellon(I°)
Mutant homeiotico
Muh In Wer din (ultra bithorax)
Y-Sc-Whe-Th-Ls
(gen material loss)

Populations
D.melanogaster - Silvestre (Wulai).
D.melanogaster - Silvestre (Villeta).
D.melanogaster - Silvestre (Col.)

UNIVERSITÄT MÜNSTER, Institut fur Strahlen biologie, 4400 Münster, FR Germany.

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

UNIVERSIDAD NACIONAL MAYOR DE SAN MARCOS, Lab. de Genetica Animal, Lima, Peru.

Chromosome 1
1. f
2. v
3. sc
4. w
5. y

Chromosome 2
6. px
7. bw
8. vg
9. sp
10. dp
11. Cy

Chromosome 3
12. st
13. e
14. tx

Multiple Chromosome
15. B; w^a
16. dp; sp
17. dp; e
18. cv; v; sc
19. y; pn
20. bw; st
21. sc; cv; f; v
22. b; cn; px; bw; sp
23. B1, L^2/SM5

SUBMITTED STOCK LISTS - Other Species

BANARAS HINDU UNIVERSITY, Cytogenetics Laboratory, Dept. of Zoology, Varanasi 221005, India.

D.simulans
- wild type
- y
- y w

anannassae
bipectinata
hydei
jambulina

kikkawai
nasuta
pseudoobscura

UNIVERSIDAD DE LOS ANDES, Instituto de Genetica, Bogota, D.E., Colombia.

D.pseudoobscura:

Markers (mutants)

△ //Ba-gl (II°)
Lor//or Bl-Sc (III°)
or-pr//or-pr (III°)
or//or (III°)
Lor//or-pr (III°)

Karyotype

Pradera SC/SC
Pradera TL/TL
Potosi SC/SC
Potosi TL/TL
Aguas Calientes SC/SC
Aguas Calientes TL/TL

Naturals populations

Potosi - II	Catedra
Potosi - III	Mosquera
Potosi - IV	Sopo
Potosi - VI	Sopo
Potosi - X	Tabio
Potosi - XVI	Chia
Potosi - XIX	Zipaquira
Potosi - XX	Torobarroso
Potosi - XXI	Torobarroso
Potosi - XXII	Aguas Calientes
Potosi - XXIII	Tenjo
Sochagota - I	El Recreo
Sochagota - II	Torobarroso
Torobarroso	Tunja
Torobarroso I (83)	Paipa
Santillana II	Lanceros
Santillana III	Pamplona
San Pedro III	Sochagota
Recreo I (83)	Duitama
Lomitas I	Sogamoso
Pradera IV	Santillana
Aguas Calientes I	Torobarroso
Aguas Calientes II	Aguas Calientes
Aguas Calientes III	

D.willistoni:

Markers (mutants): Pink//Pink

List of "semi-species" of D.paulistorum:

Angra 25 ANDINA del Brazil
Belem AMAZONIA del Brazil
C. Rey ANDINA Tierra baja, Colombia
Gigante ANDINA Tierra baja, Colombia
Fusa ANDINA Tierra baja, Colombia
Raposo TRANSICIONAL
Choco C TRANSICIONAL
Caripe 8 ORINOCA - Venezuela
Llanos 13 A TRANSICIONAL - 13 A
Guiana PAVLOUSKIANA
Marco - 1 ANDINA
Marco - 2 INTERIOR
Mitu 1A, 2A, 1B ANDINA S Tierra baja
Macarena 1 ANDINA
Macarena 2 AMAZONIA
Turbo A TRANSICIONAL
Turbo B "CARMODY"
Lancetilla CENTROAMERICAN
Umaripunta Bosque ANDINA
Umaripunta estacion #2 INTERIOR
Valparaiso 1 ANDINA Tierra baja, Colombia
Valparaiso 2 INTERIOR
Yaquaracaca A 1,2 ANDINAS Amazonas
Yaquaracaca B 1,2 INTERIOR, Amazonas

MICHIGAN STATE UNIVERSITY, Zoology Department, East Lansing, Michigan USNA.

Chymomyza amoena:

wild type stocks
East Jordan, Michigan
Grand Rapids, Michigan
Rose Lake Wildlife Research Area
(East Lansing, Michigan)
Lansing, Michigan

D.simulans:

isofemale lines
Mexico
Amherst, Massachusetts

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	Miami
	Ancash (locality: Yumpe)
D.simulans	Ancash (locality: Callejon de Huaylas)
	Ancash (locality: Monterrey)
	Lima (locality: Iguanil)
D.ananassae	Pucallpa
D.willistoni	Lima (locality: Huando)
D.immigrans	Lima (locality: Huando)
D.polymorpha	Trujillo
D.floricol	Lima
D.buzzattii	Paraguay
	Chil
	Venezuela (locality: Arroyo Escobar)
D.serid6	Venezuela (locality: Palo Labrado)
D.neohydei	Venezuela
D.hydei	Sao Paulo
D.virilis	La Merced
	Cuzco
G.Replete	Trujillo
	Lima (locality: Huando)

UNIVERSITÄT TÜBINGEN, Institut für Biologie II, Lehrstuhl für Populationsgenetik, Auf der Morgenstelle 28, D7400 Tübingen, FR Germany.

D.subobscura

Wild strains
Formia (Italy)
Tubingen (Germany)
Sunne (Sweden)
Caucasus (USSR)
Fort Augustus (Scotland)
Zurich (Switzerland)
Belgrad (Yugoslavia)
Bizerte (Tunesia)
Cinisi (Sicily)

Marker strains
Küsnacht
$(Va/Ba)^{210}$
Oc
y
vg, pp
cn, ma
ch, cu
nt

LINKAGE DATA

Report of W.W. Doane and A.G. Clark.
Arizona State University, Tempe, Arizona.

Three linkage tests independently suggest revision of the map location of the narrow (nw) locus in Drosophila melanogaster, currently placed at 2-83 according to Lindsley & Gre l (1968). The first test employed the cross: S Sp Tft nw^D adp^+ Pin^{Yt}/S^+ Sp^+ Tft^+ nw^+ adp^{60} Pin^+ ♀ x adp^{60}/adp^{60} ♂. Progeny scores were homogeneous over replicates, and a total of 41 recombinants was recovered in the nw-adp interval (N 654). Using the methods of Clark (1981), the maximum likelihood nw-adp interval is 4.21±0.78 map units. This places the nw locus at 2-79.2.

The second linkage test employed the cross: c nw^+/c^+ nw^2 ♀ x c nw^2/Cy ♂. Progeny tests were done to verify phenotypes. Replicates and sexes were homogeneous, and the phenotypic counts after pooling were: 12 curved, narrow; 444 curved; 428 narrow; and 20 wild type. This gives a c-nw interval of 3.54±0.61 map units, placing nw at 2-79.0.

Pooling these results with those of Rizki et al. (1980) who placed nw at 2-79.6, and weighting by sample size, we get a revised location for nw at 2-79.6, and weighting by sample size, we get a revised location for nw of 2-79.3, with an approximate standard error of ±0.2 units.

This work was supported by NIH grant GM 25255. The S Sp Tft nw^D Pin^{Yt} chromosome was provided by T.A.Grigliatti; the nw^2 marker derived from stock 4773 of the Bowling Green stock center.

References: Clark, A.G. 1981, Genetics 99:157-168; Lindsley,D.L. & E.H.Grell 1968, Genetic Variations of Drosophila melanogaster, Carn.Inst.Wash.Publ. No. 627; Rizki,T.M., R.M.Rizki & E.H.Grell 1980, Wilhelm's Roux's Archives 188:91-99.

NEW MUTANTS

Report of L. Craymer.
California Institute of Technology, Pasadena, California.

SM6: Second Multiple Six Craymer, 1979. SM6 is a combination of In(2LR)O and In(2LR)SM1. It was constructed through a sequence of selected single exchanges. SM6 currently exists with two marker combinations: SM6a carries al^2, Cy, dp^{lvl}, cn^{2P}, and sp^2; SM6b carries Roi in addition to the markers carried by SM6a. New Order: 21 to 22A3/60B to 58B1/42A3 to 50C10/30E to 22D2/34A1 to 42A2/58A4 to 50D1/30F to 33F5/22D1 to 22B1/60C to 60 F.

TM6B: Third Multiple Six, B structure Craymer, 1982. TM6B is In(3L)P+(3LR)HR33+(3R)Hu+C+(3)M6, where In(3)M6 is the In(3)75C; 94A inversion characteristic of TM6 [TM6B has In(3LR)HR33 in place of In(3LR)P88 and also carries Hu, but otherwise carries the same inversions as TM6]. TM6B exists with several marker combinations and usually carries either D^3 or Tb [see DIS 55:199] as a dominant marker, with various combinations of e [all current TM6B chromosomes carry e], ca, h, and Hn^P; all TM6B chromosomes carry Hu. TM6B should be the most efficient third chromosome balancer currently available: the longest unbroken region is the 63B8-11 to 72E1 region defined by In(3L)P--possibly 30 cM in length--and the centric region is bracketed by the 75C and 84B breaks so that TM6B should efficiently balance the entire third chromosome even in the presence of heterologous inversions. New Order: 61A/87B to 86C/84F to 86B/84B to 84F/84B to 75C/94A to 100F/92D to 87B/61A to 63B/72E to 63B/72E to 75C/94A to 92E/100F.

TM6C: Third Multiple Six, C structure Craymer, 1982. TM6C is identical in structure to TM6B except for the absence of In(3R)Hu. TM6C exists with various marker combinations, including cu and Sb in addition to the markers listed for TM6B. TM6C is homozygous viable, and a stock of TM6C, e ca [homozygous, and with no other markers] exists which should be useful for recovering and balancing mutagenized chromosomes [stocks of */TM6C, e ca will not become homozygous for TM6C because of the lowered viability and delayed development of the TM6C homozygotes]. TM6C has balancing properties similar to those of TM6B, but

should balance the centric region less effectively because of the absence of Hu.

Dr1: Droplet 2-51 [1 or 2 cM to the left of Tft]. Ethyl-nitroso-urea (ENU) induced on Canton S by S. Sakonju, 1982. Heterozygote has bery small, glazed eyes--like $Dr^{Mio}/+$, but more extreme. Heterozygote has excellent viability. Homozygous lethal. RK1.

qs: quicksilver 1-39 (between v and g). ENU induced by M.Crosby, 1982. Homozygous lethal, but not cell lethal. In gynandromorphs, qs autonomously depigments cuticular tissue, including chaetae. Viability of gynandromorphs may be reduced as a result of weakened cuticle. Cytologically between 10A1 and 11A7, based on coverage by $Dp(1;2)v^{65b}$.

Sta: Stigmata 3--rearrangement. X-ray induced on T(2;3)P10 by E.B.Lewis, 1978. Pigment absent from corners and mid-anterior edge of notum. Wings are held out from body, and this becomes more extreme with age. Associated with In(3LR)Sta = In(3LR)79D;94A [Lewis]. RK2A.

Ubx^{U}: Ultrabithorax^{Ultra} X-rady induced on sbd^{2} ss bx^{34e}, 1966. Has also been referred to as Ubx^{300}. Associated with $In(3LR)Ubx^{U}$ [62A2-3; 89E1-2]. $Ubx^{U}/+$ has an extreme Ubx phenotype, with haltere volume about three times that of a Df(3R)Ubx/+. RK1A

In(3L)P: Inversion (3L) of Payne Breakpoints are 63B8-11; 72E1-2 on Bridges' revised map.

Dp(3:3)S2: Duplication (3:3) Synthetic is $Dp(3;3)HR33^{P}bxd^{106D}$ and was synthesized from $In(3LR)bxd^{106}$ and In(3LR)HR33 via a sequence of selected single exchanges by Craymer, 1980. This duplication is lethal when heterozygous with a normal third chromosome, and it is kept in stock balanced over $In(3LR)HR33^{L}bxd^{106R}$. It is useful for screening for 3L deficiencies or tandem duplications. New Order: 61A1 to 72D11/89E2 to 87B4/61A2 to 100F.

The following duplications are X-ray induced derivatives (Craymer, 1981) of a Dp(3:3)S2 which carried ru Hn^{r3}--h^{2} app:

Dp(3:3)S2a1 Dp(3:3)70A-C; 71A1.
Dp(3:3)S2a2 Dp(3:3)66D; 67D-E.
Dp(3:3)S2a3 Dp(3:3)67D9-11; 68A1-2.
Dp(3:3)S2a4 Dp(3:3)88F; 89E2 plus Df(3L)72D11-E1; 73A10-B1.
New Order: 61A1 to 72D11/89E1 to 88F/73B1 to 100F.
Dp(3:3)S2a5 Dp(3:3)69D1-2; 71D2-4.
Dp(3:3)S2a8 Dp(3:3)66E6-F1; 69E7-F2.
Dp(3:3)S2a9 Dp(3:3)64C4-5; 66E.
Dp(3:3)S2a11 Dp(3:3)66B; 70A.
In(3LR)79i In(3LR)67B11-C1; 87D5-13. Spontaneous in CIT y stock which seems to have constitution y; In(3L)P/In(3LR)79i. Discovered by I.Duncan, 79i.

