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Prepared at the  
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University of Kansas  
Lawrence, Kansas



## Table of Contents

22ND ANNUAL DROSOPHILA CONFERENCE ANNOUNCEMENT. W.K. Baker . . . . .	55: 1
22ND ANNUAL DROSOPHILA CONFERENCE PROGRAM . . . . .	55: vi
21ST ANNUAL DROSOPHILA CONFERENCE REPORT. E. Murray . . . . .	55: 1
ERRATA - DIS 54 . . . . .	55: 4
BOWLING GREEN STOCK LIST. R.C. Woodruff . . . . .	55: 5
ANNOUNCEMENTS: Personal & Laboratory News, Material Requested/Available, and Other . . . . .	55: 8

## RESEARCH NOTES

ABEDIN, K., S. McNAMARA, D. OSTERBUR and W.W.M. STEINER. Studies on the effect of temperature on fitness and fecundity at the Esterase-6 locus in <i>D. melanogaster</i> . . . . .	55: 10
ALBORNOZ, J. and J. RUBIO. Location of a region controlling the suppression of normal bristles in <i>D. melanogaster</i> . . . . .	55: 11
ANDERSON, S., M. SANTOS and J. McDONALD. Comparative study of the thermostability of crude and purified preparations of alcohol dehydrogenase (EC 1.1.1.1) from <i>D. melanogaster</i> . . . . .	55: 13
BEWLEY, G.C. and S. LUBINSKY. Toxicity to the dietary administration of hydrogen peroxide in acatalasemic <i>Drosophila</i> . . . . .	55: 14
BOS, M. and A.C. BOEREMA. Oviposition preferences in six <i>Drosophila melanogaster</i> -subgroup species. . . . .	55: 15
BOULETTEAU, M. and C. BIEMONT. Reproductive potential of a Hymenopteran parasite of <i>D. melanogaster</i> . . . . .	55: 16
BOURNAIS-VARDIABASIS, N. and M. BOWNES. Cell death in the tumorous head mutant of <i>D. melanogaster</i> . . . . .	55: 17
BOWNES, M. and M. SEILER. UV irradiation of <i>Drosophila</i> embryos. . . . .	55: 18
BREGLIANO, J.C., A. BUCHETON, J.M. LAVIGE, A. PÉLISSON and G. PICARD. Hybrid dysgenesis in <i>D. melanogaster</i> : the I-R system. . . . .	55: 19
BRNCIC, D. and M. BUDNIK. Colonization of <i>D. subobscura</i> Collin in Chile. . . . .	55: 20
BRYANT, M.L. and M.R. MURNIK. The mutagenicity of herbicides in <i>D. melanogaster</i> . . . . .	55: 20
BULYZHENKOV, V.E. and V.I. IVANOV. Expression of Antennapedia <sup>50</sup> in triploid <i>D. melanogaster</i> . . . . .	55: 21
CALVEZ, C. Reduced mitotic activity in anterior and posterior follicle cells of the egg-chamber of <i>D. melanogaster</i> . . . . .	55: 22
CARLSON, E., P. FERRIOLA and E. SCHUCHMAN. Pseudoallelism at the vestigial locus. . . . .	55: 23
CHOO, J.K. Genetic change of Korean natural populations of <i>D. melanogaster</i> . . . . .	55: 24
CLYDE, M. The chromosomes of <i>D. rubra</i> Sturtevant. . . . .	55: 24
CLYDE, M. Chromosome IV variation in <i>D. albomicans</i> Duda. . . . .	55: 25
COMENDADOR, M.A. Abnormal bristles that show maternal inheritance in <i>D. simulans</i> . . . . .	55: 26
CREUS, A. and R. MARCOS. Relationship between mating speed and duration of copulation in <i>D. melanogaster</i> . . . . .	55: 26
DAVID, J.R., M. de SCHEEMAEKER-LOUIS and E. PLA. Evolution in the seven species of the <i>D. melanogaster</i> subgroup: comparison of the electrophoretic mobility of the enzymes produced by the Adh locus. . . . .	55: 28
DAVIS, B.K. Mutants which cause abnormal rotation of the abdomen or genitalia. . . . .	55: 29
DAVIS, B.K. A new twist on an old mutant. . . . .	55: 31
DE SALLE, R., J.S. YOON and L.H. THROCKMORTON. Karyotypes of two new species of the virilis group of <i>Drosophila</i> . . . . .	55: 33
DEWEES, A.A. Lethal-bearing genomes from a Texas population of <i>D. melanogaster</i> . . . . .	55: 34
DIAMANTOPOULOU-PANOPOULOU, E. Estimation of <i>N<sub>em</sub></i> by allelism method in <i>D. subobscura</i> . . . . .	55: 35
DIAMANTOPOULOU-PANOPOULOU, E. and H. BACOUILLAS. "Sex ratio" in <i>D. obscura</i> . . . . .	55: 36
DOANE, W.W. Midgut amylase activity patterns in <i>Drosophila</i> : nomenclature. . . . .	55: 36
ESPINET, S. and M.L. TRACEY. Detection of differences in the element patterning of <i>D. melanogaster</i> mating behavior. . . . .	55: 39
FADDA, S., S. SANGIORGI and E. PIERAGOSTINI. G6PD electrophoretic phenotype variation during development of <i>D. melanogaster</i> laboratory populations. . . . .	55: 41
FLEURIET, A. Analysis of a polymorphism quite common in French natural populations of <i>D. melanogaster</i> . . . . .	55: 43
FONTDEVILA, A., W.T. STARMER, W.B. HEED and J.S. RUSSELL. Differential mating activity in two co-existing species of <i>Drosophila</i> . . . . .	55: 43

GAMO, S., M. OGAKI and E. NAKASHIMA-TANAKA. Anesthetics resistance in <i>D. melanogaster</i> .	55: 45
GAUSZ, J., A.A.M. AWAD and H. GYURKOVICS. New deficiencies for the <i>kar</i> locus of <i>D. melanogaster</i> .	55: 45
GAYATHRI, M.V. and N.B. KRISHNAMURTHY. Preliminary studies on the effects of a mercurial fungicide Ceresan on fecundity in <i>D. melanogaster</i> .	55: 48
GERSHENSON, S. Additional data on putative insertion mutations in wild populations of <i>D. melanogaster</i> .	55: 46
GOLUBOVSKY, M.D. and I.K. ZAKHAROV. Simultaneous reversions of two sex-linked unstable mutations.	55: 49
GRANT, S. and E. RAPPORT. The effect of lactamide on the mutant <i>eyeless</i> <sup>2</sup> .	55: 53
GUILLET, C. Mitochondrial activity during metamorphosis in <i>D. melanogaster</i> .	55: 51
GUNAWAN, B. and J.S.F. BARKER. Adult viability of <i>D. buzzatii</i> in stress environments.	55: 53
HARDY, R.W. Crystal aggregates in the primary spermatocytes of XO males in <i>D. melanogaster</i> .	55: 54
HARDY, R.W. and J.A. KENNISON. Identification of a small Y chromosome region responsible for meiocyte and spermatid abnormalities typically observed in XO males.	55: 55
HAWLEY, R.S. Radiation-induced nondisjunction in females homozygous for <i>In(1)sc</i> <sup>8</sup> .	55: 56
HAZELRIGG, T. and T.C. KAUFMAN. Newly induced mutations of <i>doublesex</i> .	55: 56
HAZRA, S.K., J. BANERJEE and S.K. SEN. Location and nature of white-ivory ( <i>w</i> <sup>1</sup> ) in the white locus of <i>D. melanogaster</i> .	55: 57
HEDRICK, P.W. and E. MURRAY. Competition between <i>D. melanogaster</i> and <i>D. simulans</i> from natural populations.	55: 58
HEGDE, S.N. and N.B. KRISHNAMURTHY. Studies on the <i>Drosophila</i> fauna from three localities of Maharashtra State: India.	55: 60
HENIKOFF, S. A more conventional view of the "ebony" gene.	55: 61
HODGETTS, R.B. A cytogenetic description of three duplications in which portions of proximal 2L have been inserted into the Y-chromosome.	55: 63
INGHAM, P.W. Genetic analysis of <i>trithorax</i> , <i>trx</i> , a new homoeotic mutant of <i>D. melanogaster</i> .	55: 63
ITOH, K. Lack of chromosomal polymorphism and low frequencies of unique inversions in <i>D. simulans</i> .	55: 64
JALLON, J.-M. and Y. HOTTA. A proposal for a new genetic scheme to hunt for sex-appealless mutants of <i>D. melanogaster</i> .	55: 65
JHA, A.P., B.N. PANDEY and D.N. MISHRA. Substrate specificities of alcohol dehydrogenase in <i>Drosophila</i> .	55: 65
JOINER, M. and J.S. JOHNSTON. Determination of exact age in <i>Drosophila</i> .	55: 66
KAJI, S. and Y. USHIODA. The duration of cell cycle in the development of the Bar eye disc cells in vitro.	55: 68
KAMPING, A. and W. VAN DELDEN. The <i>Adh</i> <sup>nl</sup> mutant of <i>D. melanogaster</i> .	55: 69
KATOH, S. and Y. ARAI. Configurational determination of neopterin from <i>sepia</i> mutant of <i>D. melanogaster</i> .	55: 70
KAUR, P. and R. PARKASH. Ontogeny of esterase in <i>Zaprionus paravittiger</i> .	55: 69
KAUROV, B.A. The effect of trypsin on "survivability" of imaginal disks of <i>D. melanogaster</i> .	55: 71
KAUROV, B.A. Manifestation of mutation singed on the homoeotic limbs, caused by the action of homoeotic mutations <i>Nasobemia</i> and <i>aristapeda</i> at different temperatures.	55: 71
KEMPHUES, K.J. and T.C. KAUFMAN. Two-dimensional gel analysis of total proteins from X/O, X/Y, X/Y/Y, X/Ys and X/Y <sup>L</sup> testes from <i>D. melanogaster</i> .	55: 72
KERVER, J.W.M. and R. BIJLSMA. The effect of DDT on the activity of the pentose phosphate cycle in <i>D. melanogaster</i> .	55: 73
KING, R.C. and B.D. BUCKLES. Three mutations blocking early steps in <i>Drosophila</i> oogenesis: <i>fs(4)34</i> , <i>fs(2)A16</i> , and <i>fs(1)231M</i> .	55: 74
KISS, I. and J. SZABAD. Characteristics of some new X-linked pupal lethals of <i>D. melanogaster</i> .	55: 75
KÖHLER, W., J. KRAUSE and A. MICHUTTA. Heritability of phototactic behavior in <i>Drosophila</i> .	55: 77
KRAUSE, J., A. MICHUTTA and W. KÖHLER. Oviposition preferences in <i>D. melanogaster</i> .	55: 78
KRIMBAS, C.B. and M. LOUKAS. Chromosomal homologies among four <i>Drosophila</i> species.	55: 80

LAWLOR, T.A. Genetic and cytological localization of mei-9. . . . .	55: 81
LEE, T.J. Sexual isolation among four species in the <i>D. auraria</i> complex. . . . .	55: 82
LEIGH BROWN, A.J. Molecular weights of seven <i>Drosophila</i> enzymes. . . . .	55: 82
LEWIS, R.A., T.C. KAUFMAN and R.E. DENELL. Genetic analysis of the Antennapedia gene complex (ANT-C): mutant screen of proximal 3R, section 84B-D. . . . .	55: 85
LINDSLEY, D.E., L.S.B. GOLDSTEIN and L. SANDLER. Male sterility in maternal-effect mutants. . . . .	55: 84
LOVERRE, A. and R. CICCHETTI. A male-specific lethal gene in <i>D. melanogaster</i> . . . . .	55: 88
MAJUMDAR, D. and A.S. MUKHERJEE. Morphogenetic expression of a reversible Bar-inversion of <i>D. melanogaster</i> . . . . .	55: 89
MALOGOLOWKIN-COHEN, Ch. Inversion polymorphism in <i>D. subobscura</i> in Israel. . . . .	55: 90
MALOGOLOWKIN-COHEN, Ch. The distribution of <i>D. subobscura</i> in relation to other species in Israel. . . . .	55: 91
MARCOS, R. and A. CREUS. Predictive value of heritability estimates. . . . .	55: 93
MARENGO, N.P. The ultrastructure of normal and "rotated" prepupal muscles of <i>D. melanogaster</i> . . . . .	55: 94
MARONI, G. A duplication of Adh in association with Sco. . . . .	55: 96
MAROY, P., K. KOCZKA, É. FEKETE and J. VARGHA. Molting hormone titer of <i>D. melanogaster</i> larvae. . . . .	55: 98
MATHER, W.B. and G. BALWIN. Inversions in two species of <i>Drosophila</i> from the River Kwai, Thailand. . . . .	55: 99
MATHER, W.B. and P. THONGMEERKOM. Chromosome map of <i>D. albomicans</i> . . . . .	55:101
MATHER, W.B., W.R. KNIBB and G. BALWIN. Inversions in <i>D. sulfurigaster albostrigata</i> from the River Kwai, Thailand. . . . .	55:103
MGLINETZ, V.A. Bristle phenotype of split flies contradicts the hypothesis of quantal mitoses. . . . .	55:105
MGLINETZ, V.A. Interaction of homoeotic mutations <i>Pc</i> <sup>2</sup> and <i>Cbx</i> . . . . .	55:107
MGLINETZ, V.A. Temperature-sensitive period (TSP) of lethal action of split ( <i>spl</i> ) in <i>D. melanogaster</i> . . . . .	55:107
MICHINOMAE, M., S. SHINDO and S. KAJI. Patterns of acid phosphatase during the development of <i>D. melanogaster</i> . . . . .	55:108
MICHUTTA, A., J. KRAUSE and W. KÖHLER. Reproductive fitness in exchange lines of a selected photonegative strain and its control. . . . .	55:110
MIKASA, K. and T. NARISE. The relation between dispersive behavior and temperature. II. Sex difference. . . . .	55:111
MILKMAN, R. Thermostability variation in <i>D. melanogaster</i> allozymes. . . . .	55:113
NAGARAJ, H.J. and N.B. KRISHNAMURTHY. <i>Drosophila</i> fauna of Dandeli and Ambikanagar. . . . .	55:114
NAGARAJ, H.J. and N.B. KRISHNAMURTHY. Effect of crowding and temperature on the rate of development in <i>D. nasuta</i> . . . . .	55:114
NARISE, S. and M. SASAKI. Thermal stability difference among $\alpha$ Gpdh allozymes from <i>D. virilis</i> . . . . .	55:116
NISSANI, M. Brain transplantation and behavior. . . . .	55:117
NÖTHIGER, R., M. ROOST and T. SCHÜPBACH. "Masculinizer" is an allele of "doublesex". . . . .	55:118
OGAKI, M., H. NABATA, E. NAKASHIMA-TANAKA and S. GAMO. Ether sensitivity at embryonic stage of <i>D. melanogaster</i> . . . . .	55:117
OHNISHI, S. and R.A. VOELKER. Genetic mapping of hexokinase-C in <i>D. simulans</i> . . . . .	55:119
O'TOUSA, J. and P. SZAUTER. The initial characterization of non-claret disjunctional ( <i>ncd</i> ): evidence that <i>ca</i> <sup>nd</sup> is the double mutant, <i>ca ncd</i> . . . . .	55:119
PAIK, Y.K. and K.C. SUNG. Chromosome inversions in Korean populations of <i>D. melanogaster</i> . . . . .	55:120
PINSKER, W. Relation between effective population size and allozyme polymorphism in <i>D. subsilvestris</i> and <i>D. subobscura</i> . . . . .	55:120
PINSKER, W. Sterility in <i>D. subobscura</i> males homozygous in a rare allele at the $\alpha$ Gpdh locus. . . . .	55:121
PRAKASH, H.S. and SREERAMA REDDY, G. Distribution of different species of <i>Drosophila</i> in Agumbe (Western Ghats), South India. . . . .	55:122
RAHMAN, R. and D.L. LINDSLEY. Effect of proximal X-chromosome deletions on male fertility. . . . .	55:123
RAMSHAW, J.A.M. and J.A. COYNE. Is secondary modification primarily responsible for observed enzyme polymorphism? No! . . . . .	55:125

RANSOM, R. Investigation of temperature sensitivity in three eye mutations. . . . .	55:126
RASMUSON, B. and I. MONTELL. Genetic instability and the production of transposing elements in <i>D. melanogaster</i> . . . . .	55:127
RICHMOND, R.C. Temperature and dessication tolerance in four species of the <i>affinis</i> subgroup. . . . .	55:127
RICHMOND, R.C. Effects of temperature and humidity stress on genotype distribution at six allozyme loci. . . . .	55:129
ROBERTSON, A. Quantitative variation on the fourth chromosome of <i>D. melanogaster</i> . . .	55:130
ROMANS, P. Gene conversion in <i>mei-9a</i> , a crossover defective mutant in <i>D. melanogaster</i> . . . . .	55:130
ROMANS, P. Effects of purine selection on survival of <i>Drosophila</i> mosaic for Xanthine Dehydrogenase (XDH) activity. . . . .	55:132
SCHÄRER, E. Brief ecological notes on <i>Drosophila</i> collections from Berlin. . . . .	55:135
SCHUCHMAN, E., A. PORT and E.A. CARLSON. Mutagenesis of the vestigial region of <i>D. melanogaster</i> . . . . .	55:134
SCHUPPE, N.G., T.V. SYROTA and V.T. KAKPAKOV. Turnover of ribosomal RNA in the course of <i>D. melanogaster</i> development. . . . .	55:137
SCHUPPE, N.G., T.V. SYROTA, N.G. NOSOVA and V.T. KAKPAKOV. Protein-synthesizing activity of <i>Drosophila</i> ribosomes containing rRNA with hidden breaks. . . . .	55:137
SMITH, C.M., J.N. THOMPSON, Jr., and R.C. WOODRUFF. An attempt to transmit male recombination activity by non-genetic means. . . . .	55:138
STEIN, S.P. and E.A. CARLSON. Mosaicism of eye color induced in mature sperm of <i>D. melanogaster</i> . . . . .	55:139
TSAKAS, S. and E. DIAMANTOPOULOU-PANOPOULOU. Is the "hidden heat sensitive polymorphism" (crude extract) polymorphism of the structural examined locus in all cases? Experiments with <i>D. subobscura</i> . . . . .	55:140
WAKIMOTO, B.T., R.A. LEWIS and T.C. KAUFMAN. Genetic analysis of the Antennapedia gene complex: mutant screen of proximal 3R, bands 84A-84B1. . . . .	55:140
WATABE, H., E. MOMMA and M.T. KIMURA. Changes in drosophilid fauna at the University Botanical Garden in Sapporo, Japan. . . . .	55:141
WEISS, R.L. The inhibition of fertilization in <i>D. melanogaster</i> under the influence of sodium tungstate. . . . .	55:143
WELTER, R.J. and J.F. McDONALD. A search for third-chromosome ADH active loci. . . .	55:143
WICKER, C. and J.R. DAVID. Preliminary characterization of a $\beta$ -N-acetyl-glucosaminidase from <i>D. melanogaster</i> adults. . . . .	55:144
ZHIMULEV, I.F. and O.V. ILYINA. Localization and some characteristics of <i>sbr</i> in <i>D. melanogaster</i> . . . . .	55:146

## TECHNICAL NOTES

BÉLO, M. and P.M. LACAVA. Box for testing nutritional preferences (yeasts) in <i>Drosophila</i> . . . . .	55:146
BOCK, I.R. and P.A. PARSONS. Culture methods for species of the <i>Drosophila</i> ( <i>Scaptodrosophila</i> ) <i>coracina</i> group. . . . .	55:147
COBEL-GEARD, S.R. and H. GAY. A new simplified method for the preparation of neuroblast mitotic chromosomes from <i>D. melanogaster</i> . . . . .	55:148
CROSSLEY, S. and J. McDONALD. A method for permanently recording courtship song and courtship behavior simultaneously in <i>Drosophila</i> . . . . .	55:150
DE JONG, F. and G.E.W. THÖRIG. A simple test for intra-electromorphic variants of alcohol dehydrogenase in <i>D. melanogaster</i> . . . . .	55:151
GUPTA, A.P. A new technique for collecting <i>Drosophila</i> eggs. . . . .	55:152
JOHNSON, P. and D.E. COWLING. A courtship song simulator for <i>Drosophila</i> . . . . .	55:152
JOHNSTON, J.S. Hawaiian <i>Drosophila</i> with colored headlamps: a new mark-recapture technique. . . . .	55:154
KELTNER, L. Low temperature enhancement of fluorescence as an aid in chromosome banding. . . . .	55:155
LOUKAS, M. and C.B. KRIMBAS. Isozyme techniques in <i>D. subobscura</i> . . . . .	55:157
LUMME, J. An efficient instrument to measure freezing points of insects. . . . .	55:155
MAHOWALD, A.P. Improved method for dissecting late ovarian stages. . . . .	55:156
MAJUMDAR, D. and A.S. MUKHERJEE. Culture of <i>Drosophila</i> salivary glands. . . . .	55:159



MILANOVIĆ, M. and W.W. DOANE. Affinity chromatographic purification of amylase from <i>Drosophila</i> species. . . . .	55:160
MITTLER, S., K. SCHROEDER and D. WILSON. <i>Aspergillus flavus</i> producer of mutagen and carcinogen aflatoxin can contaminate laboratory cornmeal-molasses-agar media. . . .	55:162
MOLNÁR, I. and I. KISS. An easy method for rearing axenic <i>Drosophila</i> lines. . . . .	55:163
NÓTHIGER, R. and C. LABHART. A self-amplifying system for mass collection of unfertilized eggs. . . . .	55:162
PARENTE, A. and R. ARKING. A convenient method of collecting moderately large numbers of eggs from <i>D. melanogaster</i> . . . . .	55:164
PASIC, T.R. and H. NICKLA. A BASIC program for construction of fate maps. . . . .	55:165
SAWICKI, J.A. and W.W. DOANE. Preparation of "small" polytene chromosomes for cytological analysis. . . . .	55:166
SHEARN, A. Reintroduction of $y^+$ onto a TM3 chromosome. . . . .	55:167
TAKAMURA, T. Multi-roomed, multi-purpose chamber for <i>Drosophila</i> . . . . .	55:168
VALENTIN, J. A reasonably priced automatic medium dispenser. . . . .	55:168
WILLIAMSON, R.L. A notation for genetic mosaics. . . . .	55:169

## TEACHING NOTES

BRYANT, S.H. Salivary preparations from <i>D. pseudoobscura</i> . . . . .	55:169
KLUG, W.S., G. NICHOLLS and T.W. KOTTKE. <i>Drosophila</i> transmission genetics computer package. . . . .	55:169
PYE, Q. New white-eyed <i>Drosophila</i> "unknown" stocks for genetics laboratory courses. .	55:171
PYE, Q., D. KNIPPLE and R. MacINTYRE. Construction of segmental deficiency stocks from Y-autosome translocation stocks. . . . .	55:170
SVED, J.A. A computer program which saves on cooking and washing up. . . . .	55:171
WRIGHT, C.P. A method for transferring etherized flies into a container of active flies. . . . .	55:172

CLASSIFIED MUTANTS OF <i>D. MELANOGASTER</i> . S.H. Bryant and M.R. Murnik . . . . .	55:173
LINKAGE DATA . . . . .	55:191

NEW MUTANTS - *D. MELANOGASTER*. Report of:

G. Alvarez . . . . .	55:193	Y. Inoue . . . . .	55:206
M. Ashburner, J. Faithfull et al. . . . .	55:193	W.-E. Kalisch . . . . .	55:206
M. Ashburner et al. . . . .	55:196	D.T. Kuhn and F.C. Walker . . . . .	55:207
M. Ashburner and V. Velissariou . . . . .	55:196	E.B. Lewis . . . . .	55:207
B. Baker . . . . .	55:197	L.I. Mukhina and I.F. Zhimulev . . . . .	55:209
L. Craymer . . . . .	55:197	H. Nöthel . . . . .	55:208
L. Craymer and E. Roy . . . . .	55:200	C.A. Poodry . . . . .	55:210
J. Crist et al. . . . .	55:204	P.T. Shukla . . . . .	55:210
M. Crosby . . . . .	55:204	V. Vasudev and N.B. Krishnamurthy . . . . .	55:210
R. Frankham and R.K. Nurthen . . . . .	55:204	V. Vasudev et al. . . . .	55:211
M.A. Hughes and M. Shelton . . . . .	55:204	J.R.S. Whittle . . . . .	55:211
O.V. Ilyina et al. . . . .	55:205	I.F. Zhimulev et al. . . . .	55:211

NEW MUTANTS - *DROSOPHILA* SPECIES. Report of:

S.H. Bryant . . . . .	55:212	H.S. Prakash and G. Sreerama Reddy . . . . .	55:216
D. DeJianne . . . . .	55:213	R.C. Woodruff . . . . .	55:217
C.W. Hinton . . . . .	55:213		

COURTSHIP IN *DROSOPHILA MELANOGASTER*: A Song For *Drosophilists*

D.F. Sewell and J.P. Baggaley . . . . .	55:217
HERSKOWITZ BIBLIOGRAPHY: Part I . . . . .	55:218
HERSKOWITZ BIBLIOGRAPHY: Part II . . . . .	55:236

## Snowbird, Utah

## PROGRAM

Thursday, 1 May

6:00 PM Registration  
8:00 PM Combined Social and Poster Session

Friday, 2 May

8:00-9:00 AM Registration Continued  
9:00-9:45 AM Welcome Bender, Stanford Univ., "Rosy, ace, and bithorax, a walker's guide"  
9:45-10:15 AM Coffee Break  
10:15-11:00 AM Chip Quinn, Princeton Univ., "Genetic studies of learning and memory in *Drosophila*"  
11:00-11:45 AM Bruce Baker, Univ. of California, San Diego, "Sex and the single cell"  
12:00-1:00 PM Catered Lunch  
1:00-5:30 PM Concurrent Sessions (3:00-3:30 break): (a) Chromosomal, (b) Populational, (c) Developmental, (d) Molecular  
5:30-8:00 PM Dinner (on your own)  
8:00-10:00 PM Concurrent Sessions: (a) Chromosomal, (b) Populational, (c) Behavioral and Neurobiological, (d) Molecular

Saturday, 3 May

9:00-9:45 AM Joe Dickinson, Univ. of Utah, "Regulatory genes in Hawaiian *Drosophila*"  
9:45-10:15 AM Coffee Break  
10:15-11:00 AM Bill Heed, Univ. of Arizona, "Central and marginal populations revisited"  
11:00-11:45 AM John Palka, Univ. of Washington, "Neurobiology of homoeotic mutants"  
11:45-7:00 PM Free Afternoon  
7:00 PM Western Barbeque for all registrants

Sunday, 4 May

9:00-9:45 AM Jerry Coyne, Univ. of California, Davis, "Of flies and men: human blood and enzyme variation in *Drosophila*"  
9:45-10:15 AM Coffee Break  
10:15-11:00 AM Dan Lindsley, Univ. of California, San Diego, "Some chromosomal requirements for male fertility"  
11:00-11:30 AM Charlie Sing, Univ. of Michigan, "Enzymes, glycolysis, and fitness"  
11:30-12:00 Business Meeting  
12:00 Noon Conference Ends

Additional Information

For further information concerning conference arrangements, please contact Conferences and Institutes, 1152 Annex Building, University of Utah, Salt Lake City, Utah 84112, (801) 581-5809. Direct questions concerning program arrangements and the poster session to Dr. William Baker, Department of Biology, University of Utah, Salt Lake City, Utah 84112.

William K. Baker, University of Utah

The 1980 Conference will be held at the Snowbird Resort. As you may know, Snowbird is one of the most famous ski resorts in the country and is less than a 3/4 hour drive from Salt Lake City. The meeting will commence at 8:00 PM May 1 with a combination informal social and poster session. The Conference will close at noon, Sunday, May 4.

There will be the usual general sessions with invited speakers as well as concurrent sessions on more specialized subjects.

There will be one afternoon without scheduled sessions in order for the participants to more fully enjoy the scenic beauty of Snowbird. It is anticipated that the ski lifts will close that weekend. Those to wish to be assured of skiing should make reservations on their own to precede the Drosophila meetings.

This notice is being sent to those who either attended the meeting last year at Indiana University or who responded to notices of that meeting. The program and reservation forms will be sent out in February. This notice is to help you in planning your spring activities.

The living facilities at Snowbird are excellent (I've stayed there). I am assured that the food is also, and last week I checked out the meeting rooms which are more than adequate although the room for the general session is not as nice as most university lecture auditoriums. Since we are meeting at the end of the ski season, we will receive reduced room rates—but more details in the February notice. If you wish to have the final announcement and the registration blank, contact: William K. Baker, Department of Biology, University of Utah, Salt Lake City, Utah 84112.

For complete program details, see page vi (opposite).

## 21ST ANNUAL DROSOPHILA CONFERENCE REPORT

Elizabeth Murray, University of Kansas

The 21st Annual Drosophila Conference was held in Bloomington, Indiana from May 17 to 20 this year, hosted by the University of Indiana Biology Department. Some 300 drosophilists with interests ranging from molecular biology to population genetics met to attend seminars, short talks during concurrent sessions, view poster sessions and to socialize informally at the numerous social evenings culminating in a pleasant banquet at the Poplars Research and Conference Center. The local arrangements were admirably handled by Thomas Kaufman, who is heartily congratulated for an excellent meeting, albeit a year late! DIS is publishing brief summaries of the eight invited seminars for those unlucky enough not to attend these excellent meetings. Next year's meeting will be at the University of Utah in Salt Lake City. William Baker will be the host for this meeting to be held in May.

### Alcohol Adaptation in Drosophila: A Model System for the Study of Evolution

John McDonald of Iowa State University brought us up to date on some of his group's current work on alcohol dehydrogenase (ADH), an enzyme studied by drosophilists from population geneticists to molecular biologists. McDonald considers ADH ideal as a model system since its genotype, phenotype and environment can be manipulated in laboratory situations. McDonald focused his seminar on the role of the producer and regulator genes and their interaction in the adaptive process. His research method has been to use several lines made homozygous for either the fast (F) or slow (S) ADH allele, which maps to chromosome 2. This system is manipulated by substituting different third chromosomes (and with its possible regulator genes), by altering the level of several different alcohols in the environment, or by varying the level of the enzyme using  $K_m$  and  $V_{max}$ , the Michaelis Menton coefficients. This information can be used to generate hypotheses concerning the adaptive significance of the F and S alleles which can be tested in population cages. Based on this model system, McDonald has demonstrated that third chromosome regulator genes can affect competitive ability of lines made homozygous for ADH F and S alleles. He is currently testing the hypothesis that some S homozygous lines will out-compete some F lines at low alcohol concentrations even though F strains always out-compete S strains in high alcohol environments, a hypothesis suggested by his data on the enzyme activity of these alleles at different alcohol and NADH concentrations.

### In Situ Hybridization of tRNA's of D. m.

Shizu Hayashi of the University of British Columbia outlined for us her research group's methods for mapping tRNA sites to bands in polytene chromosomes using purified labeled tRNA species as probes, and presented some of their results. They have found that sites of tRNA hybridization are scattered throughout the genome, even for a single tRNA species, although the labeling can vary from intense to weak. However, certain regions are hybridized by more than one kind of tRNA probe. This could be due to hybridization by contaminant tRNA, to tRNA's sharing partially complementary regions, or to different, adjacent tRNA genes. These hypotheses are currently being tested using hybridization competition by different tRNA's for the same site, which can differentiate the first two situations from the last. In addition, recombinant DNA plasmids are being obtained which can hybridize to the tRNA probes. When two tRNA species hybridize to the same plasmid, that region of DNA can be directly sequenced to differentiate between these three possible cases. As Hayashi and her group continue to expand the number of species of tRNA purified enough to use as probes, they will broaden our total pictures of the tRNA loci in the Drosophila genome.

### Ribosomal RNA Synthesis and Ribosomal Protein Assembly in D. m.

Yean Chooi of Indiana University presented her group's current research into the transcription of rRNA genes and the isolation and purification of proteins involved in ribosomal assembly. Chooi and her group have focused on the variation in size seen in the 38S precursor rRNA. They have found some rRNA genes with an insert varying in size from 500 to 6000 bases in the third quarter of the 28S region. She offered evidence that this intervening sequence is transcribed. She argued that these longer rRNA genes exceeded the range of stretching seen in experimental situations. Distribution of rRNA genes with intervening sequences appeared random with respect to those without them. Chooi discussed evidence for an hypothesis that shorter normal rRNA genes without intervening sequences are fully transcribed while longer genes with 28S intervening sequences are partially transcribed to produce complete 18S regions and incomplete 28S regions, a situation which might be advantageous if 18S rRNA had a higher turnover rate. Chooi and her group are also interested in determining when ribosomal protein assembly begins and whether genes with intervening sequences work well in ribosomal biogenesis. Chooi's first task has been to develop techniques to separate and purify these basic, insoluble, and numerous proteins in Drosophila. Their methods has been to elute six groups of proteins and run these groups on two-dimensional gels. Smaller samples are eluted in each of these six groups and run on similar two-dimensional gels. A number of these smaller pools contain only one protein, and more contain two or three proteins separable on the gels. Antibodies can be raised to these proteins thus isolated and can be used to purify large amounts of these proteins, so now the ribosomal assembly studies can begin.

### A New Theory of Speciation by the Founder Principle

Alan Templeton of Washington University discussed the necessity of uniting models of speciation with population genetics theory to yield testable hypotheses. Templeton suggests that the current speciation models with their emphasis on geography may restrict more than expand our understanding of speciation as a genetic process. He offers instead a matrix of situations where speciation is more or less probable based on the genetic changes involved and the population structure present when these genetic changes occur. Templeton identifies four genetic mechanisms which can result in speciation: adaptive divergence, genetic transilience, chromosomal rearrangements, and disruptive and clinal selection. He also specifies three types of population structure which can lead to speciation: a founder event, the classic model of Mayr with a split in a large population, and no population split at all. Templeton considered in detail the probability of speciation due to the mechanism he terms genetic transilience in all three types of population structure as an example of the usefulness of his matrix of speciation situations. He defines genetic transilience as a rapid shift in a multi-locus complex in response to a sudden shift in the genetic environment characterized by increasing homozygosity with respect to that of the ancestral population. The conditions leading to genetic transilience include inbreeding and the restriction of the genetic background due to a founder event. The parameters important in promoting genetic transilience are in-

breeding effective size ( $N_{ef}$ ) and variance effective size ( $N_{ev}$ ) of both the original and the founder population. Genetic transience is extremely unlikely to lead to speciation in a panmictic population or one in which a large founder population is involved. However, Templeton suggests that genetic transience could lead to speciation when a very small founder population splits off from a large panmictic population which has a high  $N_{ef}$  and  $N_{ev}$ . However, organisms able to reestablish the founder population's genetic variation due to a variety of buffers against inbreeding would avoid a genetic transience, and *D. m.* may be a good example of such a species.

#### Studies on Gene Organization in Drosophila

Art Chovnick of the University of Connecticut reviewed for us the progress his group has made towards understanding the genic organization of the rosy locus, to which genes for the production of Xanthine dehydrogenase (XDH) have been mapped. They have mapped approximately 70 unquestionable XDH structural mutants detected electrophoretically within the rosy locus. They showed that the structural mutants mapped to the left end of the locus, bands 87D 12+13, but they did not detect a control region in this fashion. Chovnick then searched for lines which seemed to vary in the intensity of their enzyme staining, indicating different quantities of enzyme were present. Such inbred lines were obtained and Chovnick showed using rocket electrophoresis that variants were present which produced more or fewer molecules of XDH than normally present. Using fine structure mapping techniques the high and low XDH mutants were mapped to separate sites to the left of the crossover point within the structural gene. Chovnick and his group have determined that the rosy control region is approximately 1300 bases long while the structural region is 4100 bases long. Because the size of the structural region is close to that predicted based on the enzyme site, Chovnick suggests that the rosy locus is unlikely to contain the intervening sequences found in some eukaryote genes.

#### The Engrailed Gene: Compartments and Positional Information in Drosophila Imaginal Discs

Mike Russell of the University of Alberta presented a new model to explain the formation of compartments and the role of positional information in *Drosophila* imaginal discs. French and co-workers have published a model to account for the patterns of regeneration seen in disc fragments in culture in which specification of position in a two-dimensional sheet of cells requires two pieces of information: the shortest intercalation rule and the complete circle rule. They used a system of polar coordinates with angular and radial values. Russell proposes a modification of French's model which incorporates all features of the original except the polar coordinate system and the two rules. Russell's model uses a coordinate system based on three axes: ventral-dorsal, anterior-posterior, and proximal-distal. In addition, he offers a single rule to explain patterns of regeneration: When normally nonadjacent positional values are confronted, growth occurs at the junction until cells with all the intermediate values have been reached, and then growth ceases. Russell offered examples to show that this model was more parsimonious and less biologically complex than the earlier model and presented evidence that two mutants, the engrailed gene and the polycomb gene, represent a defect in growth on one of the three axes proposed, accounting for the mirror image symmetry of the defects.

#### The Chorion Genes of Drosophila

Allen Spradling of the Carnegie Institute described his group's progress in isolating chorion structural proteins and their mRNA's, the sequence of activation of these genes in normal egg production, and their role in chorion mutations such as ocellolus. Spradling's group has purified chorion, solubilized it, and used two-dimensional electrophoresis to isolate the more than 20 proteins, five of which were very abundant. Then they determined the sequence of appearance of chorion proteins during the 14 stages of oogenesis. Spradling's group then isolated the follicle cells which surround the oocytes and produce these proteins and purified from them a small number of distinct mRNA's. These mRNA's were transcribed in a cell free system and produced proteins close to those spots of chorion proteins except for a slightly higher molecular weight. These mRNA's appeared and disappeared in the stages of o-

ogenesis in the sequence predicted from the chorion proteins they were determined to code for. Next they used the techniques of recombinant DNA to establish a library of 20,000 clones from late stage oogenesis. Using purified labeled chorion mRNA, they isolated and characterized 16 clones which contained chorion genes. These labeled clones were used for in situ hybridization to map the chorion genes in polytene chromosomes. These researchers are now trying to understand the molecular basis of known chorion mutants with some success. They determined that the ocellolus mutant, characterized by sterile females, altered chorion morphology and a distinct head shape has fewer C36 and C38 chorion proteins, whose genes map to a single site on the X chromosome. A closer examination of the Oc gene in polytene chromosomes revealed that it was neither a point mutation nor a deletion but rather a tiny inversion including the genes for C36 and C38. It is not yet clear how this inversion resulted in the underproduction of these two proteins, but this promises to be an illuminating example of the molecular and morphological results of a chromosome rearrangement.

#### DNA Sequence Organization in Drosophila Heterochromatin

Doug Brutlag of Stanford University presented his group's recent research on the DNA sequence organization of the tandem repeat regions in D. m. heterochromatin. They discovered that although the major species of heterochromatin were 95% homogeneous according to renaturation studies, when the DNA was sequenced more than one repeating unit was encountered per species of heterochromatin. Clones of heterochromatic DNA were isolated and sequences revealed that variation in satellite DNA species was not due to a little variation at many sites but rather to a lot of variation at a few sites. Insertions and deletions were found in the repeating units of a satellite DNA species using restriction endonucleases, although hybridization between species with and without a deletion persists. Repeating sequences homologous except for a 100 base pair region are assumed to have had a common ancestor and diverged after the insertion or deletion occurred and then the region was amplified. In sequencing heterochromatic DNA, Brutlag and his group found regions of long and regular inverted repeats similar to others known to bind regulatory proteins. They searched for a protein which would bind specifically to a plasmid DNA containing such a region. They isolated and purified a protein with a high affinity for these inverted repeats and isolated and sequenced its binding site. Currently these researchers are attempting to discover whether this protein is usually associated with DNA, how it binds to DNA, and what occurs when it binds to longer strands of DNA.

#### ERRATA

Volume 54: July 1979

In the Computerized Stock List 2 by Lindsley and Zimm (DIS 54:1-266), a text-processing error and two small heading errors should be corrected.

1. In the INTRODUCTION (page i, last indented paragraph), change the text "B. Reference numbers (at right) signifying the numbers of the stocks on the preceding list that carry the particular chromosome" to "Reference numbers (at right) for use in cross-indexing to the left-hand numbers of the chromosome list".
2. In the right heading of each page, change "DIS 55" to "DIS 54".
3. On the cover, change "SIMM" to "ZIMM".

Woodruff, R. C. Report from the Mid-America Drosophila Stock Center (Bowling Green State University, Bowling Green, Ohio).

I. Acquisition of Stocks: The following D. melanogaster stocks have been added to the Mid-America Drosophila Stock Center.