The 200 series of rearrangements wereX-ray induced on Canton S by S. Ou and detected as transvecting Cbx Ubx/+:

In(3LR)206 In(3LR)65E10-12; 85A1-3 [B.S.Baker]. Homozygous viable, fertile and phenotypically wild type.
In(3LR)208 In(3LR)61E2-3; 86C1-2.
In(3LR)209 In(3LR)62F; 89D. Homozygous lethal.
In(3LR)215 In(3LR)63E; 86D.
In(3LR)216 In(3LR)63E3-6; 80-81; 84A1-2 [B.S.Baker]. Behaves genetically like an In(3LR)63E; 84A inversion; New Order is thus probably: 61A to 63E3; 81-84A1/80 to 63E6/84A2 to 100F with the location of the centromere undetermined.
In(3LR)217 In(3LR)71B2-4; 81F1.
In(3LR)220 In(3LR)63C5-D1; 85E-F.
In(3LR)222 In(3LR)62A10-12; 85F10-12.
In(3LR)223 In(3LR)64D5-E1; 84E6-11 [B.S.Baker]
In(3LR)224 In(3LR)69E7-F1; 83B7-C1. Homozygous viable, fertile, and phenotypically wild type.
In(3LR)225 In(3LR)77E1; 88E2-3.

In(3LR)226 In(3LR)64A10-B1; 87E2-4.
In(3LR)229 In(3LR)61F7-62A1; 88B.
In(3LR)230 In(3LR)67F2-68A1; 84E2-7.
In(3LR)234 In(3LR)67D6-8; 88A10-B1. Homozygous viable, fertile, and phenotypically wild type.
In(3LR)238 In(3LR)80D-F; 89B.
In(3R)221 In(3R)82F; 96 [E.B.Lewis]
T(2;3)221 T(2;3)22B; 62F discovered in association with In(3R)221. Does not transvect Cbx Ubx/+.
In(3LR)A114 In(3LR)80; 92A [E.B.Lewis]. Originally associated with T(Y;3)A114 [Lindsley-Sandler].

The f-series of rearrangements were induced with X-rays (1981) and were detected as translocations between free armed autosomes as described elsewhere in this DIS.

In(2LR)f6: Inversions (2LR) from free arms #6 In(3LR)39D3-E1; 48F6-49A1. Subsequently separated from the second chromosomal free arms.

In(3LR)f1: In(3LR)74D; 86E1-2.
In(3LR)f7: In(3LR)75A1-2; 90A1-5. Subsequently separated from the free third chromosomal arms. Homozygous viable and fertile; wings may be slightly flimsy, but otherwise phenotypically wild type.
In(3LR)f9: In(3LR)68C9; 84F8.
In(3LR)f13: In(3LR)63C5-D1; 84D4-7.
In(3LR)f19: In(3LR)62A; 98A. Subsequently separated from the free third chromosomal arms. Homozygous viable and fertile.
T(3;3)f4: T(3;3)66C8-11; 80 on 3^{P}J17; 3^{P}J139. New Order: 61A to 66C8/80.81 to 100F; B^{S}/80/66C11 to 80.81 to 82A. Has a good B^{S} phenotype, unlike the 3^{P}J139 from which it was derived.

Report of Dawson Mohler.
University of Iowa, Iowa City, Iowa USNA.

oc^{db}: ocelliless-disturbed bristles; polymorphic in the P_1 strain of Ore R. The pattern of head bristles is in variable disarray with about 80% penetrance. The ocellar, interocellar and postvertical bristles are extra, missing or misplaced. oc^{db}/oc females show nearly complete penetrance of the disturbed pattern and slighly greater space between the posterior ocelli. oc^{db}/Df(1)RA2 and oc^{db}/1(1)JA101 have the typical oc phenotype--missing bristles and ocelli. All genotypes are fertile.

Report of Dawson Mohler and Andrea Carroll.
University of Iowa, Iowa City, Iowa USNA.

Sex-linked female-sterile mutations in the Iowa collection:

Part A (Table). Mapped genes included in the study of Mohler 1977, Genetics 85:259.
Part B (Table). Genes mapped in later studies.

NOTES: * Egg development has not been studied.
(1) mutant strain isolated by Lee Engstrom and A.P.Mahowald
(2) mutant strain isolated by Lee Snyder
(3) mutant strain isolated by Pat Roman
(4) mutant strain isolated by Ed Stephenson
(5) mutant strain isolated by Ed Vysse
(6) mutant strain isolated by Lois Girton
(7) mutant strain isolated by Mary Ellen Digan.

PART A. MAPPED GENES INCLUDED IN THE STUDY OF MOHLER 1977, GENETICS 85:259.

NOTE ABBREVIATIONS USED FOR PHENOTYPE:
e.l. = egg layer; s-e.l.= sterile-egg layer; lk. = leaky; e.c. = eggs collapse; f.c. = fragile chorions
r.m-e.l.=rescuable maternal-effect lethal; m-e.l. =maternal-effect lethal; prebl. =preblastoderm; g. =gastrula; postg. =postgastrula; preg. = pregastrula

designation of locus	synonyms	location	cytology	alleles listed by strain number	phenotype of mutant females
cin: cinnamon	fs(1)M50			11-1239	e.l.; r.m-e.l., lk; brown-red eye color
dnc: dunce	fs(1)M42			11-761 (dnc^{M11}), 14-756 (dnc^{M14})	e.l.*; poor "learner"; reduced cAMP phosphodiesterase
fs(1)h:	fs(1)M16 fs(1)1456	21+	7D1·2-7D5·6	12-4102	e.l.; r.m-e.l., preg.
fs(1)K10:	fs(1)M9 "bottle"			12-3654, 13-534, 13-1121	s-e.l.; bottle-shaped eggs, misshapened chorions
fs(1)M1:		10.2±		12-5045	e.l.*
fs(1)M2:		1±		12-158, 12-2786, 12-3318, 13-1058, 13-1920	e.l.; m-e.l., prebl.
fs(1)M3:	fs(1)K646	14±	5C7-5D5·6	11-1472, 12-333, 12-1365, 13-151, 1901(1)	e.l.; m-e.l., prebl.; some eggs collapse
fs(1)M4:		0		12-521, 12-1124, 12-1611, 12-2096, 13-841, 14-835	e.l.; m-e.l., prebl.
fs(1)M5:	fs(1)147	0		11-1315, 12-698, 13-59	e.l.*, e.c.
fs(1)M8:		y - cv		12-3286	e.l.; r.m-e.l., lk.
fs(1)M11:		y - cv		13-228	e.l.*, lk.
fs(1)M13:		14±	5C7-5D5·6	11-1478, 12-115, 13-177, 13-527, 13-1942	e.l., m-e.l., lk.; some e.c., f.c.
fs(1)M14:		near cv	5A8·9-C51	12-170, 13-1347,	s-e.l.
fs(1)M15:		near cv		11-1047, 12-3580	e.l.; r.m-e.l.
fs(1)M17:		15±		13-1867	e.l.; m-e.l., prebl.
fs(1)M19:		37±	10A2-11B1·2	11-241, 11-600, 11-1501, 12-173, 12-368, 12-378, 12-2685, 12-3147, 13-423, 14-1299, 12E-92	e.l.*
fs(1)M20:		v - f	12E1-13A5	11-1157, 12-120	e.l.; r.m-e.l., prebl.
fs(1)M21:		v - f		12-196	e.l.; r.m-e.l., prebl.
fs(1)M22:	fs(1)457	v - f		12-1136	e.l.; m-e.l., g.
fs(1)M23:	fs(1)1459 fs(1)K499	v - f		12-1444, 12-5087	e.l.; m-e.l., g.
fs(1)M24:		v - f	12E1-13A5	12-2825, 12-4244, 12E-78	e.l.; r.m-e.l.

fs(1)M25:	fs(1)148	48±	12E1-13A5	11-73, 11-380, 11-432, 12-1259, 12-2252, 12-5004, 12-5262, 14-465, $5^{(2)}$, $205^{(2)}$, $L186^{(3)}$ $L193^{(3)}$, $L196^{(3)}$, $L211^{(3)}$	e.l.; eggs lack proteinaceous yolk and collapse, fragile chorions
fs(1)M26:		v - f	10A2-11B1·2	12-2331, 13-1732	e.l.; m-e.l., lk.
fs(1)M27:		v - f		12-4293	e.l.; r.m-e.l.
fs(1)M28:		v - f		12-4656	e.l.; r.m-e.l.
fs(1)M29:		53±		12-626, 12-2153, 12-2552, 13-400, 13-758, 14-632	e.l.r.m-e.l., postg.
fs(1)M30:		v - f		12-959	s-e.l., lk.; many e.c., f.c.
fs(1)M31:		near f		12-1330	e.l.*, lk.
fs(1)M32:		near f		12-1815, 12-1939	e.l.; r.m-e.l.
fs(1)M33:		near f		12-2163	s-e.l.; some e.c., f.c.
fs(1)M36:		58±		11-938, 12-273a, 12-1180, 12-3706, 13-1238, 12E-90	e.l.; m-e.l., preg.; some e.c.
fs(1)M38:		10.2±		13-1358	s-e.l.; some e.c., f.c.
fs(1)M40:		y - cv		14-989	e.l.; r.m-e.l., postg.
fs(1)M41:		y - cv		12E-90	e.l.; r.m-e.l.
fs(1)M43:		cv - v	10A2-11B1·2	14-31	s-e.l.
fs(1)M44:	fs(1)1497	cv - v		11-999, 13C82, $T573^{(4)}$	e.l.; m-e.l., g,, some e.c.
fs(1)M46:		near v	10A1-11B1·2	11-5, 13-1867	s-e.l.
fs(1)M48:		cv - v		14-402	s-e.l.; e.c., f.c.
fs(1)M49:		cv - v	9A	12C-45, $1598^{(5)}$, $6303^{(5)}$	s-e.l.; some e.c.
fs(1)M51:		v - f		12-2627	s-e.l.; small eggs, some collapse, f.c.; s-e.l., lk.
fs(1)M52:		cv - v	8EF	13-1643	s-e.l., lk.
fs(1)M53:		near v		13-1973	e.l.; m-e.l., postg.
fs(1)M54:		v - f		13-1750	e.l.; m-e.l.*
fs(1)M55:		near f		14-145	s-e.l., lk.; some e.c.
fs(1)M56:		v - f		12C-40	e.l.; r.m-e.l.
fs(1)M57:		f - spa		11-341, 11-104, 13-358, 12C-114	e.l.; m-e.l., postg.; some e.c., f.c.
fs(1)M58:		f - spa		14-213	e.l.; r.m-e.l.
fs(1)M59:		f - spa		13A-40	e.l.*
fs(1)M60	fs(1)1371 fs(1)K575	y - cv		11-1676	s-e.l.
fs(1)M63:		v - f		11-818, 14-533	s-e.l., lk; small eggs
fs(1)N:	fs(1)M6	(0.0)		12-1166, 12-1420, 14-574, 12N2X2D$^{(3)}$, 211	s-e.l.; some e.c.
gd: gastrula-tion defective	fs(1)M18 fs(1)573	37±	10A7-11B·2	11-1524(gd^2), 12-4955(gd^3), 13-935(gd^4), 13-1697(gd^5), 13-1859(gd^6), 14-743(gd^7)	e.l.; m-e.l., g.
mei-41:	fs(1)M37	35±		11-1325, 12-1007, 12-3616, 13-1272, 12C-20	e.l.; r.m-e.l.

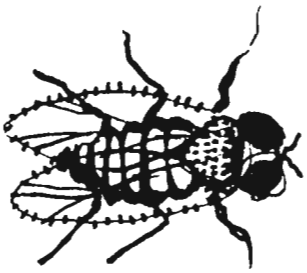
mel(1)R1:	fs(1)M10 "pecanex"	0.5±		12-1012, 12-1743, 12-3014, 12-3102, 12-3135, 12-4169, 14-567, 14-1153	e.l.; r.m-e.l., blastoderm
mk: murky	fs(1)M7	(0.8)		12-2691	e.l.; r.m-e.l.
pt: platinum	fs(1)M47	(23.1)	7E10-8A5	14-208	e.l.; r.m-e.l.; bristle tips are pale
r: rudimentary	fs(1)M34	(54.5)		11-722, 11-836, 11-992, 11-007, 12-779, 12-829, 12-1247, 12-2088, 12-2502, 12-3642, 12-4331a, 13-873, 13-1977, 14-693, 14D-73	e.l.; r.m-e.l. leaky; small wings
sn: singed	fs(1)M45	(21.0)		12-379, 13F-50, 14F-133, 6366[5]	e.l.; small eggs with mis-shapend chorions; singed bristles
fs(1)Y2	fs(1)M12 fs(1)73	1.1±		13-1970, 14-77, 13B-76	s-e.l.