A. Electrophoretic enzyme markers or null enzyme markers:

- |  |  |
|--|--|
| 1 Acph-1n1/ TM3, Sb Ser Acph-1B                      | 21 Got-27/ SM1, Got-24   |
| 2 Acph-1n2/ TM3, Sb Ser Acph-1B                      | 22 Idh <sup>2</sup>  |
| 3 v;bw;Acph-1n4                                      | 23 6-Pgd <sup>8</sup>  |
| 4 v;bw;Acph-1n5                                      | 24 hk Ddcn <sup>7</sup> pr/ Cy0  |
| 5 e Acph-1n7/ TM3, Sb Ser Acph-1B                    | 25 SM1/+;TM6,Ubx Pgm <sup>4</sup> /Pgm <sup>1</sup>  |
| 6 Acph-1n8   | 26 SM1/+;TM6,Ubx Pgm <sup>4</sup> /Pgm <sup>2</sup>  |
| 7 v f B;bw;Acph-1n8                                  | 27 SM1, Got-24 $\alpha$ -Gpdh <sup>4</sup> cMdh <sup>4</sup> Adh <sup>4</sup> Dip-A <sup>4</sup>                     |
| 8 Acph-1n10/ TM3, Sb Ser Acph-1B                     | Hex-C <sup>4</sup> $\alpha$ -Amyl / Got-26 $\alpha$ -Gpdh <sup>2</sup> cMdh <sup>6</sup>                             |
| 9 v;bw;Acph-1n11                                     | Adh <sup>2</sup> Dip-A <sup>2</sup> Hex-C <sup>6</sup> $\alpha$ -Amyl <sup>2,3</sup> (Stock Cy/IIa)                  |
| 10 Acph-1n12/ TM3, Sb Ser Acph-1B                    | 28 SM1, Pgm <sup>4</sup> Pgi <sup>4</sup> / Pgm <sup>2</sup> Pgi <sup>2</sup> ; TM6, Ubx Men <sup>4</sup>            |
| 11 Acph-1n13   | Xdh(ry) <sup>4</sup> Aldox <sup>4</sup> mMd <sup>4</sup> / Men <sup>6</sup> Xdh(ry) <sup>2</sup>                     |
| 12 Acph-1n14/ TM3, Sb Ser Acph-1B                    | Aldox <sup>8</sup> mMd <sup>6</sup> (Stock CY/IIb;Ubx/IIIb)  |
| 13 ru h th st cu sr e Pr Acph-1B/ TM3, Sb            | 29 TM3, Sb Ser Idh <sup>4</sup> To <sup>4</sup> Pgm <sup>4</sup> Ald <sup>4</sup> / Idh <sup>6</sup> To <sup>2</sup> |
| Ser Acph-1B  | Pgm <sup>6</sup> Ald <sup>2</sup> (Stock Sb Ser/IIIc)  |
| 14 Df(2L)64j L <sup>2</sup> / Cy0, Adh <sup>nB</sup> | 30 TM6, Ubx Est-6 <sup>6</sup> Est-C <sup>4</sup> Odh <sup>4</sup> Acph-1 <sup>4</sup> Tpi <sup>4</sup> /            |
| 15 Adh <sup>n1</sup>                                 | Est-6 <sup>4</sup> Est-C <sup>6</sup> Odh <sup>2</sup> Acph-1 <sup>2</sup> Tpi <sup>6</sup> Lap-D <sup>4</sup>       |
| 16 b pu <sup>76e</sup> Adh <sup>n4</sup> / Cy0       | (Stock Ubx/IIIa)   |
| 17 Adh <sup>n5</sup> pr                              | 31 Pgd <sup>4</sup> Fum <sup>6</sup> Hex-A <sup>2</sup> (=Hex-A,B) Gpt <sup>2</sup> Zw <sup>4</sup>                  |
| 18 Amy <sup>4,6</sup>                                | (=G6pd) (X tester)   |
| 19 Dip-A <sup>6</sup> / SM1, Dip-A <sup>4</sup>      | 32 FM7, Pgd <sup>6</sup> Fum <sup>4</sup> Hex-A <sup>4</sup> Zw <sup>6</sup>   |
| 20 Est-C <sup>8</sup> / TM6, Ubx                     |  |

B. Mutants and rearrangements in the 37 to 38 region of 2L (Wright et al. 1976, Genetics 84: 267-285 and 287-310):

- |                           |                            |
|---------------------------|----------------------------|
| 33 1(2)E1 rdo hk pr/Cy0   | 60 1(2)E104 rdo hk pr/Cy0  |
| 34 1(2)E2 rdo hk pr/Cy0   | 61 1(2)E105 rdo hk pr/Cy0  |
| 35 1(2)E3 rdo hk pr/Cy0   | 62 1(2)E106 rdo hk pr/Cy0  |
| 36 1(2)E8 rdo hk pr/Cy0   | 63 1(2)E129 rdo hk pr/Cy0  |
| 37 1(2)E11 rdo hk pr/Cy0  | 64 1(2)E131 rdo hk pr/Cy0  |
| 38 1(2)E13 rdo hk pr/Cy0  | 65 1(2)E134 rdo hk pr/Cy0  |
| 39 1(2)E19 rdo hk pr/Cy0  | 66 1(2)E138 rdo hk pr/Cy0  |
| 40 1(2)E20 rdo hk pr/Cy0  | 67 1(2)E142 rdo hk pr/Cy0  |
| 41 1(2)E27 rdo hk pr/Cy0  | 68 1(2)E145 rdo hk pr/Cy0  |
| 42 1(2)E29 rdo hk pr/Cy0  | 69 1(2)E146 rdo hk pr/Cy0  |
| 43 1(2)E34M rdo hk pr/Cy0 | 70 1(2)E151 rdo hk pr/Cy0  |
| 44 1(2)E35 rdo hk pr/Cy0  | 71 1(2)224 pr/Cy0          |
| 45 1(2)E41 rdo hk pr/Cy0  | 72 1(2)261 pr/Cy0          |
| 46 1(2)E42 rdo hk pr/Cy0  | 73 1(2)285 pr/Cy0          |
| 47 1(2)E43 rdo hk pr/Cy0  | 74 1(2)286 pr/Cy0          |
| 48 1(2)E47 rdo hk pr/Cy0  | 75 Df(2L)TE48-2 M/Cy0      |
| 49 1(2)E50 rdo hk pr/Cy0  | 76 Df(2L)SDRev 37/Cy0      |
| 50 1(2)E51 rdo hk pr/Cy0  | 77 Df(2L)A20 cn bw/Cy0     |
| 51 1(2)E53 rdo hk pr/Cy0  | 78 w/+;Df(2L)TE48-1/Cy0    |
| 52 1(2)E56 rdo hk pr/Cy0  | 79 Df(2L)A14 cn bw/Cy0     |
| 53 1(2)E60 rdo hk pr/Cy0  | 80 Df(2L)A16 cn bw/Cy0     |
| 54 1(2)E62 rdo hk pr/Cy0  | 81 Df(2L)119 cn bw/Cy0     |
| 55 1(2)E65 rdo hk pr/Cy0  | 82 Df(2L)137 M cn bw/Cy0   |
| 56 1(2)E68M rdo hk pr/Cy0 | 83 Df(2L)50 cn/Cy0         |
| 57 1(2)E70 rdo hk pr/Cy0  | 84 Df(2L)E71 rdo hk pr/Cy0 |
| 58 1(2)E102 rdo hk pr/Cy0 | 85 Df(2L)3, 1(2)74i/Cy0    |
| 59 1(2)E103 rdo hk pr/Cy0 | 86 Df(2L)158 cn bw/Cy0     |

87 Df(2L)130 cn bw/CyO  
 88 Df(2L)E55 rdo hk pr/CyO  
 89 Df(2L)2 Tft 1(2)74i/CyO  
 90 Df(2L)9 Tft cn/CyO  
 91 Df(2L)12 Tft 1(2)74i/CyO  
 92 Df(2L)84 Tft 1(2)74i/CyO

93 Df(2L)150 cn bw/CyO  
 94 Df(2L)65 Tft 1(2)74i/CyO  
 95 Df(2L)1 Tft 1(2)74i/CyO  
 96 Df(2L)161 M cn bw/CyO  
 97 1(2)amdH45 cn bw/SM5

## C. Others:

98 Df(2L)Gdh-A/CyO  
 99 Df(2L)Gdh-A dp L/SM1  
 100 Df(2L)J69L H56R, y<sup>+</sup>/CyO  
 101 Df(2L)J-der<sup>27</sup>/SM1  
 102 Df(3R)cu<sup>40</sup>, Sb/TM6  
 103 Df(3R)dsx<sup>+</sup>R5, bx sr es/TM3, Sb Ser  
 104 Df(3R)kar<sup>31</sup>/MKRS  
 105 Df(3R)kar<sup>31</sup>/MRS  
 106 Df(3R)126c, kar<sup>2</sup>/MKRS  
 107 Df(3R)red<sup>31</sup>/MKRS  
 108 Df(3R)red<sup>P52</sup>/TM1  
 109 Df(3R)red<sup>P93</sup>, 1(3)tr Sb/In(3L)P +  
 (3R)P18, Me Ubx e<sup>4</sup>  
 110 Df(3R)ry<sup>36</sup>/MKRS  
 111 Df(3R)ry<sup>74</sup>/MKRS  
 112 Df(3R)ry<sup>75</sup>/MKRS  
 113 Df(3R)ry<sup>81</sup>/MKRS  
 114 Df(3R)ry<sup>614</sup>/MKRS  
 115 Df(3R)ry<sup>619</sup>/MKRS  
 116 Df(2L)MdhA/T(1;2)Bld & T(1;2)OR64/T(1;2)  
 Bld  
 117 Df(3L)thss<sup>102</sup>/TM3, Sb Ser  
 118 Df(3R)P14/T(2;3)apXa  
 119 Df(3R)Ubx<sup>109</sup>/Dp(3;3)P5, Sb  
 120 Df(3L)vin<sup>6</sup>/TM3  
 121 Df(1)C246/FM6  
 122 Dp(3;Y)P5  
 123 Dp(3;1)P68, y/FM6/Dp(3;Y)P92;Hu SbSpi/pP  
 124 T(2;3)P10/Dp(3;2)P10;Sb Ubx  
 125 T(1;3)P115, e<sup>11</sup>/TM1  
 126 Tp(3)sbd<sup>104</sup>/In(3R)C, e  
 127 Tp(3)P20, Ubx<sup>P20</sup>/TM1  
 128 Tp(3)bxd<sup>100</sup>/TM1  
 129 Tp(3)S462/T92;3)apXa  
 130 Cma/TM1  
 131 cui  
 132 ry<sup>8</sup>  
 133 ry<sup>26</sup>  
 134 ry<sup>41</sup>  
 135 y<sup>2</sup> su(wa) wa yL.YS/O/C(1)M4, y  
 136 ChV red sbd/Ubx<sup>130</sup>  
 137 cn en L<sup>rm</sup> nwB Bc Pu2 PinB/SM5  
 138 C(1)M4, y<sup>2</sup>/O/FM7Y, w m<sup>2</sup>

139 In(1)pdf, wa  
 140 PinYt/In(2R)C72  
 141 Sp B1 L<sup>rm</sup> Bc Pu<sup>2</sup>/CyO  
 142 C-scan = sc f;al b sp;ve st ca  
 143 C(1)TM, X YS.YL X, y<sup>31d</sup> Tw/X.Y, In En  
 B f y.y<sup>+</sup>  
 144 In(2LR)EN/Cy  
 145 C(2)EN1, +  
 146 C(2)EN1, cn bw  
 147 C(2)EN1, bw sp  
 148 C(2)EN1, sp  
 149 B<sup>S</sup>Y y<sup>+</sup>/+;C(2)EN1, +  
 150 R(1;Y)EN, y/M-5;Dp(1;3)scJ4/+  
 151 w (sn?) 157.3/w (sn?) r (Bx)  
 152 R(1;Y)EN, y Tw/C(1)RM, yv bb/0  
 153 R(1;Y)EN, y/C(1)RM, y v bb/0  
 154 Oce  
 155 v/rs<sup>2</sup>  
 156 FM6;Sp bw<sup>D</sup>/SM5;TM3, Ser/Sb  
 157 C(1)DX, y f/1(1)mysts<sup>2</sup> su(f)1ts67g  
 158 car su(f)1ts67g (=car 1(1)su(f)ts67g)  
 159 fs su(f)1ts67g (=fs 1(1)su(f)ts67g)  
 160 fu su(f)1ts67g/FM4 (=fu 1(1)su(f)ts67g/  
 FM4)  
 161 1(1)mys/FM4(1ts)  
 162 b Tft/CyO, 1(2)100DTS  
 163 b Tft/CyO, 1(2)513DTS  
 164 FM7, 1(1)TW-9/oc ptg<sup>3</sup> 1(1)TW-1cs;CyO/  
 1(2)91DTS  
 165 FM7, 1(1)TW-24/oc ptg<sup>3</sup> 1(1)TW-1cs; CyO/  
 1(2)91DTS  
 166 FM7, 1(1)TW-9/sc ec cv ct<sup>6</sup> v g<sup>2</sup> f  
 167 FM7, 1(1)TW-24/sc ec cv ct<sup>6</sup> v g<sup>2</sup> f  
 168 DTS-2, Sb/TM3, Ser  
 169 1(2)91DTS/CyO  
 170 In(2LR)bw<sup>V1</sup>, bw<sup>V1</sup> ds<sup>33k</sup> 1(2)18DTS/CyO  
 171 T(Y;2)CyO, 1(2)513DTS  
 172 C(1)M4, y<sup>2</sup>;CyO Roi/Sp B1 L<sup>rm</sup> bw<sup>D</sup>  
 173 shifts1  
 174 y<sup>2</sup> cv v shifts<sup>2</sup>/FMA3, y<sup>2</sup>;pP/+  
 175 para<sup>ts</sup>/C(1)DX, y f  
 176 Sh<sup>3</sup>

The following species of the melanogaster subgroup have been added to the Mid-America Drosophila Stock Center:

177 D. erecta (154.1)  
 178 D. mauritiana  
 179 D. simulans (C135.20)

180 D. teissieri (128.2)  
 181 D. yakuba



II. Stocks to be Discarded: To reduce the number of redundant cultures the Mid-America Drosophila Stock Center, the following stocks will be discarded six months after publication of this issue of DIS.

Wild Stocks

a4 + Canton-S, A (iso, 1952)  
a5 + Canton-S, B (iso, 1970)  
a12 + Lausanne-S (iso, 1970)  
a14 + Oregon-R (iso, 1962)  
a15 + Oregon-R (iso, 1970)

Chromosome 1

b29 Co & y w f:=  
b31 Co rst<sup>2</sup> & y w bb.=  
b88 f BB/InAM  
b94 f fu/C1B  
b109 fan<sup>o</sup>sp1  
b111 Fl oc ptg v/y C1B v  
b135 gt wa (Oregon-R)  
b144 In49 ptg oc ct<sup>ns</sup>/y C1B v  
b146 In49 ptg oc ct<sup>ns</sup>/y C1B  
b186 nd rb  
b213 rst<sup>2</sup>/F<sup>1</sup>M1, y3ld sc<sup>8</sup> wa lzs B  
b215 rst<sup>3</sup>, In & y f:=  
b218 rst<sup>3+2</sup>, v f & y f:=;bw  
b219 rst<sup>3+1</sup>, v  
b220 rst<sup>3+88</sup>, v f & y f:=  
b221 rst<sup>3+125</sup>, v f & y f:=;bw  
b268 spx car  
b304 v<sup>+</sup>(rev.v) BM2  
b305 v<sup>+</sup>(rev.v) BM2+(rev.B,Rein) fB15(mosaic)  
b337 we<sup>m</sup>  
b371 y ac sc pn B & y f:=  
b399 y cv  
b431 y pn  
b451 y v & y f:=  
b469 y w sn<sup>3</sup> f36a & y f:=  
b474 y w -258-42/y Hw In49 m2 g4  
b499 y<sup>2</sup>su-wa walz<sup>1</sup>suw-f.y<sup>+</sup> y f:=  
b511 y<sup>2</sup> v  
b534 y3d & y f:=

Chromosome 2

g36 ap4/dptx1 Cy, Ins0 pr cn2  
g60 b pr  
g89 bw<sup>-5</sup>/Cy cn2 L4 sp2  
g114 cn (iso, 1962)  
g266 ed dp cl  
g272 ed dp<sup>ols</sup> cl/Cy B1 L  
g346 ltd bw  
g354 M(2)B/SM5, a12 Cy 1tv sp2  
g370 M(2)S10/Cy cn2 L4 sp2  
g393 net dp  
g436 rdo (iso, 1963)  
g481 shv ho  
g521 vg-C/SM5, a12 Cy 1tv sp2

Chromosome 3

h17 ca bv  
h30 cu kar<sup>2</sup>  
h57 e tx  
h73 h ri  
h74 h ri ca (iso, 1953)  
h75 h ri e<sup>s</sup> (iso, 1957)  
h94 kar<sup>2</sup> ca  
h118 ma ry/TM1, Me ri sbd<sup>1</sup>  
h144 red (Malpighans) e  
h147 ri e  
h148 ri pP  
h158 ru h e<sup>s</sup>  
h181 se h  
h192 sf<sup>2</sup> cd  
h196 sr gl  
h201 ss ca (iso, 1953)  
h202 ss ca (iso, 1953)  
h203 ss e tx  
h204 ss tx  
h213 st ry  
h214 st ry<sup>2</sup>  
h243 ve bv (iso, 1957)  
h244 ve ca (iso, 1953)  
h245 ve ca (iso, 1961)  
h252 ve st (iso, 1964)

Multiple Chromosomes

j6 car;ltd  
j7 cm;cl  
j18 ras;ltd  
j20 rb;ltd  
j32 v;bw<sup>D</sup>  
j46 y f:=;al b cn sp  
j49 y f:=dp  
j54 y f:=; net bw sp  
j55 y f:=; pr  
j58 y f:=; vg-B/SM5, a12Cy 1tv sp2  
j67 y w f;dp  
j69 y w In49 f;dpv2  
j70 y w m f;dp  
j71 y w f & fy:=dp  
j72 y w sn<sup>3</sup>;dp  
j73 y w sn<sup>3</sup> B;dp  
j74 y w sn<sup>3</sup> f36a;dp  
j82 In49 ptg oc ct<sup>ns</sup> & y f:=;ri pP  
j110 w;red  
j117 y;red  
j119 y f:=;ell  
j122 y f:=;su-ve ru ve bv (h? th?)  
j124 y w;red (Malpighians)  
j125 y w;red e  
j136 y;spa<sup>pol</sup>  
j137 y f:=;spa  
j160 b;ell

Multiple Chromosomes cont.

j161 b;pP  
 j169 bw;st (iso, 1970)  
 j174 c;e  
 j175 cn;ca  
 j177 cn;ca<sup>2</sup>  
 j178 cn;se<sup>51j</sup>  
 j180 cn;st (iso, 1968)  
 j185 cn;ve (iso, 1960)  
 j191 dp;cu  
 j192 dp;ell  
 j193 dp;ell (iso, 1970)  
 j194 dp;es  
 j195 dp;red  
 j196 dp;sbd<sup>2</sup> bx<sup>3</sup>  
 j197 dp;tx  
 j198 dp;tx (iso, 1969)  
 j199 dp;ve  
 j230 ed dp cl;tx

j234 ho dp;ri pp  
 j241 ltd;ca  
 j242 ltd;ma  
 j243 ltd;ry  
 j244 ltd;se  
 j254 pr;cd  
 j298 vg;bv  
 j399 sc<sup>8</sup>.Y/y scS1 In49 sc8 & y f:=;dp bw;st pp

Attached (Compound) Chromosomes (6-26)

m26 w;C(4)RM:ci ey<sup>R</sup>

Non-autonomous Sex-linked Lethals  
(E. Novitski) (m27-43)

m35 (glufultyrless-3) y w spl sn bb/scS1 bb<sup>+</sup>  
 B In49 oc ptg sc8

## ANNOUNCEMENTS

Department of Genetics, University of Cambridge, England: The Library has acquired E. B. Basden's collection of reprints of *Drosophila* papers. Together with those reprints already in the Library about 90% of the *Drosophila* literature to 1975 is available in the Department. Can we ask all *Drosophila* workers to send us reprints of their papers since 1975 and to put the Library's name on mailing lists. The collection is available for use by anyone. The reprints cover all aspects of the biology of *Drosophilidae*, not just genetics. Eventually we would hope to produce a computerized index to the collection. Reprints or inquiries, please, to The Librarian, Department of Genetics, Downing Street, Cambridge CB2 3EH.

Dr. Lee Ehrman and Professor Peter A. Parsons are now initiating the writing of the second edition of their 1976 *Genetics of Behavior* (Sinauer Associates, Sunderland, Massachusetts), and would appreciate reprints, preprints, etc., of appropriate materials for possible inclusion. Please send these materials to Dr. Lee Ehrman, Division of Natural Sciences, State University of New York, Purchase, New York 10577, or to Professor Peter A. Parsons, Genetics and Human Variation, LaTrobe University, Bundoora, Victoria 3083, Australia.

A new laboratory for research in *Drosophila* has been organized in the Institute of Ecology and Evolution, Austral University of Chile, Casilla 567, Valdivia. Eduardo del Solar O., Director, reports that this new laboratory will be devoted to research on ecological genetics and behavior.

The Genetics Society of Korea was founded in June, 1978 to provide a forum for scientific discussions and exchanges of ideas between Korean and foreign geneticists. GSK plans to hold periodic regional and national meetings that will provide opportunities for formal presentations and informal discussions. Your attendance at such meetings would be most welcome and perhaps cooperative research programs will result from such interactions. GSK also plans to publish a new Journal of Genetics entitled The Korean Journal of Genetics once a year. Dr. Yong K. Paik, Dept. of Genetics, School of Medicine, Hanyang University, Seoul 133, was elected first president of the society. Anyone wishing to join this fledgling society or desiring further information please contact Dr. Eun Ho Park, Secretary, Hanyang University, College of Liberal Arts and Sciences, Dept. of Biology, Seoul 133, Korea.

F. A. Lints informs us that he has some reprints of the late Dr. M. J. Hollingsworth's publications. Those publications mainly concern longevity studies in *Drosophila* and are available upon request. His address is: Laboratoire de Génétique, Université de Louvain, Place Croix du Sud 2, B-1348 Louvain-la-Neuve, Belgium.

John A. Thomson, School of Biological Sciences, University of Sydney, N.S.W., 2006, Australia, requests mutants of *D. immigrans*, or information on any being maintained in stock collections.

Y. K. Paik, Dept. of Genetics, School of Medicine, Hanyang University, Sungdong-ku, Seoul 133, Korea, would highly appreciate receiving reprints (old or new) on the genetics of *Drosophila* and humans to establish a library in the Department.

The new Department of Genetics, University of Göteborg, Stigbergsliden 14, S-414 63 Göteborg, Sweden, would be grateful for any reprints, particularly on *Drosophila* genetics.

Developmental mutants which affect the eye are needed for allele studies. If you have any mutants that affect the structure of the eye selectively, please write Ilse Schwinck, University of Connecticut, Box U-42, Storrs, Connecticut 06268, USA. Also, any other homoeotic mutants would be of interest.

John A. Thomson, formerly at the Division of Plant Industry, CSIRO, Canberra, has accepted appointment as Professor of Biology (Genetics) in the School of Biological Sciences, University of Sydney. He will be continuing work on the genetics and biochemistry of larval storage proteins in Diptera, including *Drosophila*.

Y. K. Paik has moved to the Department of Genetics, School of Medicine, Hanyang University, Seoul 133, Korea, as a professor and Chairman of Genetics (from the Department of Genetics, School of Medicine, University of Hawaii, USA).

Charles Detwiler, formerly a graduate student at Cornell University, will be a Postdoctoral Fellow in Michael Ashburner's laboratory, Cambridge, England, commencing April 1, 1979.

As of August, 1979, the laboratory of J. S. F. Barker will be moved to the Department of Animal Science, University of New England, Armidale, N.S.W. 2351, where all current research will be continued.

The *D. pseudoobscura* mutant and inversion strains which Wyatt Anderson maintained in the past have been added to the National *Drosophila* Species Resource Center. They may be requested (by Wyatt's stock numbers as given in DIS 52:8) from the National *Drosophila* Species Resource Center, Department of Zoology, University of Texas, Austin, Texas 78712.

Abedin, K., S. McNamara, D. Osterbur and W. W. M. Steiner. University of Illinois, Urbana. Studies on the effect of temperature on fitness and fecundity at the Esterase-6 locus in *D. melanogaster*.

Wright and MacIntyre (1965) found the presence of two alleles in *D. melanogaster* producing variants of esterase-6 (est-6) enzyme which had similar electrophoretic mobility. Allele est-6<sup>F</sup><sub>1</sub> was found to control production of a heat labile form in which no activity of the enzyme remained after 5 minutes at 60°C while allele

est-6<sup>F</sup><sub>2</sub> was heat stable. An electrophoretically slow migrating third allele, est-6<sup>S</sup>, was found to be heat stable also. Long (1970) has determined that temperature fluctuations may effect genetic responses in *D. melanogaster* populations but the specific response of the different est-6 alleles have remained uninvestigated in this regard. The in-vitro effects described by Wright and MacIntyre imply that under high temperature stress flies carrying the est-6 heat resistant allele may be at an advantage. In addition, the fecundity of each allelic type has remained uninvestigated under different temperature regimes, a point which is of interest with respect to the selective maintenance of the different alleles.

We have investigated these points using strains 3008 (homozygous for an est-6<sup>F</sup> allele) and 3009 (homozygous for est-6<sup>S</sup>) obtained from the University of Umea, Sweden. Heat denaturation tests were first conducted to determine if the allelic types differed in heat sensitivity. Homogenates of 10 males of each genotype were placed in a glass tube (Kimax, 6x50 mm) and subjected to incubation temperatures of 30°C or 60°C for 10 minutes, on a temp-bloc (Scientific Products). The test was repeated for flies raised at 18°C, 25°C, and 30°C and for a control raised at a varying room temperature of 20-23°C in order to determine if variation in developmental temperature might play a role in heat sensitivity. A second control raised at each of the above temperatures but not heat treated was also used. After the temperature exposure, the supernatants from each homogenized set of flies was electrophoresed on a tris-citrate gel (pH 8.45 containing 10% of electrode buffer) bridged across a set of electrode trays containing lithium hydroxide-boric acid (pH 8.2) solution. Gels were run for 8 hours at 200 volts. Allozyme bands were visualized using a standard esterase histochemical stain, and the presence or absence of bands was noted. The results shown in Table 1 clearly indicate that line 3008 carries a heat sensitive form of the est-6<sup>F</sup> allele but we are not sure it is the same as that found by Wright and MacIntyre (1965). Although we must conclude that there is no effect on the expression of sensitivity due to various developmental temperature regimes, it is interesting to note that the sensitivity of est-6<sup>F</sup> at 30°C in flies raised at a fluctuating (room) temperature and at a high (30°C) temperature is reduced. The est-6<sup>S</sup> allozyme retains activity regardless of development temperature background or heat treatment.

Table 1. The results of heat-inactivation studies on est-6<sup>F</sup> and est-6<sup>S</sup> carrying flies raised at different temperatures. The presence of enzyme activity after a treatment is indicated by a + while a - indicates no activity.

Temperature raised at	Treatment	3008 est-6 <sup>F</sup>	3009 est-6 <sup>S</sup>
room	no denaturation	+	+
room	30°C	+	+
room	60°C	-	+
18°C	no denaturation	+	+
18°C	30°C	-	+
18°C	60°C	-	+
25°C	no denaturation	+	+
25°C	30°C	-	+
25°C	60°C	-	+
30°C	no denaturation	+	+
30°C	30°C	+	+
30°C	60°C	-	+

To investigate fecundity at different temperatures for the two allelic types, flies 24 hours of age were chosen for preconditioning on sugar media for two days at room temperature. These were then split into three groups for each line and were kept for two more days at 18°C, 25°C, and 30°C, respectively. They were then transferred onto a standard yeast and cornmeal media containing red food coloring to facilitate egg counting. These vials, which contained a moist folded Kimwipe to maintain humidity in each vial, were then kept at their respective temperatures for 48 hours longer. At the end of this period, the flies were removed and the eggs in each vial were counted under a dissecting microscope. Eggs were maintained at each temperature until adults emerged. These were counted daily over an 8 day period when it was ascertained that hatching was completed.

The results of this study are shown in Table 2 which also contains the fitness coefficients for egg to adult survival. For both lines, fecundity and egg to adult survival are maximized at what should be the near-optimal temperature of 25°C. Examining the data reveals that line 3008 has a higher egg to

adult survival rate (85.8% compared to 70.5%) at 25°C, and has a slightly higher survival at 18°C. At 30°C, however, the situation is reversed and not only are over twice as many flies surviving to the adult stage in line 3009, but line 3008 has a relative fecundity almost twice that of 3009 (the difference between strains for fecundity at 25°C and 30°C is significantly different,  $t = 3.59$  and  $3.03$ ,  $p = 0.005$ ). The fitness coefficients reflect the higher survival rates in line 3009 as well.

Table 2. Relative fecundity, survival and fitness within est-6 strains in flies kept at three different temperatures.

Line	Temperature °C	No. of eggs	No. of adults	Percent Relative Survival	Relative Fecundity	Fitness
3008 (est-6 <sup>F</sup> )	18	29	8	27.6	15.3	.32
	25	190	163	85.8	100.0	1.00
	30	155	42	27.1	81.6	.29
3009 (est-6 <sup>S</sup> )	18	40	10	25.0	14.5	.35
	25	275	194	70.5	100.0	1.00
	30	116	75	64.7	42.2	.96

Thus, it appears that the heat sensitive est-6<sup>F</sup> allele in this study may be at an advantage with respect to individual survival and fecundity at temperatures around 25°C and lower but at a disadvantage in vivo at temperatures approaching 30°C. That this is an

effect associated with the est-6<sup>F</sup> heat sensitive allele is not entirely clear even though the study was initiated to determine if the ad hoc prediction would be met. The survey of additional lines fixed for est-6<sup>S</sup> and the heat sensitive est-6<sup>F</sup> alleles, which should differ in genetic background except at this locus, would aid in clarifying the relationship observed in this study. Examination of the relative allozyme activities in flies raised at different temperatures and delineation of the physiological role of esterase-6 would also help. Studies on certain aspects of these problems are continuing.

References: Long, T. 1970, Genetics 66:401; Wright, T.R.F. and R.J. MacIntyre 1965, Elisha J. Mitchell Soc. -1:17.

Albornoz, J. and J. Rubio. University of Oviedo, Oviedo, Spain. Location of a region controlling the suppression of normal bristles in *Drosophila melanogaster*.

Repeated attempts to increase the number of missing dorsocentral and scutellar bristles (dc and sc) by selective crossing of the few deviant flies found in wild populations have failed. However, we found quite a number of missing dorsocentral and scutellar bristles in a population

that had been intensively selected for increased number of bristles (dc and sc) and then let go without selection, where more than 95% of the flies had extra dc and sc bristles. The quick response to selection for increased number of missing bristles (line S) is shown in Figure 1 up to generation 11, when a plateau was reached; the 6 latest counts are included.

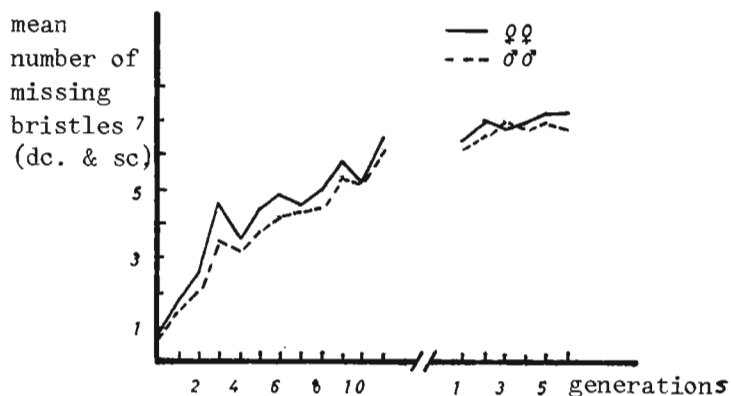


Fig. 1.

Presently all the flies in the population lack some normal bristles in the dorsocentral and scutellar areas; other bristle systems, and occasionally some microchaetae, are also affected. The proportion of flies having extra bristles was drastically reduced from 95% to 6-8% at generation 11, but even now 4-8% of the flies in every generation have extra dc and sc bristles.

Chromosome contribution analysis: Conventional crossing of line S to balanced strain J-407 carrying dominant markers in all three major chromosomes indicates that the presence of chromosome III in homozygous con-

dition is quite fundamental, although chromosomes I and II also play a relevant role. The analysis of variance is shown in Table 1, together with the mean factorial effect of each chromosome given in missing bristles number.

Table 1. Analysis of Variance

Factor	d.f.	M. S.	F	P	Mean Factorial Effects
chromosome I	1	74	81.22	<0.01	0.50
chromosome II	1	471.25	517.24	<0.01	1.25
chromosome III	1	9976.33	10949.81	<0.01	5.77
interaction I-II	1	3.83	4.23	<0.05	-0.11
interaction I-III	1	79.05	86.77	<0.01	0.51
interaction II-III	1	453.87	498.16	<0.01	1.23
interaction I-II-III	1	3.20	3.52	n. s.	-0.10
residual (error)	1192	0.91			

Table 2. Mean number of missing bristles in each chromosomal type (O = Oregon-R wild type chromosome; S = chromosome from the S line.

Chromosome II		Chromosome III		
		S / S	S / O	O / O
S / S	f.	5.16 ± 0.07	0.002 ± 0.002	0.01 ± 0.04
	m.	4.15 ± 0.08	0.01 ± 0.05	0.01 ± 0.05
S / O	f.	4.24 ± 0.08	—	—
	m.	3.22 ± 0.08	—	—
O / O	f.	1.58 ± 0.07	—	—
	m.	1.03 ± 0.05	—	—

Table 3.

Males recombinant class	Number of cultures	
	with missing bristles	without missing bristles
h st cu e <sup>S</sup> ca	1	14
++ + + +	12	0
h st cu e <sup>S</sup> +	13	0
++ + + ca	0	10
h st cu + +	11	0
++ + e <sup>S</sup> ca	3	8
h st + + +	5	0
++ cu e <sup>S</sup> ca	0	9
h + + + +	12	0
+ st cu e <sup>S</sup> ca	1	10

the reduced number of progenies observed and the short distance left between claret and the chromosome end, this location is only approximate. We are now looking for any other mutant marker located in this region of chromosome III.

References: Robertson, F.W. 1954, J. of Genetics 52(3):494-520; Thompson, J.N. Jr. and J.M. Thoday 1975, Genet. Res. 26:149-162.

In order to assess the specific effect of chromosomes II and III of line S, these chromosomes were substituted for the same chromosomes in inbred line Oregon-R, following the method used by Robertson (1954). Table 2 shows the mean number of missing bristles observed in each chromosomal type.

The phenotypic expression due to chromosome III (S) alone is low, but still this chromosome in homozygous condition causes by itself a quite distinct phenotype, so much so that the interaction with the chromosome II (S) is effective only when chromosome III (S) is homozygous.

Location of the relevant region on chromosome III from the S line: Following the method used by Thompson and Thoday (1975), females from the S line were mated to an inbred marker stock carrying hairy (26.5

cM), scarlet (44.0 cM), curled (50.0 cM), ebony-sooty (70.7 cM) and claret (100.7 cM). F<sub>1</sub> females were backcrossed to h/st/cu/e<sup>S</sup>/ca males, and the recombinants were picked up as males in the next generation. Twenty males of each recombinant class were mated individually to females from the S line. In their progeny recombinant chromosomes carrying factors contributing to bristle suppression could be identified by the occurrence of missing bristles. Table 3 gives, for each recombinant male class, the number of single-pair cultures showing missing bristles.

The S region can be located on the distal part of chromosome III, after claret. Among a total of 109 recombinant males cross-tested, only 5 are recombinant between claret and the S region; this places the S region at about 4.6 cM away from claret, or 105 cM on the map. Due to

Anderson, S., M. Santos\*, and J. McDonald.  
Iowa State University, Ames. Comparative  
study of the thermostability of crude and  
purified preparations of alcohol dehydro-  
genase (EC 1.1.1.1) from *D. melanogaster*.†

Thermostability studies are often employed as a means of uncovering enzyme variation, which may go undetected with routine electrophoretic techniques (e.g., Singh et al. 1975; Milkman 1976). In this note, we report a study to evaluate the comparability of the results of thermostability studies carried out on crude vs. purified prepa-

rations of *Drosophila* ADH. Enzymes from three electromorphically identical "fast" strains (F-1, F-2, F-3) and two electromorphically "slow" strains (S-1, S-2) were analyzed.

Crude extracts were prepared by homogenizing 30 mg of  $5 \pm 1$  day old adults in 1.0 ml of Tris-HCl Buffer, pH 8.6. After centrifugation for 20 min at 28,000Xg, a constant amount of supernatant (200  $\mu$ l) was placed into microtubes for use in the temperature stability studies. ADH was purified according to the techniques of McDonald et al. (1977) and prepared for thermostability studies as described.

Thermostability studies were carried out by placing the microtubes in a 45°C water bath. The temperature in the water bath was kept constant by means of a constant-temperature thermomix pump, model 1440. Preparations were exposed to this temperature for various periods (5-90 min) after which time they were immediately placed on ice until assayed for ADH activity (McDonald and Avise, 1976). The results are presented in Fig. 1. Crude extract ADH for all strains was extremely heat labile. No significant differences are detectable between the strains (Fig. 1a). In contrast, the thermostability of the purified preparations was generally increased. Significant variation both within and between the "fast" and "slow" forms of the enzyme are clearly evident (Fig. 1b).

Two conclusions can be drawn from this study. (1) Thermostability studies are most reliably carried out on pure enzyme. Spurious results may result from the analysis of crude preparations due to the presence and/or temperature activation of other factors affecting enzyme stability (e.g., proteases). (2) Degree of thermostability does not seem to be diagnostic of electrophoretic class; i.e., some slow strains are more heat stable than some fast strains and vice versa.

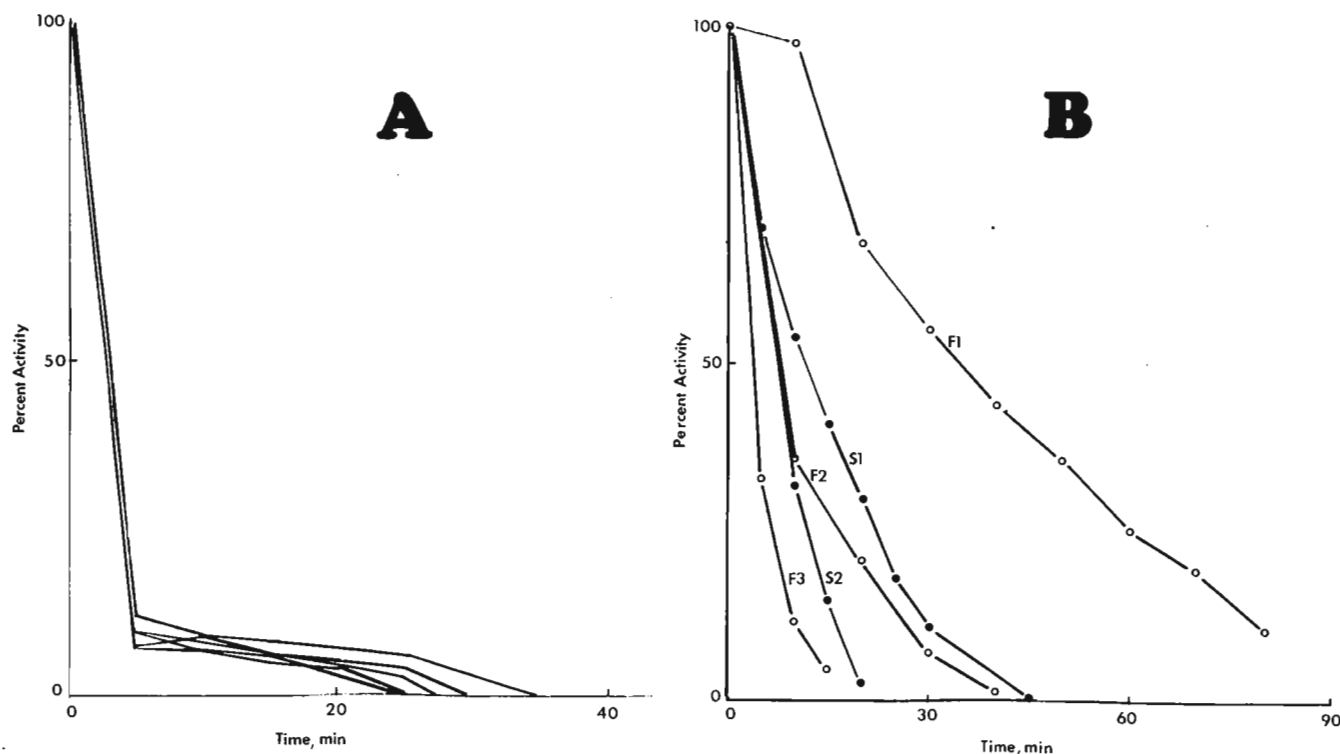


Fig. 1. Percent activity remaining after exposure of (a) crude and (b) purified ADH to 45°C for varying lengths of time in 3 Fast (○) and 2 Slow (●) strains of *Drosophila melanogaster*. See text for details.

References: McDonald, J.F. and J.C. Avise 1976, *Biochem. Genet.* 14:347-355; McDonald, J. F., G.K. Chambers, J. David and F.J. Ayala 1977, *PNAS* 74:4562-4566; Milkman, R. 1976, *Biochem. Genet.* 14:383-387; Singh, R.C., J.L. Hubby and L.H. Throckmorton 1975, *Genetics* 80:637-650.

\*Permanent address: University of Santiago, Spain.

†Journal paper No. J-9438 of the Iowa Agriculture and Home Economics Experiment Station, Ames, Iowa. Project No. 2272.

This work was supported in part by PHS/NIH Grant No. 5 505 RR07034 to John McDonald.

Bewley, G.C. and S. Lubinsky. North Carolina State University, Raleigh. Toxicity to the dietary administration of hydrogen peroxide in acatalasemic *Drosophila*.

We have recently demonstrated that the dietary administration of the catalase inhibitor 3-amino-1,2,4-triazole (AT) provides a very sensitive and simple technique for the destruction of existing catalase molecules in vivo (Lubinsky and Bewley, 1979). Adult flies are starved for 24 hours on agar, and then fed on a 5 mM AT-sucrose solution for two hours, which results in a complete destruction of catalase activity with no apparent effect on viability. This technique has provided a mechanism for examining the toxicity of the substrate  $H_2O_2$  in flies with normal catalase activity and flies made acatalasemic by the AT-method.

Adult flies with normal catalase levels appear to be relatively resistant to the dietary administration of the substrate  $H_2O_2$  (Fig. 1). However, these same concentrations of  $H_2O_2$  are extremely toxic to flies that have been made acatalasemic following the administration of 5 mM AT. In fact, as little as 0.05%  $H_2O_2$  in the diet results in 100% mortality within five days of exposure while 0.1% results in 100% mortality within three days of exposure. The threshold for  $H_2O_2$  tolerance in normal flies is apparently close to 1%  $H_2O_2$  since this concentration will eliminate a population with normal catalase levels within three days of exposure. These results indicate that  $H_2O_2$  can serve as a sensitive discriminator between CAT-positive and CAT-negative flies in a similar fashion that the substrate ethanol serves as a discriminator between ADH-negative and ADH-positive flies (Vigue and Sofer, 1976), and as such may prove useful as a positive selection agent in studies focusing on reversion, intracistronic recombination, conversion, and suppression at the Cat locus.

(Supported by PHS Grant GM-23617.)

References: Lubinsky, S. and G.C. Bewley 1979, *Genetics* 91:723-742; Vigue, C. and W. Sofer 1976, *Biochem. Genet.* 14:127-135.

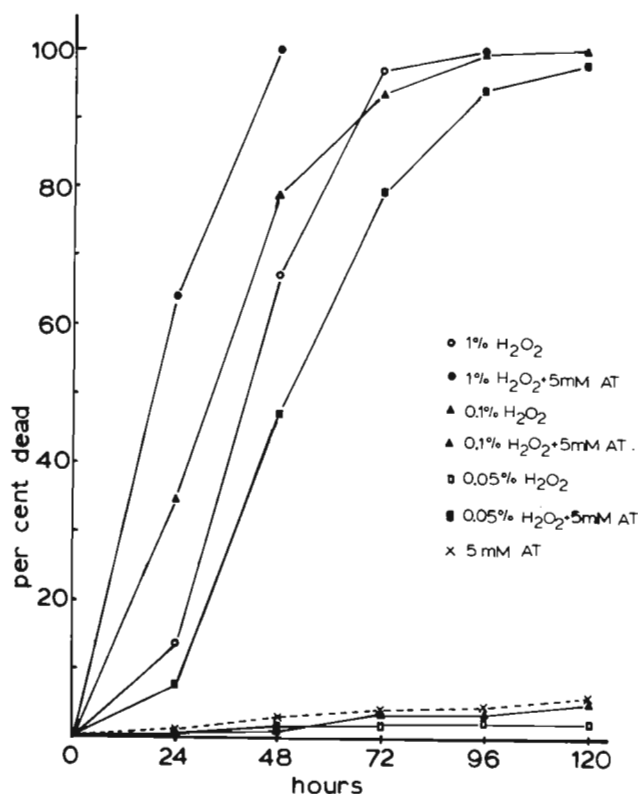


Fig. 1. The mortality rate of adult male *Drosophila* of an Oregon-R strain when fed on differing concentrations of  $H_2O_2$  with or without 5 mM AT.



Bos, M. and A.C. Boerema. University of Groningen, The Netherlands. Oviposition preferences in six *Drosophila melanogaster*-subgroup species.

Van Dijk and Van Delden (1979) calculated genetic distance and evolutionary relation by investigating 17 isozyme loci. Both papers report a phylogenetic "family tree". The six species fall into two groups: (1) *D. melanogaster*, *simulans* and *mauritiana*; (2) *D. yakuba* and *teissieri*; while *D. erecta* is rather separate from the other species.

Since Tsacas, Lachaise and David discovered some new species the *Drosophila melanogaster*-subgroup comprises at least six species.\*

Lemeunier and Ashburner (1976) studied the interrelations by analysis of the banding patterns of their polytene chromosomes. Eises,

We tested oviposition prefer-

ences of the six species on five different sites. A site consisted of two circular slices of medium. The strains we used were kindly provided by Dr. Van Delden and Dr. Tsacas and all originated from Africa: mel. collected in Tanzania, sim. in Uganda, maur. in Mauritius, yak. in Gabon, teis. in Kameroon, erecta in Ivory Coast. The oviposition sites we presented were complete (C), glucose sugar 5% (S), yeast medium 3% (Y), ethanol 1.5% (E) and pure agar 2% (A). All media were presented in 2% agar; the composition of complete medium is given in Bos and Scharloo (1974).

Fig. 1 presents the number of eggs produced by a species on a medium, given as the percentage of the total number of eggs laid on the five media together. In the choice situation the number of eggs laid on a medium is calculated from the number of eggs laid on that medium in the four possible choice situations.

From a two-factors (media and species) analysis of variance of the "no-choice" situation it is clear that there are highly significant differences between the number of eggs laid on different media, that the species laid the

Fig. 1. The percentages of eggs laid by six sibling species of the *D. melanogaster* subgroup on five different oviposition media in two situations. Upper part: no choice of media; lower part: choice between two media (see text).

same total number of eggs and that there was a small but significant species/media interaction. Looking to the data (upper part, Fig. 1), we notice that (1) all the species are laying very poorly on pure agar; (2) all species do have their optimal laying on complete media. There is especially good laying by *D. yak.*, *teis.* and *erecta*. (3) Two species are laying on ethanol agar (mel. and sim.); (4) *teis.* and *maur.* do lay very well on sugar agar.

In the "choice" situation the analysis of variance shows highly significant species, media and interaction effects. The media effect was largest. Again looking to the detailed data (lower part, Fig. 1), we notice (1) no production of eggs on agar; (2) all species have their highest production on complete medium; (3) all species do have a rather good production on sugar; (4) mel. and sim. lost their significant production on ethanol in the choice situation (in agreement with experiments of McKenzie and Parsons, 1972).

If we calculate niche breadth ( $B = 1/EP_{ij}^2$ , divided by the number of situations;  $P_{ij}$  = frequency of eggs laid by species *i* on medium *j*) on the basis of the egg productions on the different oviposition sites we get the following range:

no choice		choice	
sim.	0.82	mel.	0.63
mel.	0.68	maur.	0.55
maur.	0.65	erecta	0.52
teis.	0.50	sim.	0.48
erecta	0.39	yak.	0.44
yak.	0.34	teis.	0.43

In the "no-choice" of medium situation we find a range of the species which does agree with the position of the species in the phylogenetic trees found by Lemeunier and Ashburner (1976) and Eises et al. (1979). In the "choice" situation the range of the species is changed.

From our experiments it seems reasonable to conclude that the females from the sibling species use different components of available breeding

space and that the similarities between species sometimes do and sometimes do not fit with their genetical relationships.

References: Bos, M. and W. Scharloo 1974, Genetics 75:679-693; Eises, C., H. van Dijk and W. van Delden 1979, Evolution (in press); Lemeunier, F. and M. Ashburner 1976, Proc. R. Soc. Lond. B. 193:275-294; McKenzie, J.A. and P.A. Parsons 1972, Oecologia 10:373-388.

\*Since then *D. oreana* is added.

Boulétreau, M. and C. Biéumont. Université de Lyon I, Villeurbanne, France. Reproductive potential of a Hymenopteran parasite of *Drosophila melanogaster*.

Though natural *Drosophila* populations have been extensively studied, little attention has been paid to the possible ecological role of their hymenopterous parasites (see the partial list established by Basden, 1972). The present results show that one of these parasites (*Cothonaspis*

*boulardi*) has the fecundity, egg deposition rate and egg retention capacity capable of rendering it efficient in limiting population size of the host.

The parasite studied is a Cynipid belonging to the new species *Cothonaspis boulardi* described by Barbotin et al. (1979). Females lay their eggs in the 2nd instar larvae of *D. melanogaster* and the adult wasp emerges from the empty host pupa 18 days later at 25°C. Adult specimens were captured in southern France and the stock derived has been maintained in the laboratory by mass culture.

For determination of offspring production, couples of newly emerged imagoes were placed in 100 ml plastic cages, fed with honey and kept at 22°C under LD 12:12. Three experimental groups were tested for egg retention capacity: group (a) had hosts added from the first day on; group (b) from day 4; group (c) from day 10. Every second day each couple was provided with 0-48 hour old eggs and larvae of *D. melanogaster* in a sufficient amount to minimize superparasitization (deposition of more than one egg per host).

Fig. 1 summarizes offspring production by females of the three groups. Females provided with hosts immediately after emergence (Fig. 1a) parasitize *Drosophila* larvae right away. Afterwards their production decreases rapidly and falls to 0 on day 16 though lifespan is 21 days on the average and can reach 40 days. This early egg deposition suggested preimaginal egg formation and maturation which was confirmed by dissection.

When host availability is delayed for 4 days (Fig. 1b) or 10 days (Fig. 1c) the same phenomenon is observed: intense, brief offspring production follows introduction of the host. However, the total offspring production is lower than in the first case and varies significantly with the duration of host deprivation, as shown in Table 1.

In conclusion the main features of offspring production in this species are the following:

- preimaginal ovarian activity making adult females little dependent on food availability;
- fast deposition of the whole egg batch as soon as hosts are available;

- capacity for delaying egg laying: production is still 68% of normal after 4 days of host deprivation and 49% after 10 days.

These traits adapt the species to the exploitation of fluctuating host populations. Such parasites may be of importance in limiting seasonal demographic explosions. Moreover, the rather narrow specificity to *melanogaster* could favor other flies which compete for the same breeding sites, as suggested by Rouault (1979). This example shows that the parasitic complex associated to *Drosophila* populations should no longer be neglected in population dynamics studies.

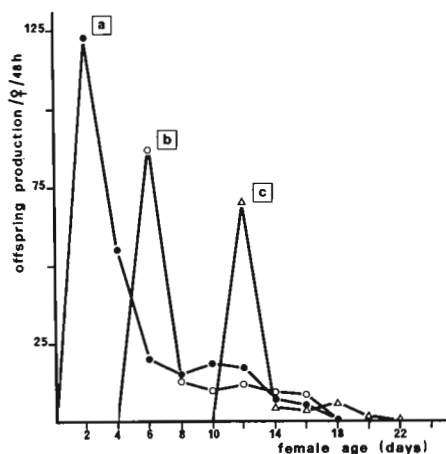


Fig. 1. Offspring production by females of *C. boulardi*. a: hosts present from the first day; b: from day 4; c: from day 10

Table 1. Total offspring production in groups a, b, c

group a:	198.3 ± 9.6 (n=32)
group b:	134.8 ± 10.6 (n=25)
group c:	96.2 ± 10.0 (n=26)

We thank O. Terrier for valuable technical assistance and R. Grantham for help with the manuscript.

References: Barbotin, F., Y. Carton, F. Kellner-Pillaut 1979, Bull. Soc. Entomol. Fr. 84:19-26; Basden, E.B. 1972, DIS 48:70-72; Rouault, J. 1979, C. R. Acad. Sci. Paris (in press).

Bournias-Vardiabasis, N. and M. Bownes.  
City of Hope National Medical Center,  
Duarte, California, and University of  
Edinburgh. Cell death in the tumorous  
head mutant of *Drosophila melanogaster*.

The tumorous head mutants of *Drosophila melanogaster* not only show homeotic transformations of head to genitalia and antenna to leg, but also have numerous duplications and deficiencies of head structures. Often the eyes are reduced or missing, and the palpi and head bristles can be absent or duplicated. The appearance of

these defects suggested that they may result from cell death. Therefore, we have investigated the relationship between cell death observed in the eye-antennal disc and the adult abnormalities of the head.

Late third instar eye-antennal discs of six stocks were examined for cell death using a trypan blue/neutral red staining procedure. The dead cells were identified by their dark blue coloration.

Oregon R, tuh-1a;  $\frac{Ubx^{130}}{TM1}$ , and y tuh-1b; mwh jv showed occasional small areas of cell death (Fig. 1a) of the eye antennal discs stained from tuh-1a; tuh-3, y tuh-1b; tuh-3 and tuh-3 stocks 26%, 16%, and 21% showed cell death in the presumptive eye facet region, the vibrissae region, the antenna arista region, or the palpus rostrulhaut region (Fig. 1a). This suggests that the cell death is the result of the activity of the tuh-3 gene in these mutants and the cell death is located so that, according to eye-antennal disc fate maps, the defects seen in the adult could result from it.

A comparison of the percentage of tumorous head eye-antennal discs showing cell death and the percentage of abnormalities found in the adult which may be attributable to cell death, from the same fly population, showed that there was a strong positive correlation between these two observations

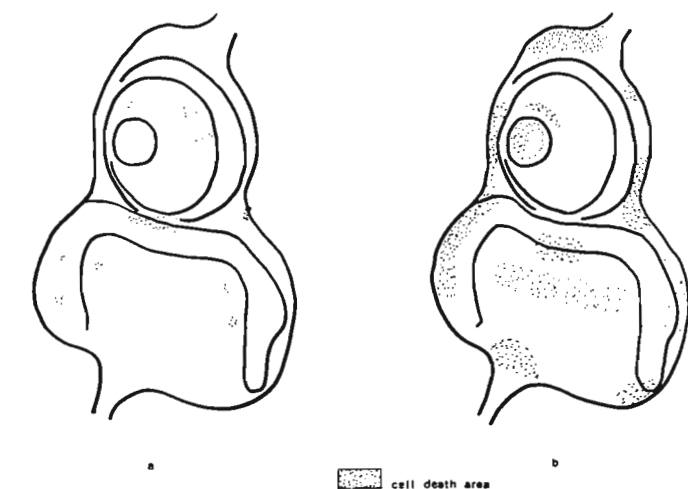


Fig. 1. Cell death areas (stippled) in whole mounts of eye-antennal discs of (a) Oregon R, (b) tuh-1a, tuh-3, y tuh-1b; tuh-3 and tuh-3 late third instar larva.

(Table 1) ( $p = 0.05$ ). Temperature did not affect the frequency of cell death in discs.

In conclusion, many of the aspects of the tumorous head phenotype probably are the result of cell death in the eye-antennal disc. Thus, the tuh-3 gene is not only responsible for some embryonic lethality and homeotic transformations, but also for cell death in the eye-antennal disc of the larva.

Table 1. Relationship between cell death in the eye-antennal discs and adult abnormalities in the head of tumorous head adults.

Abnormalities probably due to cell death	Number of abnormalities scored	Total number of all types of abnormalities scored in the population	
Antennal and arista missing	20	1367	
Eye reduced	326	Percent of abnormalities probably caused by cell death (a)	38
Eye missing	16	Expressivity for total population (i.e., number abnormalities per half head) (b)	1.45
Head to palpus	15	Calculated percent of discs that should show cell death (a/b) if our assumptions are correct	26
Palpus missing	85		
Rostrahlaut to palpus	16	Actual percent of discs showing cell death	27
Palpus malformed	51		
Total abnormalities attributable to cell death	529		

Bownes, M. and M. Seiler. University of Essex, Colchester, England and University of Freiburg, Germany. UV irradiation of *Drosophila* embryos.

Using UV irradiation it is possible to cause polarity reversals in the eggs of some insects. In *Smittia* double-abdomen embryos, where the head and thorax are replaced by a second abdomen in mirror image symmetry to the original position of the abdomen, can be induced with 100%

efficiency (Kalthoff, 1971). In *Drosophila*, however, double abdomens could not be induced by UV and the only reorganized embryos were ones containing 8 abdominal segments occupying the whole egg with no head or thorax and no loose tissue at the anterior (Bownes and Kalthoff, 1974). The maternal effect mutation *bicaudal* (*bic*) of *Drosophila* leads to the production of similar embryos (Bull, 1966; Nusslein-Volhard, 1977). It is possible that since *bicaudal* mothers can produce this defect in the organization of pattern in the embryo that eggs laid by these mothers would also be sensitive to UV irradiation-induced polarity reversals. Eggs were collected from both *bic/vgB* mothers which normally produce some *bicaudal* eggs and *bic/bic* mothers which rarely produce *bicaudal* eggs when homozygous, but often produce them when hemizygous. Eggs were UV irradiated at the anterior pole at the nuclear multiplication stage using 285nm wavelength. As can be seen in the table the proportions of abnormal embryos are not altered very much by the different genetic backgrounds, although the number of undifferentiated and abnormal embryos is significantly increased in all cases by the UV. Furthermore, no *bicaudal* embryos and no increase in the number of abdomen-only embryos were observed. Thus the genetic instability in these flies which causes them to produce double abdomens does not make them more responsive to UV irradiation. One possible explanation for this is that the *bicaudal* mutation alters the initial establishment of positional information whereas UV ir-

Survival of UV-irradiated eggs laid by females with different genotypes

Genotype	Experiment	Total	% Hatched	% Undifferentiated	% Abnormal
OrR	control	264	76	17	7
	UV irradiated	632	12	34	54
OrK	control	226	75	22	3
	UV irradiated	259	31	35	35
<i>bic/bic</i>	control	1128	81	14	5
	UV irradiated	1680	29	30	41
<i>bic/vgB</i>	control	264	29	60	11
	UV irradiated	536	1	77	22

radiation alters its interpretation. Thus the bicaudal embryos resulting from mutation and the UV-induced double abdomens found in other species of insects may well be produced by entirely different pathways at the molecular level.

References: Bownes, M. and K. Kalthoff 1974, *J. Embryol. Exp. Morph.* 31: 329-345; Bull, A.L. 1966, *J. Exp. Zool.* 161: 221-242; Kalthoff, K. 1971, *Wilhelm Roux Archiv* 168: 63-84; Nusslein-Volhard 1977, *Wilhelm Roux Archiv* 183: 249-268.

Acknowledgments: Thanks to Dr. K. Kalthoff for the use of his excellent UV irradiation facilities and for many useful discussions.

Bregliano, J.C., A. Bucheton, J.M. Lavige, A. Pélisson and G. Picard, University of Clermont-Ferrand II, France. Hybrid dysgenesis in *Drosophila melanogaster*: the I-R system.

Since 1970 we have been working on a non-mendelian female sterility in *Drosophila melanogaster*. On the basis of the fertility of  $F_1$  females, two main classes of strains, inducer and reactive, can be distinguished. Crosses between reactive females and inducer males give rise to daughters (SF females) showing a more

or less important reduction of fertility, while reciprocal crosses produce only normally fertile daughters (RSF females). SF sterility is characterized by several specific physiological features (Picard et al., 1977). A survey of more than 200 strains indicates that all wild populations are inducer, whatever may be their geographical origin, while both inducer and reactive are found among long established laboratory stocks.

We have demonstrated that SF sterility results from an interaction between a chromosomal factor (I factor) responsible for the inducer condition and a genetic state, called reactivity, responsible for the reactive condition. Both conditions show a great range of variations. According to the amount of reduction of fertility of SF females, reactive and inducer strains can be arranged from "strong" to "weak": the stronger the parental strains, the higher the reduction of SF female fertility (Bucheton et al., 1976). All wild populations tested so far are strong inducer.

Reactivity corresponds to a cytoplasmic state controlled by a polygenic system, with a long delayed effect (Bucheton and Picard, 1978). I factor is chromosomal and may be linked to any chromosome of inducer strains. Two kinds of chromosomes, inducer ( $i^+$ ) and non-inducer ( $i^0$ ) have been found in inducer strains according to their ability or not to carry I factor, respectively. Through heterozygous males bearing both  $i^+$  and reactive originating (r) chromosomes, I factor is transmitted following a strict mendelian pattern. In contrast, in heterozygous females, even in those carrying only one  $i^+$  chromosome, every r chromosome may acquire irreversibly I factor, often with a high frequency, by a process called chromosomal contamination (Picard, 1976, 1978). Several evidences indicate that I factor might be a transposable element (Pélisson, 1978). Chromosomal contamination occurs only in females in which the I-R interaction exists but not in inducer females. Indeed, although  $i^0$  chromosomes can contaminate in SF females as well as r chromosomes, some inducer strains maintain a stable  $i^+/i^0$  polymorphism. The lack of chromosomal contamination in  $i^+/i^0$  inducer females allows the mapping of I factor. The first results support the idea that there are only a few sites on each chromosome but the data do not permit a decision as to whether or not these locations are the same on all homologous chromosomes of various strains (Pélisson and Picard, 1979).

We recently showed (Picard et al., 1978) that the I-R interaction leads not only to SF sterility and chromosomal contamination but also to high levels of several dysgenic traits in the female germ line (X non-disjunction, lethal and visible mutations). Some of the mutations observed are very unstable, suggesting they might result from insertions. It is of course tempting to hypothesize that they are insertions of I factor. The I-R interaction does not seem to have any effect on the male germ line.

The I-R interaction clearly enters in the field of hybrid dysgenesis. It is now firmly established (Kidwell, 1979) that there are at least two causally independent systems displaying many common features: the I-R and the P-M systems. The latter produces dysgenic traits in both female and male germ line, especially male recombination. These observations make it necessary to take a critical look at many studies done for 40 years on *Drosophila melanogaster*, mainly on mutator effects. In most cases, the experimental schemes do not exclude the possibility that the high mutability observed results from strain interactions rather than from widespread mutator genes acting in natural populations. Moreover, we claim that it is no longer possible for *Drosophila* geneticists to neglect the I-R and P-M classifications of the stocks they use.

References: Bucheton et al. 1976, *Heredity* 36:305-314; Bucheton and Picard 1978, *Heredity* 40:207-223; Kidwell 1979, *Genet. Res.* in press; Pelisson 1978, *Genet. Res.* 32:113-122; Pelisson and Picard 1979, *Genetics* in press; Picard 1976, *Genetics* 83:107-123; Picard 1978, *Molec. Gen. Genet.* 164:235-247; Picard et al. 1977, *Biol. Cell.* 29:89-98; Picard et al. 1978, *Genet. Res.* in press.

Brncic, D. and Budnik, M. Universidad de Chile, Santiago, Chile. Colonization of *Drosophila subobscura* Collin in Chile.

In February 1978, Mr. H. Fenner of our laboratory collected for the first time in Chile *Drosophila subobscura* Collin, in an orchard near Puerto Montt (S.41°30'). A laboratory stock was established and our determination was con-

firmed by crosses with stocks from Bilbao (Spain) provided by Prof. Antonio Prevosti (Barcelona), and from Norway (Stock TX 2361-01 provided by the Univ. of Kansas). F<sub>1</sub> and F<sub>2</sub> were fully fertile in both tests. Photomicrographs of the giant salivary gland chromosomes of the larvae from the Chilean stock were studied by Prof. A. Prevosti (Barcelona), who kindly informs us that the band sequences correspond most probably to those observed in the Western Mediterranean Europe region (Meridional Spain), and in the Mediterranean coast of North Africa (Marruecos and Tunis). This first observation of *D. subobscura* in Puerto Montt (Chile) is significant, because *Drosophila* have been collected there practically every summer for the last 25 years.

Eight months later (November and December 1978), we collected *D. subobscura* in large numbers in the following places in Chile, that correspond to a north-south gradient of about 1200 km: Santiago (S.33°30'), Lake Rapel (S.34°15'), Talca (S.35°26'), Chillan (S.36°36'), Salto del Laja (S.37°10'), Los Angeles (S.37°28'), Pucon (S.39°15'), Valdivia (S.39°50') and Puerto Montt (S.41°30'). Most collections were made utilizing fermenting banana traps placed in orchards or gardens, with the exception of the Salto del Laja and Pucon localities in which the baits were placed in small natural forests of *Notofagus*. In addition, a few flies were collected in a fruit-vegetable store in Chillan City by sweeping the net over the fruits. In none of the above mentioned places was *D. subobscura* recorded before, indicating that it represents a newly introduced species coming most probably from the Palearctic zone. We have no information of the existence of the species in other places of Neotropical zone or in the Nearctic.

The quantitative data of the collections seems to indicate that the rapid invasion of *subobscura* has displaced some "domestic" species, particularly *D. simulans*, which was a very abundant species all over the central and south-central parts of Chile, and has now become a relatively rare species.

Stocks of *D. subobscura*, originated from the above indicated places, were sent to Prof. A. Prevosti of Barcelona for further research. [The authors would like to thank Mrs. Hertha Fenner and Mr. Gonzalo Gajardo from our Department and Prof. Eduardo del Solar from the Univ. Austral (Valdivia - Chile), who collected the flies at Puerto Montt, Lake Rapel and Valdivia respectively, supported by grants from PNUD/UNESCO (Proyect RLA 76/006) and Univ. of Chile (Proyect B 027 - 784).]

Bryant, M. L. and M. R. Murnik. Western Illinois University. The mutagenicity of herbicides in *Drosophila melanogaster*.

Our laboratory is interested in the potential mutagenicity of herbicides. Trifluralin (Eli Lilly Company) is an herbicide commonly used for weed prevention in soybean crops. It is a yellow liquid, miscible with water. Accord-

ding to several investigators (Andersen, Leighty, and Takahashi 1972; Shirasu, 1975), trifluralin is not mutagenic in any of four different microorganisms. Since tests in microorganisms test only for point mutations, we decided to test this herbicide in *Drosophila*. Male, wild type Oregon-R flies, fed as larvae 0.01 trifluralin (w/w in modified Carpenter's medium) were mated with virgin Basc females. The concentration of trifluralin used was the highest dose not toxic to the developing flies. The treated group produced 0.09% sex-linked recessive lethals, while the control group had 0.12% of these mutations. Thus, the results of these tests indicate that trifluralin does not produce sex-linked recessive lethals in *Drosophila*.

For a more complete testing program, we also used a chromosomal assay test system. Both larval fed and adult fed flies were used. Adult male flies (0-4 hours old) were starved for four hours, and then allowed to feed for 24 hours on 0.02% trifluralin (v/v) in a 1% sucrose (w/v) solution. The herbicide concentration used is about the LD<sub>10</sub> dose for adults. Males of the genotype  $y^2 w^1 ct^6 f/sc^8 y^+ Y B^S$  which survived the feeding were mated with two virgin  $y/y$  females for two days each. Live transfer of males was made for five subsequent broods. Progeny of these matings were scored for loss, breakage, and nondisjunction of the X and Y chromosomes. There was no significant difference between the treated and control groups for any of the aberrations scored. However, the actual number of aberrations scored in each category was always higher in the treated group.

A larval fed group of  $y^2 w^1 ct^6 f/sc^8 y^+ Y B^S$  males were mated within 24 hours of eclosion to  $y/y$  virgin females, and their progeny scored for aberrations. The results are in Table 1. The rate of XXY nondisjunction (0.12%) in the treated group is significantly different ( $P < 0.01$ ) from that of the control group (0.04%). Data were analyzed according to the tables of Kastenbaum and Bowman.

Table 1. Chromosomal assay - larval feeding

	XXY nondisjunction		X or Y loss		N
	Number	Percent	Number	Percent	
Treated	16**	0.12	22	0.17	13152
Control	11	0.04	46	0.16	28767

\*\*  $p < 0.01$

the mitotic divisions of development. The larval fed treated group had significantly more mosaics than the control group at the 95% level.

In conclusion, although trifluralin does not appear to produce point mutations in microorganisms or *Drosophila*, it does appear to induce chromosome breakage and nondisjunction in *Drosophila*. The mechanism of chromosome aberration appears to be spindle apparatus malformation (Lignowski and Scott). In this case, larval feeding of flies was more efficient in demonstrating chromosome aberrations than was adult feeding.

References: Andersen, K.J., E.G. Leighty and M.T. Takahashi 1972, J. Agr. Food. Chem. 20:649-656; Kastenbaum, M.A. and K.O. Bowman 1970, Mutation Res. 9:524-526; Lignowski, E.M. and E.G. Scott 1972, Weed Science 20:267-270; Shirasu, Y. 1975, Environ. Quality Safety 4: 226-231.

Bulyzhenkov, V.E. and V.I. Ivanov. Institute of Medical Genetics, Moscow, USSR. Expression of Antennapedia<sup>50</sup> in triploid *Drosophila melanogaster*.

Dominant homozygous lethal homoeotic mutations of Antennapedia (Antp; 3-48.) locus cause transformation of proximal antennal segments into respective leg elements. The allele-specific interaction of Antp alleles with some other homoeotic genes in transforming the antennae (Bulyzhenkov, Ginter and Ivanov, 1975) suggested the mutations to be of missense type; thus the determinative products of mutant as well as of normal alleles should appear in cells of the antennal imaginal discs. In this case, a certain influence might be expected of an extra dose of Antp<sup>+</sup> on the phenotypic expression of mutant alleles of this locus. In search for such influence the homoeotic transformation of antennae in triploid flies having in their genotype two doses of normal and a single dose of mutant allele was studied. Triploid females with normal third chromosomes and marked X-chromosomes ( $z/z/FM7(y^w lz B); +/+$ ) were crossed with diploid  $FM7/Y; Antp^{50}/T(2;3)Xa$  males. The markers employed allowed us to distinguish between triploids, intersexes, and diploids. In preliminary tests complete penetrance of  $T(2;3)Xa$  was shown. To estimate the rate of homoeotic transformation of antennae, the aver-

Another aberration recently scored in our laboratory is mosaicism. Larval fed males which produced progeny for the chromosomal assay also produced progeny of the genotype  $+/B^S$ , indicating that the long arm of the Y chromosome was broken and lost in one of



Table 1. Number of leg bristles on the antennae in Antp<sup>50</sup> mutants.

	Genotype	No. of flies	Mean $\pm$ S.E.
Diploid ♂♂ & ♀♀	XY; Antp <sup>50</sup> /+ XX; Antp <sup>50</sup> /+	60	11.6 $\pm$ 1.6
Triploid ♀♀	XXX; Antp <sup>50</sup> /+/+	40	5.6 $\pm$ 1.1
Intersexes	XX; Antp <sup>50</sup> /+/+	15	32.3 $\pm$ 5.6

is hardly the only source of variation in the Antp<sup>50</sup> expression since in the XX; Antp<sup>50</sup>/+/+ intersexes having the same Antp<sup>50</sup> to Antp<sup>+</sup> ratio as the triploid females the rate of antennal transformation is three times as large as that in the diploids and six times greater than in the triploids, thus suggesting the possible role of the balance of X-linked modifiers.

References: Bulzyhenkov, Ginter and Ivanov 1975, Genetika (USSR) 11:27-33.

Calvez, C. Université Claude Bernard Lyon I, Villeurbanne, France. Reduced mitotic activity in anterior and posterior follicle cells of the egg-chamber of *Drosophila melanogaster*.

The egg-chamber is surrounded by follicle cells derived from mesodermal cells during the migration of 16-cystocyte clusters through germarial region 2 (Brown and King 1964). Each chamber that enters the vitellarium contains approximately 80 follicle cells.

During egg-chamber growth from stage 1 to stage 6 (according to the terminology of King et al. 1956) these cells divide; during stage 6 a maximum number of follicle cells, roughly 1200, is reached and mitosis ceases (King and Vanoucek 1960). Each cell increases thereafter in surface area by a series of about four endopolyploid doublings. During stage 9, however, a group of follicle cells originating at the anterior pole of the egg-chamber migrates through the center of the nurse chamber. These follicle cells squeeze through the nurse cells and reach the surface of the oocyte (King 1970). These border cells thereafter secrete vitelline membrane and later form the micropylar complex.

Follicle cells from the posterior pole of the egg-chamber may also have a specific function and act as oocyte stabilization and growth factors (Koch and King 1969).

The question is whether during the division and differentiation of ovarian follicle epithelium the cells at the two poles have a particular behavior in relation to their particular function. To answer this question [<sup>3</sup>H] thymidine (25 to 50 pmole per animal) was injected into newly hatched females. Groups of four females were killed, the first one hour after injection and the others daily thereafter until the seventh day.

Seriated sections of ovaries were examined after autoradiography (Calvez 1978).

The percentage of labelled follicle cells and their distribution in the egg-chamber were observed daily. The percentage of labelled border cells was also monitored.

Follicle cells: One hour after injection in all the egg-chambers in the vitellarium (stage 1 to stage 7), 40% to 50% of the follicle cells were labelled. Labelled cells were regularly located around each egg-chamber. During the migration of these chambers through the vitellarium, from day 1 to day 4, the percentage of labelled follicle cells decreased because the radioactive DNA was diluted during the doublings. Labelled follicle cell distribution was irregular. The largest numbers of labelled cells were found at the two poles (photos 1 and 2); density of silver grain labelling was very high in these cells.

Although the [<sup>3</sup>H] thymidine pool was depleted in 30 minutes (Chandley 1966) a large number of labelled follicle cells appeared in egg-chambers produced from the first to the fourth day. Respective labelling percentages in stages 2 and 3 for these four days were 35%, 35%, 17%, 5%. In these chambers and during their migration the largest numbers of highly labelled cells were also observed at the two poles (photo 3). On and after the fifth day after injection isolated labelled cells were located only in these regions (photos 4 and 5).

age number of leg bristles per pair of antennae was counted. These data are shown in Table 1.

Diploid males did not differ from the diploid females so the data on both could be pooled. In triploid females the number of leg bristles on the antennae was only about half of that in the diploid Antp<sup>50</sup> heterozygotes. This decrease may be interpreted as a result of the lower Antp<sup>50</sup> to Antp<sup>+</sup> ratio in triploids. However, the Antp<sup>50</sup> to Antp<sup>+</sup> ratio



**Border cells:** The percentage of labelled border cells was very high (80% to 90%) until the second day after injection. Moreover, seven days after injection grains were detected in 12% of border cells.



Photo 1: Stage 7 two days after injection with highly labelled follicle cells at the 2 poles.  
Photo 2: Stage 9 two days after injection with a high density of silver grains at the posterior pole follicle cells.

Photo 3: Stage 7 four days after injection with labelled follicle cells at the 2 poles.  
Photo 4: Stage 8 six days after injection with an isolated labelled follicle cell at the anterior pole.

Photo 5: Stage 10 six days after injection with an isolated labelled follicle cell at the posterior pole.

From these results four conclusions may be drawn:

1. Labelling of follicle cells in egg-chambers produced a long time after injection concurs with the hypothesis proposed by King (1970) that follicle cells derive from generative profollicle cells which must function as the stem-line oogonia.

2. Since labelled follicle cells were often found a long time after injection at the anterior pole and labelled border cells were subsequently found, the number of DNA doublings (mitoses and possibly endopolyploidy) was lower in these follicle cells. The specialization of the follicle cells was therefore determined as from egg-chamber formation.

3. The follicle cells of the posterior pole also have reduced mitotic activity. Like the anterior follicle cells, they may therefore have a specific function. This result corroborates the hypothesis of Koch and King (1969) who on the basis of morphological data suggest a role for these cells in oocyte induction.

4. Since during the first four days after injection the largest number of labelled follicle cells appeared at the two poles of the newly produced egg chambers it may be supposed that different profollicle lines exist in the germarium. Some cells form the majority of follicle cells, others specifically produce pole cells.

References: Brown, E.H. and R.C. King 1964, *Growth* 28:41-81; Calvez, C. 1978, *Thèse 3ème Cycle*, Lyon; Chandley, A.C. 1966, *Exptl. Cell Res.* 44:201-215; King, R.C. 1970, In *Ovarian development in Drosophila melanogaster*, Acad. Press N.Y. and London; King, R.C., A.C. Robinson and R.F. Smith 1956, *Growth* 20:121-157; King, R.C. and E.G. Vanoucek 1960, *Growth* 24:333-338; Koch, E.A. and R.C. King 1969, *Z. Zellforsch* 102:129-152.

Carlson, E., P. Ferriola and E. Schuchman.  
State University of New York, Stony Brook.  
Pseudoallelism at the vestigial locus.

The vestigial series of alleles involves several quantitative variations in wing size and shape and some qualitatively distinct pleiotropic traits (including scutellar bristle position, body size, and viability). The vestigial alleles are difficult to work with because some complement, some are phenotypically normal as homozygotes, and some show a nicking or notching of the wings in the heterozygous condition. Furthermore, vestigial is sensitive to temperature, lower temperatures (about 18°C) having more mutant phenotypes and higher temperatures (about 28°C) having more normal phenotypes.

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Cross used (x cn vg sf <sup>2</sup> ♂)	Total counted	Confirmed non-vg	Distance (map units)
vg <sup>np</sup> /cn vg sf <sup>2</sup>	30,389	4 cn+++	0.026
vg <sup>nw</sup> /cn vg sf <sup>2</sup>	9,813	2 +++sf	0.041
vg <sup>E7</sup> /cn vg sf <sup>2</sup>	14,567	0	-
vg <sup>NO2</sup> /cn vg sf <sup>2</sup>	7,145	0	-
vg <sup>np</sup> /cn vg <sup>E7</sup> sf	4,402	0	-
vg <sup>nw</sup> /cn vg <sup>E7</sup> sf	13,257	0	-
vg <sup>NO2</sup> /cn vg <sup>np</sup> sf	16,234	0	-

$\left[ \begin{array}{c} \text{vg}^{\text{E7}} \\ \text{vg}^{\text{NO2}} \end{array} \right]$

$\leftarrow 0.026 \rightarrow \quad \leftarrow 0.041 \rightarrow$   
 $\text{vg}^{\text{np}} \quad \quad \text{vg} \quad \quad \text{vg}^{\text{nw}}$

Stocks of cn vg sf<sup>2</sup> were made to provide suitable markers for a four-point test (cn = 2,57.5; vg = 2,67.0; sf<sup>2</sup> = 2,71.5). The alleles vg, vg<sup>np</sup>, vg<sup>NO2</sup>, vg<sup>nw</sup>, and vg<sup>E7</sup> were used in a series of crosses utilizing the cn and sf markers. The results so far establish the pseudo-allelic nature of the vestigial region with three sites mapped. Two alleles, vg<sup>NO2</sup> and vg<sup>E7</sup>, have not yet been separated. The vg<sup>E7</sup> allele shows a strap allele when heteroallelic with vg. It was induced by ethyl methane sulfonate and it is phenotypically normal as a homozygote. Its failure to undergo pseudoallelic crossing-over with vg, vg<sup>np</sup>, and vg<sup>nw</sup> suggests that it might be a minute intragenic rearrangement. Similarly, vg<sup>NO2</sup> does not crossover with vg or vg<sup>np</sup> and may be a minute rearrangement. When vg<sup>nw</sup> is crossed to vg<sup>NO2</sup> the heteroallele, vg<sup>nw</sup>/vg<sup>NO2</sup>, does not appear and is thus inviable at both 18°C and 25°C.

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Choo, J.K. Chungang University, Seoul, Korea. Genetic change of Korean natural population of *Drosophila melanogaster*.

The frequency of recessive lethal genes on the second chromosomes concealed in natural populations of *D. melanogaster* has been analyzed annually. Surveys have been made since 1971 in Anyang City, since 1975 in Ulsan City, and since 1977 in the Banweol area. The frequency of lethal plus semilethal second chromosomes had been about 28.2% in the 1971-1973 period at Anyang City. It then increased directly through three years and the population maintained 48.6% of L+S1 content in 1977. In the natural population of Ulsan City, the L+S1 content has been 65.7% to 53.4% in the 1975-1977 period. On the other hand, the frequency in the Banweol population was 26.0% in 1977, low compared to other populations.

All second homozygote viability consisting of lethal genes had been 22.3% in the Anyang population in 1971, and then its rate decreased annually to 14.6% in 1977. On the contrary, the Ulsan population maintained about 15.0% on the average in 1975-1977, and 24.8% occurred at Banweol in 1977.

The allelism rate between lethals isolated from the Anyang population has maintained unchanged at about 1-2% during the past six years. However, allelism rate of the Ulsan population decreased from 5.66% in 1975 to 1.47% in 1976.

The frequency of individuals eliminated by deleterious genes in the natural population was estimated to be  $IQ^2$ , where I and Q are the L+S1 frequency and allelism rate for successive years. The elimination rate in the Anyang population increased by two times during six years, from 0.04% to 0.08%. Moreover, in the Ulsan population it occurred 2.44% in 1975.

Clyde, M. University of Queensland, Brisbane, Australia. The chromosomes of *Drosophila rubra* Sturtevant.

*D. rubra*, a member of the *D. immigrans* subgroup (Wilson et al. 1969) was described by Sturtevant (1927) from the type specimen collected at Mt. Maquiling, Luzon, Philippines. The flies are yellowish with a reddish tinge. The dull

reddish color occurs on the frons, antennae, mesonotum, scutellum and abdomen. The pleurae and legs as well as face, cheeks and mouthparts are yellow.

Five isofemale lines, from Hidden Valley Springs, Luzon (adjacent to the original collection site at Mt. Maquiling) were analyzed. The salivary chromosome configuration of *D. rubra* comprises four long arms and one short arm (Fig. 1). In one isoline a small, simple inversion in the central region of chromosome III was detected (Fig. 2).

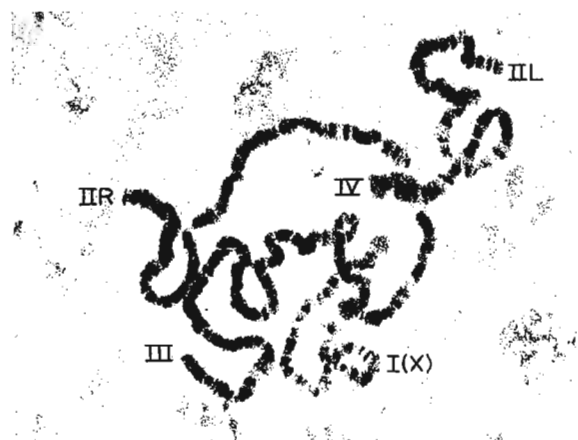


Fig. 1. Salivary gland chromosomes of *D. rubra*.



Fig. 2. Simple inversion on chromosome III in *D. rubra*. c.e. = centromere end

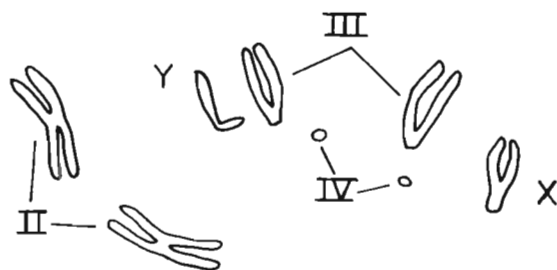


Fig. 3. Karyotype of male *D. rubra*.

*D. rubra* has a diploid chromosome number of  $2n = 8$ . The metaphase chromosomes consist of a pair of V's (chromosome II), a pair of rods (chromosome III), a pair of dots (chromosome IV) and the sex chromosomes, of which the X chromosome is rod-shaped and the Y chromosome is J-shaped with arm-ratio of approximately 1:2.5 (Fig. 3). No karyotypic variation was detected.

The isofemale lines used in this study were collected and established by Dr. Wharton B. Mather, University of Queensland.

The work reported was part of a Ph.D. thesis accepted by the University of Queensland in 1978.

References: Sturtevant, A.H. 1927, Philipp. J. Sci. 32:361-374; Wilson, F.D., M.R. Wheeler, M. Harget and M. Kambysellis 1969, Univ. Texas Publ. 6918:209-253.

Clyde, M. University of Queensland, Brisbane, Australia. Chromosome IV variation in *D. albomicans* Duda.

chromosome IV when compared to an isoline from Taiwan. This is apparent in intraspecific hybrids between the Taiwan and Chiang Mai or West Malaysia isolines (Fig. 1). In three out of eight isolines from Chiang Mai, extra heterochromatin occurred in the form of supernumerary (unattached) dots. Individuals of the same isoline may possess one extra dot or two extra dots in addition to the two short rod-shaped fourth chromosomes (Fig. 2). The presence of this extra heterochromatin in the form of dots in the karyotype appears not to have any phenotypic effect on individuals that possess it. It is conceivable that the extra dots have resulted from fragmentation of the longer rod type of chromosome IV as seen in the strain from Taiwan. As heterochromatin often carries very few or no genes, the loss of such small fragments would not have any deleterious effects on the carrier. Isolines from West Malaysia did not possess these extra dots. An alternative possibility is that extra heterochromatin has been added to the fourth chromosome in the case of the Taiwan strain, thus making it longer than the basic rod-shaped fourth chromosome exhibited by the Malaysian and Thailand strains. The "floating" dots in some of the Chiang Mai strains would then represent as yet unattached heterochromatin.

Two types of chromosome IV were detected in metaphase chromosome preparations of isofemale lines of *D. albomicans* from Southeast Asia. Isolines from Chiang Mai and West Malaysia (Penang, Kuala Lumpur) were found to have a shorter rod-shaped

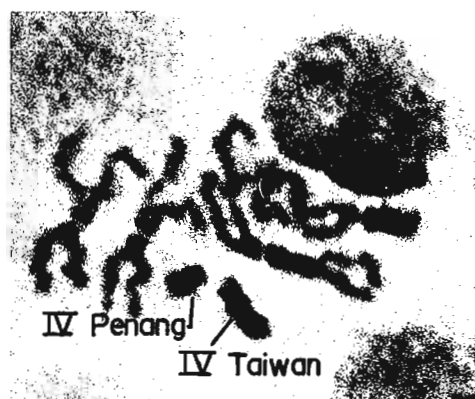


Fig. 1. Different fourth chromosomes in *D. albomicans* from Penang and Taiwan.



Fig. 2. Two extra "dots" of heterochromatin in *D. albomicans* from Chiang Mai.

The isofemale lines used in this study were collected and established by Dr. Wharton B. Mather, University of Queensland. The work reported was part of a Ph.D. thesis accepted by the University of Queensland in 1978.

Creus, A. and R. Marcos. Autonomous University of Barcelona, Bellaterra, Spain. Relationship between mating speed and duration of copulation in *D. melanogaster*.

From a survey of published data on the genus *Drosophila*, it is clear that in various species, *D. gaucha*, *D. melanogaster*, *D. persimilis*, *D. pseudoobscura* and *D. robusta*, the mating speed is an important component of fitness. However, the relation between mating speed and duration

of copulation has been the subject of very few studies. As a part of a wider analysis we present in this note the preliminary results.

The lines used in these experiments were derived from a wild type stock of *D. melanogaster* designated AR, isolated by R. Marcos in 1973 from a strain collected at the mouth of the Llobregat River, Barcelona. The flies were cultured and the experiments conducted at  $25 \pm 1^\circ\text{C}$  under standard light conditions. Samples of 50 males and 25 virgin females aged for 3 days were placed together in glass bottles of 500 ml. As soon as a pair commenced mating, they were sucked out. Mating speed and duration of copulation were scored in minutes. In each experiment the matings were scored only during the first hour. Ten replicas were done at each line.

The regression coefficients of duration of copulation with respect to mating speed were calculated. The results are summarized in the table.

Line	N (mated)	$b_{yx} \pm e_b$	F	t	d.f.
AR1	108	$-0.108 \pm 0.030$	12.55***	3.54***	106
AR2	182	$-0.138 \pm 0.061$	4.98*	2.23*	180
AR3	145	$-0.128 \pm 0.041$	11.29***	3.13***	143
AR4	132	$-0.133 \pm 0.052$	6.98**	2.58**	130

\*\*\*- significant at 0.001 level; \*\*-significant at 0.01 level;  
\*- significant at 0.05 level.

From these results we can infer that there is a negative and significant regression; that is, the flies taking a long time to mate have a shorter duration of copulation. These results are in contrast to those obtained by Spiess (1968) in *D. pseudoobscura*.

Reference: Spiess, E.B. 1968, Amer. Nat. 102:363-379.

Comendador, M.A. University of Oviedo, Spain. Abnormal bristles that show maternal inheritance in *D. simulans*.

During a routine analysis of a population of *D. simulans* recently captured in the Azores Islands, we observed an unusual proportion of flies that lack some dorsocentral and scutellar

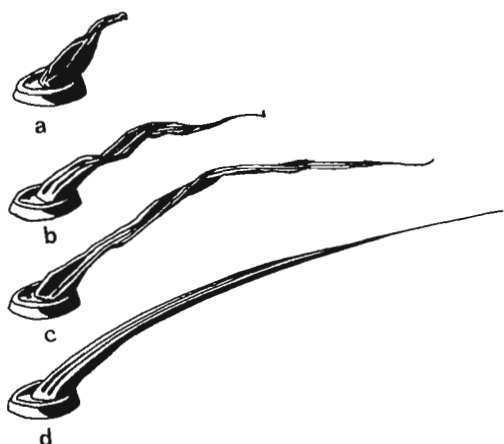


Fig. 1. Various types of bristle structures altered in size and/or shape in relation to the normal one (d), as seen under the scanning electron microscope (see text).

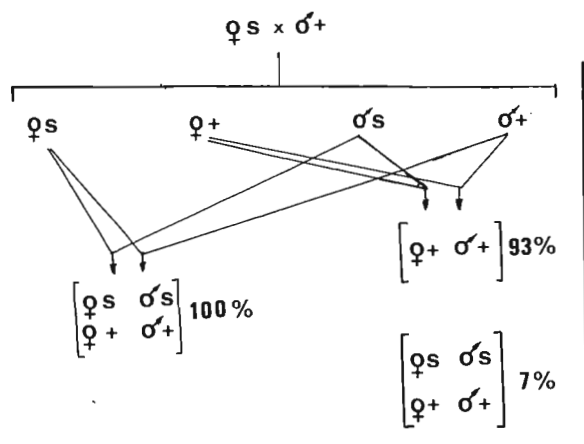


Fig. 2. Phenotypic segregation in  $F_1$  and  $F_2$  from mating S females to wild type (+) males and loss of the character in the progeny of the reciprocal cross. In percentage is given the proportion of single pair mating having each type of progeny.

probability for any location of two bristles ( $\chi^2=20.95$ ,  $p < 0.01$ ) on the same side of the body. That is, there is an appreciable strong tendency toward bilateral asymmetry. And it is so regardless of whether the two altered bristles are dosocentral or both are scutellar.

In order to establish the type of inheritance of S phenotype, 100 females and males with well expressed S phenotype were individually mated to wild type males and females taken from a natural population of Asturias. The results of these reciprocal crosses and their  $F_2$  are given in Fig. 2. Clearly no Mendelian segregation would fit these  $F_1$  and  $F_2$  segregations restricted to one type of mating. Despite the small percentage (7%) of females  $F_1+$  that, irrespective of the male parent (crosses ♀+ X ♂s and ♀+ X ♂+), give  $F_2$  segregant progenies,

bristles. The 50 females with higher number of missing bristles were selected for individual mating to males from the same population. In all the progenies but one, a very small proportion of flies showed a low number (one or two) of missing bristles, as is often found in many natural populations. The progeny of a female, however, comprised an unexpectedly high number of individuals with most or even all dorsocentral and scutellar bristles either wholly suppressed or variously altered in their structure. This deviant phenotype (S) is most apparent in the dorsocentral and scutellar regions, although it shows up also in other bristle regions; our data will refer only to both dorsocentral and scutellar regions taken together.

The following types of altered bristles are observed: (1) bristle structures wholly suppressed; (2) only the basal ring left (which under scanning electron microscope appears as in Fig. 1a); (3) shortened and distorted bristle; (4) shortened, distorted and light-colored bristle; (5) normally large but distorted and light-colored bristle (Fig. 1). For the time being and in order to clear up its mode of inheritance, we assume that all these five graded types of altered bristles result from the variable expressivity of one and the same phenotype.

The first noteworthy feature of S strain is its sex dimorphism: there are more deviant males (45.12%) than females (30.20%), and the mean number of abnormal bristles per affected fly is also significantly higher in males ( $2.32 \pm 0.06$ ) than in females ( $1.66 \pm 0.07$ ). This is contrary to most known cases of *Drosophila* strains with missing bristles, where both penetrance and expressivity for bristle suppression are greater in females than in males.

A second intriguing feature of the S phenotype appears in the spatial bristle pattern shown by flies with two altered bristles: both tend to be located more often than expected in the null hypothesis of equal

there is every reason to suggest that the trait is controlled by an extranuclear factor.

Most facts here reported might be explained if such a factor were scarce and its replication rate were slower than the cellular division rate, so that it could be lost in certain cellular lines but not in others within an individual. Further tests to probe the hypothesis are already in progress.

David, J.R., M. de Scheemaeker-Louis and E. Pla. Laboratoire de Biologie et Génétique Evolutive du CNRS, Gif-sur-Yvette, France. Evolution in the seven species of the *Drosophila melanogaster* subgroup: comparison of the electrophoretic mobility of the enzymes produced by the Adh locus.

Alcohol dehydrogenase (Adh) of *Drosophila melanogaster* is an extensively studied enzyme for several reasons. It has been possible to relate the enzymatic function with a physiological trait, ethanol tolerance. This phenotypic property has a strong adaptive significance in the ecology of wild populations. Finally the worldwide polymorphism known at the Adh locus seems to be maintained by natural selection. The F

allele, producing a fast migrating protein, has a higher activity than the S allele and is most frequent in temperate countries where the alcohol tolerance is higher (see David, 1976 for a review). Apart from the two widespread Adh<sup>F</sup> and Adh<sup>S</sup> alleles, three other rare ones have also been found in natural populations.

At the present time six other species are known in the *D. melanogaster* taxonomic subgroup; these include the cosmopolite *D. simulans* and five others, endemic in the Ethiopian region. Up to now only the Adh of *D. simulans* has been studied and the species is known to be generally monomorphic for a very slow allele, having some analogy with the US allele found in an African population of *D. melanogaster*. It seemed therefore interesting to compare the mobility of Adh found in the different species with that of the five alleles available in *D. melanogaster*.

Results are presented in Fig. 1. In order to improve electrophoretic discrimination, the various alleles were ordered according to their decreasing anodal mobility. In all cases, the electrophoretic pattern was the same: for a homozygous strain, we observe two isozymes which correspond to conformational differences of the dimer molecule; the activity of the slower isozyme is always higher. Moreover, treatment of flies with acetone (not shown) resulted in all cases in the disappearance of the slow migrating isozyme and in the increase of a third, still faster migrating, band. There is therefore almost a complete certitude that the enzymes shown in Fig. 1 are the product of the same, homologous locus, in all species.

It is well known that a single electrophoretic technique reveals only part of the effective genetic variability. In the present case, it is striking that ordinary starch gel electrophoresis was sufficient for showing a significant difference between all alleles. In some cases, the difference of migration is very small (1 mm) but it proved to be always the same in different runs.

Five alleles were found in *D. melanogaster*, the most extensively studied species. By contrast, all the other species were observed to be monomorphic. This last conclusion, however, cannot be considered as being strongly established because only a small number of laboratory strains were studied. Present data seem, however, to allow several conclusions.

First the Adh locus, at least in that group of species, can be considered as a fast evolving gene. As previously stated, the enzyme produced is involved in ethanol detoxification and big differences are observed in ethanol tolerance of the various species (David et al. 1974). Perhaps this diversification of the ecological niche with respect to environmental alcohol is related to the occurrence of different alleles in the different species.

Second, a proportion of 100% of unique alleles is observed here so that the enzyme seems to have an absolute diagnostic value for a specific identification. In a recent paper (Throckmorton, 1978) indicated that, when studying phylogenetic relationship between related species, an average of 30% of unique alleles was observed. The much higher proportion found here is probably a singularity of the locus here studied.

Third, a general problem in speciation studies is to establish whether the electrophoretic alleles occurred before or after the specific divergence. In the present case we can state that the apparition of the new alleles and their fixation almost certainly occurred after the specific separation.



Fig. 1. Electrophoregram of Adh stained with isopropanol after starch gel electrophoresis (discontinuous buffer system of Poulik, 1957). From left to right: *D. yakuba*; *D. melanogaster* allele Ultra Slow; *D. simulans*; *D. melanogaster* Slow; *D. teissieri*; *D. mauritiana*; *D. orena*; *D. melanogaster* Fast; *D. melanogaster* F<sub>1</sub>; *D. erecta*; *D. melanogaster* Ultra Fast.

Phylogenetic relationships between the various species were recently worked out using polytene chromosome structures (Lemeunier and Ashburner, 1975). Results here presented suggest that the genetic distance between the various species could be high. Of course, analysis of a single gene provides little information and studies of other loci are in progress.