PART B. GENES MAPPED IN LATER STUDIES.

designation of locus	synonyms	location	cytology	alleles listed by strain number	phenotype of mutant females
NOTE ABBREVIATIONS USED FOR PHENOTYPE: e.l. = egg layer; s-e.l. = sterile-egg layer; lk. = leaky; e.c. = eggs collapse; f.c. = fragile chorions; r.m-e.l. = rescuable maternal-effect lethal; m-e.l. = maternal-effect lethal; prebl. = preblastoderm; g. = gastrula; postg. = postgastrula; preg. = pregastrula					
dec$_1$: defective chorion-1	fs(1)C1 fs(1)M102 fs(1)384	(20.8)	7C	11-549, 12-365, 12-403, 12-1873, 12-2514, 12-3512, 12-3907, 12-4860, 13-453, 14-963, 13C-57, 13C-73, 14A-114, 14E19, 952[6], A5[7]	rarely e.l.; thin chorion & respiratory appendages
dec$_2$:	fs(1)C2	(23.1)	7E10-8A5	14-133	rarely e.l.; thin chorion, short & thin respiratory appendages
fs(1)C3:		cv - v	8BC	14-713	no eggs laid; part of chorion thin, short resp. appendages
fs(1)C4:		cv - v		13-1462	no eggs laid; thin chorion; disorganized ovary
fs(1)C5:		cv - v		13-714	no eggs laid; thin chorion; disorganized ovary
fs(1)C6:		cv - v		12-405	no eggs laid; thin chorion; disorganized ovary
fs(1)M35:		y - cv		12-1245	e.l.*
fs(1)M39:		cv - v		12-4752	e.l.*
fs(1)M62:		cv - v	10A2-11B1-2	13-638	e.l.*
fs(1)M64:		y - cv		11-209	e.l.*
fs(1)M65:		y - cv		12-3429	e.l.*
fs(1)M66:		y - cv		12-3778	e.l.*
fs(1)M68:		cv - v		11-73, 11-1487	e.l.*

fs(1)M70:		cv - v		11-726	e.l.*
fs(1)M72:		23.6±	7E10-8A5	14-677	s-e.l., no sperm stored in hemizygous female; fertile homozygous
fs(1)M73:		cv - v	7CD	12-2234	e.l.*
fs(1)M74:		cv - v		12-2929	e.l.*
fs(1)M76:		cv - v		12-3526	e.l.*
fs(1)M77:		cv - v		12-5107	e.l.*
fs(1)M78:		v - f	9E3·4-11B1·2	11-956	e.l.*
fs(1)M79:		v - f	12D	11-1172	e.l.*
fs(1)M81:		v - f		12-4696	e.l.*
fs(1)M82:		v - f		13-65	e.l.*
fs(1)M83:		near f		11-839	e.l.*
fs(1)M84:		near f		13-743	e.l.*
fs(1)M85:		v - f		13-1717	e.l.*
fs(1)M86:		near f		14-743	e.l.*
fs(1)M87:		f - spa		11-874	e.l.*
fs(1)M88:		f - spa		11-1111	e.l.*
fs(1)M89:		f - spa		13-1025	e.l.*
fs(1)M91:		cv - v		11-752	e.l.*
fs(1)M93:		cv - v	7E10-8A5	12-578	e.l.*, lk.
fs(1)M94:		cv - v	7E10-8A5	12-3648	e.l.*, lk.
fs(1)M95:		cv - v		11-917	e.l.*
fs(1)M103:		5±		11-1015, 12-2844	e.l.; m-e.l.*
fs(1)M104:		(1.8)		11-52, 12-1279, 14-926	no eggs laid; ovaries have abnormally shaped yolky eggs
fs(1)M105:		19±		11-1041, 13-1790, 14-1000	no eggs laid; ovaries have abnormally shaped yolky eggs
fs(1)M106:		19±	6E4·5-7A1	12-222, 13-558	no eggs laid; rudimentary ovaries
fs(1)M108:		52±		11-447	e.l.*
fs(1)M111:		21±	7D1·2-7D5·6	11-335, 12-1304, 12-2806, 12-4672, 13-461	no eggs laid; accumulate normal-looking stage 14 eggs
fs(1)M112:		(25.1)	7E10-8A5	12-2174, 1777(7)	no eggs laid; accumulate stage 8,9 oocytes; disturbed ocellar bristle pattern
fs(1)M114:	fs(1)K1075	65±		12-4350	no eggs laid; thin chorion & respiratory appendages
fs(1)M116:		30±	9E3·4-10A1	12-2170, 13-546	no eggs laid; accumulate stage 8
fs(1)M120:		0.5±		14-1054	e.l.*
fs(1)M121:		near f		14L-12	e.l.*
fs(1)M122:		cv - v	7CD	14F-118	e.l.*
lz: lozenge	fs(1)M69	(27.7)		11-164, 12-4669	no eggs laid; narrow, smooth eye

otu:	fs(1)M101 fs(1)231	(23.2)	7E10-8A5	11-1037(otu^5), 12-3266, 12-4474, 13-1004(otu^4), 14-97(otu^2), 14-334, 12C-129, 13F-3, 209(1)(otu^6), 1001(7), 1304(1)(otu^7), 1396(1),(otu^8), 1401(7), A3(7)	no eggs laid; some alleles characterized by agametic ovaries, others by tumorous ovaries, and a few by pseudo stage 12 egg chambers
ptg: pentagon	fs(1)M71	23.6±	7E10-8A5	13-342	e.l.*, lk.; dark thorax



Report of C. Najera.
Universidad Literaria de Valencia, Burjasot (Valencia), Espana

List of the different eye colour mutants of Drosophila melanogaster obtained in two different captures carried out in a wine cellar from Requena, Valencia (Spain).

Localized mutants

- sed^{79i} (sepiaoid-79)
- sed^{81d} (sepiaoid-81)
- se^{79i} (sepia-79). Two alleles at the same capture
- se^{81d} (sepia-81). Two alleles at the same capture
- cd^{79i} (cardinal-79)
- cd^{81d} (cardinal-81). Four alleles at the same capture
- w^{a79i} (white-apricot-79)
- w^{81d} (white-81). Two alleles at the same capture
- v^{81d} (vermilion-81). Two alleles at the same capture
- pr^{81d} (purple-81). Two alleles at the same capture
- g^{81d} (garnet-81)

Non-localized mutants

2,112. Eye colour slightly maroon, differs little from wild type, rugous eyes.
3,18,24A,33,37,71A,79. Eye colour dark chestnut.
1. Eye colour dark red.
19A,23A 27,30,34A,35A,38,42,45,50,53B,59,60,69,110,121,141. Eye colour soft dark brown.
23B,65,73. Eye colour grows dark with age.
39A,64,126. Eye colour soft, dull and dark.
48,70A,87A. Eye colour dark brown.
61A,68A,120. Eye colour darker than wild, darkening with age.

12A,19B,34B54,58A,95B. Eye colour dull chocolate.
22C,58C,68B. Eye colour deep purplish ruby.
6A,14,35B,47,70B,72,113,131. Eye colour ruby.
6B,21,24B,43. The young lighter than wild.
12B,39B,58B,87B. Eye colour light brownish, darkening with age.
44,71C. Lighter than wild.
16,31,53A. Eye colour bright red.
71B,133. Eye colour orange.
55. Eye colour between orange and yellow
52. Eye colour bright scarlet.

Report of Luisa Pilares-Guevara.
Universidad Nacional Mayor de San Marcos, Lima Peru.

D.mercatorum yellow (stock de Brasil)

Report of W.E. Ratnayake and C.N.L. Bogahawatte.
University of Sri Jayewardenepura, Negegoda, Sri Lanka.

Three new wing mutants of Drosophila ananassae from Sri Lanka: An attempt was made to isolate morphological mutants from wild populations of Drosophila ananassae and to estimate their mutation load. Seventeen mutants of spontaneous origin were isolated and we report here, the three new mutants of D.ananassae in these populations. Two of the mutants were recessive autosomal while the other was recessive sex linked and are described below.

(1) mcv: marginal cell cross vein wing. The extra cross vein is seen between 1st and 2nd longitudinal (L1 & L2) veins. Expression is variable in most individuals. Viability is excellent.

The F_2 results (Table 1) show a fit for 3:1 ratio from one parental cross only. This departure from the expected ratio at p=0.05 level in the reciprocal parental cross line may be taken to be due to rapid development of the mutant type, or perhaps even, due to misidentification. However, both crosses taken together approximate a 3:1 ratio. This mutant can, therefore, be taken to be due to a recessive autosomal gene.

Similar mutants, extra cross vein (ecv) and Trident (Tr) have been recorded by Moriwaki in 1972. In his extra cross vein mutant, the L2 and L3 veins are partly fused as a cross vein (Moriwaki & Tobari 1975).

(2) mdcv: marginal cell double cross vein wing mutant. The marginal cell double cross vein wing arose from the culture of mcv described above. Except for the presence of an additional cross vein between L1 and L2 this mutant shows a similarity to mcv. Expressivity is variable and viability is good.

The F2 results of the parental crosses (Table 1) show that they fit a 3:1 ratio which is expected for the segregation of a recessive autosomal gene. We presume that mdcv is pseudo-allelic to mcv.

Fig. 1. Marginal cell cross vein wing mutant.

Fig. 2. Double marginal cell cross vein wing.

Fig. 3. mcl.

(3) mcl: marginal cell less wing. The marginal cell of this mutant appears completely snipped off. However, with reference to expressivity it was found to be variable because the wings are seen to be cut at different depths of the marginal and even the costal cell on the upper margin of the wing. Viability is excellent.

The F2 results arising from the parental crosses P_1 and P_2 (Table 1) show a fit for the expected ratios of 2:1:1 and 1:1:1:1, respectively, for an intercross and backcross for recessive sex linked mutation, Moriwaki (1938) has described in D.ananassae, two similar mutants called Beaded (Bd) wing and cut^2 (ct)^2 wing and again in 1975^2 Beaded (Bd)^3 wing.

Table 1. Results of the F2 progeny of three new mutants isolated from D.ananassae in Sri Lanka.

Mutant & cross	wild type ♀♀	wild type ♂♂	mutant type ♀♀	mutant type ♂♂	Total no. of flies normal	Total no. of flies mutant	χ^2 values
1. marginal cell cross vein							
i) mcv/mcv ♂♂ x +/+ ♀♀	278	224	104	94	502	198	4.0304
ii) mcv/mcv ♀♀ x +/+ ♂♂	145	157	61	50	302	111	0.6307
2. marginal cell double cross vein							
i) mdcv/mdcv ♂♂ x +/+ ♀♀	158	190	51	85	348	136	2.4793
ii) mdcv/mdcv ♀♀ x +/+ ♂♂	192	179	81	57	371	138	1.2103
3. marginal cell-less wing							
i) mcl/y ♂♂ x +/+ ♀♀	2369	1270	-	1188	3639	1188	4.4260
ii) mcl/mcl ♀♀ x +/+ ♂♂	235	239	206	227	474	433	2.8610

The mutation frequency of the Sri Lankian population was calculated from the formula given by VandeHey in 1961 (modified from Spencer). In this investigation 65 recessive mutants were isolated from 119 pair matings. Analysis of this data indicates that the mutation frequency is 0.28 per individual. Compared to Spencer's value (obtained also for D.ananassae on a Pacific Island) of 0.63 per individual (VandeHey 1964), the frequency obtained by us is seen to be low.

Acknowledgement: We are grateful to Drs. K.M.Harris and B.H.Cogan of the Commonwealth Inst. of Entomology, London, for confirming the identification of D.ananassae. We also wish to thank Mr. N.Wimalaratne of the Labour Dept. for assisting with photomicrographs. This work was supported by a grant from Natural Resources Energyand Science Authority of Sri Lanka (RGB/80/5).

References: Moriwaki, D. 1938, Jap.J.Genetics 14(1&2):1-22; Moriwaki,D. & Y.Tobari 1975, IN Handbook of Genetics (King, ed), V3 on D.ananassae; VandeHey,R.C. 1964, Ann.Entomol.Soc. Amer. 57:488-495.

Report of F.Salas and J.A. Lengyel.
University of California at Los Angeles, California USNA.

Revertants of Jammed in Drosophila melanogaster:
isolation and genetic mapping of 5 new mutants.

The second chromosome of Drosophila melanogaster was screened for revertants of the dominant mutation Jammed (J), which maps to 31AF on the cytogenetic map (Mange & Sandler 1973). This region is of interest because it contains a number of female sterile and maternal effect mutations (i.e., M(2)fs, mfs 48, da).

Two different screens were carried out in which males homozygous for J were X-irradiated, mated to non-J females and the progeny screened for the absence of Jammed wings. In the first screen, irradiated (4000R) J34e males were mated to Pm/SM5 females. Crosses were maintained at 27-28°C to obtain full penetrance of J. From 8000 chromosomes screened in this cross, three revertants were obtained: J-der-77, J-der-99, and J-der 106. For the second screen, the J34e chromosome was marked with cn bw from a recently isogenized line. Irradia-

screen, the J34e chromosome was marked with cn bw from a recently isogenized line. Irradiated males were mated to Pm/CyO females. From 18,700 chromosomes screened in this way, 2 revertants were obtained: J-der-222, and J-der-233. The J-der-99 and J-der-106 chromosomes were established as balanced lines with SM5, and the J-der-222 chromosome with CyO. Flies carrying J-der-77 and J-der-233 are Minute and female sterile (presumably the M(2)fs described by Mange & Sandler 1973); therefore these stocks are maintained by mating J-der/CyO males to Pm/CyO females.

The five revertants obtained in the two screens were tested for complementation with lethal mutations in the region (see below). J-der-77, -106, and -233 behave as deletions. J-der-77 and J-der-233 are Mfs, and uncover da, mfs48, and 1(2)54, but not abo or 1(2)gd. Therefore, they resemble Mdh2J (Sandler 1977) genetically. J-der-106 uncovers da, mfs48, and 1(2)54, but not abo or 1(2)gd. This is similar to J-der-27 (Sandler 1977). J-der-222 is homozygous lethal but does not uncover any of the loci tested. We conclude that it is a small deletion or inversion which does not include da. J-der-99 is a translocation to the Y chromosome, as determined from sex linkage and crosses to attached-X females. The J-der-99 breakpoint is presumed to be at or near J. In the course of these complementation studies, we observed that 1(2)54 over the various J-der deletion mutants is viable but sterile at 18°C and lethal at 25°C.

These newly isolated J-der mutations should provide additional useful breakpoints in the 31AF region.

Complementation of various J-der mutations with:

	da	mfs 48	1(2)54	1(2)gd	abo
Jder 77	-	-	-	+	+
99	+	+	+	+	+
106	-	-	-	+	+
222	+	+	+	+	+
233	-	-	-	+	+

Acknowledgement: We thank T.Kaufman and J.Merriam for encouragement and advice.

References: L.Sandler 1977, Genetics 86:567-82; A.Mange & L.Sandler 1973, Genetics 73:73-86.

Report of L.M.Sierra and M.A. Comendador.
University of Oviedo, Espana.

A new mutant of D.melanogaster which modifies the shape and number of tarsi: ats (abnormal tarsi).

A new mutant with abnormal phenotype of tarsi has been found in a selected line to increase the adult resistance against toxic action of acrolein. This mutant which provisionally has been called ats, is recessive and is associated to a third chromosome carrier of three inversions (Figure 1).

Tarsi of the three pairs of legs display an abnormal structure in flies ats/ats although this genotype shows variable expressivity. In the more frequent phenotype second, third and fourth tarsi are lost, and first one is shorter than wildtype (Figure 2a). In smaller frequency, fifth tarsus arises from the tibia but an appendix, likely an aborted tarsus, arises from the tibia (Figure 2b) or from the fifth tarsus (Figure 2c). Other phenotypes, which appear with low frequency, are the following: only fifth tarsus (Figure 2d); fifth tarsus surrounded by three globular appendices which arise from the tibia (Figure 2e); and, finally, first and fifth tarsi with shape and size near wildtype (Figure 2f). Other phenotypic modifications have not been observed. There is not sexual dimorphism.

Females and males ats/ats are both steriles. The adult viability is reduced; more than 50.00% of imagos die in the first day. Likewise, survival preadult is decreased, varying between 20% and 100% in different single cross.

At the moment, it has not been possible to map the ats locus because the chromosomal inversions at which this locus is linked, prevent the recombination. We maintain the ats/ats mutant balanced with a TM3 chromosome.

We thank F.J. Sanchez-Refusta for polytene chromosome observation.

Fig. 1. Third homozygous polytene chromosomes which appear in ats/ats individuals. Break points of each inversion are shown. The three chromosomal inversions are: In(3L) 63C;72E; In(3R) 84E;88A and In(3R) 89C;96A.

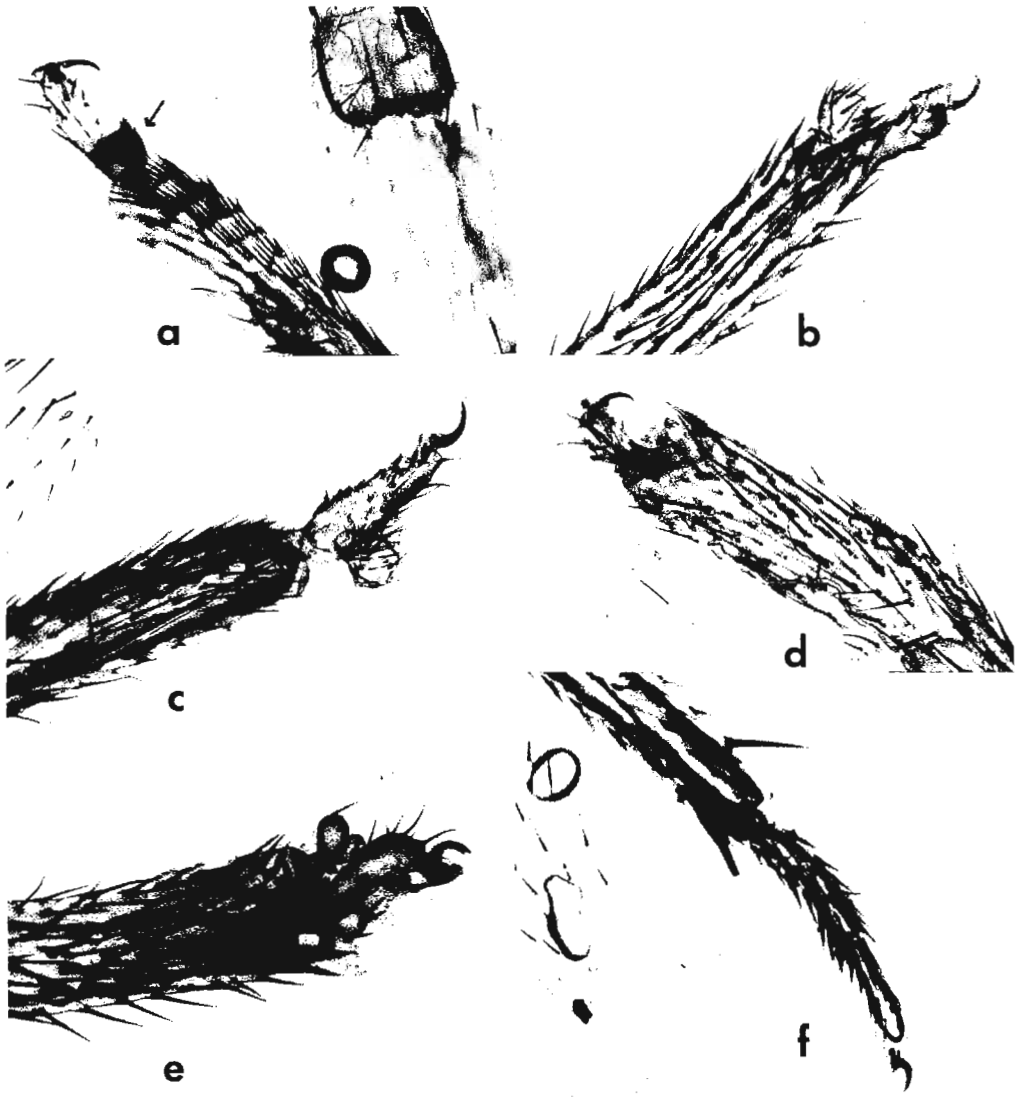


Fig. 2. Different phenotypes which can be observed in the ats/ats mutant.

In 2a, a transverse row bristles in the first tarsus, marked by an arrow, can be seen.

ALPHABETICAL DIRECTORY

June 1, 1984

There has been a great response to our mailing for updated directory listings. Approximately two-thirds of the laboratories responded. In some cases labs had been closed for quite some time and we were able to delete that information.

The following abbreviations are used only in the Alphabetical Directory:

CHINA-PRC = People's Republic of China
CHINA-ROC = Republic of China (Taiwan)
GB = Great Britain
FR GERMANY = Federal Republic of Germany

For the United States: (USNA = United States of North America)

AL	Alabama	ID	Idaho	MT	Montana	PR	Puerto Rico
AK	Alaska	IL	Illinois	NC	North Carolina	RI	Rhode Island
AR	Arkansas	IN	Indiana	ND	North Dakota	SC	South Carolina
AZ	Arizona	KS	Kansas	NE	Nebraska	SD	South Dakota
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DE	Delaware	MD	Maryland	NV	Nevada	UT	Utah
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The following names of Drosophila workers (1400 in all) are followed by the City name and Country name in which their laboratory is located. The completed address will be found in the GEOGRAPHICAL DIRECTORY according the the Country and then the listing by City name.

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Bajraktari, I. Pristina, Yugoslavia
Baker, B.S. La Jolla, USNA CA
Baker, G.T. College Park, USNA MD
Baker, K. Northridge, USNA CA
Baker, S. Jerusalem, Israel
Baker, W.K. Salt Lake City, USNA UT
Balana, J. Barcelona, Spain
Balial, M. Calcutta, India
Band, H.T. East Lansing, USNA MI
Banerjee, I. Calcutta, India
Banerjee, S. Calcutta, India
Banerjee, U. Pasadena, USNA CA
Banzatto, D. Jaboticabal, Brasil
Barker, J.S.F. Armidale, Australia
Barnes, P. Raleigh, USNA NC
Barnett, S. Berkeley, USNA CA
Barrett, T. Salt Lake City, USNA UT
Basden, E.B. Edinburgh, GB-Scotland
Battaglia, B. Padova, Italy
Bauer, G. Dusseldorf, FR Germany
Bauer, S. Downsview, Canada
Bazin, C. Paris, France
Beardmore, J.A. Swansea, GB-Wales
Beck, H. Geneva, Switzerland
Beckenbach, A.T. Burnaby, Canada
Beckendorf, S. Berkeley, USNA CA
Beckman, C. Montreal, Canada
Beermann, W. Tubingen, FR Germany
Behnel, H.J. Saarbrucken, FR Germany
Belalcazar, H. Bogota, Colombia
Belo, M. Jaboticabal, Brasil
Belote, J. La Jolla, USNA CA
Benedik, J. Brno, Czechoslovakia
Benner, D.B. Johnson City, USNA TN
Bennett, C.J. DeKalb, USNA IA
Benson, A.J. Carbondale, USNA IL
Bentley, M.M. Calgary, Canada
Benz, W. Madison, USNA WI
Benzer, S. Pasadena, USNA CA
Berg, R. St.Louis, USNA MO
Bernstein, C. Lyon, France
Bernstein, S.I. San Diego, USNA CA
Berry, T. Eugene, USNA OR
Bewley, G.C. Honolulu, USNA HI
Bewley, G.C. Raleigh, USNA NC
Bhadra, U. Calcutta, India
Bhakta, R. Calcutta, India
Biemont, C. Lyon, France
Bihari, B. Varanasi, India
Bijlsma, R. Haren, Netherlands
Bischoff, W.L. Toledo, USNA OH
Black, B.C. Charlottesville, USNA VA
Black, D. Cambridge, GB-England
Blackman, R. Cambridge, GB-England
Blair, S. Seattle, USNA WA
Blanco, G. Oviedo, Spain
Block, K. Lund, Sweden
Bock, I.R. Bundoora, Australia
Boerema, A.C. Haren, Netherlands
Bogahawatta, C.N.L. Nugegoda, Sri Lanka
Bohm, I. Tubingen, FR Germany
Bokla, L. Tubingen, FR Germany
Bonaccorsi, S. Rome, Italy
Borack, L.I. Newark, USNA NJ
Borai, F. Alexandria, Egypt
Borner, P. Zurich, Switzerland
Bos, M. Haren, Netherlands
Botella, L.M. Burjasot, Spain
Bouletreau, M. Lyon, France
Bouletreau-M., J. Lyon, France
Bourgin-Rosenberg, M. Paris, France
Bourgois, M. Louvain-la-Neuve, Belgium
Bournias-V., N. Duarte, USNA CA
Bove, C. La Jolla, USNA CA
Bownes, M. Edinburgh, GB-Scotland
Boyce, J. Bowling Green, USNA OH
Boyd, J.B. Davis, USNA CA
Boyer, P. Los Angeles, USNA CA
Bozcuk, A.M. Ankara, Turkey
Brandt-R., K. Stockholm, Sweden
Braver, G. Norman, USNA OK
Bravo, M.L. Medellin, Colombia
Bregliano, J.C. Aubiere, France
Brennan, M. Salt Lake City, USNA UT
Brennan, M. Tuscaloosa, USNA AL
Brezinsky, L. Honolulu, USNA HI
Brittnacher, J. Madison, USNA WI
Brock, H. Vancouver, Canada
Brodbeck, B. Tubingen, FR Germany
Brooks, L. Cambridge, USNA MA
Brown, W.P. Marietta, USNA OH
Bruins, B. Utrecht, Netherlands
Bryant, S. Macomb, USNA IL
Bucheton, A. Aubiere, France
Buchner, E. Wurzburg, FR Germany
Bulthoff, H. Tubingen, FR Germany
Buncik, D. Chapel Hill, USNA NC
Bundgaard, J. Aarhus, Denmark
Bunemann, H. Dusseldorf, FR Germany
Burberry, D.S. Uxbridge, GB-England
Burch-Jaffe, E. Seattle, USNA WA
Burdick, A.B. Columbia, USNA MO
Burgess, E. St.Louis, USNA MO
Burma, P. Varanasi, India
Burnet, B. Sheffield, GB-England
Bustos Parra, E. Bogota, Colombia
Butterworth, F.M. Rochester, USNA MI
Buzin, C.M. Duarte, USNA CA