References: David, J. 1977, *Année Biol.* 16:451-472; David, J., F. Fouillet and M.F. Arens 1974, *Arch. Zool. exp. gén.* 115:401-410; David, J., F. Lemeunier, L. Tsacas and C. Bocquet 1974, *Ann. Génét.* 17:235-241; Lemeunier, F. and M.A. Ashburner 1976, *Proc. R. Soc. Lond. B* 193:225-294; Poulik, M.D. 1957, *Nature* 180:1477; Throckmorton, L.M. 1978, *Beltsville Symposium in Agricultural Research*, Wiley and Sons, N.Y. 221-239.

Davis, B.K. Virginia Polytechnic Institute and State University, Blacksburg, Virginia. Mutants which cause abnormal rotation of the abdomen or genitalia.

A surprising number of loci can mutate to affect the development of the abdomen or genitalia in *D. melanogaster* such that the adult structures are out of alignment with the rest of the body. A study of the twisted locus

(Davis 1975; Davis, DIS this issue) led to a search through the "Genetic Variations of *D. melanogaster*" (Lindsley and Grell 1968), aided by "The Mutants of *D. melanogaster* Classified According to Body Parts Affected" (Braver 1956). Although this search was not exhaustive for the main source (Lindsley and Grell 1968) and did not include current literature, it revealed 15 loci with one or more alleles reported to cause rotation of the genitalia or the abdomen (Table 1).



Table 1. Mutants of *D. melanogaster* causing genital or abdominal rotation.

Mutant	Chromosome	Sex Affected*		Fertility	
		Male	Female	Male	Female
Abruptex	X	G	-	+	+
curlex-twisted genitalia	X	G	-	-	+
shifted genitals	X	G	-	-	+
twisted	X	A	A	+	+
twisted genitals	X	G	G	±	+
abrupt <sup>2</sup>	2	G	-	-	+
chaetelle	2	G	-	+	-
engrailed	2	G	-	±	+
fringed	2	A	A	+	-
rubroad	2	G	-	+	+
thickoid	2	G	-	+	+
rotated abdomen	3	A	A	-	-
rotated penis	3	G	-	-	+
Roughened	3	G	-	±	+
abdomen rotatum	4	A	A	-	±

\* A indicates abdominal rotation; G indicates genital rotation; and - indicates no rotation.

sterile or fully fertile with no apparent differences in internal anatomy or in sperm motility (Davis, DIS this issue). The mutant chaetelle has the unusual property that many males have rotated genitalia, but the infertility occurs in females (Beatty 1949).

Why do so many loci cause rotation of the abdomen or genitalia? It is known that the normal development of the male reproductive system involves a complete clockwise 360° rotation of the terminal abdominal segments (Gleitsch 1936). This results in a complete clockwise loop of the ejaculatory duct around the rectum. Following the duct from its anterior connections with the paired vas deferens and accessory glands to its posterior connection with the penis, it originates ventrally, loops from the fly's left side over the dorsal rectum, and then down the right side to a ventral position (see Fig. 38 in Miller 1950). Either there are many genes which normally control this movement, or it is a process which can be readily interfered with by mutants. It might be argued that the four loci which cause abdominal rotation belong in a different category since they affect females as well as males. However, the fact that twisted<sup>3</sup> causes both genital and abdominal rotation (Davis, DIS this issue) strongly supports an association between the two types of mutants.

There are two general questions about the developmental mechanisms which account for the observed external rotation of male genitalia in these mutants. The first is the direction of the rotation, either clockwise or counterclockwise. The second is the degree of rotation. For example, a specific male might have genitalia out of phase with the abdomen by 90° clockwise because (1) the normal clockwise rotation was incomplete, stopping at 90°; or (2) clockwise rotation continued beyond the normal 360°, to 450°; or (3) rotation was counterclockwise for 270°. Similarly, a male might have genitalia out of phase with the abdomen by 90° counter-

These loci are scattered throughout the genome with each of the four chromosomes represented. All four of the mutants which cause abdominal rotation affect both sexes, but of the 11 which cause genital rotation, all but one are male specific. Although the single exception is named "twisted genitals", the original phenotypic description merely refers to an abnormal positioning of deformed external genitalia (Fahmy 1959). It is therefore possible that the mutant did not cause rotation. Unfortunately, it cannot be examined because it has been lost.

The degree of rotation is quite variable from locus to locus and from fly to fly for some loci. For example, males homozygous for rotated penis have genital rotation varying from normal to 270°. The direction of rotation in different mutants may be clockwise or counterclockwise. As used throughout this paper, "clockwise" and "counterclockwise" refer to flies as viewed from the posterior end. The abdomen rotatum locus has one allele (ar) which causes clockwise rotation and two alleles (ar<sup>57d</sup> and ar<sup>57g</sup>) which cause counterclockwise rotation.

Rotation is often but not invariably associated with infertility or reduced fertility. Males homozygous for rotated penis are sterile even when the genitalia are not out of line. Males carrying twisted<sup>3</sup> may be



clockwise because (1) clockwise rotation stopped at  $270^\circ$ ; or (2) rotation was counterclockwise for  $90^\circ$ ; or (3) rotation was counterclockwise for  $450^\circ$ . Fortunately, the loop of the ejaculatory duct around the rectum serves as a record of the mechanism.

Table 2. Developmental rotation of the posterior abdominal segments in twisted<sup>3</sup> males and its effect on internal and external structures.

Developmental rotation <sup>1</sup>	Ejaculatory duct loop <sup>2</sup>	Relationship of genitalia to the abdomen	
		Degree out of phase	Direction out of phase
$180^\circ$ to $360^\circ$	1/2 - 1	$180^\circ$ to $0^\circ$	counterclockwise
$360^\circ$ to $450^\circ$	1 - 1-1/4	$0^\circ$ to $90^\circ$	clockwise

1. The developmental rotation is always clockwise.
2. This shows the fraction of the rectum around which the ejaculatory duct is looped. The direction is always clockwise.

spect to the rest of the body, showed the normal ejaculatory duct loop as described earlier. Apparently the twisting of the abdomen was superimposed upon the normal rotation. Those males in which the genitalia were out of phase with the tip of the abdomen showed that (1) the ejaculatory duct loop and therefore the developmental rotation is always clockwise, and (2) the loop varies from 1/2 to 1-1/4 times around the rectum and therefore the developmental rotation varies from  $180^\circ$  to  $450^\circ$  (Table 2). Thus, rotation between  $180^\circ$  and  $360^\circ$  gives the appearance of genitalia rotated counterclockwise, while rotation between  $360^\circ$  and  $450^\circ$  gives the appearance of genitalia rotated clockwise.

These results show that a mutant such as twisted<sup>3</sup> or a locus such as the abdomen rotatum locus for which rotation is apparently sometimes in one direction and sometimes in the other can be explained in terms of a single mechanism which alters the normal developmental process. Although the mutants which cause the abdomen or genitalia to be out of phase with the rest of the body are described in the literature as "rotated clockwise" or "rotated counterclockwise", this should be interpreted only in terms of morphological appearance and not as a suggested developmental mechanism.

References: Beatty, R.A. 1949, Proc. Roy. Soc. Edinburgh B 63:249-270; Braver, N.B. 1956, The Mutants of *D. melanogaster* Classified According to Body Parts Affected; Davis, B.K. 1975, Genetics 80:s25; Davis, B.K. 1979, DIS this issue; Fahmy 1959, DIS 33:93-94; Gleichauf, R. 1936, Z. wiss. Zool. 148:1-66; Lindsley, D.L. and D.H. Grell 1968, Genetic Variations of *D. melanogaster*, Carnegie Inst. Wash. Publ. 627; Miller, A. 1950, in: Biology of *Drosophila*, ed. M. Demerec.

Davis, B.K. Virginia Polytechnic Institute and State University, Blacksburg, Virginia. A new twist on an old mutant.

Among a number of male sterile mutants induced on the X chromosome of *D. melanogaster* by ethyl methane sulfonate, was one with a morphological phenotype. The abdomens of both sexes are rotated clockwise, varying from slight to  $90^\circ$ . In this report "clockwise" and "counterclockwise" refer to flies as viewed from the posterior end. Additionally, in males the genitalia and anal plate are often out of alignment with the abdomen, either in a clockwise or counterclockwise direction. This misalignment may be as much as  $180^\circ$ . At the suggestion of Jeffery C. Hall (pers. comm.), the mutant was named "tricky dicky" (td). Some features of the mutant have been reported previously (Davis 1975).

Females of the genotype w m f/y su(wa) td wa were mated to Canton S wild type males and the sons were examined to genetically map the tricky dicky locus. The number and percent of recombinants for various regions of the X chromosome are given in Table 1. The percents of recombinants for y-w, w-m, and m-f are in reasonable agreement with standard map distances (Lindsley and Grell 1968), with allowance for multiple crossing over within the two large

Table 1. Recombinant progeny in selected regions among 1157 total progeny sons from a cross of  $w^m f/y$   $td$   $su(w^a)$   $w^a$  females and Canton S wild type males.

Region	Recombinants	Percent Recombinants
y - td	0	0.0
td - w	15	1.3
y - w	15	1.3
w - m	344	29.7
m - f	229	19.8

(1/26 X 1.5). The  $su(w^a)$  locus is also at 0.1 map units and  $td$  has arbitrarily been placed proximal to  $su(w^a)$  in this discussion.

The map position and phenotype of tricky dicky are quite similar to those of twisted, a mutant which was discovered by Demerec in 1928 (Lindsley and Grell 1968). It is located at 0.4 map units and causes clockwise rotation of the abdomen in both males and females. A second allele,  $tw^2$ , was discovered by Mohr in 1932 (Lindsley and Grell 1968), but has been lost. It caused male genitalia to be out of line in a counterclockwise direction from the twisted abdomens. A complementation test showed that females heterozygous for  $td$  and  $tw$  (obtained from the California Institute of Technology stock center) all have the abdomens out of line with the rest of their bodies. Therefore,  $td$  is in fact an allele of  $tw$  and it is necessary to drop the name tricky dicky. It will henceforth be referred to as  $tw^3$ .

Both alleles cause late hatching. Following outcrossing, the  $tw$  allele showed several new phenotypic features, all with incomplete penetrance. These were etched or absent tergites, leg deformities, pointed wings and small rough eyes.

The new allele  $tw^3$  causes frequent sterility among both males and females. Indeed, as noted above, it was isolated as a male sterile mutant. However, those flies which are not sterile produce as many progeny as do their nonmutant siblings. Both the sterility and the abdominal twist are recessive in females.

The internal anatomy was examined first for 11  $tw^3$  males who failed to produce any progeny with wild type females. They all showed normal reproductive systems including motile sperm. Other  $tw^3$  males, selected on the basis of having the genitalia and anal plate out of line with the abdomen, also had normal reproductive systems in almost all cases. Most of the reproductive systems of a sample of  $tw$  males with etched or absent tergites were also normal. Apparently, the sterility in some individuals results primarily from mechanical difficulties with copulation. Of course, in those males in which the genitalia are out of line with the abdomen, the internal structures are correspondingly displaced. This point is discussed further in a second note (Davis, DIS this issue).

Preliminary tests of chromosome behavior in males showed that both alleles cause a low but significant frequency of nondisjunction. Males hemizygous for  $tw$  but with unmarked fourth chromosomes were crossed to  $C(4)RM, ci\ ey^R/0$  females. The males produced 1.5% nullo-4 sperm (3/204) which is about 37 times the control frequency (0.04% - see Table 3 in Davis 1971). Males hemizygous for  $tw^3$  were mated to  $bb$  attached-X females. These males produced 1.0% (8/798) sperm with neither an X nor Y chromosome, which is about seven times the control frequency (0.14% - see Table 3 in Davis 1971).

Females were tested for meiotic nondisjunction by crossing them to  $Y^S X.Y^L, In(1)En, v f B/0; C(4)RM, ci\ ey^R/0$  tester males (Table 2). Since the fourth chromosomes in the females

regions. This suggests that there are no chromosome rearrangements involving the X chromosome, which was confirmed by polytene chromosome analysis. The tricky dicky locus lies between 0.0 (based on no recombinants with y) and 0.2 (based on 1.3 percent recombinants with w).

A second cross was  $y\ su(w^a)\ td\ w^a/+ +$  females mated to  $y\ su(w^a)\ td\ w^a/Y$  males. Among 3746 male and female progeny were 14  $w^a$ , 11 y  $td$ , and one y, representing 25 recombinants between the tricky dicky and white loci and one recombinant between the tricky dicky and yellow loci. This would place the tricky dicky locus 1/26 of the distance between the yellow and white loci or at approximately 0.1 map units

Table 2. Disjunction data for chromosomes 1 and 4 from females with unmarked fourth chromosomes mated to  $Y^{SX} \cdot Y^L$ ,  $In(1)En$ ,  $v f B/O$ ;  $C(4)RM$ ,  $ci ey^R/O$  males.

Female X Chromosomes	Constitution of Ova Producing Recovered Progeny						Total Progeny	Percent Exceptions	
	X,4+ X,44	XX,4+ XX,44	0,4+ 0,44	X,0 X,0	XX,0 XX,0	0,0 0,0		X	4
y $tw^3$ su( $wa$ ) $wa$ / y $tw^3$ su( $wa$ ) $wa$	8094	2	2	2	1	0	8101	0.06	0.04
tw/tw	2425	4	13	3	1	0	2446	0.74	0.16
tw/y $tw^3$ su( $wa$ ) $wa$	112	0	0	0	0	0	112	0.0	0.0

0.05% nullo-4 exceptions - see Table 1 in Davis 1971). However, homozygous tw females produced exceptions at rates which were 12 times ( $18/2446 = 0.74\%$ ) and 3 times ( $4/2446 = 0.16\%$ ) the control rates for the X and fourth chromosomes respectively. Although these rates of nondisjunction are modest compared to those of meiotic mutants which have been studied, they are significantly higher than control rates.

Females heterozygous for tw and  $tw^3$  were also mated to the tester males. No exceptional progeny were recovered, although only 112 total progeny were scored. Heterozygous females mated to Canton S wild type males produced 231 progeny with no X exceptions. Fourth chromosome exceptions would not have been recognized. The low number of progeny scored prevents firm conclusions, but the data suggest that meiosis in heterozygotes is more like homozygous  $tw^3$  than like homozygous tw.

A total of 335 tw/tw females mated to  $C(3L)RM$ , se h  $rs^2$ ;  $C(3R)RM$ , sbd gl  $e^s$  males produced 7 progeny while 207 tw/tw females mated to  $C(2L)RM$ , dp;  $C(2R)RM$ , px males produced 3 progeny. Since viable euploid zygotes can only result from aneuploid ova, these females are also showing some low frequency of nondisjunction for the large autosomes. Crosses of 57  $tw^3/tw^3$  females to  $C(3L)RM$ , se h  $rs^2$ ;  $C(3R)RM$ , sbd gl  $e^s$  males produced no progeny.

Since tw/tw females show chromosome nondisjunction while  $tw^3/tw^3$  females do not, an attempt was made to separate the morphological and nondisjunctional phenotypes. One generation of free recombination between the chromosome bearing tw and a Canton S wild type X was allowed. Twenty-three recombinant chromosomes with tw and 21 recombinant chromosomes with  $tw^+$  were isolated in male progeny. Stocks were made of all 44 recombinant chromosomes, from which homozygous females were selected and tested for nondisjunction by mating to the same tester males used previously. Overall, the tw lines produced 21 X chromosome exceptions (0.31%) and 3 fourth chromosome exceptions (0.04%) among 6763 progeny while the  $tw^+$  lines produced 29 X chromosome exceptions (0.23%) and 8 fourth chromosome exceptions (0.06%) among 12,824 progeny. Individual lines varied from no exceptions to about 1% exceptions in both groups. Thus the meiotic nondisjunction in females appears to be caused by genes which are independent of the twisted locus.

References: Davis, B.K. 1971, Molec. Gen. Genetics 113:251-272; Davis, B.K. 1975, Genetics 80:s25; Davis, B.K. 1979, DIS this issue; Lindsley, D.L. and E.H. Grell 1968, Genetic Variations of *D. melanogaster*, Carnegie Inst. Wash. Publ. No. 627.

De Salle, R., J.S. Yoon\* and L.H. Throckmorton. University of Chicago, Chicago, Illinois, and \*Bowling Green State University, Bowling Green, Ohio. Karyotypes of two new species of the virilis group of *Drosophila*.

Recently, two new species have been discovered in the virilis group, one from Japan and one from western Canada. Their descriptions will be published shortly (Watabe and Higuchi, in publication; Throckmorton and Yoon, in preparation). A cytological study of them is underway. With respect to karyotypes, it shows the following. The species from Japan has a rod-shaped X, a submetacentric Y, one pair of small metacentrics, three pairs of rods, and a pair of dots. Preliminary analysis of the salivary gland chromosomes identifies the small metacentric as chromosome 2 of earlier workers, and the karyotype of this species may be the hypothetical primitive III of Hsu (1952) or very near to it. Approximately half of the male lar-

were not marked, diplo-4 and mono-4 ova were indistinguishable. Homozygous  $tw^3$  females produced 0.06% X exceptions and 0.04% nullo-4 exceptions, rates which are remarkably close to control rates (0.06% X exceptions and

shaped X, a submetacentric Y, one pair of small metacentrics, three pairs of rods, and a pair of dots. Preliminary analysis of the salivary gland chromosomes identifies the small metacentric as chromosome 2 of earlier workers, and the karyotype of this species may be the hypothetical primitive III of Hsu (1952) or very near to it. Approximately half of the male lar-

vae of our stock carry an extra Y chromosome, with the two Y's identical to each other in all respects. The females may also carry a supernumerary Y, but that condition occurs less frequently than in the males.

The species from western Canada proves to have a rod-shaped X, a submetacentric Y, one pair of small metacentrics, one large metacentric pair, one pair of larger rods and one pair of small rods. With the exception of the small rods in place of dots, this karyotype is very similar to that published for the European *D. littoralis*. We are grateful to Dr. E. Momma for providing us with the stock of the new Japanese species and to the National Drosophila Species Resource Center, the University of Texas, for providing us with a strain of the new species from Canada. This work was supported in part by National Institute of Health grant GM 23007 to L. H. Throckmorton.

References: Hsu, T.C. 1952, Univ. of Texas Publ. 5204:35-72; Watabe, H. and C. Higuchi (in publication), submitted to Annot. Zool. Japan. (2); Throckmorton, L.H. and J.S. Yoon (in preparation).

Deweese, A.A. Sam Houston State University, Huntsville, Texas. Lethal-bearing genomes from a Texas population of *D. melanogaster*.

Second and third chromosomes were sampled directly from a natural population near Huntsville, Texas, and analyzed simultaneously for the presence of lethal genes. The  $A_1B_{18}$  marker stock, supplied by Dr. Bruce Wallace, was used to produce flies isogenic for both second and

third chromosomes (Wallace, Zouros and Kimbras 1966). This stock contains two reciprocal translocations, designated (Cy L; Ubx)/(Pm; Sb), and allows second and third chromosomes to be handled simultaneously. The mating scheme was initiated by crossing single wild-caught males to virgin (Cy L; Ubx)/(Pm; Sb) females in shell vials. From each parental mating a single  $F_1$  male (Cy L; Ubx)/(+;+) was mated with a (Cy L; Ubx)/(Cy; Pm) female. Virgin  $F_2$  (Cy L; Ubx)/(+;+) brothers and sisters were mated in half pint bottles to produce an  $F_3$  generation (in some  $F_2$  matings (Pm; Sb)/(+;+) flies were used). The expected proportion of viable  $F_3$  flies, assuming lethal-free wild type chromosomes, is 2 (Cy L; Ubx)/(+;+): 1 +/+;+/.

Second and third chromosomes, "genomes", from a total of 78 wild-caught males were carried through to the  $F_3$  generation. An average of 105  $F_3$  flies was examined for the 78 genomes. The distribution of genomes into viability classes is presented in Table 1 for each of two collecting periods. The results from a 2x4 contingency chi-square analysis ( $\chi^2_3 = 0.42$ ;  $0.9 < P < 0.975$ ) indicate no difference in distributions over the two months. The total percentage of lethal plus semilethal genomes was 89.7% (70/78), based on pooling the three lowest viability classes. These results are similar (2x2 contingency  $\chi^2_1 = 3.4$ ;  $0.05 < P < 0.1$ ) to genome lethal plus semilethal frequencies reported by Wallace et al. (1966). Using the same balancer stock for the detection of viability differences in a Bogota, Colombia, population, they reported 79.8% (95/119) lethal plus semilethal genomes. Although no effort was made in the present study to localize the lethal effects of a genome to the individual chromosomes, some predictions can be made based on the findings of Wallace et al. (1966). They reported that their genome lethals were nearly equally distributed between second and third chromosomes. Band and Ives (1963) also reported similar lethal plus semilethal frequencies for these two chromosomes tested separately. Under the assumption of equal chromosome frequencies, lethal plus semilethal frequencies in the Huntsville, Texas, population can be individually determined for the second and third chromosomes as follows: Let  $p$  = frequency of lethal plus semilethal chromosomes; then  $(1-p)^2$  = expected frequency of genomes having both second and third chromosomes free of lethal and semilethal genes. Eight of 78 genomes were in this category; therefore, the estimate of  $p$  is  $1 - \sqrt{8/78} = 0.68$ .

Ives (1945) reported second chromosome lethal plus semilethal frequencies from 34% to 67% for eastern U.S. populations of *D. melanogaster*, with the highest frequencies obtained for Florida populations. The southeast Texas population sampled in the present study appears to be quite similar in its frequency of lethal plus semilethal chromosomes to Florida and Colombia populations.

Table 1. Observed (and expected) numbers of genomes in different viability classes.

Collecting Period	Relative Frequency of Wild-Type $F_3$ Flies				Total Number Genomes
	0.0	0.01-0.083	0.084-0.166	0.167-0.333	
May 1977	22 (23.2)	9 (8.8)	7 (6.6)	5 (4.4)	43
June 1977	20 (18.8)	7 (7.2)	5 (5.4)	3 (3.6)	35
Totals	42	16	12	8	78

References: Band, H.T. and P.T. Ives 1963, Can. J. Genet. Cytol. 5:351-357; Ives, P.T. 1945, Genetics 30:167-196; Wallace, B., E. Zouros and C.B. Kimbras 1966, Am. Naturalist 100: 245-251.

Diamantopoulou-Panopoulou, E. Agricultural College of Athens, Votanicos, Greece. Estimation of  $N_{em}$  by allelism method in *D. subobscura*.

In an attempt to estimate the effective population size,  $N_e$ , by the allelism method (Wright, Dobzhansky and Howanitz 1942) the parameters  $Q$ ,  $P$ , and  $P_{\infty}$  have been determined in two Greek natural populations of *D. subobscura* from Mt. Parnes and Crete. ( $Q$  = frequency of lethal chromosomes;  $P$  and  $P_{\infty}$  = frequencies of lethal chromosomes in and between populations.)

For Parnes 145 and 218 O chromosomes, collected at different times, were analyzed by the  $Va\ ch\ cu/Ba\ O_{3+4+8}/O_{ST}$  balanced strain, and for Crete 150 and 261, respectively. The frequency  $Q$  had no significant difference in two samples for each population, so they have been considered as one.

Table 1. Estimation of  $Q$  in Crete-Parnes population.

Population	Total $Q$	Corrected $Q$	
		$O_{3+4}$	$O_{ST} \ \& \ O_{3+4+8}$
Crete	$0.187 \pm 0.032$	$0.136 \pm 0.073$	$0.242 \pm 0.037$
Parnes	$0.248 \pm 0.036$	<u><math>0.284 \pm 0.042</math></u>	<u><math>0.414 \pm 0.073</math></u>

The underlined data were considered as more representative of each population.

with reference to O inversion (Tables 1, 2, 3). By some relations of the method, it was possible to estimate the parameters:  $p$ , frequency of allelism of lethal genes' O chromosomes in a population;  $p_{\infty}$ , frequency of allelism of lethal genes' O chromosomes between two populations;  $n$ , number of genes subject to lethal mutation. Then the quantity  $N_{em}$  was estimated, where  $m$  = migration rate (Table 4).

The two populations differ at O chromosome inversions. The  $O_{3+4}$  and  $O_{3+4+\phi}$  (where  $\phi = 1, 2, 2, 7$ ) are the most common in the Parnes population, while in Crete the most common is the  $O_{3+4+8}$  inversion. Because the balanced strain (it has been analyzed) does not cover the O chromosome near the  $O_{3+4}$  end, all the estimated frequencies were corrected

Table 2. Estimation of  $P$  in Crete-Parnes population.

	$O_{3+4} \times O_{3+4}$		$O_{3+4} \times \begin{Bmatrix} O_{ST} \\ O_{3+4+8} \end{Bmatrix}$		$O_{ST} \times \begin{Bmatrix} O_{ST} \\ O_{3+4+8} \end{Bmatrix}$		Total	
	Crete	Parnes	Crete	Parnes	Crete	Parnes	Crete	Parnes
total no. of crosses	3	138	71	67	284	5	358	210
no. allelic crosses	0	2	0	0	2	0	2	2
allelism frequency	0	$0.0145 \pm 0.0102$			$0.0070 \pm 0.0049$		$0.0054 \pm 0.0038$	$0.0095 \pm 0.0069$

Table 3. Estimation of  $P_{\infty}$  between Crete-Parnes populations.

	$\begin{matrix} 0_{ST} \\ 0_{3+4+8} \end{matrix} \times \begin{matrix} 0_{ST} \\ 0_{3+4+8} \end{matrix}$	$\begin{matrix} 0_{3+4} \\ 0_{3+4+8} \end{matrix} \times \begin{matrix} 0_{ST} \\ 0_{3+4+8} \end{matrix}$	$0_{3+4} \times 0_{3+4}$	Total
total no. of crosses	174	913	417	1553
no. allelic crosses	1	2	0	3
allelism frequency	$0.00575 \pm 0.00573$			$0.0019 \pm 0.0011$

Table 4. Estimation of Q, P,  $P_{\infty}$ , p,  $p_{\infty}$ , n and  $N_{em}$  in Crete-Parnes population.

		Q	P	$P_{\infty}$	p	$p_{\infty}$	n	$N_{em}$
Crete	uncorrected Q, P, $P_{\infty}$	0.187	0.00540	0.00190	0.00505	0.00155	645	344
	corrected Q, P, $P_{\infty}$	0.242	0.00704	0.00575	0.00568	0.00439	228	573
	corrected Q, P	0.242	0.00704	0.00190	0.00659	0.00145	689	175
Parnes	uncorrected Q, P, $P_{\infty}$	0.248	0.00950	0.00190	0.00904	0.00144	694	115
	corrected Q, P	0.284	0.01449	0.00190	0.01396	0.00137	730	59

Diamantopoulou-Panopoulou, E. and H. Bacoulas. Agricultural College of Athens, Votanicos, Greece. "Sex ratio" in *D. obscura*.

One isofemale line of *D. obscura* from a Greek natural population (Mt. Parnes) produced offspring of only female sex; this continued for many generations (the male parent was taken from an *obscura* stock). A treatment was undertaken to clarify if this condition was similar

to that of "sex ratio" in *D. bifasciata*. After penicillin G was given "per os" for one or two generations, the culture produced both sexes (males and females vs. females only) progressively to fifty-fifty percent. After enough time the culture began to produce again only female flies.

An attempt to find the causal factor, spirochaete in the haemolymph of the female fly, gave no results.

Doane, W.W. Arizona State University, Tempe, Arizona. Midgut amylase activity patterns in *Drosophila*: nomenclature.

A control gene for tissue specific expression of  $\alpha$ -amylase in the adult posterior midgut (PMG) in *D. melanogaster* was located at 2-80 $\pm$  by Abraham and Doane (1976, 1978). This gene, called map for midgut activity pattern, lies approxi-

mately two crossover units to the right of the structural gene(s) for the enzyme (Amy). Strain specific differences in the regional expression of amylase in the PMG were attributed to allelic differences at the map locus. Three spatially different PMG patterns were found in an initial survey of isogenic laboratory strains. These patterns, which reflect the cellular dis-

tribution of amylase, were designated A, B and C (top three patterns in Fig. 1); they apparently correlate with alleles  $\text{map}^A$ ,  $\text{map}^B$  and  $\text{map}^C$ , respectively. While clear-cut recombination data exist for  $\text{map}^A$  and  $\text{map}^C$ , an exact definition of the postulated  $\text{map}^B$  allele has not yet been achieved. Recombination data suggesting the existence of a  $\text{map}^B$  allele (Abraham and Doane, unpublished) are somewhat ambiguous because of overlapping phenotypes between heterozygotes bearing the so-called  $\text{map}^B$  allele and homozygous classes for it and other  $\text{map}$  alleles. An attempt is being made to resolve this situation.

Subsequent to the initial survey which revealed the above three PMG patterns, a search was made (Doane 1977a, b) for variability in the expression of amylase activity in the anterior midgut (AMG). (The highly acidic middle midgut region does not display activity.) Included in the search were 7 laboratory "wild" strains received from Dr. Bruce Wallace; these had been maintained at Cornell University for varying lengths of time up to 20 years. The strains derived from flies collected around the world, including the following localities: Capetown (CA 1), Chile (SC 1), Kentucky (4 B), New York (1 A, 1 B), South Africa (KSA 2), and Spain-France (PYR 3). An additional 49 newly established strains from single females collected in Puerto Rico in February, 1976, were provided by Dr. Donald F. Poulson of Yale University. The array of midgut activity patterns displayed by adults from these "wild" strains is diagrammed in Fig. 1.

A total of 15 different patterns is shown in Fig. 1, including five different AMG patterns. The three PMG patterns originally described were again found, but in various combinations with the AMG patterns. As pointed out by Abraham and Doane (1978), the AMG may be subdivided into three activity regions and the PMG into two such regions. The different patterns reflect these subdivisions, the precise boundaries of which remain to be clarified. Polymorphism for midgut activity patterns was common among the various strains surveyed, especially those from Cornell. All strains displayed  $\text{Amy}^1$  phenotypes for the structural gene region, except the SC 1 strain (=  $\text{Amy}^{2,3}$ ).

Because of the regional variability in expression of amylase activity in adult flies, a system of nomenclature for midgut patterns was developed, based on known phenotypes (Doane 1978). That system is presented in Fig. 1. Phenotypic classes are listed on the left. The three subdivisions in the AMG are numbered 1, 2 and 3 in an anterior-posterior direction. A strain with activity in all three subdivisions is classified as AMG-123, while another with activity in the first two subdivisions but little or none in the third subdivision is typed as AMG-120. "0" thus indicates no activity or almost no activity on the basis of the whole-mount starch-iodine technique used to prepare the patterns (Abraham and Doane 1978). The numerals 1, 2 and 3 indicate not only well defined amylase activity but the regional location of that activity as well. Five different AMG classes are so defined: AMG-123, AMG-120, AMG-103, AMG-100 and AMG-000. The PMG displays only two potentially active subdivisions according to this system of nomenclature. Three patterns were found: PMG-12, PMG-10 and PMG-00. These classes correspond to the A-, B- and C-patterns, respectively, of Abraham and Doane. The three PMG patterns were found in all possible combinations with the five AMG patterns, accounting for the total array of 15 adult patterns in the above survey. It seems likely that additional AMG patterns may still be found in *D. melanogaster*, such as AMG-023 or AMG-003, unless some unknown developmental restriction limits their appearance.

In most isogenic strains examined, the larval midgut pattern differs from that of the adult for a given strain. Nevertheless a nomenclature similar to that in Fig. 1 may be used for larval patterns as well, although the relative proportions for larval patterns differ somewhat from those of adults. In *D. melanogaster*, the most common larval pattern is AMG-003, PMG-12 among strains surveyed (e.g., see Doane 1969). The nomenclature in Fig. 1 may also be used for other species of *Drosophila*, c.f. *D. hydei* (Doane 1969) and *D. pseudoobscura* (Powell and Lichtenfels 1979).

Care must be taken in classifying strains according to their adult patterns because of an age-dependent "switch" which may occur in regions designated as having little or no activity in young flies. These "0" regions typically become fully active in older flies (Doane, unpublished). Thus, in *D. melanogaster*, the AMG-123, PMG-00 pattern (= "C-pattern") changes to the AMG-123, PMG-12 pattern ("A-pattern") at approximately 14 days of age in adult females. This change-over at about two weeks of age is typical for most of the isogenic strains of *D. melanogaster* examined, but not all. The time of the "switch" in amylase expression is strain-specific, some strains showing it earlier in adult life than others (e.g., 3-4 days or 7-8 days). In an isogenic line derived from the Cornell CA 1 strain, the "switch" does not seem to occur. Here, flies as old as 21 days retain the AMG-000, PMG-00 pattern; midgut preparations beyond that age are technically not feasible to make. The strain-specific "switch"



lends further credence to the hypothesis (Abraham and Doane 1978) that the midgut amylase activity patterns may be controlled by "temporal genes". A similar age-dependent transition in midgut patterns was independently described for *D. pseudoobscura* by Powell and Lichtenfels (1979) who noted that the exact time of the change-over is temperature dependent. No strain-specific differences in the age of onset of the transition were reported thus far in that species.

At least two control loci appear to control the midgut activity patterns in adults of *D. melanogaster*. The original map locus for the PMG has now been distinguished by recombination analysis from another locus which controls the AMG (Doane, unpublished; Table 1). Accordingly, the genetic symbols indicated on the right side of Fig. 1 are proposed. The first map gene described may now be called map-PMG with superscript numbers for alleles complementing the phenotypic classes, i.e., map-PMG<sup>12</sup> (= map<sup>A</sup>), map-PMG<sup>10</sup> (= map<sup>B</sup>), and map-PMG<sup>00</sup> (= map<sup>C</sup>). The second locus, map-AMG, is tentatively placed to the right of map-PMG on chromosome 2R, based on the preliminary data shown in Table 1. Recombination data exist for those genotypes indicated by an asterisk in Fig. 1. All other proposed genotypes have been deduced from these data and the 15 adult midgut phenotypes diagrammed and, accordingly, must be considered speculative until additional data are available.

Figure 1. Diagram of adult midgut amylase activity patterns found among isogenic and "wild" strains of *D. melanogaster*. Each horizontal line represents a midgut extending from its anterior or cardia end (left) to its posterior end (right) where Malpighian tubules attach. Thickened, black regions within the anterior midgut (AMG) and posterior midgut (PMG) are subdivisions expressing amylase activity. Other regions display little or no activity in young flies (including the MMG). Phenotypic classes are listed on the left, with corresponding homozygous genotypes on the right. Asterisks indicate which of the proposed genotypic symbols are supported by genetic data. The linear order of the two loci is not implied.

Phenotypic Class	AMG			MMG	PMG		Proposed Genotype
	1	2	3		1	2	
AMG-123, PMG-12	■	■	■		■	■	map-AMG <sup>123</sup> map-PMG <sup>12</sup> *
AMG-123, PMG-10	■	■	■		■		map-AMG <sup>123</sup> map-PMG <sup>10</sup> *
AMG-123, PMG-00	■	■	■				map-AMG <sup>123</sup> map-PMG <sup>00</sup> *
AMG-103, PMG-12	■		■		■	■	map-AMG <sup>103</sup> map-PMG <sup>12</sup>
AMG-103, PMG-10	■		■		■		map-AMG <sup>103</sup> map-PMG <sup>10</sup>
AMG-103, PMG-00	■		■				map-AMG <sup>103</sup> map-PMG <sup>00</sup>
AMG-120, PMG-12	■	■			■	■	map-AMG <sup>120</sup> map-PMG <sup>12</sup>
AMG-120, PMG-10	■	■			■		map-AMG <sup>120</sup> map-PMG <sup>10</sup>
AMG-120, PMG-00	■	■					map-AMG <sup>120</sup> map-PMG <sup>00</sup>
AMG-100, PMG-12	■				■	■	map-AMG <sup>100</sup> map-PMG <sup>12</sup>
AMG-100, PMG-10	■				■		map-AMG <sup>100</sup> map-PMG <sup>10</sup>
AMG-100, PMG-00	■						map-AMG <sup>100</sup> map-PMG <sup>00</sup>
AMG-000, PMG-12					■	■	map-AMG <sup>000</sup> map-PMG <sup>12</sup>
AMG-000, PMG-10					■		map-AMG <sup>000</sup> map-PMG <sup>10</sup>
AMG-000, PMG-00							map-AMG <sup>000</sup> map-PMG <sup>00</sup> *



Table 1. Recombination analysis revealing two separable loci, *map-AMG* and *map-PMG*, which control the tissue-specific expression of amylase in the adult AMG and PMG, respectively, in *D. melanogaster*.

$  \begin{array}{c}  \text{Amy}^1 \text{ map-AMG}^{000} \text{ map-PMG}^{00} \text{ ♀} \times \text{Amy}^{1,6} \text{ map-AMG}^{123} \text{ map-PMG}^{12} \text{ ♂} \\  (= \text{iso-CA 1}) \qquad \qquad \qquad (= \text{Amy}^{1,6} \text{ map}^A) \\  \downarrow \\  F_1 \text{ ♀} \times \text{Amy}^1 \text{ map-AMG}^{000} \text{ map-PMG}^{00} \text{ ♂} \\  \downarrow \\  \text{Female Progeny}  \end{array}  $			
	Amylase	Midgut Pattern	Number (N = 215)
Parental:	1,6	AMG-123, PMG-12	106
	1	AMG-000, PMG-00	102
Recombinant:	1,6	AMG-000, PMG-00	1
	1	AMG-123, PMG-12	2
	1,6	AMG-000, PMG-12	1
	1	AMG-123, PMG-00	3

Amy	map-PMG	map-AMG

This work was supported by NIH grants GM-18729 and GM-25255; it was mostly carried out at Yale University. I wish to thank Drs. Wallace and Poulson for the strains surveyed and Dr. J. R. Powell for providing a preprint of the paper on *D. pseudoobscura*.

References: Abraham, I. and W.W. Doane 1976, *Genetics* 83:sl; Abraham, I. and W.W. Doane 1978, *Proc. Natl. Acad. Sci. USA* 75:4446; Doane, W.W. 1969, in *RNA in Development*, E.W. Hanley, ed., p. 73, Univ. Utah Press, Salt Lake City; Doane, W.W. 1977a, *Genetics* 86:sl5; Doane, W.W. 1977b, *J. Cell Biol.* 75(2, pt. 2):147a; Doane, W.W. 1978, *Int. Conf. Mol. & Devel. Biol.* Insects, Crete, Abstracts, p. 45; Powell, J.R. and J.M. Lichtenfels 1979, *Genetics*, in press.

Espinet, S. and M.L. Tracey. Florida International University, Miami. Detection of differences in the element patterning of *D. melanogaster* mating behavior.

The difficulties inherent in quantitatively measuring differences in the behavior of *Drosophila* are well known (Bastock and Manning 1955; Bastock 1956). However, careful description of behaviors, including an account of their patterning in time permits quantification

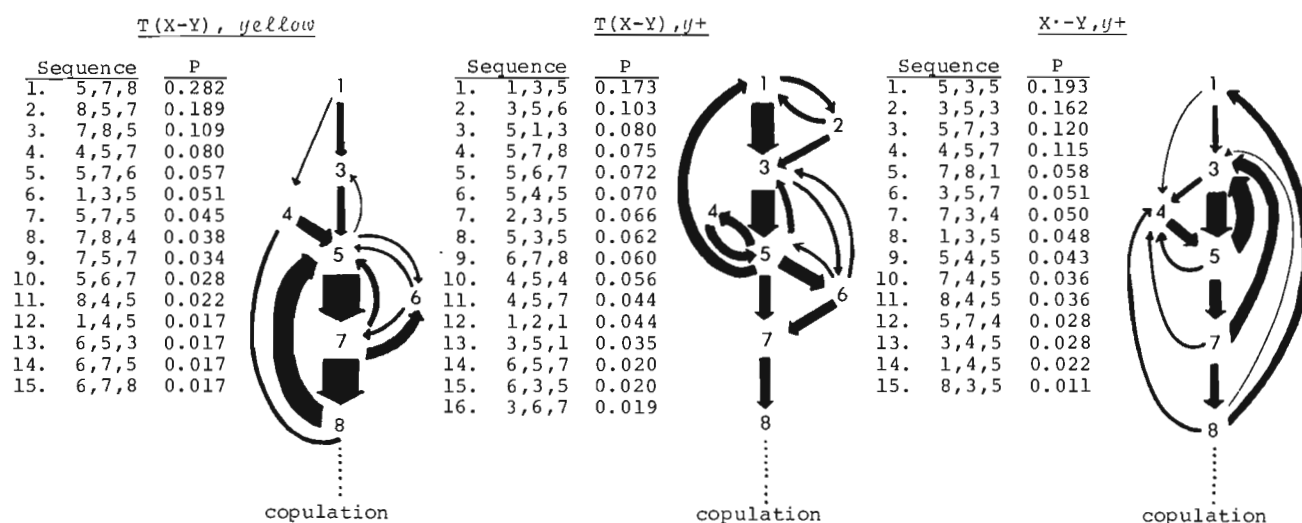
of within and between strain variation. We report a technique in which all possible triplets of behavioral elements which occur at frequencies greater than 1% are graphically overlain to produce a standard sequence. Comparisons of graphical representations indicate both inter-population and intrapopulation variation.

X.Y<sup>L</sup>/Y<sup>Sy</sup>+ males outcompete X.Y<sup>L</sup>/Y<sup>S</sup> yellow males in female choice experiments where all females are X.Y<sup>L</sup>/X.Y<sup>L</sup> (P<0.001); and X.Y<sup>L</sup>+/Y<sup>S</sup> males outcompete X./Y males (Tracey and Espinet 1976). To test the hypothesis that the observed differences reflect alterations in the sequence and frequency of courtship elements, pairs of flies were observed and courtship elements were recorded. Recordings were made with a Bell telephone push-button key board coupled

to a 20-pen Esterline-Angus event recorder at chart speeds of 3.8-4.0 cm per minute. Twenty elements were recorded, but some were eliminated by the 1% triplet criterion employed. Those involved in the final analysis were: (1) male standing approximately 1 mm directly behind female, (2) female preening, (3) male scissoring, (4) male following female, (5) male vibrating, (6) male circling female, (7) male licking female's genitalia, (8) male abdomen curling or attempted copulation. Note that successful mating did not always follow attempted mating; compare T(X-Y),yellow and T(X-Y),y+ in Fig. 1. Courtship behavior was identified as the sequence of elements preceding successful copulation. Thus a shift from more or less passive behaviors such as walking to active alteration was used to define the onset of courtship.

The frequencies of all possible combinations of triplets of behavioral elements were calculated. Triplets of elements that occurred at a frequency less than 0.01 were eliminated from the analysis for each trial. The normalized probability for each triplet of elements in a trial was obtained by setting the sum of the frequencies of each element triplet observed more often than one in 100 equal to 100%. Since only two of the recording sessions involving Xy+//Y males terminated in successful copulation two trials of recordings from each of the

Figure 1. Temporal pattern and element triplet performance probabilities of courtship behaviour in T(X-Y),yellow, T(X-Y),y+ and X-Y,y+ *Drosophila melanogaster* stocks. Behavioural elements are represented numerically (see text for descriptions); sequences of triplets are listed in the first column of each study, followed by their associated probability of performance (P). Triplets having observed frequencies of less than 0.01 are not included. Diagrams of the probable sequences of performance of courtship behaviour elements for each pair of flies are presented. The probability that one behavioural element is followed by another is indicated by the width of the arrows.



three males were analyzed and the within-strain data pooled to obtain an average probability of performance for each triplet of elements. The results are presented in Fig. 1.

The standard sequence of behavior in X-Y,y+ males is 1-3-5-7-8. In the event the "next" element is not elicited at any point in the sequence, interruption results in the individual starting again at the initial elements (1, 3 or 4) of the sequence. The triplet sequence 3-5-7 is most frequent and perhaps most important in the behavioral repertoire of these males. It should be noted that circling (6) does not play an important role in the mating behavior of these males (Manning 1955).

In T(X-Y)y+, circling plays a more significant role; 1-3-5-6-7-8 is the standard sequence in this case. The initial elements of the sequence are more frequently elicited when these males are wild body-colored, and female preening (2) appears to stimulate male scissoring. When males are yellow, male scissoring is not affected by element 2, and circling (6) does not fit as neatly into the standard sequence as it does in y+ bearing males. The standard sequence in the case of T(X-Y) yellow males is 1-3-5-7-8 with the subset 5-7-8 being most frequently elicited.

These results are consistent with the threshold theory of behavioral elicitation, where each behavioral element in the sequence has a higher threshold than the element directly preceding it. In the event this threshold level of stimulation is not achieved the particular behavioral element is not elicited; rather a preceding element with a lower threshold appears in the sequence (Bastock 1956).

References: Bastock, M. and A. Manning 1955, Behavior 8:7-111; Bastock, M. 1956, Evolution 10:84-92; Manning, A. 1965, Viewpoints in Biology 4:125-169; Tracey, M.L. and S.A. Espinet 1976, Nature 263:321-323.

Fadda, S., S. Sangiorgi and E. Pieragostini. Istituto di Genetica, Università di Bologna, Italy. G6PD electrophoretic phenotype variation during development of *D. melanogaster* laboratory populations.

Developmental pattern of G6PD isozymes in *D. melanogaster* was investigated from egg deposition up to the 48th hour by Wright and Shaw (1970). When they crossed parents exhibiting different electromorphs,  $F_1$  embryos showed both maternal and paternal forms of the enzyme by the 23rd hour; they concluded that genetic and mole-

cular models, proposed for adult G6PD (Young et al. 1964; Steele et al. 1968), could hold also for the developmental expression of the enzyme.

This does not seem to be the case in our lab populations: when investigated through electrophoresis of mass homogenates, they exhibited different electrophoretic phenotypes, that did not reflect adult variation nor were inherited following any simple Mendelian scheme (Pieragostini et al. 1978; Fadda et al. 1979).

These observations agree with a good deal of evidence about complex determination of G6PD in *D. melanogaster* adults of our populations (Pieragostini et al. 1978) and others studied by several authors (Komma 1968; Giesel 1976). With the present communication we complete the picture of G6PD developmental polymorphism through electrophoretic observations of two day old pupae and discuss them in relation to larval and adult stages.

We sampled pupae from Canton strain, from an unrelated strain carrying the vg marker and from six populations having a Canton x vg cross as their common origin, plateaued for a quantitative trait after about 70 generations of selection (Palenzona and Alicchio 1973). We maintained these populations in mass culture at 25°C for several generations and then took random samples of about 100 individuals in the proper developmental phase. Cello-gel electrophoresis was carried out on multiple homogenates of the above samples, following the procedure detailed in Pieragostini et al. 1978. We also calculated experimental errors affecting relative mobilities in order to base our homology statements upon statistical testing. For all cases where we suspected the existence of mobility differences, we examined electrophoretically mixed samples and took single band patterns as evidence of homology.

The results obtained analyzing electrophoretically our lab populations are presented in Fig. 1, which summarizes published observations of adult samples (from Pieragostini et al. 1978), of larval samples (Fadda et al. 1979) and original data from pupal samples. In general, we may point out that both larval and adult stage exhibit several differences between populations, while pupal stage has a single variant common to all populations (Fig. 2); however, the pupal electromorph is slower than any other observed variant.

For larval stage in particular it is worth noticing that the variants from parental populations (Canton and vg) disappear in the progeny (selected lines), where bands of intermediate mobility are present; parental variants of adult stage behave differently, because they are maintained in the progeny and are rearranged in patterns typical of vg and winged populations. Since genetic analyses, performed on each of the two stages separately (Fadda et al. 1979 for larvae; Pieragostini et al. 1978 for adult flies), provided evidence that these variants are not inherited in simple Mendelian fashion, the authors suggested regulatory hypotheses for these phenomena. However, as these phenomena exhibit no similarities in larval and adult stages, we might add to the regulatory hypothesis that the mechanisms controlling the expression of adult and larval variants depend on the specific developmental stage, whether the structural genes are the same or not.

As for the pupae, they seem to differ "non-specifically", that is, depending on the very developmental stage rather than on the genetic complement of each population. We suggest the expression of pupal G6PD to depend on the physiological state, either because the electromorph actually has a peculiar function in pupal metabolism, or because it is modified as a metabolic side effect. Examples of such epigenetic developmental variation, due to modifiers present in

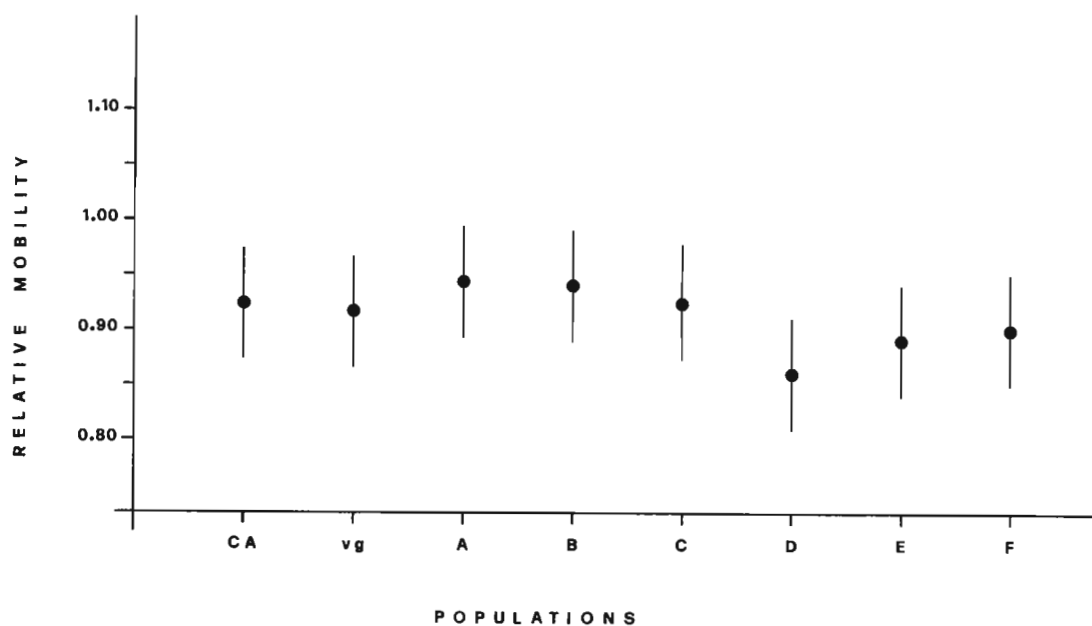


Fig. 1. Summary of polymorphic patterns for G6PD throughout development of *D. melanogaster* populations. Bands are listed in order of decreasing electrophoretic mobility at distances proportional to the real ones. Horizontally in the diagram we list variant populations or population groups: for larval and adult stages we represent, from left to right, Canton and vestigial parental strains (Ca and vg), winged selected lines ( $vg^+L$ ) and vestigial selected lines ( $vgL$ ) mobilities; for pupal stage we represent the only variant common to all populations.

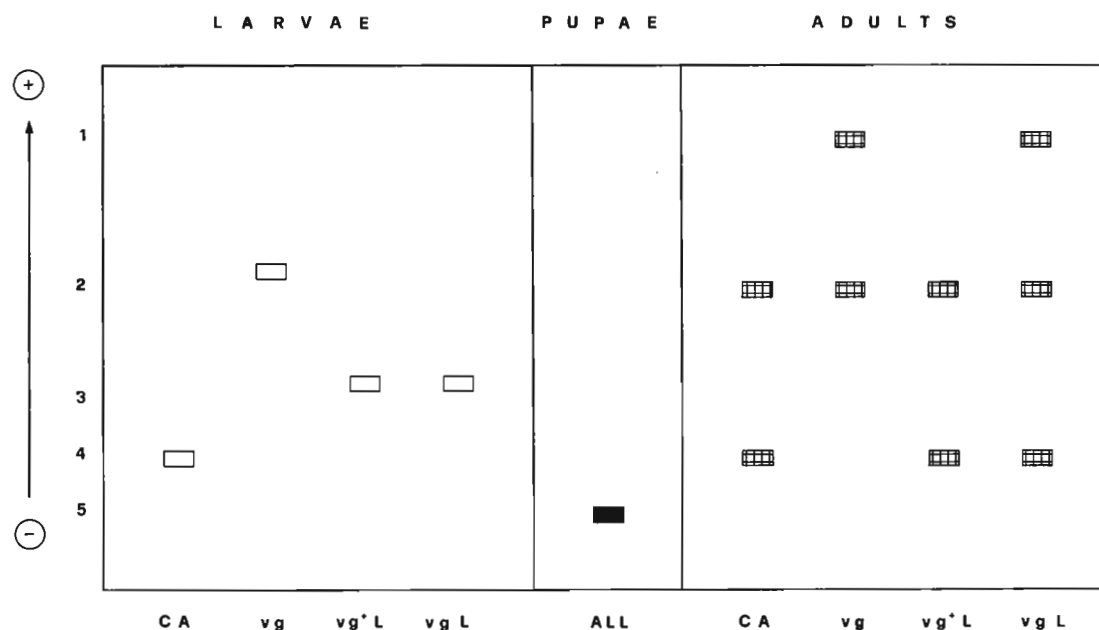


Fig. 2. Relative mobilities of two day old pupae from our 8 populations. From left to right: Canton strain (Ca), vg strain (vg),  $PMvg^+$ ,  $PFvg^+$ ,  $PKvg^+$  winged selection lines (indicated by A, B, C) and  $PMvg$ ,  $PFvg$ ,  $PKvg$  vestigial selection lines (indicated by D, E, F); mobilities, which were averaged over 5 determinations per population, are reported with their "t95" confidence intervals.

crude extracts, are reported for other gene-enzyme systems, such as  $\alpha$ -GPDH (Bewley and Lucchesi 1977) and alkaline phosphatase (Schneidermann 1967; Wallis and Fox 1968).

Experiments are in progress to detect post-translational modifiers, if any; as a preliminary test, we mixed crude extract of larvae or adults to pupal extracts in vitro: upon electrophoresis, the components migrated independently and formed bands of dissimilar mobility. No modifiers seemed to be present; however, these observations do not provide a definite answer, because we cannot be sure that our homogenization procedure simulates adequately the in vivo situation.

In any case, the determination of these isozymes throughout development seems to be very complex and the Mendelian models proposed for adult and embryo G6PD do not apply to our populations. These experiments emphasize how dangerous it is to generalize structural models to whole species on the basis of electrophoretic observations. G6PD gene enzyme system throughout development reveals a remarkable polymorphism of regulatory origin mainly (Steele et al. 1969; Komma 1968; Giesel 1976; Pieragostini 1978; Fadda et al. 1979); in our opinion, such systems deserve to be studied in further detail because they can draw more attention to the importance and the evolutionary significance of regulatory variation in respect to structural one.

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Fleuriet, A. University of Clermont-Ferrand II, France. Analysis of a polymorphism quite common in French natural populations of *Drosophila melanogaster*.

From a survey made since 1969, it has been established that French natural populations of *Drosophila melanogaster* are polymorphic for two features. First of all, 10 to 20% of the flies are infected by a Rhabdovirus called "sigma".

It has been known for years that this virus is not contagious but transmitted from fly to fly only through gametes and is responsible for CO<sub>2</sub> sensitivity of infected flies. This situation is presently arousing more interest since the discovery that some pathogenous viruses of vertebrates are transmitted transovarially in their insect vectors. When experimental populations of flies are raised in cages, the sigma virus usually infects most of the individuals. Further experiments are now being performed to explain the discrepancy between natural and experimental populations.

A second feature, very constant at least in French populations, is a polymorphism for two alleles of a gene for resistance to the sigma virus: *ref(2)P<sup>0</sup>* and *ref(2)P<sup>P</sup>*. The respective frequencies of these two alleles are very similar among all the populations studied and they are quite the same in experimental populations, whether the sigma virus is present or not. The strong selective forces working on this equilibrium are now being analyzed.

From a few other observations, it seems that these two features may, at least, exist in populations of flies living in other countries.

References: Fleuriet, A. 1976, *Evolution* 30:735-739; Fleuriet, A. 1978, *Genetics* 88: 755-759.

Fontdevila, A\*, W.T. Starmer, W.B. Heed and J.S. Russell. \*Universidad de Santiago, Santiago de Compostela, Spain, and University of Arizona, Tucson, Arizona. Differential mating activity in two co-existing species of *Drosophila*.

Ecologists have disclosed many cases of character displacement as a means to avoid species competition (see Margalef 1974 for a revision). This seems particularly true among closely related species occurring together and less so when species are genetically different. However, under certain conditions where convergence of non-related species is favored by natural selection, character displacement may be established. The present work provides a new example of

tion, character displacement may be established. The present work provides a new example of

behavioral character displacement between two coexisting desert species which are very similar in morphology, but not closely related phylogenetically.

*D. nigrospiracula* inhabits the Sonoran Desert, which extends in the USA through southern Arizona and in Mexico through Baja California and Sonora. Its biology is well understood (Fellows and Heed 1972). For example, it is known to be an oligophagous species feeding on several cacti, but utilizes the Saguaro cactus (*Carnegiea gigantea*) in the region of Tucson, Arizona, where this study was done. Occupying the same distribution area, there is a species (*Drosophila mettleri*) that resembles *D. nigrospiracula* very closely. Both species differ externally in male genitalia and in the pattern of bristles on their frons, being almost equal in size, shape and morphology. Most interesting are their breeding niche differences; *D. nigrospiracula* larvae feed upon the necrotic tissue of the cactus, while *D. mettleri* larvae feed upon the soil flooded with the juices of the necrotic tissue. Adults of both species feed upon liquid exudates at the cactus surface. This is the second case of such a fine separation in breeding sites between two species of *Drosophila*. Kaneshiro et al. (1973) have described a similar situation in two Hawaiian *Drosophila* which are closely related.

These desert species of *Drosophila* can be very abundant on the Saguaro cacti and provide the unique opportunity of collecting in their natural habitat a sufficient number of mating pairs to make up a workable sample to study. During two consecutive days we aspirated a total of 321 mating pairs, of which 277 were *D. nigrospiracula* matings and the remainder matings of the other species. A mating pair was aspirated as soon as actual copulation started, the time was recorded and each pair was kept in an individual vial for later species identification.

The results of investigating mating activity throughout the day are shown in Fig. 1. At this time of the year (November) *D. nigrospiracula* shows a bimodal distribution of mating activity with one peak at about 11 a.m. and a second peak at about 4 p.m. Obviously, those periods correspond to two moments of the day when the temperature is optimal (approximately 16°C)

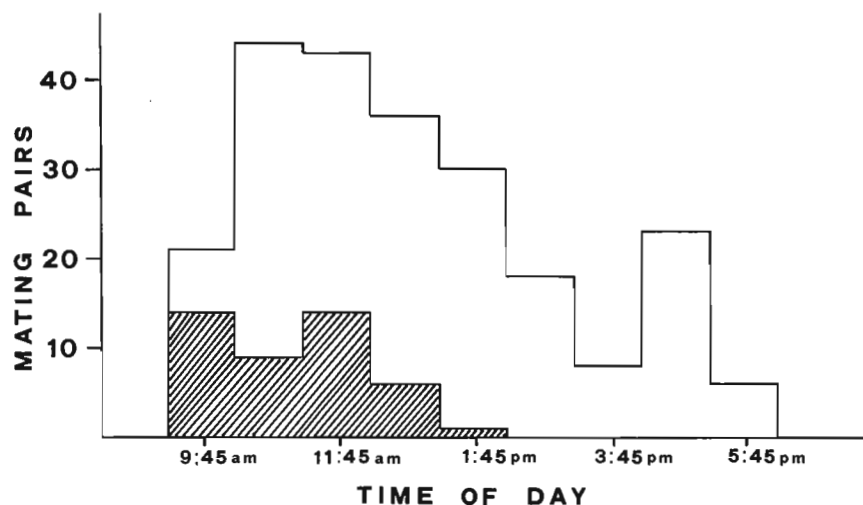


Fig. 1. Distribution of numbers of mating pairs observed throughout the day in *D. nigrospiracula* (blank) and *D. mettleri* (crossed).

this test is highly significant ( $p < 0.001$ ), which demonstrates that mating occurrence before and after 2 p.m. is dependent on species.

We do not know the physiological reasons which determine niche separation in mating behavior. Light may play an interesting role in inducing differential mating among species, since temperature appears to be similar at both peaks of maximum activity of *D. nigrospiracula*. The interesting thing is that this difference in behavior reduces the probability of interspecific courtships and this may be relevant to avoid reproductive interference between species. Under severe desert conditions species must adapt to exploit the few available niches. Obviously, mating time is reduced to a short period of the day during most of the year. (S.

for activity of flies. On the other hand, *D. mettleri* shows also a maximum of mating activity in the morning at about the same time as *D. nigrospiracula*, but its period of activity does not extend into the afternoon. In several days of sampling for this and other experiments we never observed a mating of *D. mettleri* later than 1:40 p.m. (local MST), and even this was an extreme case since matings usually stop at about noon for this species.

We have grouped the data in two classes corresponding to numbers of matings collected before and after 2 p.m. and performed a G-test for independence. As expected,

Johnston has done extensive research on these desert species and has reported to us that mating always stopped when temperature rose above 25°C or when humidity fell below 25%, usually at 10 a.m.) November is a rather mild month and provides the right temperature conditions for prolonged mating activity. Yet we have seen that *D. mettleri* does not use afternoon time efficiently. Apparently this would reduce the reproductive potential of this species but since numbers of *D. mettleri* are high, although not as high as of *D. nigrospiracula*, this may be irrelevant. As we mentioned above, both species have a very similar morphology, but the more we study their ecology, the more distinct we find they are. This suggests that most of their differentiation has occurred at the level of behavior, and morphology only plays a role on the characters involved directly with sexual isolation. More investigation is needed to know the adaptive (or non-adaptive) meaning of the differential mating activity reported here. However, as far as we know, this is the first time that such differential behavior has been described from a natural population of *Drosophila*.

We gratefully acknowledge the field assistance of R. Mangan, M. Jefferson, D. Vacek and D. Jurgenson.

References: Fellows, D.P. and W.B. Heed 1972, *Ecology* 53:850-858; Kaneshiro, K.Y., H.L. Carson, F.E. Clayton and W.B. Heed 1973, *Amer. Natur.* 107:766-774; Margalef, R. 1974, *Ecologia*, Ediciones Omega, Barcelona.

Gamo, S., M. Ogaki and E. Nakashima-Tanaka. University of Osaka Prefecture, Japan. Anesthetics resistance in *D. melanogaster*.

We reported that ether resistance of Eth strain at 24 hours age of adult flies was completely dominant over sensitivity and that maternal or cytoplasmic effects were negligible. The locus of the major gene(s) for the ether resistance is around 61 on the third chromosome, and the

minor genes are on both the X and the fourth chromosomes (Ogaki et al. 1967).

We have investigated effects of chloroform and halothane on the Eth and the bw;st;sv<sup>n</sup> strains. Mortality of the Eth strain was 1.3% in females and 1.7% in males, and that of the bw;st;sv<sup>n</sup> strain was 82% in females and 90% in males in 30 seconds treatment with chloroform. The LT<sub>50</sub> (50% lethal time) of the Eth strain for halothane treatment was 7 minutes in females and 9.5 minutes in males; that of the bw;st;sv<sup>n</sup> strain was 3.5 minutes in females and 3 minutes in males. Thus, it can be said that a cross-resistance to ether, chloroform and halothane is found in the Eth strain. Reciprocal crosses between the Eth and bw;st;sv<sup>n</sup> strains showed that the resistance to chloroform, as well as to ether, was completely dominant over sensitivity and that maternal or cytoplasmic effects were negligible. But the resistance to halothane was an intermediate trait with no maternal or cytoplasmic factors. A major gene with respect to chloroform resistance is located on the X chromosome, and a minor gene(s) on the second chromosome. However, the major gene(s) for halothane resistance is located on the right end of the third chromosome, which may be different from the major gene of ether resistance, and minor genes are on the X and second chromosomes. Therefore, resistances to ether, chloroform and halothane may be controlled by different genes.

Gausz, J., A.A.M. Awad and H. Gyurkovics. Biological Research Center of the Hungarian Academy of Sciences, Szeged, Hungary. New deficiencies for the kar locus of *D. melanogaster*.

The heat-shock inducible puff regions of 87A and 87C on the right of the 3rd chromosome have a special importance in studying gene regulation. A detailed genetic analysis of these loci is going on in our laboratory. As a rather limited number of deficiencies were only available for these loci (Ish-Horowicz 1977; Costa 1977), we

screened for new deficiencies in the region. Males homozygous for bw eye color mutation on the 2nd chromosome were irradiated with 4000 r X-rays (150 kV, 0.5 mm Al-filter, 1000 rads/minute). After irradiation the flies were immediately mated en mass to virgin females homozygous for bw on the 2nd and cu and kar on the 3rd chromosome. The interaction of bw and kar results in a very characteristic light brown eye color, making it possible to distinguish the putative deficiencies and point mutations from the wild type progeny. After scoring 63,000



F<sub>1</sub> progeny 28 kar and 8 cu stocks were recovered over the TM3 balancer. The kar stocks were crossed to Df(3R)kar<sup>3J</sup> and Df(3R)kar<sup>3Q</sup> deficiencies deleting the kar locus and 10 of the established stocks proved to be single kar point mutations. All the isolated cu stocks produced cu progeny when crossed to cu point mutation, i.e., they represented new cu alleles. It is in accordance with the presence of a minute locus nearby to cu locus (Lindsley et al. 1972).

All the putative kar deficiencies were crossed to ru cu ca flies and 3rd instar larvae from the progeny were dissected and salivary gland chromosome preparations were made (Yoon et al. 1973) to determine the breakpoints of the deficiencies. With the aid of these deficiencies, we mapped the coding locus for kar to 87C8 band.

The new stocks carrying a deficiency in the 87A-C region were mated to In(3R)Na flies and 3rd instar larvae from the progeny were heat-treated (30 minutes at 37°C) to show either the presence or absence of 87A and 87C puffs. The data are listed in Table 1.

Table 1. List of new kar deficiencies and their breakpoints.

Deficiency	Breakpoints		Formation of puffs	
	Proximal	Distal	87A	87C
Df(3R)kar <sup>Sz-5</sup>	86E20-F1 ;	87F3-4	-	-
Df(3R)kar <sup>Sz-8</sup>	87C1-3 ;	87D14-15	+	+
Df(3R)kar <sup>Sz-11</sup>	87C7-8 ;	87E5-6	+	+
Df(3R)kar <sup>Sz-12</sup>	87B1-3 ;	87C8-9	+	-
Df(3R)kar <sup>Sz-13</sup>	86E6-7 ;	87C9-D1	-	-
Df(3R)kar <sup>Sz-15</sup>	87B1-2 ;	87E1-2	+	-
Df(3R)kar <sup>Sz-16</sup>	87C1-2 ;	87C9-D1	+	+
Df(3R)kar <sup>Sz-21</sup>	87C6-7 ;	87C8-9	+	+
Df(3R)kar <sup>Sz-23</sup>	86E6-7 ;	87C9-D1	-	-
Df(3R)kar <sup>Sz-27</sup>	87C7-8 ;	87E12-13	+	+
Df(3R)kar <sup>Sz-29</sup>	87C3-4 ;	87C9-D1	+	+
Df(3R)kar <sup>Sz-28</sup>	87C7-8 ;	87E9-10	+	+
Df(3R)kar <sup>Sz-30</sup>	87B2-4 ;	87D2-3	+	-
Df(3R)kar <sup>Sz-31</sup>	86C6-7 ;	87C9-D1	-	-
Df(3R)kar <sup>Sz-33</sup>	87C1-2 ;	87E4-5	+	+
Df(3R)kar <sup>Sz-37</sup>	87C5-6 ;	87D14-E1	+	+
Df(3R)kar <sup>Sz-40</sup>	87B2-3 ;	87D1-3	+	-
Df(3R)kar <sup>Sz-72</sup>	87E1-3 ;	87F13-14	+	+

References: Costa, D. et al. 1977, DIS 52:140; Ish-Horowicz, D. et al. 1977, Cell 12:643-652; Lindsley, D.L. et al. 1972, Genetics 71:157; Yoon, J.S. et al. 1973, Experientia 29:639.

Gershenson, S. Institute of Molecular Biology and Genetics, Academy of Sciences of the Ukrainian SSR, Kiev 252627, USSR. Additional data on putative insertion mutations in wild populations of *D. melanogaster*.

In several recently published papers (Golubovsky 1977, Golubovsky et al. 1977, Green 1975, Ising and Ramel 1976, Ivanov and Golubovski 1977, Rasmussen and Green 1974) interesting data have been presented on unstable heredity variations in *D. melanogaster* presumably caused by insertion mutations. The significance of these data is increased by the fact that at least one of these

mutations, the sex-linked recessive mutation singed bristles (sn), was found in several wild populations of this insect. In this connection I think it worthwhile to draw attention to similar findings (seemingly the first of this kind) made by me more than 40 years ago. These findings were described in a report of the genetical laboratory of the Institute of Zoology (Academy of Sciences of the Ukrainian S R) which was published in May or June, 1941. Because of the subsequent invasion of Hitler's army, this book reached only very few libraries in the USSR and no reprints have been prepared of the papers contained therein so that they remain unknown to most geneticists. Here follows a brief summary of a part of my paper (Gershenson 1941) published in this book.



Table 1. Sex-linked mutants in  $F_1$  of females caught in nature.

No. of ♀♀ which gave mutants in $F_1$	Mutations	No. of $F_1$ ♀♀	No. of wild-type $F_1$ ♂♂	No. of mutant $F_1$ ♂♂
16	yellow	27-59	22-47	1-4
1	yellow-2	33	28	1
2	yellow-ruby	34+29	19+34	1+1
1	white	57	61	2
1	mottled	40	34	2
	(allele of w)			
1	singed	39	37	1

Table 2. Results of inbreeding wild-type descendants of female No. 515 caught in nature.

Generation	Total no. of crosses	No. of crosses showing mutants in offspring	Segregation among ♂♂	
			♂♂ +	♂♂ yellow
$F_1$	1	1	45	2
$F_2$	32	5	28	1
			44	1
			26	22
			31	1
			24	1
$F_3$	28	3	35	1
			37	1
			52	1
$F_4$	66	5	16	11
			36	3
			18	1
			29	1
			32	2

Table 3. Results of inbreeding wild-type descendants of female No. 103 caught in nature.

Generation	Total no. of crosses	No. of crosses showing mutants in offspring	Segregation among ♂♂	
			♂♂ +	♂♂ yellow
$F_1$	1	1	61	2
$F_2$	26	2	44	5
			19	3
$F_3$	31	3	105	9
			41	32
			51	5

Two sex-linked recessive mutants were found among 723 *D. melanogaster* males caught in the fall of 1937 in an orchard near Kiev: one male showed yellow body color (y), another had ruby eye color (rb). Out of 547 phenotypically normal females caught at the same time, 22 produced some recessive sex-linked mutant males in their  $F_1$  (Table 1).

As seen from Table 1, in the progeny of each individual female only a single or very few mutant sons appeared among a much greater number of wild-type brothers.

In subsequent generations obtained from females which gave mutants in their  $F_1$ , the same mutations always appeared in some of the cultures. As typical examples, in Tables 2 and 3 are shown the results of several generations of inbreeding of wild-type descendants of two of the females caught in the wild.

Similar results were obtained in the progeny of all the other 20 wild females which gave mutant males in their  $F_1$ . In subsequent generations the majority of crosses between wild-type descendants produced only wild-type flies, some crosses gave a few mutant sons and in rare cases a typical 1:1 segregation took place among the male offspring. Throughout all the generations only the same kind of mutants appeared as were initially observed in the  $F_1$  of a given female. Only in the lines which gave yellow-ruby males,

non-yellow ruby males sometimes appeared, probably as a result of crossing over in their mothers, and in one of the lines which gave yellow males, a yellow-2 male was found in one of the later generations.

The instability of certain genes located in the X chromosome derived from females caught in the wild was maintained after all the autosomes were substituted by autosomes from laboratory stocks, so it is caused evidently by some factor inher-

ent to the wild X chromosomes, probably insertions.

All the mutant males found in the experiments described above bred true when crossed to attached-X females and the mutant stocks thus established remained stable in the following generations.

An analysis of salivary-gland chromosomes in one of the lines in which ruby-eyed males sometimes appeared showed that the X chromosome of this line contained a large duplication including the locus of ruby.

Besides the 22 females which produced mutant males in their  $F_1$ , the progeny was studied of 102 wild females the  $F_1$  of which consisted only of wild-type flies. In the  $F_2$  of three of these females several yellow males were found and such males continued to appear in later generations of these lines.

The same wild population of *D. melanogaster* was again investigated in 1938 and 1939 and both times the results closely resembled those of 1937.

References: Golubovsky, M.D. 1977, *Genetika* (Moscow) 13:1030-1041; Golubovsky, M.D., Yu.N. Ivanov and M.M. Green 1977, *Proc. Natl. Acad. Sci. USA* 74:2973-2975; Green, M.M. 1975, *Mutat. Res.* 29:77-84; Ising, G. and C. Ramel 1976, In: *The Genetics and Biology of Drosophila*, ed. M. Ashburner and E. Novitski, 1b, 947-954; Ivanov, Yu.N. and M.D. Golubovsky 1977, *Genetika* (Moscow) 13:655-666; Rasmuson, B. and M.M. Green 1974, *Mol. Gen. Genet.* 132:265-289; Gershenson, S. 1941, In: *Memoirs on Genetics* (Inst. of Zoology, Acad. Sci. of the Ukrainian SSR) 4-5:3-39.

Gayathri, M.V. and N.B. Krishnamurthy.  
University of Mysore, Mysore, India.  
Preliminary studies on the effects of a mercurial fungicide Ceresan on fecundity in *D. melanogaster*.

It is well known that both life span and fecundity in *Drosophila* are extremely sensitive to a great variety of direct environmental factors (Lints 1971). Indeed, the activity and the number of ovarioles, in turn fecundity of a fly depend on age, genotype and the conditions to which larvae have been submitted (Gruwez et al.

1971). So, investigations were undertaken to evaluate the effects of a residual mercury fungicide Ceresan on fecundity of *D. melanogaster*.

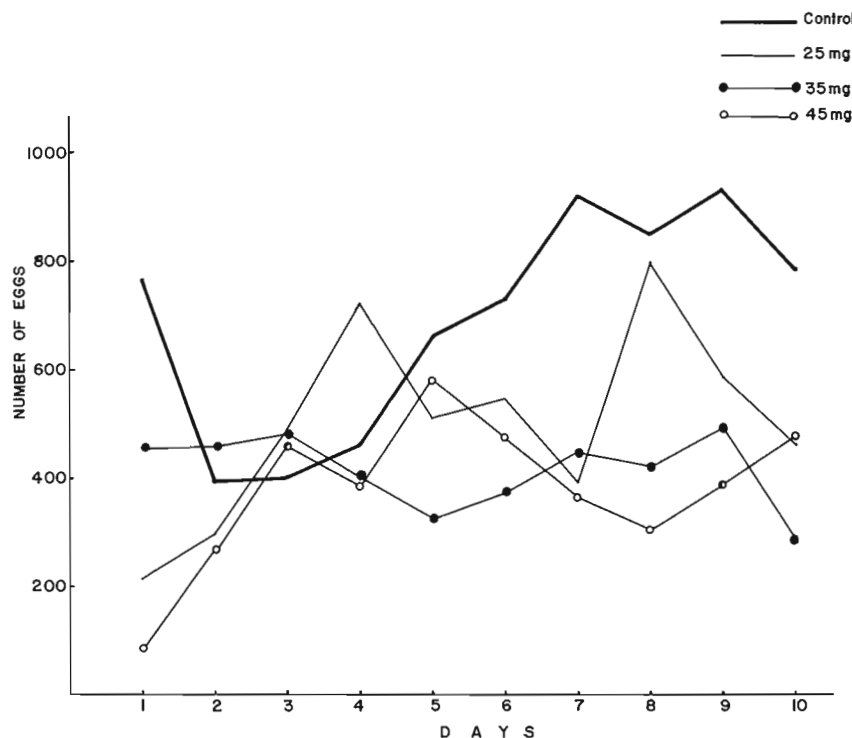


Fig. 1. Egg-laying pattern of Ceresan-treated and control flies.

Ceresan (Universal dry seed dressing; active ingredient: 1% Phenyl mercury acetate, Bayer) in concentrations of 25, 35 and 45 mg was mixed with 100 ml wheat cream agar medium. *D. melanogaster* flies of Oregon K strain were allowed to lay eggs on this chemical supplemented and normal food media so that the emerging larvae were exposed to Ceresan supplemented and control diets throughout the development. Parents were removed after 3-4 days; virgin flies (males and females) emerging from treated and control food media were isolated, aged for 5-6 days, and pair matings were made. The number of eggs laid by the control and treated flies during the following 10 days were scored. From this data, the pattern of egg laying, total fecundity and mean daily egg production were calculated and presented in Fig. 1 and Table 1.

Table 1. Fecundity of Ceresan treated and control *D. melanogaster* flies.

Concentrations	Total fecundity	Mean daily egg production/female
Control	6910	34.55 $\pm$ 0.23
25 mg	5016	25.08 $\pm$ 0.23*
35 mg	3934	19.67 $\pm$ 0.22*
45 mg	3736	18.68 $\pm$ 0.23*

\*  $P < 0.05$ , by Analysis of Variance.

computed to compare the fecundity of treated flies with that of controls has shown that fecundity is significantly reduced even by the lowest concentration of 25 mg of Ceresan tested ( $P < 0.05$ ). A dose-related reduction in fecundity is also evident from Table 1. Such an effect of mercury on fecundity is also known in other animals (Heinz 1974; Spann et al. 1972).

Ramel and Magnusson (1969), analyzing the genetic effects of mercurial compounds on *D. melanogaster*, state that "Mercurials given in the food to *Drosophila* larvae or adult flies obviously reach the gonads, where they cause chromosome disturbances presumably of the similar nature as the ones observed cytologically in plant cells". A chromosome breaking action of mercury has also been shown by Levan (1945). Further, Ramel (1969) also points out that Phenyl mercury causes more chromosome breakage than methyl mercury compounds. Phenyl mercury is also shown to cause somatic mutations, pollen sterility and chromosome fragmentation in plants (Mac Farlane 1950). The authors are of the opinion that chromosome disturbances caused by Phenyl mercury in the gonads and the germinal cells may be one of the major causes for the effects on reproduction in *Drosophila*.

**Acknowledgements:** The authors are grateful to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology, Manasa gangotri, Mysore for his encouragement. One of us (M.V.G.) is thankful to U.G.C. for the financial assistance.

**References:** Gruwez, G., C. Hoste, C.V. Lints and F.A. Lints 1971, *Experientia* 27:1414-1416; Heinz, G. 1974, *Bull. Environm. Contamination Toxicol.* 11:386-392; Heinz, G.H. 1976, *J. Wildl. Manage* 40:710-715; Levan, A. 1945, *Nature* 156:167-177; Lints, F.A. 1971, *Gerontologia* 17:39-51; Mac Farlane, E., W.E. 1950, *Genetics* 35:122-123; Ramel, C. 1969, *Hereditas* 61:208-230; Ramel, C. and J. Magnusson 1969, *Hereditas* 61:231-254; Spann, J.W., R.G. Heath, J.F. Kreitzer and L.N. Locke 1972, *Science* 175:328-331.

Golubovsky, M.D. and I.K. Zakharov. Institute of Cytology and Genetics, Novosibirsk 90, USSR. Simultaneous reversions of two sex-linked unstable mutations.

Since 1973 an outbreak of mutability of singed bristle locus was observed in natural populations of *D. melanogaster*. Most of the new alleles were unstable and putatively interpreted as insertions (Berg 1974, Ivanov 1974, Golubovsky et al. 1977, Golubovsky 1978). In 1975 in

the progeny of one male from a wild Far East population we found a remarkable case of simultaneous appearance of two mutations in the same X chromosome: singed and club wing (clw). In all male progeny the original sn49 allele had strong mutant expression, but club wing phenotype varied in penetrance and clearly was expressed in only 10-11% of males carrying the clw allele (at 25°C). In special tests 225 males with phenotypically normal wings crossed with  $\bar{X}\bar{X}/Y$  females produced 1408 club-wing  $F_1$  males out of 13829 tested, or 10.2%. At the same time, males with mutant club-like wings produced 11.7% of club-like sons among 4839 tested. It is evident that the rate of expression of clw phenotype in the progeny does not depend on parental phenotype. Instability of the two mutations was tested in successive generations. We isolated some allelic derivatives of the original sn49 alleles, as stable and unstable (Table 1). The revertants from sn49<sup>s</sup> (strong mutant expression) to sn<sup>+</sup> occurred with a frequency of  $3 \times 10^{-3}$ . They had absolutely normal bristles and wings, but some were stable (as sn49<sup>+</sup> 1-1), others unstable (1-4 and 7-3) (see Table 1). The last ones in turn were also capable of producing two types of "contra-revertants": (1) with original bi-mutant condition and (2) with mutant singed bristle phenotype and normal wing (clw<sup>+</sup>) (as sn49<sup>s</sup> 18-1). The moderate sn<sup>m</sup> derivatives were discovered to be quite unstable. The total scheme of allelic transitions is given in Fig. 1.

Table 1. The mutation frequency of unstable sn49 allele and its derivatives in the progeny of crosses with  $\overline{XX}/Y$  females.

Allele sn49 and its derivatives	Wing phenotype	Bristle phenotype of $F_1$			Direction and frequency of mutations
		sn <sup>s</sup>	sn <sup>m</sup>	sn <sup>+</sup>	
sn49 <sup>s</sup> *	club-like	14434	5	47	sn <sup>s</sup> - sn <sup>+</sup> $3.2 \times 10^{-3}$ sn <sup>s</sup> - sn <sup>m</sup> $3.4 \times 10^{-4}$
sn49 <sup>s</sup> (18-1)	normal	9223	0	0	stable
sn49 <sup>+</sup> (1-1)	normal	0	0	10172	stable
sn49 <sup>+</sup> (1-4)	normal	2	3	11152	sn <sup>+</sup> - sn <sup>s</sup> $1.8 \times 10^{-4}$ sn <sup>+</sup> - sn <sup>m</sup> $2.7 \times 10^{-4}$
sn49 <sup>+</sup> (7-3)	normal	11	1	12042	sn <sup>+</sup> - sn <sup>s</sup> $9.1 \times 10^{-4}$ sn <sup>+</sup> - sn <sup>m</sup> $0.8 \times 10^{-4}$
sn49 <sup>m</sup> (1-23)	normal	0	3465	36	sn <sup>m</sup> - sn <sup>+</sup> $1.0 \times 10^{-2}$

\* "s" means strong singed bristle phenotype; "m" - moderate; "+" - normal.

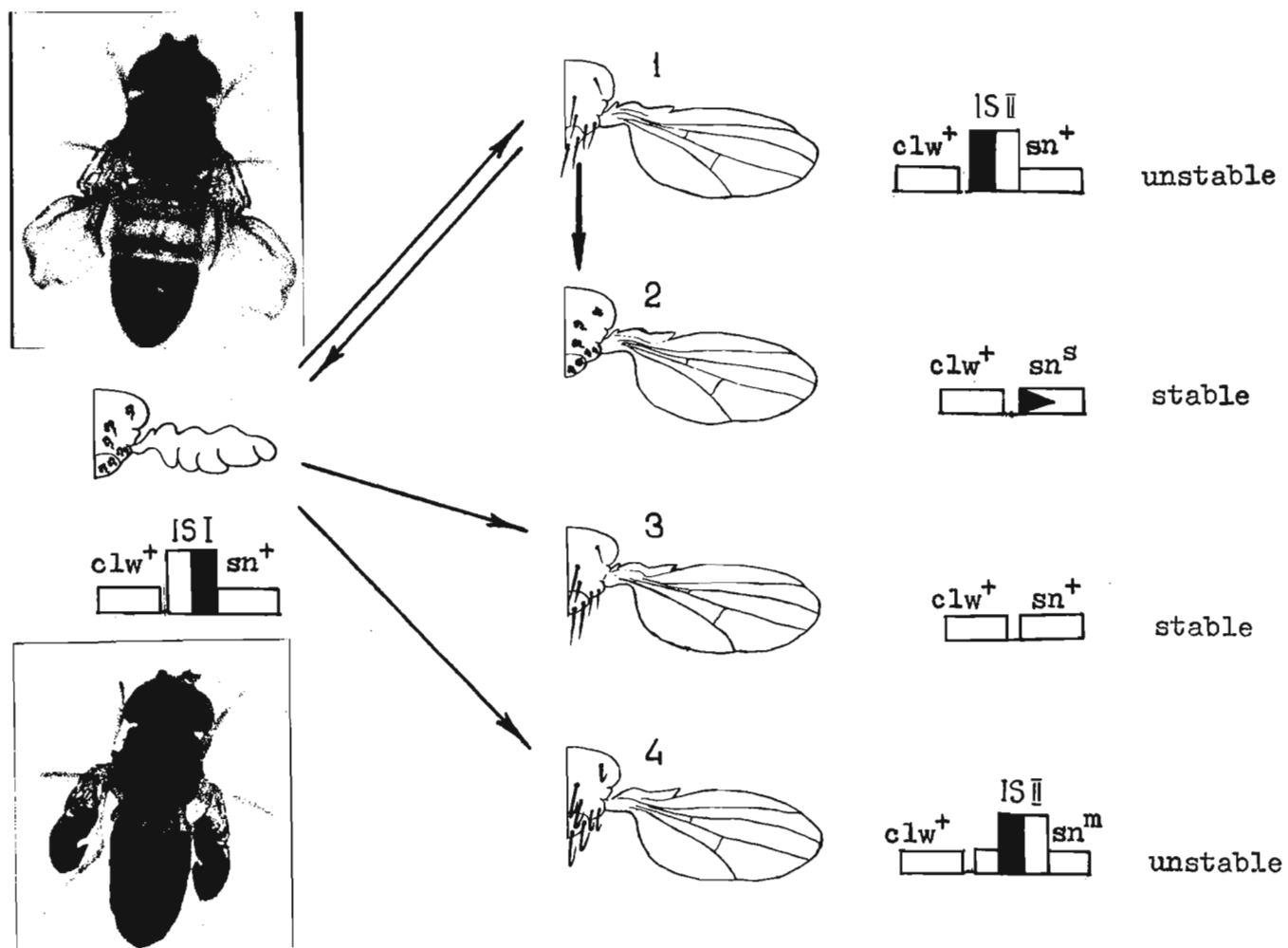


Fig. 1. Simultaneous reversions of two closely linked *sn* and *clw* mutations and possible explanation on the basis of insertion. Left - photo of original double mutant (two expressions of *clw* phenotype); right - observed allelic transitions to stable and unstable derivatives. For details see text.

It appears that *clw* mutant expression is possible only in combination with unstable singed-strong alleles. But normal *clw*<sup>+</sup> phenotype is expressed in *sns*, *sn*<sup>m</sup> and *sn*<sup>+</sup> alleles. The recombination in double mutant X-chromosome is free; the polytene chromosomes seem normal. We tried to "divide" two mutations by crossing over in *sn*49 *clw*/ct 12 females but failed; each time crossover with *sn*49 allele had *clw* expression. To explain this unusual situation we assumed that mutant state for two closely linked genes is related to insertion of a hypothetical IS-like segment into the region of these loci. The insertion segment is capable of changing its orientation or excision from the host chromosome. As shown in Fig. 1, insertion of the IS into orientation "1" blocks the normal expression of *sn* and *clw*. According to this suggestion, it is possible to predict all observed allelic transitions:

- (1) Regularly recurring transitions from the normal state to the original double mutant are due to the capacity of the IS to change its orientation, remaining in the same site;
- (2) Incorrect excision of the IS from the chromosome gives rise to a stable mutant *sn* allele and normal wing phenotype;
- (3) Precise excision of the IS produces stable wild type;
- (4) Intralocus transposition of IS segment is possible, resulting in the appearance of a novel *sn*<sup>m</sup> derivative and *clw*<sup>+</sup> state.

Unfortunately we couldn't identify *clw* mutation with known club-like wing mutations in the X chromosome. In the region of the *sn* locus (21.0) there are two mutations acting on wings: cut (20.0) and kinked femur (lost). But ct/*sn*49 *clw* flies are normal.

A similar case of simultaneous changing of two mutations was described earlier (Demerec and Slyzinska 1937). In T(1;4) *w*<sup>mt</sup> 258-18 translocation the distal region of the X chromosome is transposed to the heterochromatin area of chromosome 4 with unstable mutant expression of white and roughest genes. Here mutant condition of the white gene (*w*, *w*<sup>ch</sup>, *w*<sup>cr</sup>) was observed each time in *rst* facets, but in *w*<sup>+</sup> sectors *rst* and *rst*<sup>+</sup> facets were observed. So there is definite similarity between some position-effect inducing factors and instability phenomena, as Demerec suggested (Demerec and Slyzinska 1937).

References: Berg, R.L. 1974, DIS 51:100; Ivanov, Yu.N. 1974, DIS 51:71; Golubovsky, M. D., Yu.N. Ivanov and M.M. Green 1977, Proc. Nat. Acad. Sci. USA 74:2973; Golubovsky, M.D. 1978, DIS 53:171; Demerec, M. and H. Slyzinska 1937, Genetics 22:641.

Guillet, C. Université Claude Bernard  
Lyon-I, Villeurbanne, France. Mitochondrial activity during metamorphosis in *D. melanogaster*.

Oxygen consumption shows drastic changes during development in *Drosophila* (Fourche 1969). Recently, it has been stated that the variations in respiration at the larval stage may partly be explained by changes in either mitochondrial content or mitochondrial activity (Rezvoy,

Fourche and Guillet, in press). Both were found to correspond to the feeding periods (Fourche 1967a) and the modifications in hormone balance. The present paper investigates the role of mitochondria in the control of respiration during metamorphosis.

The strain used in these experiments was a wild strain Algeria. Each batch included about 1000 pupae isolated within three hours of puparium formation to provide homogeneous batches. Age was counted from the middle of the isolation period. The pupae were kept at 25°C until they were used at the appropriate age. Mitochondrial isolation and oxygen measurement by means of a Clark electrode are described elsewhere (Rezvoy, Fourche and Guillet, in press). Mitochondrial proteins were estimated by the Folin-phenol method of Lowry et al. (1951).

In the 98 hour old larvae, the mitochondrial protein content was 8.3 µg per larva; two hours after puparium formation it was only 5.5 µg. It increased after 60 hours and reached 7.6 µg at emergence (Fig. 1).

The Q O<sub>2</sub> (µl O<sub>2</sub>/hr/mg mitochondrial protein) was measured at state 3; the substrate was sodium succinate. In the 98 hour old larvae, the Q O<sub>2</sub> was 71 µl hr<sup>-1</sup>mg<sup>-1</sup>. After puparium formation Q O<sub>2</sub> followed a U-shaped curve; the lower value was 15 µl hr<sup>-1</sup>mg<sup>-1</sup> after 36 hours. Then it increased until emergence: 71 µl hr<sup>-1</sup>mg<sup>-1</sup> (Fig. 1).

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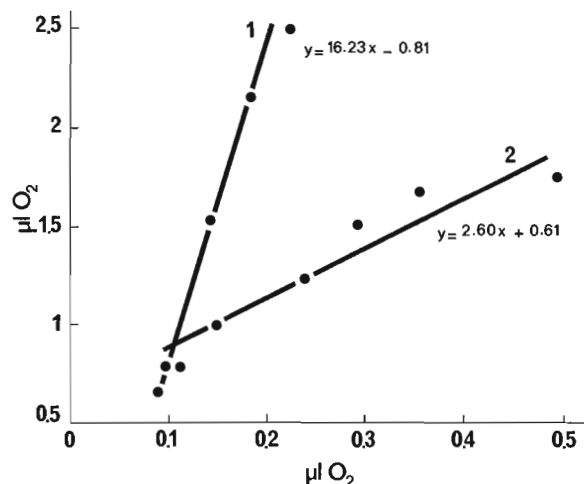


Fig. 1. Evolution of mitochondrial content (a) and mitochondrial activity (b). 1 - puparium formation, 2 - larvo-nymphal apolysis.

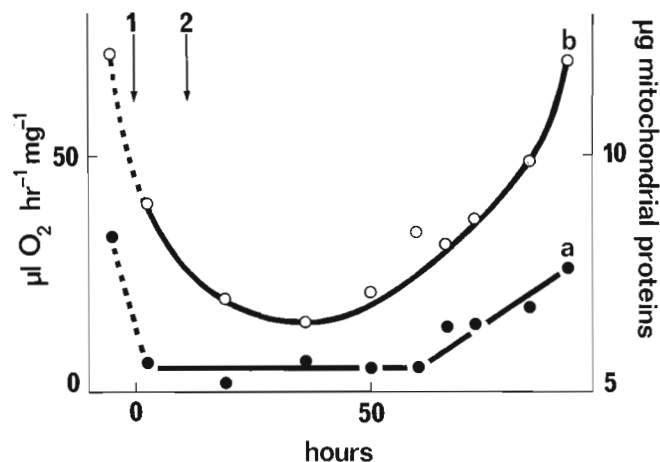


Fig. 2. Abscissa: oxygen consumption of the mitochondrial population. Ordinate: oxygen consumption of the whole pupa. 1 - descending phase, 2 - ascending phase.