Cabot, E. Burnaby, Canada
Cabre, O. Bellaterra, Spain
Cadieu, N. Toulouse, France
Cambissa, V. Padova, Italy
Campos, A-R. Waltham, USNA MA
Capy, P. Gif-sur-Yvette, France
Cardenas, H. Bogota, Colombia
Cariou, M.L. Gif-sur-Yvette, France
Carpenter, A.T. La Jolla, USNA CA
Carpenter, J.M. Lexington, USNA KY
Carracedo, M.C. Oviedo, Spain

Carson, H.L. Honolulu, USNA HI
Carton, Y. Gif-sur-Yvette, France
Cartwright, I.L. St.Louis, USNA MO
Casares, P. Oviedo, Spain
Casper, K.A. Atlanta, USNA GA
Castaneda, H.C. Bogota, Colombia
Castineiras, O. Santiago de Compostela, Spain
Castro, J. Burjasot, Spain
Casu, R. Brisbane, Australia
Cavicchi, S. Bologna, Italy
Cavolina, P. Palermo, Italy
Celniker, S. Pasadena, USNA CA
Cerda, H. Bellaterra, Spain
Cetl, I. Brno, Czechoslovakia
Chabora, P.C. New York, USNA NY
Chambers, G. Cambridge, USNA MA
Champion, L.E. Research Triangle Park, USNA NC
Chandrashekaran, S. New Delhi, India
Chang, H-Y. Nankang, Taiwan, China-ROC
Chang, H.W. Seoul, Korea
Chang, L. Honolulu, USNA HI
Charles, M. Marseille, France
Charles-Palabost, L. Paris, France
Charton-S., U. Bochum, FR Germany
Chatterjee, K. Calcutta, India
Chatterjee, R.M. Calcutta, India
Chatterjee, S. Varanasi, India
Chaudhuri, G. Calcutta, India
Chawla, S. Quebec, Canada
Chen, H. Shanghai, China-PRC
Chen, P.S. Zurich, Switzerland
Cheney, C. Baltimore, USNA MD
Cherbas, L. Cambridge, USNA MA
Chigusa, S.I. Tokyo, Japan
Chihara, C.J. San Francisco, USNA CA
Cho, C.S. Seoul, Korea
Cho, I.S. Seoul, Korea
Choi, Y. Seoul, Korea
Choo, J.K. Seoul, Korea
Chovnick, A. Storrs, USNA CT
Christensen, A.C. Chapel Hill, USNA NC
Christopoulou, A. Patras, Greece
Chumchong, C. Bangkok, Thailand
Chung, Y.J. Seoul, Korea
Church, K.K. Tempe, USNA AZ
Clare, M. Detroit, USNA MI
Clark, A.G. University Park, USNA PA
Clark, G. Lawrence, USNA KS
Clark, J.M. Atlanta, USNA GA
Clark, S. Storrs, CT
Clark, W. Piscataway, USNA NJ
Claxton, J.H. Armidale, Australia
Clayton, F.E. Fayetteville, USNA AR
Clyde, M.M. Bangi, Selangor, Malaysia
Cochrane, B.J. Tampa, USNA FL
Cockburn, A. La Jolla, USNA CA
Cohen, L. Philadelphia, USNA PA
Collett, J.I. Brighton, GB-England
Comendador, M.A. Oviedo, Spain
Concha-Frisardi, M. Waltham, USNA MA
Connolly, K.J. Sheffield, GB-England
Cook, J. Raleigh, USNA NC
Correa, M.C. Medellin, Colombia
Costa, R. Padova, Italy
Costas, E. Madrid, Spain
Costlow, N. Ithaca, USNA NY
Cote, J. Quebec, Canada
Couderc, J.L. Clermont-Ferrand, France
Counce, S.J. Durham, USNA NC
Couper, D.G. Lewiston, USNA ME
Cowan, T. Los Angeles, USNA CA
Coyne, J.A. College Park, USNA MD
Craddock, E. Purchase, USNA NY
Crawford, G. Duarte, USNA CA
Craymer, L. Pasadena, USNA CA
Creus, A. Bellaterra, Spain
Crosby, M. Pasadena, USNA CA
Crossley, S.A. Clayton, Australia
Crow, J.F. Madison, USNA WI
Crowle, A.J. Denver, USNA CO
Cumming, A.M. Aberdeen, GB-Scotland
Cummings, M.R. Chicago, USNA IL
Curtsinger, J.W. St.Paul, USNA MN

Dai, Zouhua. Beijing, China-PRC
Dalle Carbonare, B. Basel, Switzerland
Danieli, G.A. Padova, Italy
Darvey, N.L. Sydney, Australia
Das, P.C. Calcutta, India
Dastugue, B. Clermont-Ferrand, France
David, J.R. Gif-sur-Yvette, France
Davis, B.K. Morristown, USNA NJ
Davis, G. Tuscaloosa, USNA AL
Davis, M.B. Ithaca, USNA NY
Davis, R.S. East Lansing, USNA MI
Davring, L. Lund, Sweden
Dawood, M.M. Alexandria, Egypt
De Frutos, R. Burjasot, Spain
De Jong, G. Utrecht, Netherlands
De Ruiter, B.L.A. Utrecht, Netherlands
DeBanzie, J. Ithaca, USNA NY
Debise, R. Aubiere, France
DeGroote, F. Aubiere, France
DeMarco, A. Rome, Italy
Demopoulos, N. Patras, Greece
Dempster, M. Edinburgh, GB-Scotland
Denell, R.E. Manhattan, USNA KS
Deol, J.U.R. London, GB-England
Desalle, R. St.Louis, USNA MO
Devlin, B.H. Atlanta, USNA GA
Devlin, R. Vancouver, Canada
Devlin, R.B. Atlanta, USNA GA
Dewees, A.A. Huntsville, USNA TX
Dey, A. Calcutta, India
Dhingra, G. Hissar, India
Di Pasquale Paladino, A. Palermo, Italy
Dickinson, W.J. Salt Lake City, USNA UT
Diffley,J. New York, USNA NY
Dimitri, P. Rome, Italy
Dixon, L.K. Denver, USNA CO
Doane, W.W. Tempe, USNA AZ

Dominguez, A. Oviedo, Spain
Dong, K.W. New York, USNA NY
Dover, G. Cambridge, GB-England
Drysdale, R. Madison, USNA WI
Dubendorfer, A. Zurich, Switzerland
Dumapias, F.E. Tempe, USNA AZ
Duncan, I. St.Louis, USNA MO
Durand, R. Aubiere, France
Durrant, B. Cambridge, GB-England
Dusenbery, R.L. Atlanta, USNA GA
Dutta, P.K. Calcutta, India
Duttagupta, A. Calcutta, India
Duttaroy, A. Calcutta, India
Dutton, F.L. Storrs, USNA CT
Dwivedi, Y.N. Najibabad, India

East, P.D. Armidale, Australia
Eberlein, S. Pasadena, USNA CA
Economos, A. Louvain-la-Neuve, Belgium
Edwards, K. Leeds, GB-England
Eggleston, P. Liverpool, GB-England
Eggleston, W. Madison, USNA WI
Ehrman, L. Purchase, USNA NY
Eissenberg, J.C. St.Louis, USNA MO
Eisses, K.T. Utrecht, Netherlands
Ekstrom, K. Umea, Sweden
El-Masry, A. Alexandria, Egypt
Elens, A. Namur, Belgium
Elgin, S.C.R. St.Louis, USNA MO
Elkins, T. Madison, USNA WI
Emerson, C.P. Charlottesville, USNA VA
Emmerich, H. Darmstadt, FR Germany
Emmett, M. Denver, USNA CO
Enciso, J. Bogota, Colombia
Endow, S.A. Durham, USNA NC
Engeln, H. Berlin, FR Germany
Engels, W.R. Madison, USNA WI
Englert, D.C. Carbondale, USNA WI
Engstrom, L.E. Muncie, USNA IN
Epler, J.L. Oak Ridge, USNA TN
Erickson, J. Bellingham, USNA WA
Espinoza, J. Saltillo, Mexico
Evans, M.K. Sydney, Australia

Faccio-Dolfini, S. Milan, Italy
Falk, R. Jersulaem, Israel
Fargnoli, J. Piscataway, USNA NJ
Farmer, J.L. Provo, USNA UT
Fattig, W.D. Birmingham, USNA AL
Fausto-Sterling, A. Providence, USNA RI
Fehon, R. Seattle, USNA WA
Felger, I. Tubingen, FR Germany
Fernandez, P.E. Buenos Aires, Argentina
Ferre, J. Burjasot, Spain
Ferro, A.M. Salt Lake City, USNA UT
Fessler, J. Los Angeles, USNA CA
Fifis, T. Sydney, Australia
Finnegan, D.J. Edinburgh, GB-Scotland
Finnerty, V.M. Atlanta, USNA GA
Firth, J. Vancouver, Canada
Fischbach, K.F. Wurzburg, FR Germany
Fitzpatrick, K. Vancouver, Canada
Fjose, A. Basel, Switzerland
Fleming, R. Waltham, USNA MA
Fleuriet, A. Aubiere, France
Fogleman, J. Denver, USNA CO
Fontdevila, A. Bellaterra, Spain
Ford, S. Philadelphia, USNA PA
Fornili, P.N. Baltimore, USNA MD
Fouillet, P. Lyon, France
Fowler, T. Berkeley, USNA CA
Fox, D.J. Knoxville, USNA TN
Fox, D.P. Aberdeen, GB-Scotland
Frankel, A.W.K. Iowa City, USNA IA
Frankham, R. Sydney, Australia
Franklin, I.R. Sydney, Australia
Franz, G. Cambridge, GB-England
Freeman, D. Vancouver, Canada
Frei, H. Zurich, Switzerland
Friedman, T.B. East Lansing, USNA MI
Fritz-Niggli, H. Zurich, Switzerland
Frohnhofer, H.G. Tubingen, FR Germany
Fryxell, K. Pasadena, USNA CA
Fu, L-J. Nankang, Taiwan, China-ROC
Fu, Y. Shanghai, China-PRC
Fuerst, T. Ithaca, USNA NY
Fuger, M. Tubingen, FR Germany
Fukatami, A. Sakado, Japan
Fukunaga, A. Osaka, Japan
Futch, D.G. San Diego, USNA CA

Gai, P.G. Mysore, India
Gailey, P. Waltham, USNA MA
Galin, R. Tuscaloosa, USNA AL
Galissie, M. Toulouse, France
Ganetzky, B.S. Madison, USNA WI
Garber, R. Seattle, USNA WA
Garcia, E. Oviedo, Spain
Garcia, M. Bellaterra, Spain
Garcia, P. Burjasot, Spain
Garcia-H., M.E. Saltillo, Mexico
Garcin, F. Quebec, Canada
Garcini, F.J. Chestnut Hill, USNA MA
Garen, A. New Haven, USNA CT
Garen, S. New Haven, USNA CT
Gartner, L.P. Baltimore, USNA MD
Gateff, E. Mainz, FR Germany
Gatti, M. Rome, Italy
Gauger, A. Seattle, USNA WA
Gawin, R. Sheffield, GB-England
Geer, B.W. Galesburg, USNA IL
Gehring, W.J. Basel, Switzerland
Gelbart, W.M. Cambridge, USNA MA
Gemmill, R.M. Tempe, USNA AZ
Geng, Z. Shanghai, China-PRC
Gerresheim, F. Aachen, FR Germany