Before puparium formation the respiratory rate of the larvae decreased (Fourche 1967b); a concomitant disappearance of mitochondria and a decrease in mitochondrial activity were observed. The oxygen consumption of the pupa was also shown to follow a U-shaped curve (Fourche 1969). During the descending phase of this curve, there was only a decrease in the mitochondrial activity without any change in the mitochondrial content. In contrast, the ascending phase was followed first by an increase in activity and subsequently by increases in both mitochondrial activity and content in order to answer a greater energy demand.

The results may be summarized as follows. The oxygen consumption of the mitochondrial population of a pupa (specific respiratory activity  $\times$  mitochondrial content of a pupa) also follows a U-shaped curve. If these values are compared with the respiratory rate of the whole pupa at 25°C (Guillet and Fourche 1973), a strong correlation between them can be seen. There are two linear relationships (Fig. 2), one for the descending phase of the U-shaped curve and one for the ascending phase. However, in the first case the mitochondrial oxygen consumption constitutes a smaller fraction of the total respiration. One of the most likely interpretations involves the presence of degenerated mitochondria in the pellets which may already be observed at the end of the larval stage: less than 1% (Rezvoy, Fourche and Guillet, in press). It is likely that the degenerated mitochondria have a very low oxidative activity as was shown for cytochrome C oxidase (Bulos et al. 1972). These degenerated mitochondria are probably connected with histolysis processes which begin after ecdysone release (Hodgetts et al. 1977).

Finally, it may be said that the mitochondria play a role in the control of respiration both in larva and pupa. They are able to modulate oxygen consumption either by changes in mitochondrial activity or the quantity of mitochondrial proteins or both.

References: Bulos, B., S. Shukla and B. Sacktor 1972, Arch. Biochem. Biophys. 149:461; Fourche, J. 1967a, Arch. Anat. Microsc. Morph. Exp. 56:141; Fourche, J. 1967b, J. Insect Physiol. 13:1269; Fourche, J. 1969, Ann. Biol. 8:334; Guillet, C. and J. Fourche 1973, Ann. Zool. Ecol. Anim. 5:533; Hodgetts, R., B. Sage and J. O'Connor 1977, Dev. Biol. 60:310; Lowry, O.H., N.J. Rosebrough, A.L. Farr and R.J. Randall 1951, J. Biol. Chem. 60:310.

Grant, S. and E. Rapport. University of Toronto, Canada. The effect of lactamide on the mutant *eyeless*<sup>2</sup>.

*less*<sup>2</sup> (*ey*<sup>2</sup>), a fourth chromosome, recessive mutant which also reduces eye facet number.

Table 1. The effect of lactamide on facet number in *ey*<sup>2</sup>/*ey*<sup>2</sup> flies.

percent lactamide	sex	no. of eyes	average number	± std. deviation
0	♂	36	248.02	27.65
0	♀	32	261.43	32.58
2	♂	23	212.06	23.14
2	♀	28	233.79	28.49
3	♂	17	207.26	16.92
3	♀	24	211.40	26.25
4	♂	25	197.89	29.17
4	♀	25	209.18	29.98
5	♂	28	185.33	21.72
5	♀	30	201.16	16.16

pounds used by Kaji) caused a reduction of protein synthesis, the concomitant reduction in the synthesis of lysozyme could result in less cell death in the Bar mutant (Michinomae and Kaji 1973, DIS). In the *ey*<sup>2</sup> stock, however, the reduction in protein synthesis could retard eye development even more than normal. This hypothesis is now being tested.

Acknowledgements: We thank Y.C. Wong, who initially suggested that amide treatment reduced eye facet number in *eyeless*<sup>2</sup> flies.

Kaji (1954-1959) demonstrated that several organic compounds, especially lactamide, increased eye facet number in the mutant Bar. To determine if this effect was specific to the Bar mutant we tested the effect of lactamide on *eyeless*<sup>2</sup>.

We transferred 60 hour larvae from a yeast-seeded cream of wheat-molasses medium to a similar medium containing 0 to 5% lactamide by weight. After 30 hours of treatment larvae were removed to vials containing a yeast-seeded agar-sucrose medium to complete development. Facet number was determined using a compound microscope equipped with a grid ocular. Under conditions in which Bar eyed flies showed up to a four-fold increase in facet number (data not shown) *ey*<sup>2</sup> had reduced facet numbers (approximately a 20% reduction).

We suggest that a unitary hypothesis can account for the disparate effects of lactamide on the two different mutants.

If lactamide (as well as the other com-

Gunawan, B. and J.S.F. Barker. University of Sydney, N.S.W., Australia. Adult viability of *D. buzzatii* in stress environments.

*D. buzzatii* (a species of the *mulleri* subgroup of the *repleta* group) is known to breed and feed in rotting cladodes of a number of species of the cactus genus *Opuntia*, and is apparently specific to the cactus niche (Barker and Mulley 1976). In Australia, *Opuntia* species (mainly

*O. inermis*) occur as isolated patches, usually in open sclerophyll forest or largely treeless grazing areas. During the day in summer, adult flies are not found in cladode rots, where temperatures as high as 44°C have been recorded. However, adults can sometimes be located on the underside of fallen cladodes, so that during the day they presumably take refuge in the plant litter on the ground where the temperature will be lower, but where relative humidity often will also be low. Normally, they become active during summer afternoons when the temperature drops to about 24-26°C. On some occasions, however, when summer collections were being made, the temperature at sunset was at least 32°C, and flies started coming to bait buckets from just prior to sunset. They remained quite active until dark when the temperature was still at least 29°C.

The temperature below which flies are active clearly depends on other factors, one of which would appear to be light intensity. Apparently *D. buzzatii* will be active and feeding in early morning and in the evening, practically regardless of temperature. Thus during summer, while their behavior and activity patterns will act to reduce temperature and/or desiccation stress, they will be exposed to such stresses for a large proportion of each day. Also, if adults do migrate between *Opuntia* patches, they would be exposed not only to these stresses, but also to a nutritional stress resulting from lack of access to cactus-specific yeasts.



Response to such stresses has been measured as days to 50% mortality at  $25 \pm 0.5^\circ\text{C}$  and 65-70% relative humidity in 3 x 1 inch glass vials with polyurethane foam stoppers for three stress treatments: (a) empty vials; (b) agar + sucrose - 7 ml of 0.15% w/v agar and 0.4% w/v sucrose medium; (c) paper + sucrose - a 20 x 13 cm piece of absorbent paper (Kleenex tissue) pressed into the bottom of the vial and 2.5 ml saturated sucrose solution added and absorbed by the paper. Additional treatments imposed were: sex - males only, females only, males and females in 1:1 ratio; density - 10, 20 or 30 flies per vial.

This 3 x 3 x 3 factorial was set up with 2 replicates. The flies used were progeny of a sample taken from a stock population cage which derived from 96 females captured at Yarrowonga, N.S.W. (locality 5 of Barker and Mulley 1976). These progeny emerged during a 12 hour period, and were aged for 3 days in well-yeasted vials before allocation to treatment vials. Mortality in each vial was recorded daily.

Analysis of variance of days to 50% mortality (i.e., from 3 days of age) showed a significant effect only for stress treatment ( $P < 0.001$ ). The means, which were significantly different from each other, and the maximum number of days survived, were: (a) empty vials - 3.8 days, 8 days; (b) agar + sucrose - 14.9 days, 23 days; (c) paper + sucrose - 18.6 days, 25 days. The significantly longer average survival in the paper + sucrose treatment, as compared with agar + sucrose, was presumably due to an initial higher humidity.

For comparison, in other experiments using medium containing dead yeast, but for a different strain of *D. buzzatii*, mean age at 50% mortality was 34.7 days, and maximum survival was to 90 days of age.

Clearly, *D. buzzatii* shows high tolerance to these environmental stress treatments (see also Parsons and McDonald 1978), which would be adaptive in their natural habitat, and the results do not preclude the possibility of survival for many days in a non-cactus environment, such as during migration from one cactus patch to another. It is hoped that current field studies will determine whether such migration does occur.

Work supported by Australian Research Grants Committee.

References: Barker, J.S.F. and J.C. Mulley 1976, *Evolution* 30:213-233; Parsons, P.A. and J. McDonald 1978, *Experientia* 34:1445-1446.

Hardy, R.W. University of California, San Diego. Crystal aggregates in the primary spermatocytes of XO males in *D. melanogaster*.

In another note in this issue we describe a small region in the middle of the Y chromosome which when missing results in formation of the crystal aggregates in the primary spermatocyte described by Meyer et al. (1961) in XO males.

These aggregates are seen in live preparations

with phase contrast optics. In the present note we locate a gene on the X chromosome which determines the shape of the aggregates.

Crystal aggregates occur in either of two forms, needle-shaped or star-shaped (Meyer et al., 1961). Aggregates are found in both nucleus and cytoplasm but the latter are larger and more easily seen. They persist through the meiotic divisions and can also be found in developing spermatids. Their exact molecular composition is not known, but they seem to contain both protein and lipid (Cox et al., 1976).

Meyer et al. (1961) reported that in XO males whose X chromosome is FM4, the needle-shaped crystal aggregates normally found are replaced by star-shaped ones. Additionally, Cox et al. (1976) report the occurrence of star-shaped aggregates in spermatocytes of both FM4/0 and FM6/0 males and further suggest that the change in morphology may be due to a specific inversion (In(1)3C;4E-F which is superimposed on In(1)sc<sup>8</sup> + In(1)d1-49 in both of these chromosomes).

We have found star-shaped crystal aggregates in two stocks with X chromosomes of normal sequence. We designate the locus responsible for modifying the shape of the crystal aggregates as stellate (*ste*). The allele responsible for the needle-shaped aggregates is designated *ste*<sup>+</sup> since this allele seems to be the one more commonly found in laboratory stocks. The allele responsible for the star shape is designated as *ste*. It is not known which, if either, allele is dominant.

In one instance the stellate phenotype was found in XO males carrying an X chromosome marked with *y w*<sup>a</sup>, and in the second it was found segregating in our Canton-S wild type stock. Females heterozygous for *ste* from both sources and *sc ec cv ct*<sup>6</sup> *v g*<sup>2</sup> *f* were crossed to XY/O males, and recombinant sons were dissected and the phenotype of the crystals in the spermatocytes noted. The crystals were star-shaped when the *g-f* region came from the X chromosome being tested and needle-shaped when it came from the marked X chromosome. Furthermore the recombinants between garnet and forked gave the results tabulated below.

Origin of <i>ste</i> - bearing X chromosome	Recombinants				Total males
	<i>g ste f</i> <sup>+</sup>	<i>g</i> <sup>+</sup> <i>ste</i> <sup>+</sup> <i>f</i>	<i>g ste</i> <sup>+</sup> <i>f</i> <sup>+</sup>	<i>g</i> <sup>+</sup> <i>ste f</i>	
Canton-S	3	2	10	7	258
<i>y w</i> <sup>a</sup>	0	2	5	12	---

8.5% recombination was observed between *g* and *f* compared to a standard map distance of 12.1 units. The distribution of *ste* among the recombinants between *g* and *f* indicates that the stellate locus is 17% (7/41) of the distance from *g* to *f* which places it at 46.5 on the standard map of the X chromosome.

References: Meyer, G.F., O. Hess and W. Beerman 1961, *Chromosoma* 12:676-716; Cox, G.N., J.D. White and B.I. Kiefer 1976, *Genetics* 83:S17.

Hardy, R.W. and J.A. Kennison. University of California at San Diego, La Jolla, California. Identification of a small Y chromosome region responsible for meiocyte and spermatid abnormalities typically observed in XO males.

A most striking effect of deletion of the Y chromosome from the primary spermatocyte is replacement of the lampbrush loop structures with crystals of a proteinaceous nature (Meyer et al. 1961). In addition, recent studies of XO males demonstrated abnormal meiotic organelle and chromosome distribution probably as a consequence of aberrant meiotic spindle formation

(Lifschytz and Hareven 1977; Lifschytz and Meyer 1977). Furthermore, as with crystal formation (Meyer et al. 1961), the distribution of meiocyte cellular components is more nearly like wild type in spermatocytes having the long arm of the Y chromosome (*Y*<sup>L</sup>) present (Lifschytz and Hareven 1977).

We have investigated these phenotypes in males carrying deficiencies for small regions of the Y chromosome, that is to say, regions which are thought to contain only a single fertility factor. A deficiency for such a small region is generated by combining specific segregants from different male-fertile XY translocations. In particular, segmental aneuploidy for one small region, a region approximately in the middle of *Y*<sup>L</sup>, results in spermatocyte and spermatid abnormalities closely resembling those seen in the light microscope for XO and XY<sup>S</sup> males (Lifschytz and Hareven 1977; Lifschytz and Meyer 1977) and in the electron microscope for XO males (Kiefer 1973). Sixteen primary spermatocytes are formed which contain crystals characteristic of XO males. Additionally, mitochondria and chromosomes are distributed abnormally during meiosis resulting in the formation of abnormal nebenkerne and micronuclei in the sperm-

atids similar to those observed in XO males. Two deviations from the XO phenotype have been observed. Lampbrush loop structures which are not obvious in XO primary spermatocytes can be seen in those carrying the deficiency. Furthermore, in electron micrographs of cross sections through the primary spermatocytes both crystals and the nuclear structures shown by Meyer et al. (1961) and Tates (1971) to be present in wild type (XY) spermatocytes are seen. The second deviation from the XO phenotype is that counts of spermatid tails in cross sections of cysts close to the middle of the testis indicate a mean number of tails per bundle of 54.5, considerably higher than the 31 reported in XO males by Kiefer (1973). Additionally, many of the axonemes and mitochondrial derivatives exhibit cross sections like those seen in XO males.

Preliminary observations of small deficiencies totaling virtually all of the Y chromosome except for the proximal regions around the kinetichore suggest that only the small region noted above leads to crystals, aberrant nebenkerne and micronuclei. Males carrying some of the other small deficiencies do not have normal ultrastructure in their spermatocytes or spermatids but the extent of such aberrations is not known.

At present work is under way to further characterize the Y chromosome deficiencies both genetically and cytologically (Kennison) and to study their effects on germ line development using both light and electron microscopy (Hardy).

References: Kiefer, B.I. 1973, in: Genetic Mechanisms of Development (Ruddle, F.H., ed.) pp. 47-102, Academic Press; Lifschytz, E. and D. Hareven 1977, Developmental Biology 58:276-294; Lifschytz, E. and G.F. Meyer 1977, Chromosoma 64:371-392; Meyer, G.F., O. Hess and W. Beerman 1961, Chromosoma 12:676-716; Tates, A.D. 1971, Ph.D. Thesis, Transitorium voor Gneeskunde, The Netherlands.

Hawley, R.S. University of Washington, Seattle, Washington. Radiation-induced nondisjunction in females homozygous for  $In(1)sc^8$ .

males, 2  $wa^+$   $B^+$  females, 1  $y^+$   $B^+$  female, and 1  $v^+$   $B^+$  female. The frequencies of B males (.22), which result from nullo-X ova, and  $B^+$  females (.005), which result from diplo-X ova, are identical to published values obtained following similar treatment of wild-type females (Hawley 1975). The recovery of 4 females homozygous for recessive markers confirms the observation of Savontaus (1975) that radiation-induced nondisjunction is not restricted to Eo tetrads.

In a second experiment,  $In(1)sc^8, y^{wa} / In(1)sc^8, f v cv$  females treated with 3000 R were mated to  $Y^S In(1)EN.Y^L, v f B / O$ ;  $C(4)RM, ci ey^R$  females and 27  $v f B$  males and 6  $B^+$  females were selected from among the progeny. By crossing the  $v f B$  males to  $C(1)RM, y f / Y$ ;  $ci ey^R / ci ey^R$  females, 7 (24%) were shown to have resulted from eggs which were also diplo-4. Of the 6  $B^+$  females, 4 (66%) were homozygous for  $ci ey^R$ . Following similar treatment of wild-type females, 19% of the nullo-X exceptions were also diplo-4 and 38% of the diplo-X exceptions were also nullo-4 (Hawley 1975).

These data suggest that the associations between the X and 4th chromosomes that dictate the frequency and manner of radiation-induced nondisjunction are not influenced by the location of the pericentric heterochromatin.

References: Hawley, R.S. 1975, Mut. Res. 33:391-394; Savontaus, M.-L. 1975, Hereditas 80: 195-204.

Hazelrigg, T. and T.C. Kaufman. Indiana University, Bloomington, Indiana. Newly induced mutations of doublesex.

Previous work (Duncan and Kaufman) has shown that the homoeotic gene doublesex (*dsx*) is located in region 84F of the polytene chromosome map. Both a recessive allele (which yields an intersexual phenotype in males and females) and

a dominant allele (which transforms only females into intersexes) are known to be associated with this locus. In the present work, an EMS mutagenizing (Lewis and Bacher) screen has been performed to uncover new alleles of *dsx*, and also recessive lethals located in this region of the chromosome. The screen utilized a deficiency,  $dsx^{D+R2}$ , recovered as a revertant of  $dsx^D$

(Duncan and Kaufman). This deficiency extends from 84D9-12 to 84F16, and exposes the intersexual phenotype when heterozygous with the recessive *dsx*. The results of this screen are summarized in the following table.

# Chromosomes Tested	# Sterile	# Viable	# Lethal	% Lethality
2815	406	2345	64	2.3

Among the viable chromosomes, 4 recessive mutations exhibiting an intersexual phenotype were recovered. These

have been crossed to *dsx*. Their failure to complement with *dsx* demonstrates that these mutants are indeed new alleles of this locus. Further detailed analysis of the morphological characteristics of these new alleles is in progress.

It is hoped that mapping of the recovered lethals will make possible the construction of a fine structure map of the *dsx* region. By utilizing 3 overlapping deficiencies recovered in Duncan and Kaufman's study, the positions of the lethals in 3 segments of region 84E-F is currently being ascertained. Inter se crosses following the deficiency mapping should yield a picture of the genetic structure of the region to which *dsx* has been localized, an area of the chromosome comprising about 15 bands.

References: Duncan, I. and T.C. Kaufman 1975, *Genetics* 80:733; Lewis, E.B. and F. Bacher 1968, *DIS* 43:193.

Hazra, S.K., J. Banerjee and S.K. Sen.  
Bose Institute, Calcutta, India. Location and nature of white-ivory (*w<sup>i</sup>*) in the white locus of *D. melanogaster*.

It was earlier reported (Hazra et al. 1978) that white-ivory (*w<sup>i</sup>*) does not affect recombination in the region further away from its location in the white locus of *D. melanogaster*. The present investigation was designed for a critical analysis of this feature in its vicinity as well as

to locate the mutant more precisely in the white locus. The relevant *w<sup>i</sup>* mutant was obtained from Pasadena Stock Center, USA. Other white locus mutants employed here are white-apricot (*w<sup>a</sup>*), white-cherry (*w<sup>ch</sup>*), white-eosin (*w<sup>e</sup>*), white-coral (*w<sup>co</sup>*), white-honey (*w<sup>h</sup>*) and pure white (*w*). Yellow body color (*y*), split bristle (*spl*) and echinus eyes (*ec*) served as flanking markers. Attached-X females of the genotypes *y w<sup>a</sup> spl/w<sup>iec</sup>*; *y w<sup>co</sup> spl/w<sup>iec</sup>*; *y w<sup>ch</sup> spl/w<sup>iec</sup>*; *y w<sup>e</sup> spl/w<sup>iec</sup>* and *y w<sup>h</sup> spl/w<sup>iec</sup>* were constructed according to Lindsley and Sandler (1963) and mated individually to *y w spl ec* males. All heterozygous females carried *SM1/+* and *Ubx<sup>130</sup>/+* rearrangements in the 2nd and 3rd chromosomes respectively to increase crossing over in the distal portion of the X chromosome (Judd 1959). The couple mutants were confirmed by their ability to yield respective single mutants due to reversion of *w<sup>i</sup>* to *w<sup>+</sup>*. The association of *w<sup>i</sup>* with *w<sup>a</sup>* in the coupling phase is indistinguishable from *w<sup>a</sup>* phenotypically. Accordingly, this putative couple mutant was confirmed by the recovery of *w<sup>i</sup>* as results of crossing over in between *w<sup>a</sup>* and *w<sup>i</sup>* mutant sites. Out of 27140, 28500, 112860, 292000 and 552400 flies screened respectively from the crosses as mentioned above in the serial order, 6, 6, 4, 4 and 4 recombinants were recovered. Recovery of *y w<sup>a</sup> w<sup>iec</sup>* and *y w<sup>co</sup> w<sup>iec</sup>* flies as recombinants guaranteed that *w<sup>i</sup>* is located to the right of *w<sup>a</sup>* and *w<sup>co</sup>*. Emergence of *w<sup>i</sup> w<sup>ch</sup> spl* flies from the third cross confirmed the earlier indication made by Lewis (1959) that *w<sup>i</sup>* is located to the left of *w<sup>ch</sup>*. Since the mutants *w<sup>e</sup>* and *w<sup>h</sup>* were shown to share a common location with *w<sup>ch</sup>* by earlier workers, it was anticipated that they would yield similar results as that of *w<sup>ch</sup>*. On the contrary, the emergence of *y w<sup>e</sup> w<sup>iec</sup>* and *y w<sup>h</sup> w<sup>iec</sup>* flies as recombinants from fourth and fifth cross respectively refuted such a working hypothesis. The location of *w<sup>i</sup>* to the right of *w<sup>e</sup>* and *w<sup>h</sup>* led us to think that a subsite of white locus could further be split through recombination. This suspicion was found to be along the right lines as evident from the results obtained from the subsequent experiment.

This experiment was expected to serve a dual purpose. First, the relative location of *w<sup>i</sup>* and *w* could be determined; and second, the indication obtained from previous tests that the mutants of a subsite have distinct spatial locations could be checked with respect to *w* and *w<sup>ch</sup>*. Free-X females of the genotype *w<sup>i</sup> w<sup>ch</sup> spl/ y w ec*; *SM1/+*; *Ubx<sup>130</sup>/+* were constructed and mated to *y w spl ec* males. In a total of 426000 flies screened, 6 *y w<sup>ch</sup> spl* exceptionals were obtained as the result of crossing over in the genetic interval marked by *w<sup>i</sup>* and *w<sup>ch</sup>*. A most interesting observation was the emergence of two *y spl* males through reciprocal recombination in the genetic interval marked by *w<sup>ch</sup>* and *w*. The complementary crossover, *w<sup>i</sup> w<sup>ch</sup> w*, could not

be recognized. Nevertheless, it was realized that  $w^{ch}$  is located to the left of  $w$ , which in turn signifies that  $w^i$  is located to the left of  $w$ .

In the present crossing over tests with  $w^i$  and other mutants of a subsite, the frequencies of recombinational derivatives were comparatively low in general when compared to that of the mutants of two different subsites. This may be explained most plausibly due to close sharing of location of  $w^i$  with the employed mutants, rather than that  $w^i$  inactivates recombination in this genetic interval. This supplements our earlier findings (Hazra et al. 1978) that  $w^i$  is a point mutation rather than a duplication as hypothesized by Bowman (1965).

References: Bowman, J.T. Jr. 1965, Genetics 52:1069; Hazra, S.K., J. Banerjee and S.K. Sen 1978, Heredity 40:299; Judd, B.H. 1959, Genetics 44:34; Lewis, E.B. 1959, Genetics 44:522; Lindsley, D.L. and L. Sandler 1963, in: Methodology in Basic Genetics, p. 390.

Hedrick, P.W. and E. Murray. University of Kansas, Lawrence, Kansas. Competition between *D. melanogaster* and *D. simulans* from natural populations.

Lawrence, Kansas, *melanogaster* was the most common of the two species in both 1977 and 1978 (Table 1). At all sampling times, the proportion of *melanogaster* was around 90% and the overall proportion is exactly 90.0%.

Table 1. The number of *melanogaster* and *simulans* males trapped in Lawrence, Kansas.

Date	mel	sim	% mel
7/77	292	25	92.1
9/77	218	35	86.2
7/78	50	1	98.0
9/78	86	11	88.7
Total	646	72	90.0

*D. melanogaster* and *D. simulans* are sympatric over much of their distributions and they appear to have similar ecological niches. Generally, *melanogaster* is the more common species where they coexist although there are some exceptions. In samples captured in a single location in

The proportions of two species observed in nature may be a reflection of a number of factors, such as predation, interspecific competition, sampling techniques, habitat selection, etc. Therefore, an interspecific competition experiment was set up to examine whether the results of interspecific competition in the laboratory were consistent with the field data. As a result only males could be scored, since females of the two species are very difficult to distinguish. At least 50 males were scored for each replicate every generation except in a few generations where there were slightly less than 50 males in a rep-

licate. The two lines of *melanogaster*, mel 1 and mel 2, were randomly selected isofemale lines caught at the Lawrence location and had been in culture for approximately six months. One *simulans* line, sim 1, was initiated from approximately 10 females caught in a Kansas City, Kansas park about 35 miles from Lawrence and had been in culture for approximately 30 months. The other *simulans* line, sim 2, was an isofemale line that was caught at the Lawrence location and had been in culture for approximately 18 months.

Since *melanogaster* was in higher proportion in local natural populations and generally outcompetes *simulans* in laboratory tests, the experiments were initiated with 6 pairs of *melanogaster* and 24 pairs of *simulans*, giving an initial frequency of 20% *melanogaster*. Four replicates of each of the four combinations of the *melanogaster* and *simulans* lines were set up. Generations were discrete and kept at 14-day intervals with the adults allowed to lay eggs for four days. Other details are as in Hedrick (1973).

The results of competition for sim 1-mel 1 and sim 1-mel 2 are given in Figures 1A and 1B, respectively. In only one replicate did *simulans* outcompete *melanogaster* and become fixed, replicate (b) of the sim 1-mel 1 competition. In all other replicates, *melanogaster* eventually became 100% of the culture. There is variation between replicates, however, with replicates (a) and (d) of the mel 1-sim 2 competition containing a few *simulans* even after 12 generations. When sim 2 was competing with mel 1 and mel 2, *simulans* was eliminated within five generations in five of the replicates. In the other three replicates, no *melanogaster* were ever scored. It appears that for some unexplained reason, the initial six females in these replicates did not produce any progeny.

One can measure the relative competitive ability of these two species for the different replicates by finding the "best" numerical fit of the change in proportions over time. Since *melanogaster* was the winner in all but one replicate, the relative competitive ability of

Table 2. Relative competitive ability estimated from the best fit to the observed change in species composition for each replicate.

Replicate	sim 1		sim 2	
	mel 1	mel 2	mel 1	mel 2
a	0.70	0.80	0.25	0.70
b	1.30	0.45	-	-
c	0.65	0.30	0.45	0.65
d	0.70	0.80	0.25	-
Mean	0.84	0.59	0.32	0.68

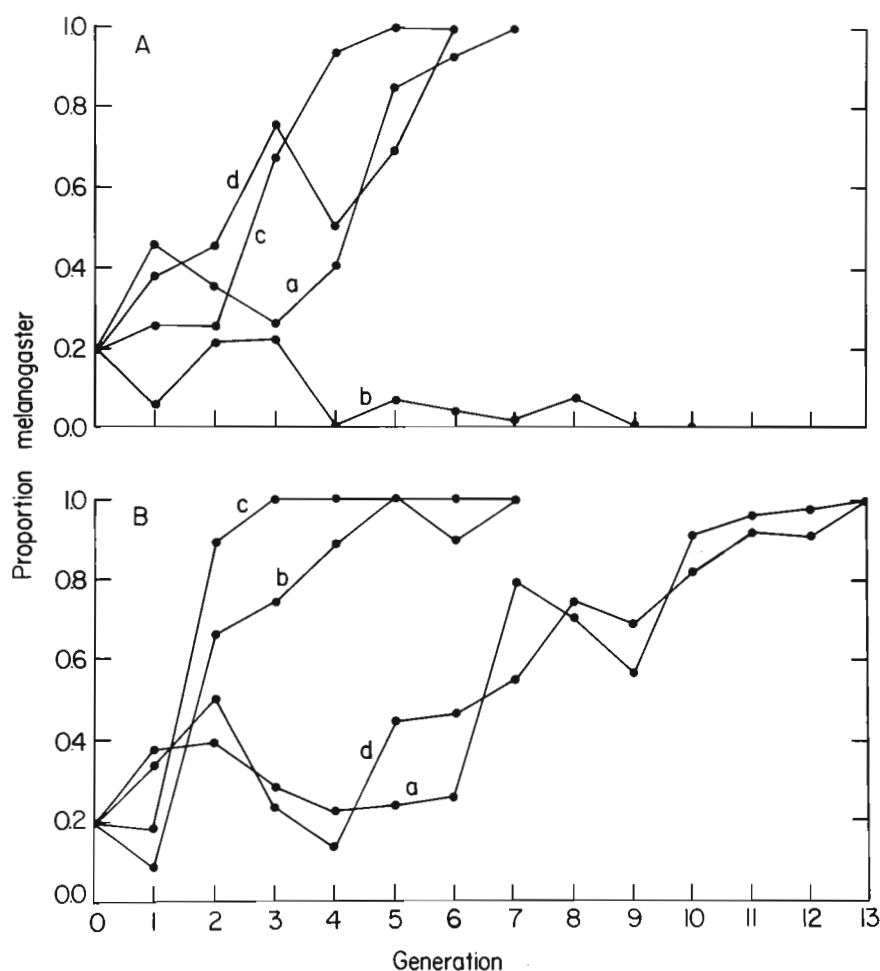
melanogaster was assumed to be unity and the simulans competitive ability ( $\bar{x}$ ) was varied at 0.05 increments until the smallest sum of squared deviations was found. More specifically, competition was assumed to be analogous to selection in a haploid where  $p_i$  and  $q_i$  are the proportions of melanogaster and simulans in generation  $i$ , respectively. Then the proportion of melanogaster in generation  $i+1$  is

$$p_{i+1} = p_i / (p_i + \bar{x}q_i)$$

The difference between these proportions and those estimated in the replicates was squared and summed for different values of

$\bar{x}$ . The value of  $\bar{x}$  which gave the minimum sum of squared deviations is given in Table 2. The relative competitive ability for the melanogaster lines can be calculated as  $1/\bar{x}$ .

An interesting finding from this analysis is that the estimated "relative competitive ability" for the two melanogaster lines are quite similar while those for the simulans lines are quite different. The mean relative competitive abilities for mel 1 and mel 2 were 1.64 and 1.61, respectively, and for sim 1 and sim 2, they are 0.72 and 0.46, respectively. The similarity of the melanogaster lines might have been expected since these lines were established from single females at the same time. On the other hand, sim 1 was different from sim 2 in that it was established earlier and with a larger initial number of females. Either of these factors may have contributed to the greater competitive ability of sim 1. The earlier establishment date would have allowed



Figs. 1A and 1B

more time for adaptation and the larger initial sample could have given more genetic variation for factors important to competitive ability.

References: Hedrick, P.W. 1973, *Evolution* 26:513-522.





Hegde, S.N. and N.B. Krishnamurthy. University of Mysore, Mysore, India. Studies on the *Drosophila* fauna from three localities of Maharashtra State: India.

leswar, all of which belong to Maharashtra state. Collections from 3 wild and 3 domestic localities made from the above places are presented in Table 1. The collections yielded a total of 21 different species in addition to a single species of the genus *Phorticella*. Out of the 21 species, 14 belong to the subgenus *Sophophora*, 3 to subgenus *Drosophila*, 3 to *Scaptodrosophila*, and one to *Dorsilopa*. The striking feature of the collection data is that in these areas only members of *melanogaster* and *immigrans* species groups dominated both in abundance and variety over others. This agrees with the suggestion made by Bock and Wheeler (1972) that "if collection made in any part of the southeast Asian or New Guinea area reveals that although many species may be collected at any particular locality, only two species groups comprise all or practically all of the catch, i.e., the *melanogaster* species group of the subgenus *Sophophora* and *immigrans* species group of the subgenus *Drosophila*".

Numerical variation in different localities under study are striking. The highest number of flies (1186) as well as species (17) has been collected in the wild localities of Mahabaleswar. In domestic localities, Lonavala has yielded a maximum number of flies (729) as well as species (4). *D. jambulina*, *D. malerkotliana* and *D. nasuta* are the 3 species which dominate numerically in these localities. It is interesting to note that *D. bipectinata* and *D. malerkotliana* which are essentially species that inhabit the wild localities have also been captured in the domestic localities of Lonavala. It is likely that these two wild species are attempting to invade and colonize the domestic locality as is evidenced by their presence in Lonavala.

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References: Bock, I.R. and M.R. Wheeler 1972, in: *Studies in Genetics*, VII, Univ. Texas Publ. 7213, pp. 1-102.

Sustained collections are essential in order to take census of the number and composition of *Drosophilid* fauna in several unexplored parts of India. The present report incorporates the results of the survey made by the authors in unexplored parts of Poona, Lonavala and Mahabaleswar.

Henikoff, S. University of Washington, Seattle, Washington. A more conventional view of the "ebony" gene.

Two reports suggest that ebony has unusual properties. Unable to find position effects for the gene, Brosseau (1970) speculated that the locus might be immune to the effects of heterochromatin. More recently, Scalenghe and Ritos-

sa (1977) reported that heat shock can induce cuticular glutamine synthetase I which is presumably the product of the ebony locus. Therefore, they proposed that the ebony gene, in addition to its role in normal development, is the site of the major heat shock puff at 93D. This communication describes ebony mutants that do not fit with the above suggestions: one mutation is a variegating position-effect, and another is a deficiency that does not remove the 93D heat shock puff locus.

Approximately 70,000 progeny of kar red e females mated to irradiated (3500r) red males were screened for mutant body and eye color. Of the 32 fertile mutant ebony chromosomes found, six carried aberrations involving the 93D region on the irradiated chromosome. Two translocations were broken in the 93D region and in the centromeric heterochromatin. One was a viable T(2;3) with a fully mutant phenotype even in the presence of an extra Y chromosome. The other was a homozygous lethal T(1;3) with a mottled phenotype that was much more strongly ebony in XX females than in the nearly wild-type XY males and XXY females. XO males were fully mutant.





Fig. 1. Ebony-variegated flies from the same  $18^{\circ}$  culture:  $XXY=T(1;3)20;93D$ , red  $e^{H2}/+$ ;  $In(3R)C$ ,  $Sb\ e\ 1(3)e / B^sY$ ,  $XX=T(1;3)e^{H2}/+$ ;  $In(3R)C$ ,  $XY=T(1;3)e^{H2}/B^sY;In(3R)C$ . An  $XO=T(1;3)e^{H2}/0$ ;  $In(3R)C$  male is also shown.

Such Y-suppressibility accords with the interpretation that this mutation is a position effect that variegates for the ebony gene. Typical examples are shown in Fig. 1.

One inversion and three deficiencies, all fully ebony, were also found in this screen. Their cytological locations agree with the assignment of ebony to the 93D2-3 region from earlier reports (Scalenghe and Ritossa 1977; Korge 1972). In salivary squashes of heat-shocked larvae, it is clear that the 93D puff is outside of both the inversion,  $In(3R)84C;93D2-3$ , and one of the deletions,  $Df(3R)93B10-13;93D2-3$  (Fig. 2). The existence of a deficiency that uncovers ebony but has no apparent effect on the heat-shock puff argues against either the proposal that glutamine synthetase I is one of the heat-shock proteins, or the presumption that the enzyme is the product of the ebony locus (Scalenghe and Ritossa 1977).

Thus, no special hypotheses are needed to explain unusual aspects of ebony gene function.

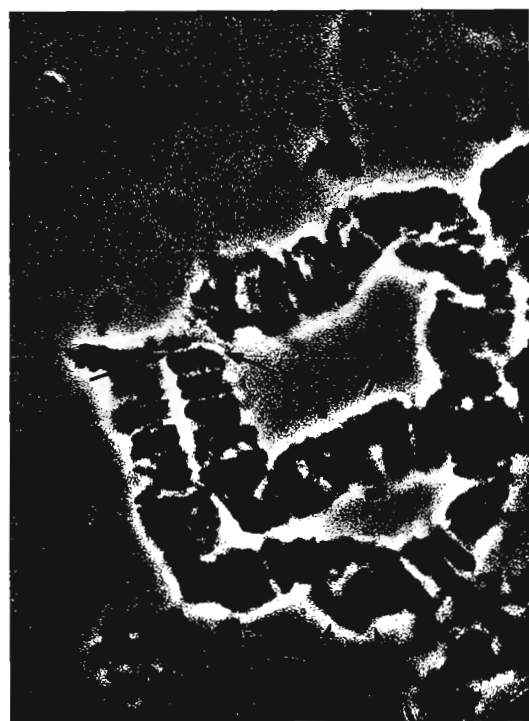


Fig. 2. a) Part of chromosome 3R from a larva of genotype  $In(3R)84C;93D2-3$ , red  $e^{H3}/Df(3R)93B10-13;93D2-3$ , red  $e^{H5}$  squashed immediately after dissection. b) Example of 3R from the other salivary gland of the same larva held 15 min at  $37^{\circ}$  before squashing.  $Df$  and  $In$  indicate the 93D puff sites on deficiency and inversion chromosomes respectively. For references, A and C indicate the sites of the heat-shock puffs in 87A and 87C.

References: Brosseau, G.E. Jr. 1970, DIS 45:100; Scalenghe, F. and F. Ritossa 1977, *Chromosoma* (Berl.) 63:317; Korge, G. 1972, DIS 48:20.

Hodgetts, R.B. University of Alberta, Edmonton, Alberta, Canada. A cytogenetic description of three duplications in which portions of proximal 2L have been inserted into the Y-chromosome.

Three derivatives of Dp(2;Y)G, the aneuploid segregant from T(Y;2)G (Lindsley and Grell 1968) have been constructed. In Dp(2;Y)G, a region of 2L between 36B4-5 and 40F has been inserted into an arm (unknown) of the Y-chromosome. In the derivatives, most of the material in the inserted segment between the dopa decarboxylase

locus (Ddc<sup>+</sup>) and the heterochromatin has been deleted. As a result, the new duplications can be carried in either sex without the serious effects on viability and fertility experienced with Dp(2;Y)G. While the arm of the Y-chromosome into which the 2L fragment is inserted is not known, Y-fertility is unaffected by the translocations.

(a) Dp(2;Y)H1 rdo<sup>+</sup> hk<sup>+</sup> Ddc<sup>+</sup> pr<sup>-</sup> lt<sup>+</sup>. This derivative was obtained following  $\gamma$ -irradiation of a stock carrying Dp(2;Y)G and contains a large deletion of the purple (pr) locus. The breakpoints of the deletion fall between the following limits: distal 37F4 - 38A1; proximal 29C2 - 39D1. Thus, the new order in Dp(2;Y)H1 is: 36B4 - 37F/39C - 40F.

(b) Dp(2;Y)H2 rdo<sup>+</sup> hk<sup>+</sup> Ddc<sup>+</sup> pr<sup>-</sup> lt<sup>+</sup>. This derivative was also obtained from Dp(2;Y)G and like Dp(2;Y)H1, contains a  $\gamma$ -ray induced pr deficiency. The breakpoints of this deficiency fall between the following limits: distal 38B2 - 38C1; proximal 39E2-3. Thus, the new order in Dp(2;Y)H2 is: 36B4 - 38B/39E3 - 40F.

(c) Dp(2;Y)H3 rdo<sup>+</sup> hk<sup>+</sup> Ddc<sup>+</sup> pr<sup>-</sup> lt<sup>-</sup>. This derivative was obtained from Dp(2;Y)H1 and contains a  $\gamma$ -ray induced deficiency for the light (lt) locus. The breakpoints of this deficiency fall between the limits: distal 37E2 - 37F1; proximal 40B2 - 40F. Thus, the new order in Dp(2;Y)H3 is: 36B4 - 37E/40.

We have used one of these duplications, Dp(2;Y)H1, in a recent work (Clark et al. 1978) where it was referred to as Dp(2;Y)Ddc<sup>+</sup>.

References: Clark, W.C., P.S. Pass, B. Venkataraman and R.B. Hodgetts 1978, Molec. Gen. Genet. 162:287-297; Lindsley, D.L. and E.H. Grell 1968, Carnegie Inst. Wash. Publ. 627.

Ingham, P.W. University of Sussex, U.K. Genetic analysis of trithorax, *trx*, a new homoeotic mutant of *D. melanogaster*.

A spontaneous recessive allele, *trx*, defining a new locus on chromosome 3 has been isolated. The incomplete penetrance of the allele necessitated its being mapped by the selection and test crossing of individual recombinant chromosomes. An

approximate localization was achieved by generating several different sets of reciprocal recombinants from 'rucuca'/*trx* ♀♀. This enabled the identification of *cu* (50.0 cMs) and *Sb* (58.2 cMs) as proximal and distal flanking markers respectively. 124 recombinants between these two loci were tested for the presence or absence of *trx*. The results are consistent with *trx* mapping to a single locus at 54.2 cMs ( $\pm 0.4$ ).

Penetrance and expressivity are highly variable. The phenotype consists of the homoeotic transformation of various adult structures. The most extremely affected individuals exhibit the following morphological changes. In the ventral prothoracic segment sternopleural bristles appear between the humerus and the first leg coxa. Transverse rows in the tibia are reduced in number or abolished; large apical and pre-apical bristles are present on the distal tibia (Fig. 1). In the basitarsus, there is a reduction in the number of transverse rows, and in males a concomitant decrease in the number of sex comb teeth. Similar changes in segment specific landmarks also occur in the third leg. The derivatives of the haltere disc are replaced distally by wing blade material and more proximally by notal and scutellar structures (Fig. 2).

The range and variety of the metathoracic transformations are closely analogous to the effects produced by ether phenocopying (Bowman and Seiler 1977; Capdevila and Garcia-Bellido 1978). The ventral prothoracic transformation has not previously been reported. The dorsal prothorax is apparently unaffected. Rotated genitalia and disruption of tergite pigmentation are also common in males.

At 25° the penetrance (P) of the selected homozygous line is 85%. However, there is a substantial maternal influence on penetrance; thus for homozygotes generated by crossing *trx*/+ ♀♀ with *trx*/*trx* ♂♂, P = 9%, while for the reciprocal cross of *trx*/*trx* ♀♀ with *trx*/+ ♂♂, P = 85%.



Fig. 1. SEM of transformed female. Right: lateral thorax showing sternopleural bristles (SP) above first and second legs. Top left: Detail of prothoracic sternopleurals. Bottom left: Apical and pre-apical bristles on distal tibia.



Fig. 2. SEM of dorsal surface of transformed fly showing extra notum and scutellum (arrowed) between scutellum and first abdominal segment.

Flies of the genotype *trx/Df(3)red*, produced by crossing *trx/trx* ♀♀ with *Df(3)red/+* ♂♂ always show the transformation, and with high expressivity. This is suggestive of this deficiency including the *trx* locus, and is consistent with the map position obtained by recombinational analysis, red mapping at 53.6 cMs.

It is interesting that the mutant tetrapter, which is described as having at least some phenotypic characteristics in common with *trx*, was also mapped to a similar region of the genome (Lindsley and Grell 1968).

References: Bownes, M. and M. Seiler 1977, *J. Exp. Zool.* 199:9-24; Capdevila, M.P. and A. Garcia-Bellido 1978, *Wilhelm Roux. Archiv.* 185(2):105-126; Lindsley, D.L. and E.H. Grell 1968, *Carnegie Inst. Wash. Publ.* 627.

Itoh, K. St. Mariana University School of Medicine, Kawasaki, Japan. Lack of chromosomal polymorphism and low frequencies of unique inversions in *D. simulans*.

somes of one larva from each line were cytologically examined within half a year to find any changes in gene sequences. Although polymorphic inversions were not detected even in a heterozygous condition, three unique inversions, one for each of the X, 2nd and 3rd chromosomes, were found. Since *D. simulans* has similar banding patterns of *D. melanogaster*, except for a large inversion in 3R, Bridges' map of *D. melanogaster* was used for the location of their breakpoints. They were:

In(1), 15A; 18D  
In(2LR), 24F; 57F  
in(3R), 87B; 90C

It is not certain whether these inversions had been carried by the original wild females or were newly produced during the maintenance of lines in the laboratory. At any rate, the frequencies of unique inversions in *D. simulans* can be calculated as  $1/448 = 0.00223$

for the X chromosome (224 female larvae were tested) and  $2/1796 = 0.00111$  for the major autosome. These low frequencies seem to be consistent with the lack of polymorphism in this species.

Four hundred and forty-nine wild inseminated females of *D. simulans* were collected in a month from August to September of 1975, on the campus of Kyushu University, Fukuoka, Japan. They were individually kept in vials to establish iso-female lines. Salivary gland chromo-

Jallon, J.-M. and Y. Hotta. University of Tokyo, Japan. A proposal for a new genetic scheme to hunt for sex-appealless mutants of *D. melanogaster*.

We have recently reported genetic and behavioral studies of female sex appeal in *D. melanogaster* (Jallon and Hotta 1979). The sex appeal was defined as the stimulus (or set of stimuli) which induces wing vibration in courting males. It is most likely to be a measure of sex pheromone re-

leased from a female.\* Sexual behavior of gynandromorphs was analyzed by means of the blastoderm fate map method (Hotta and Benzer 1972), and we were able to localize the sex appeal focus in the ventroposterior region of the blastoderm fate map. Moreover, studying ontogeny of male courtship, we found that on the first day after eclosion males possess as much sex appeal as females. However, males lose it within a day, while females retain it indefinitely.

Hunting for specific mutations affecting the presence of sex appeal in females is another way to extend the study of sex appeal and its control. A difficulty expected in isolating such mutants is that they will not reproduce. The existence of young males' sex appeal leads us to propose a novel way to overcome this difficulty. The procedure is to look for X-chromosomal sex-appealless mutants among  $F_1$  male progeny of chemically mutagenized males which are mated with attached X-chromosome females. By this genetic scheme, mutagenized paternal X-chromosome is transmitted to sons, so that sex-appealless mutations may be found among  $F_1$  males at their immature stage. If such mutants without ephemeral sex appeal could still function as normal males in later sexual activities, it would be possible to find such genes rather easily. We have so far examined about 4500 such males a few hours after their emergence and tested their ability to induce wing vibrations of male testers. None of them turned out to be a mutant. There are several possible reasons for the present difficulties. It may be that such genes happen to be rare on the X-chromosome. It may also be because such genes become lethals when they are mutated. Finally there might be at least two alternative biochemical pathways to produce sex appeal.

This method is particularly simple and allows a mass screening of sex-appealless mutant candidates. Using the same progeny, one may also investigate male mutants which would maintain female-like sex appeal beyond their usual immature period or ones which would court the wild-type male testers.

References: Hotta, Y. and S. Benzer 1972, *Nature* 240:527; Jallon, J.-M. and Y. Hotta 1979, *Behav. Genetics* (in press); \*Venard, R. and J.-M. Jallon 1979, submitted to *Experientia*.

Jha, A.P., B.N. Pandey and D.N. Mishra. Mithila University, Darbhanga, Bihar, India. Substrate specificities of alcohol dehydrogenase in *Drosophila*.

Alcohol dehydrogenase isoenzymes have been studied qualitatively (Ursprung and Leone, 1965; Jacobson et al., 1970) and quantitatively (Sofer and Ursprung, 1968; Ward, 1974) irrespectively of different substrates in *D. melanogaster*. Singh (1976) studied substrate speci-

ficities in *D. pseudoobscura*. Here we report on the substrate specificity in relation to developmental changes in *D. ananassae*, *D. malerkotliana* and *D. bipectinata*.

ADH activity was recognized by reduced tetrazolium deposition on 5% polyacrylamide gel. We used ten substrates in our experiment. In *D. ananassae*, larvae show activity with all ten substrates used in the experiment, pupae with only seven substrates and adults with nine substrates. Third instar larvae of *D. malerkotliana* and *D. bipectinata* exhibit activity with all ten substrates. Late pupae of *D. malerkotliana* reveal activity with all substrates, and those of *D. bipectinata* with only six. Adults of both species exhibit activity with all ten substrates. Differences in reactivity with substrates indicate that ADH isoenzymes must have some different physiological functions which are stage-specific.

Substrates	<i>Drosophila ananassae</i>			<i>Drosophila malerkotliana</i>			<i>Drosophila bipectinata</i>		
	Larva	Pupa	Adult	Larva	Pupa	Adult	Larva	Pupa	Adult
Ethanol	+	+	+	+	+	+	+	+	+
Methanol	+	-	+	+	+	+	+	-	+
Butanol	+	+	+	+	+	+	+	+	+
n-Propanol	+	+	+	+	+	+	+	+	+
2-Propanol	+	-	+	+	+	+	+	+	+
Benzyl alcohol	+	+	+	+	+	+	+	-	+
Allyl alcohol	+	+	+	+	+	+	+	-	+
Amyl alcohol	+	+	+	+	+	+	+	-	+
Cyclohexanone	+	-	+	+	+	+	+	-	+
Octanol	+	+	+	+	+	+	+	-	+

Presence (+) or absence (-) of activity for alcohol dehydrogenase enzyme in three species of *Drosophila* acting on a variety of alcohol substrates.

References: Ursprung, H. and J. Leone 1965, J. Exp. Zool. 160: 147; Jacobson, K.B., J.B. Murphy and F.C. Hartman 1970, J. Biol. Chem. 245: 1075; Sofer, W. and H. Ursprung 1968, J. Biol. Chem. 243: 3110; Ward, R.D. 1974, Biochem. Genet. 12: 449; Singh, R.S. 1976, Genetics 82: 507.

Joiner, M. and J.S. Johnston. Baylor University, Waco, Texas. Determination of exact age in *Drosophila* (1).

In 1971, Van Valen and Van Valen (2) reported failure to find daily growth layers in *D. melanogaster*. However, in 1973 Van Valen (3) encouraged *Drosophila* workers to try the aging methods of Schlein and Gratz (4). By following

these methods, we found limited areas of banding in thoracic muscle attachments (apodemes). As was shown in other Diptera (4), apodemes of *Drosophila* exhibit daily growth layers on regions of postmetamorphic growth. The apodemal growth layers are highly variable both in contrast and in maximum number. The best specimens show banding under transmitted light. Phase contrast microscopy improves the contrast, as does staining with Heidenhain's Hematoxylin. None of these methods, however, provide a reliable or repeatably good aging tool. After experimentation with a wide variety of techniques, stains, and counterstains, we developed the following simple and effective method: A fly with legs and head removed is placed into hot 4% KMnO<sub>4</sub> for 5 minutes. After the fly is rinsed in distilled water, the apodemes are pulled from the thorax with forceps and placed into Paragon (5) or other water soluble mounting media under a cover slip. Nemarski differential interference contrast (DIC) microscopy shows areas of growth along the apodemes. The first, second, and third furca (ful, fu2, fu3) and the second thoracic phragma (phl, shown in Fig. 1) developed growth layers. However, the third furca shows the most distinct banding (Fig. 2).

The correspondence between fly age and growth band was tested using three homozygous strains of parthenogenetic *D. mercatorum* supplied by A. Templeton (6). The results are shown in Table 1. We analyzed this by a 3-way ANOVA with unequal subclasses using BMDP. Genetic differences between strains do not affect the banding ( $F=0.68$ ,  $P>.50$ ). Temperature, however, has an effect ( $F=4.15$ ,  $P<.05$ ). The banding was most distinct, and age correspondence closest, with a 22 to 14.5°C day to night temperature fluctuation. It may be important that the 22 to 14.5°C range is close to that experienced by the strains in nature. The actual fly age corresponded closely with the number of bands ( $F$  to fit the age effect = 48.89,  $p<.01$ ). Fifty percent of the flies from the 22 to 14.5°C regime were correctly aged. The other 50% were only one day too high or too low. An experiment testing correlation between age and bands on 7-12 day old flies was not as successful. While a 12 day old fly was correctly identified once, most flies had a maximum of 8 growth bands.

The aging method is thus limited to young flies. Yet, the method is important because it permits age determination up to and into sexual maturity. The method is now being applied to ask a variety of questions about age structure of natural populations. In particular, we are interested in age-related dispersal. To date, 10 species have been successfully aged, including 8 repleta species, *D. melanogaster*, and *D. mimica* (a Hawaiian species).

Table 1. Comparison of actual age with estimated age in three strains of *D. mercatorum* maintained in three day:night temperature regimes.

Strain	Actual age (days)	Estimated age		
		22°C	22°-14.5°C	22°-5°C
O3-IM	1	2	2	4
	2	5	2	3
	4	6	3	5
	6	7	6	7
RSS18-IM	1	1	1	2
	2	5	3	3
	4	5	5	4
	6	7	6	6
S11-IM	1	2	2	2
	2	3	2	3
	4	5	5	6
	6	6	6	6

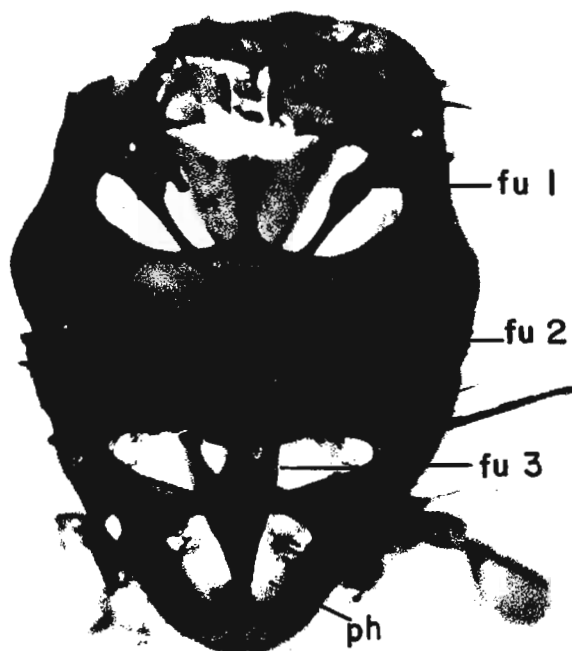


Fig. 1. Dorsal aspect of ventral thorax of *D. mercatorum* showing furcas 1,2,3 (fu1,fu2, fu3) and thoracic phragma (ph).

Fig. 2. 800X DIC micrograph of a portion of furca 3. 12 bands are visible beyond the eclosion layer (E).

References: (1) Supported in part by N.S.F. DEB-76-19879 to J.S. Johnston; (2) Van Valen, L. and P. Van Valen 1971, DIS 46:125; (3) Van Valen, L. 1973, DIS 50:110; (4) Schlein, Y. and N.G. Gratz 1972, Age determination of some flies and mosquitos by daily growth layers of skeletal apodemes, Bull. W.H.O. 47:71-76; (5) Paragon C. & C. Co., Inc., Bronx, NY 10454; (6) Templeton, A.R., H.L. Carson and C.R. Sing 1976, Genetics 82:527-542.

Kaji, S. and Y. Ushioda, Konan University, Kobe, Japan. The duration of cell cycle in the development of the Bar eye disc cells in vitro.

The experiments were carried out to determine the cell growth cycle of Bar and Oregon-R eye disc cells in the different larval stages under in vitro culture.

Schneider's culture medium. Then, they were placed in EDTA-trypsin-collagenase solution (80 mg NaCl, 3 mg KCl, 3 mg NaHCO<sub>3</sub>, 1 mg NaH<sub>2</sub>PO<sub>4</sub>, 5 mg glucose, 2 mg EDTA, 5 mg trypsin, 25 mg collagenase per 10 ml distilled water) for 30 minutes at room temperature. Dissociated cells were rinsed in culture medium, and then treated with colcemid (0.06 µg/ml) for 90 minutes. Colcemid is used in mitotic inhibitors for synchronization of the cultivated cells. After this treatment, the cell suspensions were carefully rinsed with culture medium, and then exposed with <sup>3</sup>H-thymidine (sp. act. 5.0 Ci/mM, 10 µCi/ml) for 40 minutes. After incubation with the tracer the cells were rinsed and further incubated for 1, 2, 4, 8, 12, 24 and 48 hrs in culture medium. Thereafter, cells were examined by means of autoradiography as described previously (Kaji and Ushioda 1979).

Table 1. <sup>3</sup>H-thymidine incorporation into the 70 h Oregon-R and Bar eye disc cells after exposed to colcemid in vitro.

		Duration of culture (hr)							
		0	1	2	4	8	12	24	48
No. grains per cell	Oregon-R	6.02	7.21	7.35	7.66	4.02	2.30	1.16	0.33
	Bar	5.24	6.49	6.78	6.65	3.15	1.89	0.92	0.41

Table 2. <sup>3</sup>H-thymidine incorporation into the 85 hr Oregon-R and Bar eye disc cells after exposed to colcemid in vitro.

		Duration of culture (hr)							
		0	1	2	4	8	12	24	48
No. grains per cell	Oregon-R	5.48	6.10	7.18	6.52	3.76	2.50	1.28	0.68
	Bar	4.22	5.14	5.13	5.06	3.71	2.28	1.01	0.62

Table 3. <sup>3</sup>H-thymidine incorporation into the 95 hr Oregon-R and Bar eye disc cells after exposed to colcemid in vitro.

		Duration of culture (hr)							
		0	1	2	4	8	12	24	48
No. grains per cell	Oregon-R	2.90	3.78	4.22	3.17	2.90	2.79	1.38	1.16
	Bar	3.12	3.52	4.40	3.36	3.17	2.51	1.63	1.49

have almost no division in the mature larval stage.

Results from these in vitro cell culture experiments indicate that in the Bar eye disc cells, dividing ability and duration of cell growth cycle of the photoreceptor cells is the same as that of the wild type.

References: Kaji, S. and Y. Ushioda 1979, Annot. Zool. Japon. 52:1.

Table 1 shows the rate of grain number in the 70 hr Oregon-R and Bar eye disc cells after incorporation of <sup>3</sup>H-thymidine. The number of grains in the cells decreased to about 50% for 8 hr, 25% for 12 to 24 hr, 12.5% for 24 to 48 hr culture. These results suggest that the duration of cell cycle in the 70 hr Oregon-R and Bar cells has an interval of about 8 hrs under in vitro conditions.

In the 85 hr cells, the incorporation rate decreased to about 50% for 12 hrs after tracer exposure (Table 2). It can be assumed that the 85 hr cell growth cycle was a longer interval than that of the 70 hr cells.

In the 95 hr mature larval eye disc cells, incorporation was about half the amount of the 70 hr cells. The grain number decreased to 50% for 24 hr culture. Thereafter, no change was seen until the 48 hour post-labeling time (Table 3). These results suggest that eye disc cells



Kamping, A. and W. van Delden. University of Groningen, The Netherlands. The  $Adh^{nl}$  mutant of *Drosophila melanogaster*.

homozygous for the  $Adh$ -positive alleles F, S and D. The banding patterns for homozygotes and heterozygotes, obtained after polyacrylamide electrophoresis (Van Delden et al. 1975) are given in Figure 1. All hybrids between the  $nl$  strain and

Several alcohol dehydrogenase-negative mutants, which lack detectable ADH activity, have been isolated in *Drosophila melanogaster*. We investigated the  $Adh^{nl}(nl)$  mutant (Grell et al. 1968) by crossing homozygous  $nl$  mutants with strains

the  $Adh$ -positive strains show two bands: the parental band of the  $Adh$ -positive parent and a hybrid band. From the position of the hybrid bands the electrophoretic mobility of the homozygous  $nl$  mutant can be deduced. This position is indicated in Figure 1 by "00". Though the  $nl$  mutant is derived by EMS treatment from an SS positive strain (Grell et al. 1968), the position of the  $nl$  band is not S-like, but differs from all three parental  $Adh$ -positive bands (F, S and D). Such a

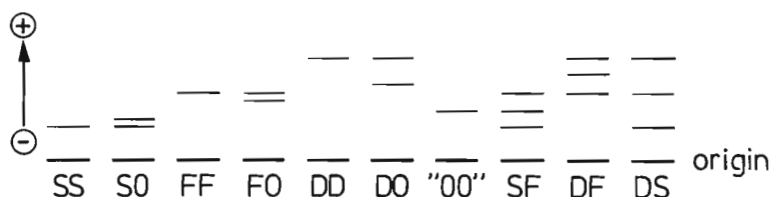


Figure 1. Position of electrophoretic banding patterns of  $Adh$  variants.

position, different from the original strain, was also observed by Schwartz and Sofer (1976) for the  $Adh^{nl}$  mutant. It must be assumed that the  $nl$  mutation has caused, in addition to loss of ADH activity, a change in charge.

References: Grell, E.H., K.B. Jacobson and J.B. Murphy 1968, Ann. New York Acad. Sci. 151:441-455; Schwartz, M. and W. Sofer 1976, Genetics 83:125-136; Van Delden, W., A. Kamping and H. van Kijk 1975, Experientia 31:418-419.

Kaur, P. and R. Parkash. G.N.D. University, Amritsar, India. Ontogeny of esterase in *Zaprionus paravittiger*.

Esterase isozymes have revealed stage specificity during different developmental stages in *Zaprionus paravittiger* (Fig. 1). Developmental esterase patterns of *Z. paravittiger* form two groups: Group I (Est-1 to Est-4) and Group II (Est-5 to Est-7). The Group I esterases persist throughout all the developmental stages and are localized in the different regions of the gut. These are suggested to participate in the breakdown of lipids with smaller fatty acid chains or other externally derived plant or animal material rich in sterols and organic compounds having ester bonds, thereby producing energy for other metabolic processes. Group II esterases are confined to late larval and pupal stages and fail to persist in the adult. These are suggested to control metamorphic events by modification of the specific insect hormones. One of the esterases (Est-5) has been recovered in the integument of the 3rd instar larva as well as in the puparium. It is suggested that this esterase might modify cuticle or may hydrolyze the puparium wall thereby helping in escape of the imago. This esterase has been found to be highly thermostable and retains enzyme activity for over a week period.

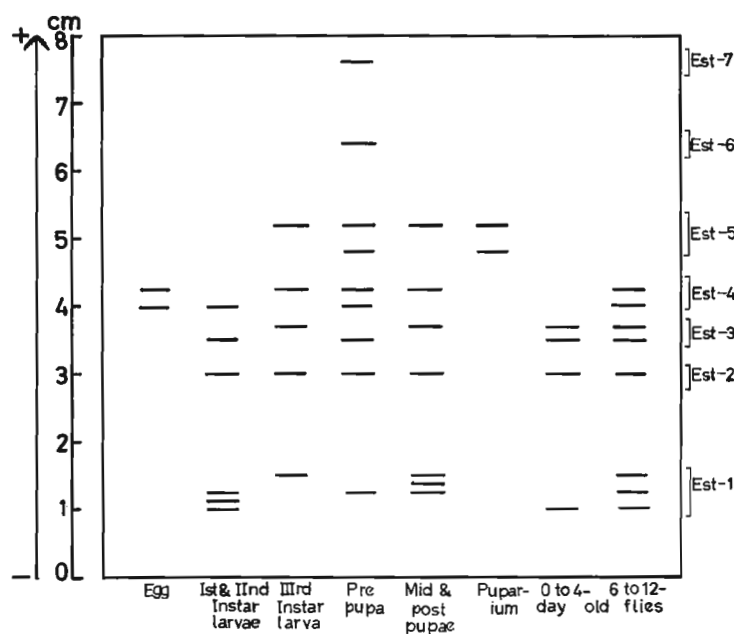


Fig. 1. Esterase isozyme patterns during development of *Zaprionus paravittiger*.



Kato, S. and Y. Arai, Josai Dental University, Sakado-shi, Saitama-ken, Japan. Configurational determination of neopterin from sepia mutant of *D. melanogaster*.

was reported in *D. melanogaster* (Fan and Brown 1976) as in *E. coli* (Burg and Brown 1968) and golden hamster (Fukushima et al. 1975), which catalyzes the formation of dihydro neopterin (2-amino-4-hydroxy-6-trihydroxypropyl-pteridine) triphosphate from GTP. And in insects, neopterin-compound might also be a key intermediate in the formation of pteridine from purine, though neopterin was not found in insects except in honey bees (Rembold and Bushmann 1963).

We have found and isolated neopterin from the sepia mutant of *D. melanogaster* as reported in DIS 51 (Kato and Arai 1974). There are four stereoisomers of neopterin: D- and L- erythro-neopterin and D- and L- threo-neopterin (Fig. 1).

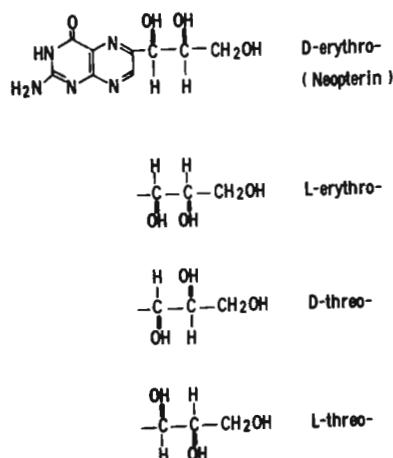


Fig. 1. Four isomers of neopterin.

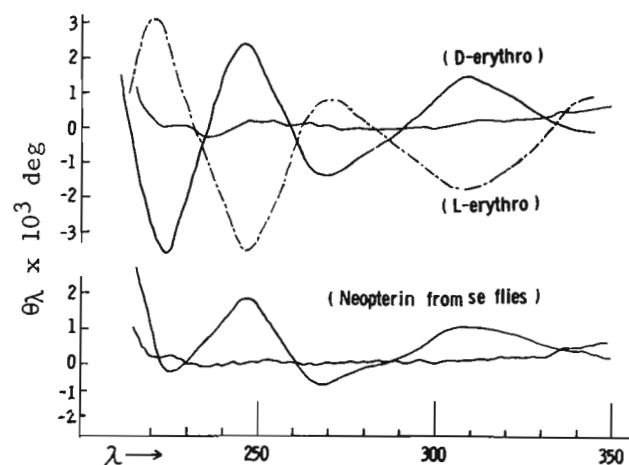


Fig. 2. CD-spectra of authentic D- and L-erythro-neopterin ( $1.4 \times 10^{-4} M$ ) and of neopterin from se flies. (in 0.1 N HCl, 20°C, 5 mm cell lightpath, scale 1)

Quite a large amount of unconjugated pteridines are found in *D. melanogaster*, and some of these pteridines were observed to be transformed from purine. But it is not fully clarified yet by what pathways these pteridines are synthesized in insects. Recently an enzyme, cyclohydrolase,

was reported in *D. melanogaster* (Fan and Brown 1976) as in *E. coli* (Burg and Brown 1968) and golden hamster (Fukushima et al. 1975), which catalyzes the formation of dihydro neopterin (2-amino-4-hydroxy-6-trihydroxypropyl-pteridine) triphosphate from GTP. And in insects, neopterin-compound might also be a key intermediate in the formation of pteridine from purine, though neopterin was not found in insects except in honey bees (Rembold and Bushmann 1963).

We have found and isolated neopterin from the sepia mutant of *D. melanogaster* as reported in DIS 51 (Kato and Arai 1974). There are four stereoisomers of neopterin: D- and L- erythro-neopterin and D- and L- threo-neopterin (Fig. 1). Neopterin obtained from sepia flies was shown to be erythro- form by paper chromatography (Kato and Arai 1974). We tried, in this study, to determine the configuration (D- or L-) of neopterin isolated from se flies by using circular dichroism (CD). Neopterin was isolated from se flies in higher purity than in a previous report (Kato and Arai 1974) by the modified method. 100 g of se flies were extracted with 5 vol of 50% ethanol at 90°C. After the column chromatographies on Ectercel-cellulose (7x30 cm, pH 7.5) and phospho-cellulose (5x48.5 cm), the blue fluorescent fraction mainly containing biopterin was collected and was applied to paper chromatography using 36 sheets of Toyo filter paper No. 50 (40x40 cm) in the ascending method with n-propanol/ethylacetate/water (7:1:2 v/v). The neopterin area was cut off and suspended in distilled water. The extract was then applied to ethanolized-cellulose column (3x24 cm) as reported (Kato and Arai 1974). The neopterin fraction was obtained from the last column chromatography on Sephadex G-25 fine (2x50 cm). About 40 µg of neopterin was finally yielded.

CD measurements were obtained with Automatic Recording Spectropolarimeter J-20 (Japan Spectroscopic Co. Ltd.). The measurements were made at 20°C in cells with 5 mm lightpaths on Scale 1. Authentic neopterins ( $1.4 \times 10^{-4} M$ ) and neopterin from se flies were assayed in final 0.1 N HCl solution. Fig. 2 shows their CD spectra. D-erythro-neopterin has positive bands at 247 nm and 310 nm, and negative bands at 223 nm and 269 nm. On the other hand, L-erythro-neopterin shows the mirror image of the CD pattern of D-erythro- form (Fig. 2). Therefore, neopterin in se flies is determined to be D-erythro-neopterin from this measurement.

L-threo-neopterin besides D-erythro-neopterin is reported in naturally occurring materials. It is the D-erythro- form of dihydroneopterin triphosphate that is synthesized from GTP by cyclohydrolase in *D. melanogaster*, and this product is further converted to sepiapterin by other enzyme systems (Fan et al. 1975). The finding of D-erythro-neopterin in *D. melanogaster* in this study really supports that neopterin

(D-erythro-dihydroneopterin triphosphate) is an important intermediate when unconjugated pteridines are formed from GTP.

References: Burg, A.W. and B.M. Brown 1968, J. Biol. Chem. 243:2439; Fan, C.L., G.C. Krivi and G.M. Brown 1975, Biochem. Biophys. Res. Commun. 67:1047; Fan, C.L. and G.M. Brown 1976, Biochem. Genet. 14:259; Fukushima, K., I. Eto, D. Saliba and T. Shiota 1975, Biochem. Biophys. Res. Commun. 65:644; Kato, S. and Y. Arai 1974, DIS 51:70; Rembold, H. and L. Bushmann 1963, Justus Liebigs Annln. Chem. 662:1406.

Kaurov, B. A. Institute of Medical Genetics AMS USSR, Moscow, USSR. The effect of trypsin on "survivability" of imaginal disks of *D. melanogaster*.

of the "Berlin wild" line at the age of 72 hours and were put into trypsin solutions of different dilutions. 0.25% trypsin solution was used as an initial material to prepare the working solutions of 1:1, 1:2, and 1:8 in physiologic Ringer's solution (NaCl 7.500 g, KCl 0.287 g,  $\text{CaCl}_2 \cdot \text{H}_2\text{O}$  0.287 g, distilled water to 1.0 liter). After 5 minutes' treatment of imaginal disks by trypsin solution at room temperature they were washed for one minute with 20% solution of bull serum prepared on the basis of Ringer's solution. Using the standard techniques of transplantation of imaginal disks (1), the latter were transplanted into larvae of the same age and line. In the control, selected disks of the same age were exposed for 5-10 minutes in Ringer's solution and then transplanted into the larval hosts.

For further analysis only those larvae were used that survived for 4 hours after transplantation, since the mortality in the first hours is presumed to be conditional on the imperfection of surgical techniques (2). The larval hosts with implanted disks were placed in tubes with the standard forage for *Drosophila* (agar, raising, treacle) and kept at room temperature. Part of these larvae were allowed to pupate and imago emerged. The adult flies were dissected and the presence of disk-implants was established. These disks were examined under a microscope to determine the elements of leg tissue. Results are given in Table 1.

Table 1. "Survivability" of leg imaginal disks after treatment with trypsin solution of different dilution.

Dilution of 0.025% trypsin solution	Number of surviving larvae	Number of adult flies (%)	Number of "survived" disks with regard to imago (%)	Number of "survived" disks with regard to larvae (%)
1:1	48	28.6	0.0	0.0
1:2	114	10.5	8.3	0.9
1:8	337	23.7	56.2	16.0
Control	791	40.0	55.0	22.3

This table shows that the increase of dilution of trypsin solution led to the increase of "survivability" of disks, both with regard to adult flies and with regard to surviving larvae; but at the same time this increase did not yield a concrete result in respect to the number of flies concerning surviving larvae. We are inclined to explain all this by unregistered technical conditions of operation rather than by peculiarities of the given trypsin dilution. In the selected disks we did not discover any significant qualitative difference (for example, appearance of allotypical elements) between the experimental and control groups.

References: (1) Ephrussi, B. and G. Beadle 1936, Amer. Nat. 70:218-225; (2) Shivertaker, L. 1970, DIS 45:188-189.

Kaurov, B.A. Institute of Medical Genetics AMS USSR, Moscow, USSR. Manifestation of mutation *singed* on the homoeotic limbs, caused by the action of homoeotic mutations *Nasobemia* and *aristapedia* at different temperatures.

The mutation *singed* (ns,1-21.0) (twisted bristles) is manifested unequally on bristles of different sizes. Specifically, its expressivity is more marked on large bristles in comparison with small ones. As a result of the effects of some mutations on the homoeotic structures (1,2,

3,4) it was interesting to find out whether the feature of behavior of mutation *sn* on the homoeotic structures is retained at different temperatures. For this purpose homoeotic mutations *Ns* (3-48.0) and *aristopedia* (*ss<sup>a</sup>*, 3-58.5) (its three alleles: *ss<sup>ak</sup>*, *ss<sup>ax</sup>* and *ss<sup>a40a</sup>*) (transform antennae to the legs of mesothoracic type) were used. The number of large bristles was significantly more in mutants *Ns*. The double mutants *sn;Ns* and *sn;ss<sup>a</sup>* obtained at 17, 25 and 28°C and single mutants *sn*, *Ns* and *ss<sup>a</sup>* were studied by binocular microscope (increase 12.5 x 4.0). 100 individuals of every genotype were tested to estimate penetrance, 64 to estimate expressivity.

The effect of temperature on the degree of twisting of bristles in mutants *sn* was not detected. Twisting of bristles in mutants *Ns* and *ss<sup>a</sup>* was not found at different temperatures. Twisting of bristles on thoracic legs and antennal homoeotic limbs was observed at the interaction of homoeotic mutations *Ns* and *ss<sup>a</sup>* (its three alleles) with the mutation *sn* at all temperatures (16, 25 and 28°C). The complete penetrance of this effect was observed. The degree of its expression on homoeotic structures was identical to that on the corresponding segments of thoracic legs. In particular, twisting of bristles was significantly more marked in double mutants *sn;Ns* on the homoeotic structures than in mutants *sn;ss<sup>a</sup>*. It can be explained by the predominance of large bristles on homoeotic limbs in mutants *sn;Ns*. The effect of temperature on the degree of twisting of bristles in double mutants was not found. The coincidence of expressivity of mutation *sn* on homoeotic structures with that on the corresponding segments of thoracic legs independent of temperature points out the morphogenetic relationship of elements (bristles) of homoeotic and normal structures.

References: (1) Braun, W. 1940, *Genetics* 25:143-149; (2) Ouweneel, W. 1970, *Genetics* 41: 1-20; (3) Kaurov, B.A., V.I. Ivanov and V.A. Mglinetz 1976, *Genetika* (USSR) 12:75-81; (4) Kaurov, B.A., V.I. Ivanov and V.A. Mglinetz 1978, *Genetika* (USSR) 14:306-312.

Kemphues, K.J. and T.C. Kaufman. Indiana University, Bloomington, Indiana. Two-dimensional gel analysis of total proteins from X/O, X/Y, X/Y/Y, X/Y<sup>s</sup> and X/Y<sup>L</sup> testes from *D. melanogaster*.

An attempt to identify proteins specific to the Y chromosome of *D. melanogaster* utilized the NEPHGE two-dimensional gel electrophoresis system of O'Farrell (1977), as modified by Waring (1978), to display total proteins extracted from testes. These gels offer the best one-step method for visualizing a large array of proteins. In

these experiments, resolution of more than 300 spots from total proteins of [<sup>35</sup>S] methionine labeled adult testes was possible.

Careful comparisons of autoradiographs of gels of labeled proteins from X/O, X/Y, X/Y/Y, X/Y<sup>s</sup> and X/Y<sup>L</sup> testes were made. These comparisons revealed no consistent differences in the two-dimensional pattern of proteins from testes of the various genotypes, other than an increase in the intensity of labeling of five spots from X/Y/Y testes relative to other genotypes. No protein differences, either qualitative or quantitative, were found between the autoradiographs of X/O and X/Y testes.

One basic protein of 55,000 daltons, however, did fail to appear in gels of testes from two of the four X/O stocks examined. A two-dimensional gel analysis of protein from X/Y testes at various developmental stages (24 hour intervals, starting at 72 hours after egg deposition) showed that the 55,000 dalton protein was not synthesized until late in development (220 hours; late pupa). The late synthesis of this protein suggests that some triggering event is necessary prior to synthesis. Because X/O spermatids degenerate before maturation (Kiefer 1970), it is possible that the 55,000 dalton protein appears in some X/O testes and not in others because this degeneration could begin either before or after this triggering event due to differences in genetic backgrounds or culture conditions.

These results indicate that if the Y chromosome fertility factors are expressed as proteins, the detection of these proteins will require more elaborate techniques than those described here. These experiments also demonstrate that the Y chromosome functions in a subtle manner which does not involve the regulation of synthesis of the major protein components of spermiogenesis. However, it may be possible that the Y specific proteins contain no methionine and that other labeling procedures will allow for their detection.

References: Kiefer, B.I. 1970, *J. Cell Sci.* 6:117-194; O'Farrell, P.Z. et al. 1977, *Cell* 12:1133-1142; Waring, G.I. et al. 1978, *Dev. Biol.* 66:197-206.

Kerver, J.W.M. and R. Bijlsma. University of Groningen, The Netherlands. The effect of DDT on the activity of the pentose phosphate cycle in *D. melanogaster*.

The problem that insects develop resistance to insecticides has gained much attention in the years 1950-1970. Especially the resistance to chlorohydrocarbon insecticides—of which DDT (1,1,1-trichloro-2,2-bis(p-phenyl)-ethane) is the best known representative—is well studied.

(For a review see Perry and Agosin 1974). The resistance can be obtained by means of different mechanisms such as: avoidance of the insecticide by the insect, changes in penetration through the cuticula and increased detoxification by degradation of the insecticide. The degradation of chlorohydrocarbon insecticides is found to occur in many different ways in insects. In *D. melanogaster* two major ways of DDT degradation are regarded as important: degradation via hydroxylation to dicofol (2,2-bis(p-chlorophenyl)-1,1,1-trichloroethanol) and via dehydrochlorination to DDE (2,2-bis(p-chlorophenyl)-1,1-dichloroethylene). These reactions both require energy in the form of NADPH which is thought to be supplied by the pentose phosphate cycle. This may imply that treatment of *Drosophila* with DDT accelerates the activity of the pentose cycle. This was investigated by studying the effect of DDT treatment on flies by measuring the activity of two enzymes of the pentose cycle: glucose-6-phosphate-dehydrogenase (G6PD) and 6-phosphogluconate-dehydrogenase (6PGD).

Adult flies (5-10 days old) were transferred to food supplemented with 0.005% DDT and the in vitro enzyme activities of G6PD and 6PGD were measured (for method see Bijlsma 1978) at subsequent days and compared with the activities of flies on food without DDT (control). Fig. 1 shows the ratio of the activity of flies on DDT food and the activity of flies on control food (DDT/control) in the course of the experiment. This figure also shows the percentage dead flies on DDT-supplemented food in the course of time (on control food hardly any flies died). It is evident that the activity of both enzymes increases in flies on DDT, especially in females, and after 96 hours this increase is significant ( $P < .01$ ) in all cases. During the experiment quite a number of flies died and it is therefore possible that the change in activity of both G6PD and 6PGD is due to selection in favor of flies with high activity, rather than to an increase of enzyme activity in the individual flies. Therefore the experiment was repeated for lower concentrations of DDT: 0.0025% and 0.001%. On these concentrations a lower number

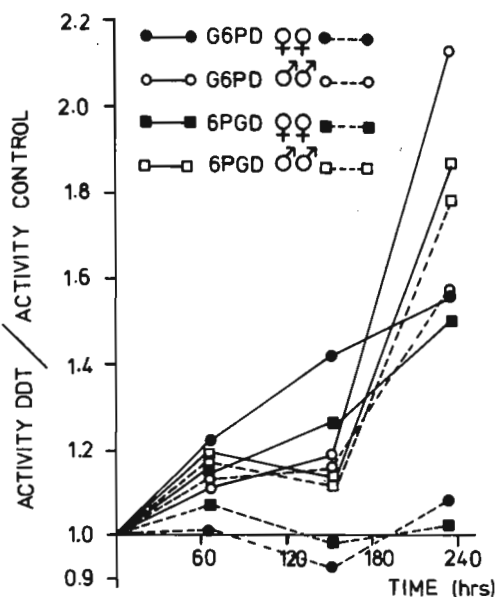


Fig. 1. The ratio of activity of flies on DDT-supplemented food (0.005% DDT) and the activity of flies on normal food at subsequent times of treatment (solid lines), together with the percentage flies that died at the same times (broken lines).

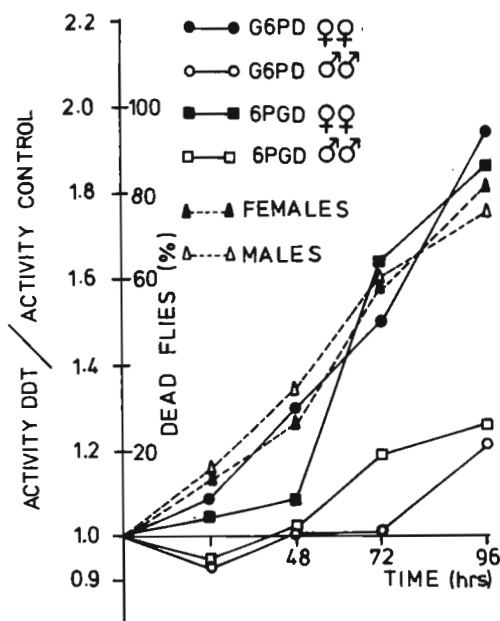


Fig. 2. The ratio of the activity of flies on DDT-supplemented food and the activity of flies on normal food at subsequent times of treatment. The data are given for two concentrations of DDT: 0.0025% (solid lines) and 0.001% (broken lines).

of flies died during the experiment, less than 40% on 0.0025% DDT and less than 10% on 0.001% DDT. The results are shown in Fig. 2. Also in this experiment a significant increase in the activity of G6PD and 6PGD is found in flies on DDT, though at the lowest concentration only the males respond. In spite of some differences in reaction between males and females it is evident that DDT treatment can strongly increase the activity of G6PD and 6PGD in adults. Preliminary experiments suggest that this also holds for larvae.

References: Bijlsma, R. 1978, Genet. Res. Camb. 31:227-237; Perry, A.S. and M. Agosin 1974, The Physiology of Insecta, vol. VI, Academic Press, New York.

King, R.C. and B.D. Buckles, Jr. Northwestern University, Evanston, Illinois. Three mutations blocking early steps in *Drosophila* oogenesis: *fs(4)34*, *fs(2)A16*, and *fs(1)231M*.

The *fs(4)34* mutation was recovered by B. Hochman from a male collected in December of 1963 from a natural population of *Drosophila melanogaster* living in Lake County, Florida. Hochman (1972) reported that the mutant resided on the 4th chromosome, and it is still the only female sterile gene known for this microchromosome.

Two major deficiencies, *Df(4)M* and *Df(4)G*, allow the right arm to be partitioned into three unequal parts (Hochman, 1974). Hochman showed that *fs34* is not included in either deficiency, and therefore it probably resides somewhere between the middle of subdivision 102B and the beginning of subdivision 102E (see King, 1975, his Fig. 1). The heterozygotes used for the maintenance of the stock population are of genotype *fs(4)34/ci<sup>D</sup>*. The *fs(2)A16* mutation was induced by Bakken (1973) using EMS. She studied ovarian whole mounts from homozygotes and concluded that the ovarioles lacked clear cut germaria. The mutation has not yet localized on chromosome 2. Heterozygotes have the genotype *fs(2)A16/SM1*.

The results of our electron microscopic studies are summarized in the accompanying illustration of sections from 2 day ovaries (Fig. 1). The *fs34* ovariole begins with a normal terminal filament which is anchored to the tubular epithelial sheath. The anterior end of the germarium contains abnormally large numbers of oogonial cells and lacks clusters of cysto-

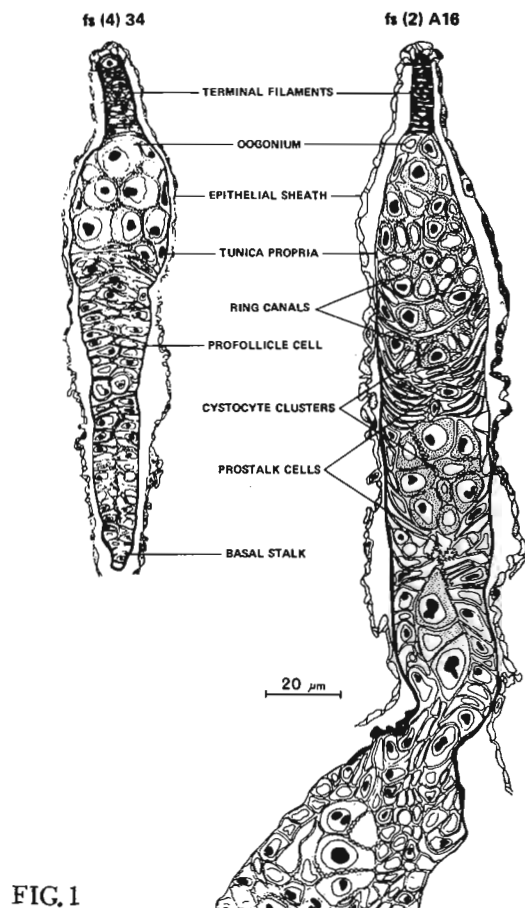


FIG. 1



FIG. 2

cytes. The remainder of the germarium consists of a solid cylinder made up of profollicle cells which ends blindly as a basal stalk. The entire structure is coated by an acellular tunica propria. In the fsAl6 germarium clusters of cystocytes are abundant, and the ring canals that connect them are often included in sections. Clustered cystocytes are surrounded by wedge-shaped follicle cells. However, adjacent clusters are not connected by inter-follicle stalks, and consequently a conventional moniliform vitellarium is not observed. The fault seems to lie with the prostalk cells which fail to interdigitate. The posterior end of the ovariole is filled with fusing follicles. Thus in the case of the fs34, oogenesis appears to be blocked at the point where oogonia are converted to cystoblasts. The developmental block is later in fsAl6 ovaries and seems to involve the mesodermal cells at the base of each germarium. Since these cells fail to form the stalks that allow egg chambers to bud off continuously from the germarium, the mutant has been nicknamed "stalkless".

The fs(1)231 mutation was induced by Gans et al. (1975). The fs(1)14-97 mutation was recovered subsequently by J.D. Mohler using previously described techniques (Mohler, 1977). King et al. (1978) reported that fs231 belongs to the ovarian tumor class. Cystocytes require the product of the fs231<sup>+</sup> gene for cytokinesis to be arrested. Cystocytes that cleave completely undergo supernumerary divisions and generate ovarian tumors. The B138L/B170R deficiency includes fs231. According to Kambyzellis (1977) females homozygous for fsl4-97 have rudimentary ovaries and accumulate large amounts of vitellogenin in their hemolymph.

The cross fs231 v<sup>24</sup>/FM3 ♀ X y cv fsl4-97 v f ♂ produces sterile daughters of genotype y cv fsl4-97 v f / ++ fs231 v<sup>24</sup>+. The cross B138L/B170R/FM7 ♀ X y cv fsl4-97 v f ♂ produces fsl4-97/B138L/B170R females that are also sterile. Fig. 2 shows a light micrograph of a section through the ovary of an 11 day old 231/14-97 female. Tumors containing hundreds to thousands of cystocytes are present. Thus fs231 and fsl4-97 are alleles, and we refer to them as fs231G and fs231M, respectively.

References: Bakken, A.H. 1973, Dev. Biol. 33:100-122; Gans, M., C. Audit and M. Masson 1975, Genetics 81:683-704; Hochman, B. 1972, DIS 48:17; 1974 Cold Spring Harbor Symp. Quant. Biol. 38:581-589; Kambyzellis, M.P. 1977, Am. Zoologist 17:535-549; King, R.C. 1975, Handbook of Genetics 3:625-652; King et al. 1978, Int. J. Morphol. Embryol. 7:359-375; Mohler, J.D. 1977, Genetics 85:259-272.

Kiss, I. and J. Szabad. Institute of Genetics, Biological Research Center of the Hungarian Academy of Sciences, Szeged, Hungary. Characteristics of some new X-linked pupal lethals of *D. melanogaster*.

A detailed study on lethal mutants which show a normal larval development but a lack or a delay in puparium formation has been made recently in our laboratory (Kiss et al. 1978). Similar experiments were made also with other mutants having a significantly longer than normal larval

development time. In these experiments we made a general characterization of the mutant phenotypes and tested the autonomous expression of the non-pupariating character in gynander mosaics and by implanting wild-type ring glands into mutant larvae. For the technical details, see Kiss et al. (1978). The mutant chromosomes were marked with y and w.

Table 1. General characteristics of the mutants

Mutant	Developmental time <sup>1</sup> (in days)	Frequency of puparium formation (%)	Imaginal discs		Ring gland size <sup>2</sup>	Metamorphic capabilities			Map position
			Size	Folding		Pre-pupal molt	Pupal molt	Histo-lysis	
ℓ/1/1-43	10;12	25	very small	∅	small	+	∅	+	26.3
ℓ/1/1-45	7;8	33	very small	∅	small	+3	+3	+	58.7
ℓ/1/1-48	8;10	4	small	undeveloped	small	+3	+3	+	66.4
ℓ/1/1-74	11;15	2	small	undeveloped	normal	+	+	+	18.5

<sup>1</sup>Days until reaching the size of a mature wild-type larva; days until the beginning of puparium formation.

<sup>2</sup>At the time of puparium formation.

<sup>3</sup>Data refer to the abdomen only. No differentiation of the head and thorax was observed (see also Fig. 1)

Abbreviations used: + = yes; ∅ = no.



General characteristics of the mutants are summarized in Table 1. Common features of these mutants were the delayed larval development, the rudimentary appearance of their imaginal discs and the formation of abnormal puparia. The puparia never contracted properly, and tanning and sclerotization of the cuticle was unequal and insufficient. The undeveloped imaginal discs seemed to prevent the normal differentiation of head and thorax regions in  $\ell/1/1-45$  and  $\ell/1/1-48$  puparia: the pupal molt occurred only on the abdomen and a small, undifferentiated rudiment was found in the place of the head and thorax (Fig. 1).  $\ell/1/1-74$  larvae had an extremely small amount of fat body.

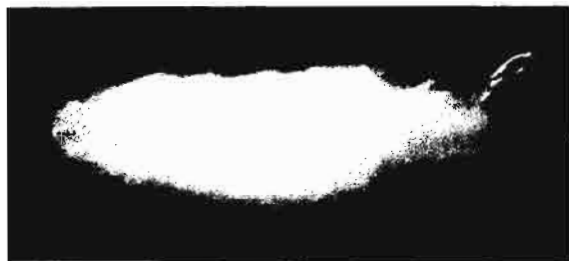


Fig. 1. Four day old "pupa" of  $\ell/1/1-45$ . The abdomen is properly formed and covered by prepupal and pupal cuticles. There are no identifiable structures differentiated in the head and thorax region and the degenerating rudiments have no cuticle.

Autonomous expression of the mutant phenotype was studied in gynander mosaics and in ring implantation experiments. The results of these tests are shown by Tables 2 and 3.

As for the action of the mutant genes, the observations suggest the following interpretation: the genes are already expressed during the larval period; therefore, the larval development is slowed down in the mutants (Table 1). The lack of larval-pupal mosaics (Table 2) suggests that the delayed pupariation character is not autonomous in the larval epiderm, probably being an indirect consequence of the mutant gene action in all the four mutants.

The implantation of normal ring glands (the main source of ecdysone in the larva) into mutant larvae caused a significant "acceleration" of puparium formation only in the case of  $\ell/1/1-43$  (Table 3); this probably means that in this mutant the ecdysone concentration is too low to induce pupariation of the mature larva. However, it cannot be an "ecdysoneless"

Table 2. Results of the gynander mosaicism test

Mutant	$\frac{y}{R/1/2} \frac{w}{gynander\ mosaics}$		
	Found		Expected <sup>1</sup> Viability <sup>2</sup>
	Adult	Larval-Puparial	
$\ell/1/1-43$	0	0	34 <0.029
$\ell/1/1-45$	0	0	61 <0.016
$\ell/1/1-48$	0	0	46 <0.022
$\ell/1/1-74$	0	0	48 <0.021

<sup>1</sup> Number of the adult gynanders expected =  $1.7 \times$  no. of  $\frac{Binsn}{R/1/2}$  gynanders found (Kiss et al. 1978).