Gersh, E.S. Philadelphia, USNA PA
Gese, E. Pasadena, USNA CA
Ghosh, A. Calcutta, India
Ghosh, M. Calcutta, India
Ghosh, S. Calcutta, India
Ghosh, Saswati. Calcutta, India
Ghosh, Shyamasri. Calcutta, India
Ghosh, Sushmita. Calcutta, India
Giddings, V. St.Louis, USNA MO
Gill, K.S. Ludhiana, India
Gillam, I. Vancouver, Canada
Gillings, M.R. Sydney, Australia
Gilmour, D. Ithaca, USNA NY
Giorgi, G. Bologna, Italy
Girard, P. Paris, France
Girton, J.R. Lincoln, USNA NE
Glatzer, K-H. Dusseldorf, FR Germany
Gloor, H. Geneva, Switzerland
Goetz, K. Tubingen, FR Germany
Gogarty, J. Sheffield, GB-England
Goldberg, M. Ithaca, USNA NY
Goldstein, E.S. Tempe, USNA AZ
Goldstein, L. Cambridge, USNA MA
Golic, K. Seattle, USNA WA
Gomez-Gomez, F. Bogota, Colombia
Gonzalez, R. Barcelona, Spain
Gonzalez-B., M.C. Burjasot, Spain
Gonzalez-G., A. Burjasot, Spain
Gorczyca, M. Waltham, USNA MA
Gottlieb, J.F. Purchase, USNA NY
Goux, J.M. Paris, France
Graf, U. Zurich, Switzerland
Grainger, R.M. Charlottesville, USNA VA
Grana, C. Oviedo, Spain
Gratecos, D. Marseille, France
Gray, M. Waltham, USNA MA
Green, B.I. Purchase, USNA NY
Green, R. Cambridge, GB-England
Greenberg, R.M. Charlottesville, USNA VA
Greenleaf, A.L. Durham, USNA NC
Grell, E.H. Oak Ridge, USNA TN
Grell, R.F. Oak Ridge, USNA TN
Gribbin, C. Brighton, GB-England
Griffin-Shea, R. Marseille, France
Grigliatti, T. Vancouver, Canada
Gromko, M. Bowling Green, USNA OH
Grossfield, J. New York, USNA NY
Gu, D. Shanghai, China-PRC
Gubb, D. Cambridge, GB-England
Guerra, D. Bologna, Italy
Guest, F.E. Fayetteville, USNA AR
Guild, G.M. Philadelphia, USNA PA
Gunaratne, P. Ithaca, USNA NY
Gupta, A.P. Rio de Janeiro, Brasil
Gupta, J.P. Varanasi, India
Gutierrez, M.C. Bogota, Colombia
Gutierrez-B., L.F. Bogota, Colombia
Ha, Y.M. Seoul, Korea
Haaland, R. Tempe, USNA AZ
Haapala, O. Turku, Finland
Hackstein, J.H.P. Nijmegen, Netherlands
Hagele, K. Bochum, FR Germany
Hahn, E. Giessen, FR Germany
Halfer, C. Milan, Italy
Halfer-Mosna, G. Milan, Italy
Hall, J. Waltham, USNA MA
Hall, L.M. Bronx, USNA NY
Hallstrom, I. Stockholm, Sweden
Hammond, M. Seattle, USNA WA
Hanna, P.J. Geelong, Australia
Hannah-Alava, A. Turku, Finland
Hansen, B. Vancouver, Canada
Hansson, L. Umea, Sweden
Hard, R.W. La Jolla, USNA CA
Hardy, D.E. Honolulu, USNA HI
Harmsen, R. Kingston, Canada
Harrington, G. Cambridge, GB-England
Harris, D. Chestnut Hill, USNA MA
Harshman, L.G. Davis, USNA CA
Hartl, D. St.Louis, USNA MO
Hartmann-G., I.J. Sheffield, GB-England
Hartshorn, J.N. Manchester, GB-England
Harvengt, J. Louvain-la-Neuve, Belgium
Hasson, E. Buenos Aires, Argentina
Hatzopoulos, M.P. New York, USNA NY
Hauschteck-J., E. Zurich, Switzerland
Hawley, R.S. Bronx, USNA NY
Hawley, S. Tempe, USNA AZ
Hayashi, S. Vancouver, Canada
He, C. Shanghai, China-PRC
Healy, M.J. Canberra, Australia
Hed, H. Umea, Sweden
Hedrick, P.W. Lawrence, USNA KS
Heemskerk, J. Berkeley, USNA CA
Hegde, S.N. Mysore, India
Heim, W.G. Colorado Springs, USNA CO
Heinstra, P.W.H. Utrecht, Netherlands
Heisenberg, M. Wurzburg, FR Germany
Hellack, J.J. Norman, USNA OK
Henderson, D. Vancouver, Canada
Henderson, E.M. Edinburgh, GB-Scotland
Henderson, N.R. Auckland, New Zealand
Hengstenberg, B. Tubingen, FR Germany
Hengstenberg, R. Tubingen, FR Germany
Henikoff, S. Seattle, USNA WA
Hennig, W. Nijmegen, Netherlands
Henrich, V. Waltham, USNA MA
Herrera, O.L. Bogota, Colombia
Herrera, S.P. Bogota, Colobmia
Herskowitz, I.H. New York, USNA NY
Hess, O. Dusseldorf, FR Germany
Hevert, F. Giessen, FR Germany
Hextger, W.M. Amherst, USNA MA
Higginbotham, J. Canberra, Australia
Hihara, F. Matsuyama, Japan
Hildreth, P.E. Charlotte, USNA NC
Hill, R.J. Sydney, Australia
Hilliker, A. Guelph, Canada

Hillman, R. Philadelphia, USNA PA
Hiniton, C.W. Wooster, USNA OH
Hiraizumi, K. Raleigh, USNA NC
Hiromi, Y. Basel, Switzerland
Hirsch, J. Champaign, USNA IL
Hochman, B. Knoxville, USNA TN
Hoekstra, R.F. Haren, Netherlands
Hoenigsberg, H.F. Bogota, Colombia
Hoffmann, F.M. Cambridge, USNA MA
Hoikkala, A. Oulu, Finland
Holden, J.J.A. Kingston, Canada
Holliday, M. Champaign, USNA IL
Holm, D. Vancouver, Canada
Holmgren, P. Lund, Sweden
Hood, R. Tuscaloosa, USNA AL
Hooper, G.B. Poughkeepsie, USNA NY
Hori, S.H. Sapporo, Japan
Horvath, D. Vancouver, Canada
Hotchkiss, S.K. Potsdam, USNA NY
Hotta, Y. Tokyo, Japan
Houtchens, K. Honolulu, USNA HI
Howard, K.R. London, GB-England
Howe, K.M. London, GB-England
Hsieh, T. Durham, USNA NC
Hu, Kai. Xian, China-PRC
Hubby, J.L. Chicago, USNA IL
Huet, C. Orsay, France
Huet, M. Orsay, France
Huh, M.K. Seoul, Korea
Huijser, P. Nijmegen, Netherlands
Hultmark, D. Basel, Switzerland
Hunt, J. Honolulu, USNA HI
Husa, L-M. Oulu, Finland
Hussain, A.F. Baghdad, Iraq
Hutchinson, E.W. Halifax, Canada
Hyde, J. Piscataway, USNA NJ

Ikeda, H. Matsuyama, Japan
Ikeda, K. Duarte, USNA CA
Ileri, N. Ankara, Turkey
Imberski, R.B. College Park, USNA MD
Inaba, A. Sakado, Japan
Inagaki, H. Gif-sur-Yvette, France
Inahara, J. Tempe, USNA AZ
Inestrosa, N.C. Santiago, Chile
Ingham, P.W. London, GB-England
Inoue, Y. Misima, Japan
Inoue, Y. Chapel Hill, USNA NC
Irick, H. Cambridge, USNA MA
Ish-Horowicz, D. London, GB-England
Ishikawa, E. Tokyo, Japan
Ising, G. Lund, Sweden
Isono, K. Sendai, Japan
Israelewski, N. Bochum, FR Germany
Ives, P.T. Amherst, USNA MA
Izquierdo, J.I. Oviedo, Spain

Jackson, R. London, GB-England
Jacobs, M.E. Goshen, USNA IN
Jacobson, J. St.Louis, USNA MO
Jacobson, K. Madison, USNA WI
Jacobson, K.B. Oak Ridge, USNA TN
Jacq, B. Marseille, France
Jaenike, J. Rochester, USNA NY
Jaime, B. Saltillo, Mexico
James, A. Waltham, USNA MA
James, J.W. Sydney, Australia
James, T.C. St.Louis, USNA MO
Janssen, W. Nijmegen, Netherlands
Jarry, B. Strasbourg, France
Jayaleela, R. Mysore, India
Jeffery, D.E. Provo, USNA UT
Jelisavcic, B. Belgrade, Yugoslavia
Jenkins, J.B. Swarthmore, USNA PA
Jennings, N.J. Cedar Falls, USNA IA
Jensen, R. La Jolla, USNA CA
Jeung, M-G. Seoul, Korea
Jiang, C. Shanghai, China-PRC
Jing, J. Chestnut Hill, USNA MA
Joeksema-Du Pui, M. Haren, Netherlands
Johnsen, R.C. Garden City, USNA NY
Johnson, G.G. St.Louis, USNA MO
Johnson, M.B. Rochester, USNA MI
Johnson, T.K. Manhattan, USNA KS
Johnson, W.W. Albuquerque, USNA NM
Jones, J.S. London, GB-England
Jones, R. Cambridge, USNA MA
Jones, W.K. Lexington, USNA KY
Jongens, T. Berkeley, USNA CA
Joslyn, G. La Jolla, USNA CA
Juan, E. Barcelona, Spain
Jungen, H. Zurich, Switzerland
Jurgens, G. Tubingen, FR Germany

Kafatos, F.C. Cambridge, USNA MA
Kafer, K. Vancouver, Canada
Kaguni, L. East Lansing, USNA MI
Kaji, S. Kobe, Japan
Kalfayan, L.J. Baltimore, USNA MD
Kalick, H. Garden City, USNA NY
Kalisch, W.-E. Bochum, FR Germany
Kalmus, H. London, GB-England
Kambysellis, M.P. New York, USNA NY
Kamping, A. Haren, Netherlands
Kaneshior, K.Y. Honolulu, USNA HI
Kang(Song), S.J. Seoul, Korea
Kang, Y.S. Seoul, Korea
Kankel, K. New Haven, USNA CT
Kanost, M. Kingston, Canada
Kaplan, M.L. New York, USNA NY
Kar, A. Calcutta, India
Karpen, G. Seattle, USNA WA
Kato, H.K. Tokyo, Japan
Kauffman, S.A. Philadelphia, USNA PA
Kawakami, J. Fukuoka, Japan
Kawanishi, M. Misima, Japan
Kaya, H.K. Davis, USNA CA

Keigher, E. Baltimore, USNA MD
Keith, T. Cambridge, USNA MA
Kekic, V. Belgrade, Yugoslavia
Keller, E.C. Morgantown, USNA WV
Kerridge, S. Marseille, France
Kerver, J.W.M. Haren, Netherlands
Khalil, M. Alexandria, Egypt
Kibart, M. Sheffield, GB-England
Kidwell, J.F. Providence, USNA RI
Kidwell, M.G. Providence, USNA RI
Kiehle, C. Seattle, USNA WA
Kilbey, B.J. Edinburgh, GB-Scotland
Kim, S.K. Seoul, Korea
Kimura, K. Okazaki, Japan
King, R.C. Evanston, USNA IL
Kitthawee, S. Bangkok, Thailand
Klarenberg, A.J. Utrecht, Netherlands
Klemenz, R. Basel, Switzerland
Kloter, U. Basel, Switzerland
Knibb, W.R. Armidale, Australia
Knoppien, W. Nijmegen, Netherlands
Kobayashi, H. Tokyo, Japan
Kobel, H-R. Geneva, Switzerland
Koehler, M.M.D. Cambridge, USNA MA
Koenig, J. Duarte, USNA CA
Koepfer, H.R. New York, USNA NY
Koga, K. Fukuoka, Japan
Kohler, W. Giessen, FR Germany
Kongsuwan, K. Los Angeles, USNA CA
Kosuda, K. Sakado, Japan
Kramers, P.G.N. Bilthoven, Netherlands
Kreber, R.A. Madison, USNA WI
Kreitman, M. Cambridge, USNA MA
Krider, H. Storrs, USNA CT
Krimbas, C. Athens, Greece
Krishnamurthy, N.B. Mysore, India
Kroeger, H. Saarbrucken, FR Germany
Kroman, R.A. Long Beach, USNA CA
Ksanz, J.G. Toulouse, France
Kubli, E. Zurich, Switzerland
Kuhn, D.T. Orlando, USNA FL
Kulkarni, S. Waltham, USNA MA
Kumar, A. Varanasi, India
Kumari, A. Mysore, India
Kumura, M.T. Sapporo, Japan
Kunz, W. Dusseldorf, FR Germany
Kuroda, Y. Misima, Japan
Kuroiwa, A. Basel, Switzerland
Kurokawa, H. Sakura-Mura, Japan
Kwan, H. Chestnut Hill, USNA MA

Lachaise, D. Gif-sur-Yvette, France
Lacky, L.R. Lexington, USNA KY
Laha, S. Calcutta, India
Lai, Hui-Chen. University Park, USNA PA
Laird, C. Seattle, USNA WA
Lake, S. Stockholm, Sweden
Lakhotia, S.C. Varanasi, India
Lakovaara, S. Oulu, Finland
Lamb, M.J. London, GB-England
Lambert, D.M. Auckland, New Zealand
Lambertsson, A. Umea, Sweden
Lande, R.S. Chicago, USNA IL
Lane, E. Blacksburg, USNA VA
Langevin, M.L. Galesburg, USNA IL
Langley, S.D. Atlanta, USNA GA
Lankinen, P. Oulu, Finland
Larochelle, C. Quebec, Canada
Lastowski-P., J.C. Chapel Hill, USNA NC
Latorre, A. Burjasot, Spain
Latter, B.D.H. Sydney, Australia
Lau You Hin, G. Quebec, Canada
Lauge, G. Orsay, France
Laurie-Ahlberg, C. Raleigh, USNA NC
Laval, M. Marseille, France
Lavery, K.J. Clayton, Australia
Lavige, J.M. Aubiere, France
Le Bourg, E. Toulouse, France
Learn, J. St.Louis, USNA MO
Lechien, J. Namur, Belgium
Lee, C.C. Seoul, Korea
Lee, H.K. Seoul, Korea
Lee, J.S. Seoul, Korea
Lee, R. Kingston, Canada
Lee, S.H. Seoul, Korea
Lee, T.J. Seoul, Korea
Lee, Y.R. Seoul, Korea
Lefevre, G. Northridge, USNA CA
Lehmann, M. Paris, France
Lehmann, R. Tubingen, FR Germany
Leibenguth, F. Saarbrucken, FR Germany
Lemeunier, F. Gif-sur-Yvette, France
Lengyel, J. Los Angeles, USNA CA
Leonard, J.E. Purchase, USNA NY
Lepot, F. Toulouse, France
Leung, J. Vancouver, Canada
Levan, G. Goteborg, Sweden
Levine, J.B.F. Baltimore, USNA MD
Levine, L. New York, USNA NY
Levinger, L.F. Newark, USNA DE
Levitan, M. New York, USNA NY
Levy, J.N. Tempe, USNA AZ
Lewgoy, F. Porto Alegre, Brasil
Lewis, E.B. Pasadena, USNA CA
Lewontin, R. Cambridge, USNA MA
Lebion-Mannaert, M. Namur, Belgium
Lichtenstein, P.S. Denver, USNA CO
Liebowitz, D.N. Atlanta, USNA GA
Liebrich, W. Dusseldorf, FR Germany
Lietaert, M-C. Namur, Belgium
Lin, F-J. Nankang, Taiwan, China-ROC
Lin, H-O. Tempe, USNA AZ
Lindquist, S. Chicago, USNA IL
Lindsley, D.L. La Jolla, USNA CA
Lineruth, K. Umea, Sweden
Lints, C.B. Louvain-la-Neuve, Belgium
Lints, F.A. Louvain-la-Neuve, Belgium
Lis, J. Ithaca, USNA NY
Liu, T. Shanghai, China-PRC
Llop, P. Burjasot, Spain
Lockette, T.J. Sydney, Australia
Loerre, A. Rome, Italy
Long, G. Blacksburg, USNA VA

Lopez, N. Medellin, Colombia
Loughlin, S.A.R. Oxford, GB-England
Loughney, K. Madison, USNA WI
Louis, C. St.Christol-les-Ales, France
Louis, J. Gif-sur-Yvette, France
Loukas, M. Cambridge, USNA MA
Loukas, M. Athens, Greece
Lower, W.F. Columbia, USNA MO
Lucchesi, T.M. Chapel Hill, USNA NC
Luckinbill, L. Detroit, USNA MI
Lumme, J. Oulu, Finland
Lundqvist, A. Lund, Sweden
Luning, K.G. Stockholm, Sweden
Lyttle, T.W. Honolulu, USNA HI

MacGaffey, J. Berkeley, USNA CA
Machova, H. Brno, Czechoslovakia
MacIntyre, R. Ithaca, USNA NY
MacKay, T.F.C. Edinburgh, GB-Scotland
Maekawa, H. Sapporo, Japan
Magalhaes, L.E. Sao Paulo, Brasil
Magnusson, J. Stockholm, Sweden
Mahoney, P. Los Angeles, USNA CA
Majumdar, D. Rochester,USNA MI
Majumdar, S.K. Easton, USNA PA
Manseau, L. Madison, USNA WI
Mansukhani, A. Ithaca, USNA NY
Marcey, D.J. Salt Lake City, USNA UT
Marchal-Segault, D. Orsay, France
Marchant, G. Vancouver, Canada
Marcos, R. Bellaterra, Spain
Marenco, M.J. Toulouse, France
Marengo, N.P. Greenvale, USNA NY
Mariani, B. Cambridge, USNA MA
Marien, D. New York, USNA NY
Marinkovic, D. Belgrade, Yugoslavia
Markow, T.A. Tempe, USNA AZ
Maroni, G.P. Chapel Hill, USNA NC
Marques, E.K. Porto Alegre, Brasil
Marthas, M. Madison, USNA WI
Martin, A. Piscataway, USNA NJ
Martin, P. Piscataway, USNA NJ
Martinez, J.C. Cambridge, USNA MA
Martinez, R.M. Hamden, USNA CT
Martinez-S., M.J. Burjasot, Spain
Marunouchi, T. Tokyo, Japan
Mason, J.M. Research Triangle Park, USNA NC
Massie, H.R. Utica, USNA NY
Mather, W.B. Brisbane, Australia
Matsuda, M. Madison, USNA WI
Matsuura, E.T. Tokyo, Japan
Mayer, P. College Park, USNA MD
Mazar-Barnett, B. Buenos Aires, Argentina
McCarron, M. Storrs, USNA CT
McCarthy, P.C. New Wilmington, USNA PA
McClish, B. Berkeley, USNA CA
McCormick, S.C. Atlanta, USNA GA
McCrady, E. Greensboro, USNA NC
McElwain, M.C. Salt Lake City, USNA UT
McEvey, S.F. Johannesburg, South Africa
McGill, S. Cambridge, GB-England
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McInnis, D. Honolulu, USNA HI
McKee, B. La Jolla, USNA CA
McKeown, M. La Jolla, USNA CA
McNabb, S. Berkeley, USNA CA
McRobert, S. Philadelphia, USNA PA
Mechler, B. Mainz, FR Germany
Medioni, J. Toulouse, France
Mensua, J.L. Burjasot, Spain
Mercot, H. Paris, France
Merrell, D.J. Minneapolis, USNA MN
Merriam, J. Los Angeles, USNA CA
Mester, M. Lyon, France
Mettler, L.E. Raleigh, USNA NC
Meyer, H. Madison, USNA WI
Meyerowitz, E. Pasadena, USNA CA
Michalopoulou, E. Patras, Greece
Michinomae, M. Kobe, Japan
Miglani, G.S. Ludhiana, India
Mikasa, K. Sakado, Japan
Miklos, M.J. Canberra, Australia
Milanovic, M.M. Belgrade, Yugoslavia
Mildner, A. Detroit, USNA MI
Milkman, R.D. Iowa City, USNA IA
Millar, C. Auckland, New Zealand
Miller, D.D. Lincoln, USNA NE
Miller, J. Blacksburg, USNA VA
Miller, O.L. Charlottesville, USNA VA
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Minato, K. Misima, Japan
Mindek, G. Zurich, Switzerland
Mindrinos, M. Chestnut Hill, USNA MA
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Miyake, T. Tokyo, Japan
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Mohler, J.D. Iowa City, USNA IA
Moisand, R. Buffalo, USNA NY
Monclus, M. Barcelona, Spain
Montague, J. Miami Shores, USNA FL
Montchamp, C. Paris, France
Moore, B.C. Riverside, USNA CA
Moore, J.A. Riverside, USNA CA
Moore, K. Blacksburg, USNA VA
Morea, H. Brighton, GB-England
Mori, I. St.Louis, USNA MO
Morin, K. Vancouver, Canada
Mortensen, M. Copenhagen, Denmark
Morton, R.A. Hamilton, Canada
Moscoso del Prado, J. Los Angeles, USNA CA
Moses, K. Cambridge, GB-England
Mout, H.C.A. Bilthoven, Netherlands
Moya, A. Burjasot, Spain
Muckenthaler, F.A. Bridgewater, USNA MA
Mueller, L.D. Pullman, USNA WA
Mukherjee, A.S. Calcutta, India

Munoz, A. Cordoba, Spain
Munoz, E.R. Buenos Aires, Argentina
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Murakami, A. Misima, Japan
Murray, A.C. Geelong, Australia
Murray, M. Seattle, USNA WA
Murthy, S.K. Ahmedabad, India
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Mutsuddi, M. Calcutta, India
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Nagaraj, H.J. Mysore, India
Nahmias, J. Raleigh, USNA NC
Najera, C. Burjasot, Spain
Nalcaci, O.B. Ankara, Turkey
Napp, M. Porto Alegre, Brasil
Narise, S. Sakado, Japan
Narise, T. Sakado, Japan
Nash, W.G. Frederick, USNA MD
Nath, B.B. Varanasi, India
Naveira, H. Bellaterra, Spain
Nawa, S. Misima, Japan
Nelson, R. Seattle, USNA WA
Nero, D. Ithaca, USNA NY
Newton, C. Vancouver, Canada
Nicod, I. Tubingen, FR Germany
Nigro, L. Padova, Italy
Nilson, L-R. Umea, Sweden
Nix, C.E. Oak Ridge, USNA TN
Nokkala, C. Turku, Finland
Nokkala, S. Turku, Finland
Norman, R. Raleigh, USNA NC
Norman, R.A. Tempe, USNA AZ
Nothiger, R. Zurich, Switzerland
Nouaud, D. Paris, France
Novitski, E. Eugene, USNA OR
Nunney, L. Riverside, USNA CA
Nusslein-Volhard, C. Tubingen, FR Germany
Nutt, E.M. Atlanta, USNA GA
Nygren, J. Umea, Sweden

O'Brien, S.J. Frederick, USNA MD
O'Connor, J.D. Los Angeles, USNA CA
O'Dell, K.M.C. Sheffield, GB-England
O'Donnell, J.M. Pittsburgh, USNA PA
Obin, M. Purchase, USNA NY
Ochoa-R., A.M. Saltillo, Mexico
Oglesby, M. Tuscaloosa, USNA AL
Oguma, Y. Sakura-Mura, Japan
Ohnishi, S. Misima, Japan
Ohta, A.T. Honolulu, USNA HI
Oishi, K. Kobe, Japan
Okada, M. Sakura-Mura, Japan
Okamoto, H. Tokyo, Japan
Olivarez-S., G. Saltillo, Mexico
Oliveira, A.K. Porto Alegre, Brasil
Omar, A. Alexandria, Egypt
Ordonez, M. Bogota, Colombia
Orr, W. Cambridge, USNA MA
Orr-Weaver, T.L. Baltimore, USNA MD
Orssaud, L. Orsay, France
Ortiz, C. Saltillo, Mexico
Osgood, C.J. Lexington, USNA KY
Oshima, C. Misima, Japan
Oster, P. Bowling Green, USNA OH
Ostrowski, R.S. Charlotte, USNA NC
Otake, K. Misima, Japan

Pacheco, M. Lisbon, Portugal
Palka, J. Seattle, USNA WA
Palmgren, B. Stockholm, Sweden
Panigraphy, K.K. Varanasi, India
Papaceit, M. Barcelona, Spain
Park, E.K. Seoul, Korea
Park, J.H. Seoul, Korea
Park, T.D. Seoul, Korea
Parkash, R. Amritsar, India
Parks, S. Baltimore, USNA MD
Parris, R. Newcastle, Australia
Parsons, P.A. Bundoora, Australia
Pascual, L. Burjasot, Spain
Paterson, H.E. Johannesburg, South Africa
Payant, V. Gif-sur-Yvette, France
Paz, C. Buenos Aires, Argentina
Pechan, P.A. Miami, USNA FL
Pelecanos, M. Patras, Greece
Pelisson, A. Aubiere, France
Pelliccia, J. Lewiston, USNA ME
Peng, T. Guangzou, China-PRC
Pereira, A. Waltham, USNA MA
Pereira, M.A.Q.R. Sao Paulo, Brasil
Perez, M. Burjasot, Spain
Perez-Chiesa, Y. Rio Piedras, USNA PR
Peris, F. Bellatera, Spain
Petit, C. Paris, France
Petkovic, D. Belgrade, Yugoslavia
Petri, W.H. Chestnut Hill, USNA MA
Pezzoli, M.C. Bologna, Italy
Pfriem, P. Tubingen, FR Germany
Phelps, R.W. San Diego, USNA CA
Philips, G.H. Lawrence, USNA KS
Phillips, J. Guelph, Canada
Picard, G. Aubiere, France
Pilares, G.L. Lima, Peru
Pimpinelli, S. Rome, Italy
Pimpinelli, S. Seattle, USNA WA
Pinchin, S.M. London, GB-England
Pineda, N. Medellin, Colombia
Pineiro, R. Oviedo, Spain
Pinol, J. Bellaterra, Spain
Pinon, H. Aubiere, France
Pinsker, W. Tubingen, FR Germany
Pisano, C. Rome, Italy

Pla, C. Bellaterra, Spain
Place, A.R. Philadelphia, USNA PA
Plehn, C. Bochum, FR Germany
Plus, N. St.Christol-les-Ales, France
Pohlod, D. Detroit, USNA MI
Polanco, M.M.E. Bogota, Colombia
Poole, J.H. Denver, USNA CO
Pope, A.K. Brisbane, Australia
Portin, P. Turku, Finland
Possin, D. Madison, USNA WI
Potter, J.H. College Park, USNA MD
Potts, A.D. Johannesburg, South Africa
Poulson, D.F. New Haven, USNA CT
Powell, J.F. New Haven, USNA CT
Powers, N.R. Fresno, USNA CA
Prasad-Sinha, J. Calcutta, India
Preston, C. Madison, USNA WI
Prevosti, A. Barcelona, Spain
Prout, M. Seattle, USNA WA
Puro, J. Turku, Finland
Pyshnov, M. Toronto, Canada

Quintana, A. Barcelona, Spain

Rabasco, A. Cordoba, Spain
Raghaven, K.V. Pasadena, USNA CA
Rajput, P.S. Amritsar, India
Ramachandra, N.B. Mysore, India
Ramel, C. Stockholm, Sweden
Ramesh, S.R. Mysore, India
Ranganath, H.A. Mysore, India
Ranganathan, S. Tuscaloosa, USNA AL
Rapport, E. Toronto, Canada
Rasmuson, A. Umea, Sweden
Rasmuson, B. Umea, Sweden
Rasmuson, M. Umea, Sweden
Ratnayake, W.E. Nugegoda, Sri Lanka
Rawls, J.M. Lexington, USNA KY
Ray, R. Berkeley, USNA CA
Real, M.D. Burjasot, Spain
Reardon, J.T. Atlanta, USNA GA
Reddy, P. Waltham, USNA MA
Reguly, M.L. Porto Alegre, Brasil
Reid, S. Clayton, Australia
Reiling, H. Bochum, FR Germany
Remondini, D.J. Bethesda, USNA MD
Renaud, M. Aubiere, France
Rhodes, S. Tempe, USNA AZ
Ribeiro, M.E. Lisbon, Portugal
Ribo, G. Barcelona, Spain
Richter, M. Vancouver, Canada
Ricker, J. Champaign, USNA IL
Rigby, D. Cambridge, USNA IL
Riihimaa, A. Oulu, Finland
Rinehart, R.R. San Diego, USNA CA
Riverso, A.S. Bogota, Colombia
Roark, M. Los Angeles, USNA CA
Robbins, L.G. East Lansing, USNA MI
Roberts, D.B. Oxford, GB-England
Roberts, P.A. Corvallis, USNA OR
Robertson, A. Edinburgh, GB-Scotland
Robertson, F.W. Aberdeen, GB-Scotland
Robertson, J.P. Davis, USNA CA
Roca, A. Oviedo, Spain
Rocha-Pite, M.T. Lisbon, Portugal
Rockwell, R.F. New York, USNA NY
Rodero, A. Cordoba, Spain
Rodino, E. Padova, Italy
Rodrigues, V. Tubingen, FR Germany
Rodriguez, J-C. Bogota, Colombia
Rokop, S. La Jolla, USNA CA
Romero, I.F. Bogota, Colombia
Ronnau, K.C. Giessen, FR Germany
Roote, J. Cambridge, GB-England
Rosbash, M. Waltham, USNA MA
Rose, M.R. Halifax, Canada
Rosewell, J. Leeds, GB-England
Rosset, R. Marseille, France
Rouault, J. Gif-sur-Yvette, France
Rowan, R. Salt Lake City, USNA UT
Rowe, A. Cambridge, GB-England
Roy, I. Calcutta, India
Rubio, A. Oviedo, Spain
Ruddell, M. Vancouver, Canada
Rudkin, G.T. Philadelphia, USNA PA
Ruiters, M. Nijmegen, Netherlands
Ruiz, A. Bellaterra, Spain
Russell, R.J. Canberra, Australia
Rutherford, P. Aberdeen, GB-Scotland

Sagarra, E. Barcelona, Spain
Sakaguchi, B. Fukuoka, Japan
Salas, F. Los Angeles, USNA CA
Salceda, V.M. Merida, Mexico
Salkoff, L. St.Louis, USNA MO
Salvaterra, P. Duarte, USNA CA
Sampsell, B. Buffalo, USNA NY
Samuels, L. Tempe, USNA AZ
San Miguel, E. Leon, Spain
Sanchez, A. Bellaterra, Spain
Sanchez, F. Oviedo, Spain
Sanchez, J.A. Oviedo, Spain
Sandler, L. Seattle, USNA WA
Sang, J.H. Brighton, GB-England
Santiago, E. Oviedo, Spain
Santos, M. Santiago de Compostela, Spain
Sarkar, D.N. Varanasi, India
Sato, T. Manhattan, USNA KS
Satou, T. Kobe, Japan
Savakis, C. Cambridge, GB-England
Savic, G. Pristina, Yugoslavia
Savontaus, M-L. Turku, Finland
Scavarda, N. St.Louis, USNA MO
Schafer, M. Dusseldorf, FR Germany
Schafer, U. Dusseldorf, FR Germany
Schaffer, H.E. Honolulu, USNA HI
Schaffer, H.E. Raleigh, USNA NC
Schalet, A. Leiden, Netherlands

Scharloo, W. Utrecht, Netherlands
Scherer, L.J. Chestnut Hill, USNA MA
Schlote, F.S. Aachen, FR Germany
Schmid, H. Fribourg, Switzerland
Schnewly, S. Basel, Switzerland
Schouten, S.C.M. Utrecht, Netherlands
Schubiger, G. Seattle, USNA WA
Schubiger, M. Seattle, USNA WA
Schulare, G. Mainz, FR Germany
Schulz, R. Cambridge, USNA MA
Schwartz, P. Tempe, USNA AZ
Schweizer, P. Zurich, Switzerland
Schwinck, I. Storrs, USNA CT
Schwochau, M. Dusseldorf, FR Germany
Scott, N. La Jolla, USNA CA
Seager, R.D. Cedar Falls, USNA IA
Sederoff, R.R. Raleigh, USNA NC
Segal, D. Cambridge, USNA MA
Seiger, M.B. Dayton, USNA OH
Semeriva, M. Marseille, France
Semionov, E.P. Sofia, Bulgaria
Sene, F.M. Ribeirao Preto, Brasil
Serban, N. Belgrade, Yugoslavia
Serra, L. Barcelona, Spain
Service, P.M. Halifax, Canada
Sha, Z. Shanghai, China-PRC
Shadravan, F. Honolulu, USNA HI
Shaffer, J.B. Raleigh, USNA NC
Sharma, A.K. Ludhiana, India
Sharma, R.P. New Delhi, India
Shearn, A. Baltimore, USNA MD
Sheldon, B.L. Sydney, Australia
Shen, N. Piscataway, USNA NJ
Shermoen, T. Berkeley, USNA CA
Shiba, T. Tokyo, Japan
Shimakawa, E. Honolulu, USNA HI
Shorrocks, B. Leeds, GB-England
Sick, K. Copenhagen, Denmark
Siddaveere, G.L. Mysore, India
Siegel, R.W. Los Angeles, USNA CA
Siegfried, E. St.Louis, USNA MO
Sierra, L.M. Oviedo, Spain
Silber, J. Paris, France
Sillans, D. Lyon, France
Silva, F. Burjasot, Spain
Simcox, A. Baltimore, USNA MD
Simkins, M. Sheffield, GB-England
Simmons, M.J. St.Paul, USNA MN
Simon, J. Ithaca, USNA NY
Simpson, P. Strasbourg, France
Simpson, P.R. Canberra, Australia
Sinclair, D. Vancouver, Canada
Sinclair, J. Brighton, GB-England
Singh, A.K. Varanasi, India
Singh, B.N. Varanasi, India
Singh, O.P. Varanasi, India
Singh, R.S. Hamilton, Canada
Sirotkin, K. Knoxville, USNA TN
Skibinski, D.O.F. Swansea, GB-Wales
Slatko, B. Williamstown, USNA MA
Smit, Z. Belgrade, Yugoslavia
Smith, B.R. Aberdeen, GB-Scotland
Smith, D. La Jolla, USNA CA
Smith, P.D. Atlanta, USNA GA
Smith, T. Edinburgh, GB-Scotland
Smolik-U., S. Cambridge, USNA MA
Snyder, L.A. St.Paul, USNA MN
Snyder, M. Eugene, USNA OR
Sobrier, M.L. Clermont-Ferrand, France
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Sokoloff, A. San Bernardino, USNA CA
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Solignac, M. Gif-sur-Yvette, France
Soliman, M.H. Armidale, Australia
Soll, D. New Haven, USNA CT
Somerson, N. Purchase, USNA NY
Sondergaard, L. Copenhagen, Denmark
Sonnenblick, B.P. Newark, USNA NJ
Sonobe, H. Kobe, Japan
Sparrow, J.C. York, GB-England
Spence, G.E. Bundoora, Australia
Spencer, H. Auckland, New Zealand
Sperlich, D. Tubingen, FR Germany
Spiegelman, G. Vancouver, Canada
Spiess, E.B. Chicago, USNA IL
Spivey, W.E. Norman, USNA OK
Spoerel, N. Cambridge, USNA MA
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Spradling, A.C. Baltimore, USNA MD
Springer, R. Vienna, Austria
Stacey, S. Vancouver, Canada
Stamatis, N. Patras, Greece
Stamenkovic-R., M. Belgrade, Yugoslavia
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Stark, W.S. Columbia, USNA MO
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Steinmann-Z., M. Zurich, Switzerland
Stephen, G. Aubiere, France
Stephenson, E.C. Rochester, USNA NY
Stewart, G. Quebec, Canada
Stewart, M.I. Sydney, Australia
Stocker, R.F. Fribourg, Switzerland
Stogerer, K. Tubingen, FR Germany
Streck, R. Berkeley, USNA CA
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Stumm-Z., E. Zurich, Switzerland
Stursa, I. Vienna, Australia
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Sutton, C. Ithaca, USNA NY
Suyo, T.M. Lima, Peru
Suzuki, D. Vancouver, Canada
Swift, H.S. Chicago, USNA IL
Syoji, E. Misima, Japan

Tai, H.J. Seoul, Korea
Tai, S. Vancouver, Canada
Takada, H. Sapporo, Japan
Takada, Y. Misima, Japan
Takikawa, S. Kitasato, Japan
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Tanimura, T. Okazaki, Japan
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Templeton, A.R. St.Louis, USNA MO
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Thompson, V. Chicago, USNA IL
Thorig, G. Utrecht, Netherlands
Threlkeld, S.F.H. Hamilton, Canada
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Wilson, S.D. Bronx, USNA NY
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Number of (Laboratories) and Drosophila Workers:

ASIA	(57 labs)	227 Workers	Australia, China-PRC, China-ROC, India, Japan, Korea, Malaysia, New Zealand, Sri Lanka, Thailand
CANADA	(11 labs)	57 Workers	
EUROPE	(86 labs)	476 Workers	Austria, Belgium, Bulgaria, Czechoslovakia, Denmark, Finland, France, FR Germany, Greece, Great Britain, Italy, Netherlands, Portugal, Spain, Sweden, Switzerland, Yugoslavia
LATIN AMERICA	(13 labs)	55 Workers	Argentina, Brasil, Chile, Colombia, Mexico, Peru
MID-EAST & AFRICA	(6 labs)	22 Workers	Egypt, Iraq, Israel, South Africa, Turkey
	(173 labs)	834 Workers	
UNITED STATES OF NORTH AMERICA	(144 labs)	564 Workers	50 states, plus Washington DC and Puerto Rico
TOTALS	(817 labs)	1,401 Workers	

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- Larochelle, C BSc asst enzyme polymorphism
- Lau You Hin, G GSc grad student subcellular distribution of enzymes, genetic regulation of ethanol metabolism
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Enciso, J MSc biochemical genetics of D.pseudoobscura
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Gutierrez-B, L F MSc population genetics of D.pseudoobscura from the Altiplano
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Hoenigsberg, H F PhD population genetics & evolution of Drosophila, speciation
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de Polanco, M M E MSc cytogenetics of D.pseudoobscura
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Khalil, M PhD AsstProf competition
Omar, A PhD AsstProf population genetics
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Lankinen, P Phil.lic photoperiodism, circadian rhythms
Lumme, J Phil.lic photoperiodism, phenology, circadian rhythms
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Nokkala, S Ph-lic research assoc cytogenetics
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Puro, J PhD AssocProf mechanism of segregation
Savontaus, M-L PhD AsstTeacher cytogenetics, Curator of stocks
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Bucheton, A Charge de Recherches)
DeGroote, F assistante) hybrid dysgenesis in
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Pelisson, A Chge de Rech) (I-R system)
Picard, G Maitre de Rech)
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Lachaise, D Charge de Rech ecological genetics & ecology
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Louis, J Charge de Rech ecology & ecophysiology
Payant, V Attache de Rech colour polymorphism
Rouault, J Charge de Rech interactions between Drosophila & parasitic wasps
Solignac, M Maitre asst mitochondrial DNA
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