<sup>2</sup> Gynander viability <  $\frac{1}{no. \text{ of gynanders expected}}$

mutant; otherwise it should be lethal around the first larval molt (Garen et al. 1977). In the other two mutants tested the cause of the delay in pupariation is not simply a low, subthreshold ecdysone titre.

The lack of the adult gynanders in all the mutants (Table 2) suggests that viable mosaic combinations are very rare or missing and might be explained by assuming the lethal focus being very large or multiple.

References: Garen, A., L. Kauvar and J.-A. Lepesant 1977, Proc. Nat. Acad. Sci. (Wash.) 74:5099-5103; Kiss, I., J. Szabad and J. Major 1978, Molec. Gen. Genet. 164:77-83.

Table 3. Effect of implanting wild type ring glands into mutant larvae

Mutant host	No. of larvae injected		Time (in days) till the beginning of puparium formation		% pupariation in operated animals until the beginning of spontaneous pupariation in the control	% pupariation within 5 days following the operation	
	Experi- mental	Control	Experi- mental	Control		Experi- mental	Control
<i>ℓ</i> /1/1-43	17	16	1	5	59	59	6
<i>ℓ</i> /1/1-45	39	26	1	1	-	15	4
<i>ℓ</i> /1/1-48	44	28	2	4	14	16	11

Köhler, W., J. Krause and A. Michutta.  
Institut für Genetik der Freien Universität, Berlin, Germany. Heritability of phototactic behavior in *Drosophila*.

Heritability is an estimate of the genetic component of the observed phenotypic variance and may be computed in a number of different ways. We chose the estimates of the realized heritability ( $h^2_r$ , Table 1) of 6 selection lines of *D. melanogaster* and *D. simulans*, the heritability from regression of offspring on mid-parent values ( $h^2_{PO}$ , Table 2), and the heritability in the narrow and broad sense ( $h^2_n$ ,  $h^2_b$ , Table 3) from a  $4 \times 4$  diallel analysis with two selected photonegative and two unselected photopositive strains (Falconer, 1970; Mather and Jinks, 1971).

The base populations of all selection lines were established by reciprocally crossing a wild-type laboratory strain and a marker strain carrying the X-chromosomal markers vermilion and garnet, or white. These markers were fixed in the photonegative lines during the first 3 or at most 7 generations (Köhler, 1977). Estimating the realized heritabilities after 7 and 10 generations of selection, respectively, these varied between 3.5% and 21.7% in *D. melanogaster* and between 6.1% and 11.5% in *D. simulans*. After 22 and 30 generations of selection respectively the estimated values generally show a slight decrease. Above all this may depend on ceasing of the selection response and the artificial selection limit in a Hirsch-Hadler maze.

The heritabilities of phototactic behavior which result from the regression of offspring on mid-parent values (Table 2) are lying in the same range but their standard errors (SE) are very high so that there is no significant difference from zero.

Table 1. Realized heritabilities ( $h^2_r$ ) of negative phototactic behavior estimated from 6 different selection lines in *D. melanogaster* (1-4) and *D. simulans* (5-6).

Strain	Marker	Fixed in gener.	$h^2_r$ (%)		Number of gen.	$h^2_r$ (%)		Number of gen.
			♂	♀		♂	♀	
1 K1NEG	v g	6	11.4 ***	21.7 ***	7	1.9 *	0.9 n.s.	22
2 K2NEG	v g	7	6.4 **	11.9 ***	7	4.3 ***	5.9 ***	22
3 N2	w	3	5.9 **	3.5 *	10	7.4 ***	5.1 ***	30
4 N3	w	3	3.7 n.s.	5.5 *	10	2.6 ***	2.8 ***	30
5 N24	w	3	10.9 ***	11.3 ***	10	6.0 ***	7.8 ***	30
6 N34	w	3	6.1 *	11.5 **	10	5.2 ***	7.9 ***	30

Significant deviation from zero (t-test) is indicated by asterisks for an acceptance probability of 5% (\*), 1% (\*\*), or 0.1% (\*\*\*).

Table 2. Heritability of phototactic behavior estimated by the mid-parent - offspring regression method for 2 unselected wild-type strains of *D. m.*

	$h^2_{PO}$ (%)	SE
KOK	6.2	3.0
K2KON	3.2	4.5

All these values are in accordance with those summarized by Grossfield (in: Ashburner and Wright, 1978). He noted that the overall picture is "that heritabilities are low, decrease with the number of generations of selection and seem to be somewhat higher for maze studies than for open field designs". We would like to remark that it could in addition depend on the method of estimation because in our third design we got heritabilities much higher than those of Hadler, for example.

We used in our diallel analysis the selected lines 1 and 2 of Table 1 after about 90 generations of selection and 2 unselected photopositive laboratory strains. The various tests described in Mather and Jinks (1971) provided no reason to doubt the adequacy of the simple model and therefore the components of variation could be estimated.



Table 3. Components of variation and heritability of phototaxis in *D. melanogaster* estimated from a 4x4 diallel with 3 replicates in 6 different Hirsch-Hadler mazes with 4 light-dark choices. In the last row the pooled data were used (4x4 diallel with 18 replicates). Nomenclature according to Mather and Jinks (1971).

Maze	D	F	H1	H2	E	$\bar{uv}$	$h_n^2$	$h_b^2$	$h_{CA}^2$
KK1	2.50	-0.18	0.55	0.39	0.09	0.17	0.88	0.94	0.69
KK2	1.88	-0.11	0.46	0.32	0.08	0.17	0.87	0.94	0.68
KK3	1.46	-0.15	0.32	0.21	0.09	0.17	0.86	0.91	0.64
KK4	1.31	-0.46	0.18	0.13	0.13	0.17	0.85	0.88	0.53
KK5	1.50	-0.09	0.16	0.10	0.13	0.15	0.84	0.86	0.66
KK6	1.62	-0.23	0.76	0.52	0.09	0.17	0.83	0.94	0.55
Total	1.71	-0.21	0.39	0.27	0.10	0.17	0.86	0.91	0.63

$h_n^2$ ,  $h_b^2$  heritability in the narrow and broad sense,  $h_{CA}^2$  heritability of Crumpacker and Allard (computer program of Lee and Kalisikes, Univ. of Manitoba, Winnipeg)

It is clear cut that the additive component D compared with E (error) and H<sub>1</sub> and H<sub>2</sub> (dominance components) is extremely high. This leads to heritability estimates between 83% and 88% in the narrow sense and between 86% and 94% in the broad sense. There exist only slight differences between the estimates from the data in the 6 mazes so that these could be handled as one diallel with 18 replicates. The negative F values indicate that there are more recessive alleles present in the 4 lines than dominant alleles, irrespective of whether these have photopositive or photonegative effects. The average frequencies of the alleles for increasing or decreasing phototactic behavior are about 40% if we assume that they are equally distributed ( $\bar{uv} > 0.16$ ).

These extremely high heritabilities of a behavioral trait are probably a result of using selected and control lines in a diallel cross and therefore these estimates are not valid to describe the heritability of phototactic behavior in a normal population. Similarly the estimates of Hadler (1964) were computed from differences in the variances of selected ("isogenic") lines and their control. Furthermore, there exists a scale effect in the Hirsch-Hadler mazes (variance depends on the mean in a binomial distribution). In summary, heritabilities of phototactic behavior in *Drosophila* are low in general and the higher estimates may depend on the computational method in connection with the strains used.

References: Ashburner, M. and T.R.F. Wright 1978, *The Genetics and Biology of Drosophila*, Academic Press, New York; Falconer, D.S. 1970, *Quantitative Genetics*, Ronald Press, New York; Hadler, N.M. 1964, *Genetics* 50:1269-1277; Köhler, W. 1977, *Genetica* 47:93-104; Mather, K. and J.L. Jinks 1971, *Biometrical Genetics*, Chapman and Hall, London.

Krause, J., A. Michutta and W. Köhler.  
Institut für Genetik der Freien Universität, Berlin, Germany. Oviposition preferences in *D. melanogaster*.

Mainardi (1968) has shown that the females of *D. melanogaster* (Oregon) are able to (1) distinguish between food sites "scented" by previous exposure to adult males of their strain and intact ones, and (2) prefer to lay eggs in the former ones.

Ayala and Ayala (1968) have repeated Mainardi's experiment. They found that the females are able to recognize the previous presence of males, but their females showed an egg-laying

Table 1. Number of eggs scored during 12 hours by females and virgins of 2 different strains of D.m. In each experimental unit females could choose between 4 food vials, 2 scented by male presence and 2 unscented ones.

Strain	No.	Number of ♀♀ tested	Number of eggs			
			Scented		Unscented	
			1	2	3	4
Berlin wild ♀♀	1	10	12	215	60	141
	2	10	323	169	6	8
	3	10	266	132	46	60
	4	10	114	218	88	40
	5	9	195	196	0	11
Berlin wild ♂♂	1	8	40	21	7	0
	2	9	32	6	14	0
	3	6	58	1	0	0
	4	7	8	58	0	0
	5	6	21	0	0	0
Grunewald ♀♀	1	10	38	199	9	222
	2	10	342	70	105	24
	3	10	144	140	73	108
	4	10	111	209	58	83
	5	10	113	51	98	227
Grunewald ♂♂	1	9	39	0	0	0
	2	8	30	66	0	0
	3	9	55	0	0	0
	4	10	88	142	0	0
	5	10	17	17	0	0

Table 2. Pooled data of Table 1. Each distribution of eggs between scented and unscented vials was tested for a 1:1 relation.

Strain	Number of ♀♀ tested	Number of eggs		Chi <sup>2</sup>
		Scented	Unscented	
Berlin wild ♀♀	49	1840	460	828,0
	36	245	21	188,6
Grunewald ♀♀	50	1417	1007	69,4
	46	454	0	454,0

preference of scented cups is very strong for the virgins of both strains, whereby in nearly all cases, even within the two scented cups, one is clearly preferred. These results support the findings of Mainardi although our experimental design was different from hers.

References: Ayala, F.J. and M. Ayala 1969, DIS 44:120; Mainardi, M. 1968, Boll. Zool. 35:135-136; Mainardi, M. 1969, Boll. Zool. 36:101-103.

preference for the intact food vials. Mainardi (1969) could reproduce her results as well for Oregon and D.m. wild strains from Italy as for D.s.

We have recently investigated the egg-laying behavior of D.m. Our experiment was conducted with the strains Berlin wild (old laboratory wild strain) and Grunewald (freshly captured wild strain). Directly after emergence 10 virgin females and 10 pairs, respectively, were put into fresh food vials. They remained therein without removal of dead ones and without changing the food vials for 4 days. After this period the surviving virgins or inseminated females were used for the test. They were shortly anesthetized with CO<sub>2</sub> and then put into a glass container (11 cm diameter, 6 cm height). Into these glasses 4 small food cups (3.5 cm diameter, 2.5 cm height) were placed, filled with standard medium, which was covered with carbon paper and sprinkled with a thin yeast solution. Two of these cups had been "scented" and 2 served as controls. Each had previously been coupled with glass cylinders for 24 hours. The cylinders either contained 10 males for "scenting" the cups or were empty for the controls.

The females were allowed to lay eggs for 12 hours in complete darkness at 25°C. For every group of females 5 replicates were made. The results are shown in Table 1. Table 2 shows the pooled data of every group of females and the Chi<sup>2</sup> values calculated for an expected 1:1 relation. In each case they are highly significant. The females of both strains prefer to lay eggs into the food vials which had been scented by the previous presence of males. This tendency is stronger for "Berlin wild" than for "Grunewald". The

Krimbas, C.B. and M. Loukas. Agricultural College of Athens, Greece. Chromosomal homologies among four *Drosophila* species.

The following table gives the chromosomal homologies among four species of the *Sophophora* subgenus of *Drosophila*: *D. subobscura*, *D. pseudoobscura* (both of the *obscura* group), *D. melanogaster* and *D. willistoni*.

General symbol	<i>D. subobscura</i>	<i>D. pseudoobscura</i>	<i>D. melanogaster</i>	<i>D. willistoni</i>
A	A	XL	X	XL
B	J	XR	3L	XR
C	U	4	2L	2
D	E	3	2R	2
E	O	2	3R	3
F	dot	5	4	?

These homologies have been established by mapping visible mutants considered homologous. At least two good maps of visible markers are available for *D. subobscura*, one for the A (Spurway 1945; Bird 1946, 1947) and one for the O chromosome (Koske and Maynard-Smith 1954). Similar maps for *D. pseudoobscura* are given by Sturtevant and Tan (1937) and others (for a review see Patterson and Stone 1952, Dobzhansky and Powell 1975, Anderson and Norman 1977) and for *D. willistoni* by Spassky and Dobzhansky (1950). The maps of *D. willistoni* contain rather few markers except for the X chromosome.

The use of biochemical markers, as well as visible landmarks on the giant chromosomes, have confirmed these homologies (Patterson and Stone 1952, Hipsch 1952, Buzzati-Traverso and Scossiroli 1955, Sondhi 1957, Loukas, Krimbas, Mavragani-Tsipidou and Kastritsis 1979).

The order of the genes in the X chromosome (element A) is completely different in the four species (see Table 42 of Patterson and Stone 1952, Bird 1946, Spassky and Dobzhansky 1950). For 10 homologous markers at least six inversions are needed to obtain the gene order of *subobscura* from that of *pseudoobscura* and with more markers this number of inversions as well as of transpositions would undoubtedly increase. Thus it is worth noting that for such distant species as *D. melanogaster* and *D. subobscura* a relatively small number of rearrangements is needed for obtaining the gene order of the O chromosome of *subobscura* from the corresponding one of *melanogaster*. Let us consider the following loci as homologous: Aph, aliest=Est-3, Odh, ma, cu, Xdh, ME, Ao,  $ss^a=ar$ ,  $Dl=Va$ ,  $H=Ba$ ,  $cd=ch$ , Lap, Acph. The *melanogaster* gene order is (3L arm): centromere - Aph - aliest - Odh - ma - cu - Xdh - ME - Ao -  $ss^a$  -  $Dl$  - H -  $cd$  - Lap - Acph; whereas the *subobscura* one is for the O chromosome: centromere - Va - ar - Odh - cu - Ba - Ao - ME - Xdh - ch - Lap - Acph (the genes ma, Aph and Est-3 are located on chromosome J). The data concerning *D. melanogaster* have been compiled by Cavener (1977) except for ME (Franklin and Rumball 1971) and those concerning *D. subobscura* are taken from Loukas et al. (1979). A first inversion including Odh and ma and a second pericentric (3L-ma) are postulated. In this way ma, Aph and Est-3 could be placed on the 3L arm (homologous to J). Furthermore we can explain the great resemblance of the centromere end of 3L with that of the O chromosome. Three more overlapping inversions are needed to establish the gene order of *subobscura* (one of the segment Odh-DL, a second of the segment Odh-Ao and a third of the segment Ba-Xdh). The three distant genes of *subobscura* (ch, Lap, Acph) are in the same order as in *D. melanogaster*. For some 13 markers this seems to be a remarkably small number of inversions needed. Unfortunately we cannot try the same for the other species since the number of homologous mapped genes is indeed small.

References: Anderson, W.W. and R.A. Norman 1977, DIS 52:11-12; Bird, B.J. 1946, DIS 20:84; Bird, B.J. 1947, DIS 21:83; Buzzati-Traverso, A.A. and R.E. Scossiroli 1955, Adv. Genet. 7:47-92; Cavener, D.R. 1977, DIS 52:120-121; Dobzhansky, Th. and J.R. Powell 1975, in Handbook of Genetics, vol. 3 (Ch. 20) ed. by R.C. King, Plenum Press NY, 537-587; Franklin, I.R. and W. Rumball 1971, DIS 47:37; Hipsch, R. 1952, DIS 26:106; Koske, Th. and J. Maynard-Smith 1954, J. Genet. 52:521-541; Loukas, M., C.B. Krimbas, P. Mavragani-Tsipidou and C.D. Kastritsis, The Journal of Heredity, 70:17-26; Patterson, J.T. and W.S. Stone 1952, Evolution in the genus *Drosophila*, McMillan NY, p. 610; Sondhi, K.C. 1957, DIS 31:162; Spassky, B. and Th. Dobzhansky 1950, Heredity 4:201-215; Spurway, H. 1945, J. Genet. 46:268-286; Sturtevant, A.H. and C. C. Tan 1937, J. Genet. 34:415-432.

Lawlor, T.A. University of California, San Diego, La Jolla, California. Genetic and cytological localization of mei-9.

Alleles of the X-linked meiotic mutant mei-9 have been mapped on the basis of both the somatic effect (monitored by an increase in frequency of multiple wing hair (mwh) clones in mwh/+, Baker et al. 1978) and the meiotic effect

(monitored by an increase in frequency of X and 4th chromosome nondisjunction, Baker and Carpenter 1972).

y mei-9<sup>a</sup>/w<sup>e</sup> ec rb females were crossed to +/Y ; mwh/mwh males; all sons were scored for the visible markers and a subset of sons were tested for the presence of mei-9<sup>a</sup> by removing one wing and scoring the number of mwh clones present. Results are in Table 1.

Table 1. Number of mwh clones per wing among sons of y<sup>+</sup> w<sup>e</sup> ec rb/y mei-9<sup>a</sup> x +/Y ; mwh/mwh.

Total mwh clones	Number of progeny scored from:																			
	Parentals					Recombinants														
	y	y <sup>+</sup>	w <sup>e</sup>	ec	rb	y <sup>+</sup>	y	w <sup>e</sup>	ec	rb	y <sup>+</sup>	w <sup>e</sup>	y	ec	rb	y <sup>+</sup>	w <sup>e</sup>	ec	y	rb
0	1		14										6			3			8	
1	2		5										4			3			7	
2			4						1				2			8			6	
3																4			2	
4	1					1					1					2				
≥5	4					18					22					15			7	
Total wings	8		23			19		1			23		12			35			30	
Total ♂ progeny	1949		1660			19		14			58		38			36			31	

Although mwh clones turned out to be difficult to score in phenotypically yellow wings, the results are clear: mei-9<sup>a</sup> maps between echinus (1-5.5) and ruby (1-7.5; map positions from Lindsley and Grell 1968). If, as seems reasonable from the other crossover classes, wings with 0-2 clones are considered mei-9<sup>+</sup> whereas wings with 5 or more clones

are considered mei-9, then mei-9<sup>a</sup> maps 15/29 (from the ec rb<sup>+</sup> recombinants) to 21/28 (from the ec<sup>+</sup> rb recombinants) of the distance from ec to rb. The somatic effect of mei-9<sup>a</sup> therefore maps in the 6.5-7.0 interval, in agreement with the earlier rough genetic mapping of the meiotic effect (Carpenter and Sandler 1974).

Deficiency/mei-9 females were tested for the mei-9 phenotype in two separate experiments: (1) wings of deficiency/mei-9<sup>a</sup> ; mwh/+ females were scored for mwh clones; (2) deficiency/mei-9 ; spa<sup>pol</sup> females were tested for meiotic nondisjunction in crosses to B/Y; C(4)RM, ci ey<sup>R</sup> males. Deficiencies HC 224, HC 163, and RC 40 were obtained from Dr. George Lefevre; GA 56 was obtained from Dr. Mel Green; HC 163B was one of two lethal segregants from my HC 163 stock (there is no obvious deficiency in the other segregant). The deficiency stocks were examined cytologically to determine breakpoints and crossed to ec rb stocks to confirm deletion extents roughly. All four deficiencies uncover rb (4C6-8) and HC 244 uncovers ec (3F1-2). In somatic effect experiments, HC 244 (3D6-3E1); 4F7-8) uncovers mei-9<sup>a</sup> (11.6 mwh clones/wing, 17 wings scored). HC 163B, which has breakpoints of 4B1-2 ; 4F1-2 and is therefore likely to be a contaminant RC 40 (4B1-2; 4F1), also uncovers mei-9<sup>a</sup> (10.8 mwh clones/wing, 12 wings scored). HC 244/+, HC 163B/+, and +/mei-9<sup>a</sup> control females had background frequencies of 2.5, 1.0, and 3.2 mwh clones/wing, respectively. Based on the genetic and deficiency experiments above, the somatic effect of mei-9<sup>a</sup> must be located between 4B1-2 and 4C6-8; that is, between the distal breakpoint of HC 163B and the bands for rb.

Three deficiencies were tested for uncovering the mei-9<sup>a</sup> meiotic phenotype. mei-9<sup>a</sup>/mei-9<sup>a</sup> control frequencies were 27.0% X nondisjunction (ND) and 15.2% 4th ND (3422 total progeny; all frequencies are gametic frequencies). Two deficiencies uncovered the meiotic effect. mei-9<sup>a</sup>/HC 244 gave 31.7% X ND and 30.5% 4th ND (631 total progeny); mei-9<sup>a</sup>/HC 163B gave 28.5% X ND and 23.4% 4th ND (4038 total progeny). Two independently isolated mei-9 alleles, mei-9<sup>b</sup> and mei-9AT1 (the latter obtained from Dr. P. Dennis Smith), are also uncovered by HC 163B. However, mei-9<sup>a</sup>/GA 56 (4C1-5; 4D1-2 - breakpoints approximate) did not expose mei-9<sup>a</sup>, giving 0.4% X ND and 0% 4th ND (2330 total progeny). Background frequencies are ca. 0.1% X and 4th ND, and mei-9 is recessive with respect to nondisjunction (Baker and Carpenter 1972).

The meiotic and somatic effects therefore most likely co-map to salivary region 4B1-2; 4C1-5; this region is defined by the distal breakpoints of HC 163B (which does uncover mei-9 alleles) and the proximal breakpoint of GA 56 (which does not uncover mei-9<sup>a</sup>). This salivary

location is in agreement with the genetic map position of 6.5-7.0 determined above.

Acknowledgements: Thanks to Dr. Adelaide T. C. Carpenter for helpful criticism and advice; thanks also to SJB, IE, KP, and KC for stimulating and productive support.

References: Baker et al. 1978, Genetics 90:531; Baker, B.S. and A.T.C. Carpenter 1972, Genetics 71:255; Carpenter, A.T.C. and L. Sandler 1974, Genetics 76:453; Lindsley, D.L. and E.H. Grell 1968, Carnegie Inst. of Wash. Publ. No. 627.

Lee, T.J. Chungang University, Seoul, Korea. Sexual isolation among four species in the *Drosophila auraria* complex.

The *D. auraria* complex was divided into four species, *D. auraria*, *D. biauraria*, *D. triauraria* and *D. quadraria* (Bock and Wheeler 1972). The sexual isolation among three species, *D. auraria*, *D. biauraria*, and *D. triauraria*, was significantly demonstrated (Kurokawa 1960; Lee 1970).

Table 1. Sexual preference tests among four species.

Crosses		Homo-gamic (%)	Hetero-gamic (%)	Isolation index	Coefficient of joint isolation
♀♀	♂♂				
A, D	X A	45	51	0.063	0.338
A, D	X D	71	17	0.614	
B, D	X B	69	50	0.160	0.088
B, D	X D	64	62	0.016	
C, D	X C	50	63	0.115	0.162
C, D	X D	78	51	0.209	

(A: *D. auraria*, B: *D. biauraria*, C: *D. triauraria*, D: *D. quadraria*)

to the morphological difference between their genitalia. It can hardly be concluded from morphological, physiological and distributional studies (Lee 1974a, 1974b) that, of the members belonging to species *D. auraria* complex, *D. quadraria* would be the ancestral species.

References: Kurokawa, H. 1960, Jap. J. Gen. 35:161-166; Lee, T.J. 1974a, Rev. Tech. & Sci., Chungang Univ. 1:9-16; Lee, T.J. 1974b, Theses Collection, Chungang Univ. 19:63-73.

Leigh Brown, A. J. National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina. Molecular weights of seven *Drosophila* enzymes.

Recent work in our laboratory (Leigh-Brown and Langley 1979; Leigh-Brown and Voelker 1979) has involved the estimation of the native molecular weight of several *Drosophila* enzymes for which such data were not previously available (O'Brien and MacIntyre 1978). As our earlier report gave

only the results, I present here the methods used and the data on which those estimates were made.

Determination of sedimentation constants ( $s_{20,w}$ ) by sucrose density gradient sedimentation was carried out according to the procedure of Martin and Ames (1961). Gradients were made in 5 ml cellulose nitrate tubes by layering 1.15 ml of each of 20%, 15%, 10% and 5% solutions of sucrose in 0.05M Tris-HCL pH 7.5 with 1 mM dithiothreitol (Sigma). They were then stored at 4°C for 24 hours. Crude fly homogenate was prepared in the same buffer by homogenising 0.5 g cn bw; ri e flies, centrifuging in the homogenate for 20 minutes at 15,000 rpm, and filtering through glass wool. The extract was then passed through a 40%/80% ammonium sulphate precipitation step and was diluted until the protein concentration, measured by O.D.<sub>260</sub>/O.D.<sub>280</sub>, was less than 20 mg/ml. Rabbit muscle Ldh was added (800 units/ml) and 0.1 ml was layered on top of each gradient. Three such gradients were centrifuged for 15.5 hr at 39,000 rpm in a Beckman SW 51 rotor at 4°C. After the run, two-drop fractions were collected on ice and assayed. Rabbit muscle lactate dehydrogenase and *D. melanogaster* alcohol dehydrogenase were used as standards.

Gel filtration was performed according to Andrews (1965). 5 g of frozen flies of the same strain were homogenized in cold 0.1M Potassium phosphate buffer, containing 1mM DTT and 1mM EDTA. The extract was spun, filtered and passed through a 40%/80% ammonium sulphate cut as before. After dialysis against 1 liter of buffer for 24 hours it was concentrated by ultrafiltration. One ml was applied to a column of "Sephacryl" S-200 (Pharmacia) of dimensions 88.0 x 2.0 cm. A flow rate of 1 ml/minute was maintained with a peristaltic pump and 20 drop fractions were collected.

Table 1.

		Sucrose density gradients $s_{20,w}$	Gel filtration ( $-\ln K_{av}$ ) <sup>1/2</sup> (Laurent & Killander, 1964)	Stokes radius $\bar{a}$	Molecular weight ( $\times 10^{-3}D$ )
Pgk	Mean	3.5	1.0343	27.3	39
	Se	0.11			
Tpi	Mean	4.0	1.0672	28.6	47
	Se	0.13			
Gpt	Mean	5.9	1.2526	35.9	87
	Se	0.19			
Pgi	Mean	6.6	1.2440	35.5	97
	Se	0.23			
Adh		3.9			
Ldh		7.45			
Chymotrypsinogen			0.8764	22.6	
Ovalbumin			1.0693	27.6	
Aldolase			1.4796	47.4	

The column was first calibrated with  $\alpha$ -chymotrypsinogen (M.W. 25,000), ovalbumin (45,000) and rabbit muscle aldolase (158,000), all assayed by measuring O.D.280. D. melanogaster Adh and aldolase were used as internal standards during the run. All operations were performed at 4°C.

In Table 1 is given the mean sedimentation constant ( $s_{20,w}$ ) obtained from sucrose density gradients for Phosphoglycerate kinase (Pgk), Triose phosphate isomerase (Tpi), Glutamate pyruvate transaminase (Gpt) and Phosphoglucose isomerase (Pgi), with the standard errors from six estimations against the standards Adh and Ldh. The Stokes radii of these four enzymes were estimated from "Sephacryl" S-200 gel fil-

tration after the method of Laurent and Killander (1964). These are also given in Table 1, with the elution data on the standards. From these data the molecular weights were calculated via the relationship:

$$M = \frac{6\pi\eta N a s}{1 - \bar{v}\rho}$$

where  $N$  = Avogadro's number ( $6.022 \times 10^{23}$ );  $\bar{v}$  = the partial specific volume (assumed to be  $0.725 \text{ cm}^3\text{g}^{-1}$  for globular proteins);  $\eta$  and  $\rho$  correspond to the viscosity and density respectively of the medium; and  $a$  = Stokes radius. The following values were obtained: Pgk, 39,000D (monomer); Tpi, 47,000D (dimer);

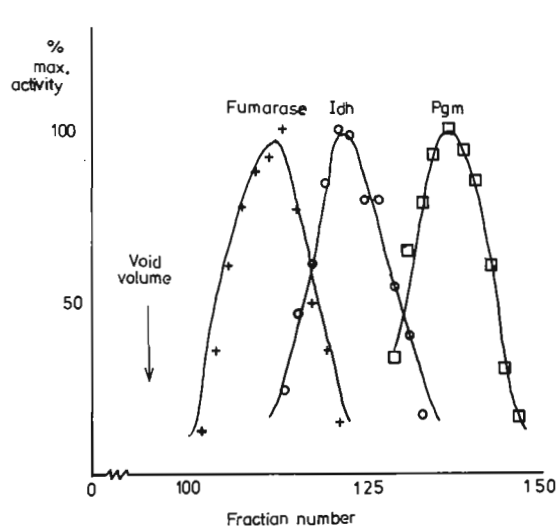


Fig. 1a

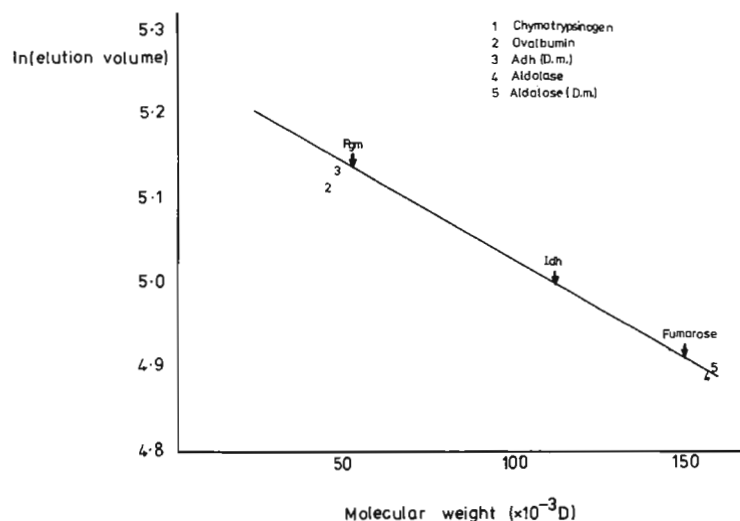


Fig. 1b

Gpt, 87,000D (prob. dimer); Pgi, 97,000D (dimer).

The molecular weights of three enzymes were estimated directly from the gel filtration data by the method of Andrews (1965). Their elution profiles and position relative to the standards are shown in Figure 1a and 1b. Estimates of 51,000, 112,000 and 150,000 were obtained from Pgm (monomer), Idh (dimer), and fumarase (tetramer), respectively, using the least-squares regression from the five standards:  $y = 5,257 - 0.002319x$ . Fractions from this column were also assayed for activity of dipeptidase isozymes by starch gel electrophoresis. By this means it was possible to assign approximate molecular weights of 120,000 and 170,000 Daltons to the Dip-A and Dip-B isozymes, respectively (Voelker, Ohnishi and Langley 1979).

References: Andrews, P. 1965, *Biochem. J.* 96:596-606; Laurent, T.C. and J. Killander 1964, *J. Chromat.* 14:317-330; Leigh Brown, A.J. and C.H. Langley 1979, *Nature* (in press); Leigh Brown, A.J. and R.A. Voelker 1979, *Biochem. Genet.* (in press); Martin, R.G. and B.N. Ames 1961, *J. Biol. Chem.* 236:1372-1379; O'Brien, S.J. and R.J. MacIntyre 1978, *In Genetics and Biology of Drosophila*, Ashburner, M. and T.R.F. Wright (Eds.), Vol. 2a, pp. 396-551, Academic Press, London; Voelker, R.A., S. Ohnishi and C.H. Langley 1979, *Biochem. Genet.* (in press).

Lindsley, D.E., L.S.B. Goldstein and L. Sandler. University of Washington, Seattle, Washington. Male sterility in maternal-effect mutants.

Seven mutations have been isolated in region 30-31 of chromosome 2 -- the so-called da-abo region. Of the five mutants that have been analyzed, all cause a maternal effect resulting in sex-specific embryonic lethality. In all cases, the severity of the maternal effect is sensitive

to the heterochromatic constitution of the zygote, and the severity of the maternal effect is reduced if the experiments are carried out at 19° instead of 25°. The proposal was made that these five mutations define a cluster of functionally related genes (Sandler 1977).

We here report evidence that another of the seven mutations, mfs48, is also a member of the da-abo cluster. This evidence takes two forms: (1) mfs48 maps within the cluster, and (2) mfs48 exhibits phenotypic similarities to hup, one of the five known mutants in the cluster. On the basis of phenotype data presented here, we suggest a site of action for the genes in the da-abo cluster.

mfs48 was initially characterized as a male and female sterile with thin bristles; hup as a maternal-effect mutant which had held-up wings. hup was mapped to the right of da and tightly linked to abo; mfs48 was deficiency mapped to the left of abo and near da. We placed mfs48 to the right of da in the following way. Recombinant  $J^+mfs48^+$  chromosomes from J da / mfs48 females were progeny tested for the da allele carried. Of 23  $J^+mfs48^+$  recombinants, 19 carried da and 3 da<sup>+</sup>. Thus, the gene order is J-da-mfs48, with the da-mfs48 distance probably shorter than the J-da distance. This means that mfs48 is between da and abo and thus clearly maps within the cluster.

Both hup and mfs48 are lethal over a deficiency; we have found that in addition both are recessive semi-lethals. Thus, only 25% of mfs48 and 20% of hup homozygotes survive at 25°. We have also found that the fertility of mfs48 and hup homozygous males and females is temperature sensitive. At 23° both mutants are fertile (to some degree) in both sexes but at 28.5°, males and females are sterile (mfs48 females were only tested at 25° where they were found to be sterile). We have examined spermatogenesis in mfs48 and hup males raised at 23° and 28.5°. Males raised at 23° rarely show visible abnormalities in spermatogenesis. The testes are full of cells and motile sperm are observed in large numbers in the seminal vesicle. Males raised at 28.5° have no motile sperm and show a variety of defects during the later stages of spermiogenesis. The earliest defect which we have found is at the stage just after meiosis, the clew stage (Tates 1971). Cells at this stage and later contain micronuclei as well as macro-nuclei (see Fig. 1). In addition we find occasional spermatid nuclei with two basal bodies



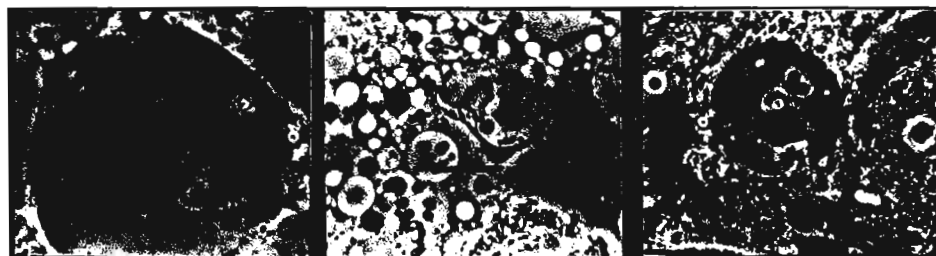


Fig. 1

Fig. 2

Fig. 3

Fig. 1. Nuclei from 28.5° mfs48. Single arrow indicates micro-nucleus. Double arrow indicates nucleus with two basal bodies attached (one is barely in the plane of focus). Magnification bar represents 10  $\mu$ m.

Fig. 2. A spermatid nucleus with two axonemes in mfs48 was followed until the cell lysed. Here, the free nucleus is distorted by the drag from the two axonemes as it moves across the preparation. Magnification bar represents 10  $\mu$ m.

Fig. 3. Two basal bodies with two axonemes attached to one nucleus in 28.5° hup spermatid. A single nebenkern (arrow) is associated. Magnification bar represents 10  $\mu$ m.

(and two associated axonemes) attached to them (see figures). This is in striking contrast to wild type raised at 28.5° where spermatids contain single nuclei of uniform size and each spermatid receives only a single centriole which attaches to the nucleus and functions as a basal body. As yet, we have found no visible defects at earlier stages.

These observations suggest that the primary lesion in spermatogenesis in mfs48 and hup raised at 28.5° may be in the system which is responsible for the segregation of centrioles during division. Micronuclei may be caused by the occurrence of abnormal meioses in nuclei where the spindle poles and centrioles are not properly positioned.

Our observations on female sterility have as yet been uninformative because the female germ line is refractory to light microscopic investigation. We think, though, that the sterility in females could be caused by a defect in the accumulation of centrioles in the presumptive oocyte (Mahowald and Strassheim 1970). Clearly, abnormalities in centriole behavior could also have somatic effects resulting in the observed semilethality of mfs48 and hup. These observations lead us to wonder whether the loci in the da-abo region are all involved in the control of centriole movement during the development of soma and germ line. We are now in the process of testing this proposition and extending the cytological analysis.

References: Mahowald and Strassheim 1970, J. Cell Biol. 45:306; Sandler 1977, Genetics 86:567; Bates 1971, Thesis 'S-Gravenhage: Drukkerij, J.H. Pasmans.

Lewis, R.A., T.C. Kaufman and R.E. Denell\*. Indiana University, Bloomington, Indiana and \*Kansas State University, Manhattan, Kansas. Genetic analysis of the Antennapedia gene complex (ANT-C): mutant screen of proximal 3R, section 84B-D.

The apparent localization to section 84B of several homoeotic loci involved in the determination and differentiation of anterior structures has led us to postulate a possible correlation between this series of developmental lesions and the well described, more distal bithorax complex (BX-C) (Lewis 1979).

A mutant screen designed to genetically dissect this region has been conducted, employing a deficiency (Df(3R)Antp<sup>Ns+R17</sup>) spanning section 84B1,2-D11,12 which was generated as a revertant of the dominant homoeotic lesion Nasobemia (Duncan and Kaufman 1975). A total of 3133 chromosomes were treated either with EMS (.0125M) (Lewis and Bacher 1968) or X-rays (4000R). Chromosomes bearing lethal or visible mutations in the region of interest were recovered and maintained in balanced stocks with In(3LR)TM3,Sb Ser.



Recovered mutations were named by the following alphanumeric code. A capital letter E or X indicates the inductive agent and is followed by a lower case letter designating the particular marked chromosome on which the lesion was induced: "a" (Dfd pP), "b" (Ki roe pP), "c" (Ki pP), "d" (pP cu) or "e" (Ki pP bx sr e<sup>s</sup>). These two letters are followed by the initial of the discoverer and finally, a number to identify the particular mutation by its order of discovery.

85 lethal, 3 semi-lethal, and 6 visible mutations were recovered. Subsequent to balancing, each of these was crossed to four additional deficiencies (Df(3R)Scr, Df(3R)Antp<sup>+</sup>R1P, Df(3R)-Antp<sup>+</sup>R2, and Df(3R)dsx<sup>D</sup>+R2). (For cytological limits see Duncan and Kaufman 1975; Kaufman 1978; and Fig. 1). These four deficiencies serve to subdivide the 84B-D interval into 7 regions and allow the identification of those mutations which reside in 84B1,2, the site of the Antennapedia complex (ANT-C) (Denell 1973; Duncan and Kaufman 1975). The results of this initial deficiency mapping are shown in Fig. 1.

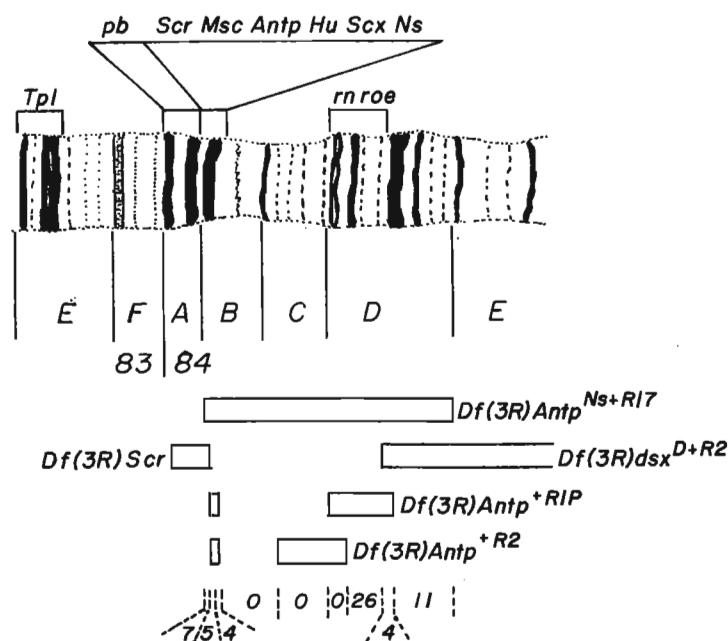


Fig. 1. Distribution of induced mutations obtained by deficiency mapping. (Tpl=Triplo-lethal; pb=pro-boscipedia; Scr=Reduced sex combs; Msc=Multiple sex comb; Antp=Antennapedia; Hu=Humeral; Scx=Extra sex comb; Ns=Nasobemia).

The deficiency analysis established three complementation groups in the vicinity of the 84B1,2 doublet. The distal-most of these is defined by failure to complement Df(3R)Antp<sup>+</sup>R1P and Df(3R)Antp<sup>+</sup>R2 and is comprised of four mutations. The proximally neighboring group fails to complement Df(3R)Scr as well as the above two deficiencies. There are five of this class of mutation. The most proximal collection fails to complement only Df(3R)Scr and contains seven lesions.

Inter se combinations within each of these deficiency defined regions have shown that the members of the distal-most group (EdR16, EdR17, Ebr27, Xbd2) form a single complementation group. The middle group (Ecr10, Ebd7, Eck5, Xbk4, Eer4) also forms a single unit. Finally, combination of members of the most proximal group reveal two separate complementation units. One (EdK6, EdD8, EdR18) is further characterized by a dominant reduction in the number of sex comb teeth similar to that seen in male heterozygotes of either Df(3R)Scr or Df(3R)Antp<sup>+</sup>Ns+R17. The other complementation unit (Ebr11, XaK2, XaK5, XaK26) is complex and is shown in Fig. 2.

The most distal complementation group, defined by its lethal interaction with Df(3R)dsx<sup>D</sup>+R2, consists of 11 members. Inter se crosses reveal four functional groups among the 11 mutations - two of two members each, one of one, and one of seven members.

Four mutations fail to complement Df(3R)dsx<sup>D</sup>+R2 and Df(3R)Antp<sup>+</sup>R1P. Inter se crosses define two functional groups, one of which is further divided into two subsites. This more complex group is characterized by a rotation of the male genitalia in surviving individuals.

26 mutations were recovered that fail to complement Df(3R)Antp<sup>+</sup>R1P. A complementation analysis among all of these members reveals an extremely complex circular pattern.

37 mutations fail to complement only the original screening deficiency. The great majority of them have been shown by a preliminary recombination analysis to reside quite far from 84B-D. Several members of this group in various heterozygous combinations with one another, confer a marked swelling on the mesothoracic and metathoracic legs. They are currently being mapped more precisely.

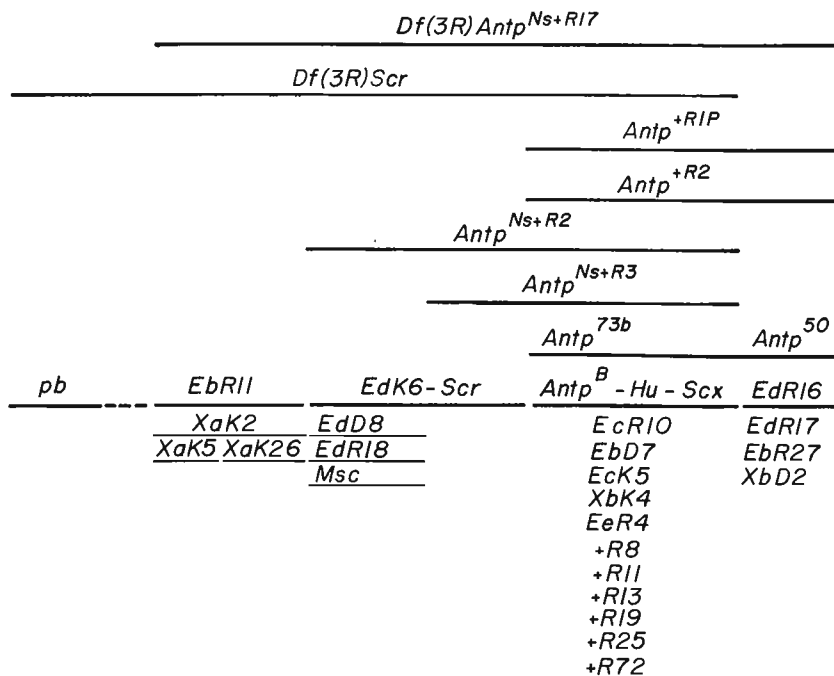


Fig. 2. Complementation map of induced mutations within Antennapedia complex. Localizations determined by complementation patterns of new mutations with deficiencies and extant homoeotic lesions resident in the 84B1,2 doublet. (Note: +R8, +R11, etc., refer to revertants of Antp<sup>Ns</sup>.)

demonstrate that the EbR11 group is proximal to the other three groups (Fig. 2).

A most interesting addition to the list of homoeotic transformations attributable to lesions in 84B1,2 has been revealed by a mutation in the Sex Combs Reduced (Scr) group. As previously mentioned the number of sex comb teeth produced in males heterozygous for these lesions and the TM3 balancer ranges from 5 to 8 as compared to the normal 8 to 12. Rare surviving individuals of the genotypes EdR18/Df(3R)Scr and EdR18/Df(3R)Antp<sup>Ns+R17</sup> frequently possess no sex combs at all. Those teeth which are occasionally present are not rotated. Further aspects of the chaetotaxy of these legs (e.g., the absence of transverse rows on the tibia) indicate that the prothoracic leg is transformed into a mesothoracic leg. An additional transformation can also be seen in EdR18/Df(3R)Scr and in EdR18/Df(3R)Antp<sup>Ns+R17</sup> individuals. The first three rows of pseudotracheae of the labial palps are transformed into what appears to be maxillary palpus. We interpret this as indicating an interaction of the Scr locus with a site proximal to it and contained within the limits of the Df(3R)Scr lesions. The possibility that this site is the proboscipedia locus is currently being investigated.

References: Denell, R. 1973, Genetics 75:279; Duncan, I.W. and T.C. Kaufman 1975, Genetics 80:733; Kaufman, T.C. 1978, Genetics 90:579; Lewis, E.B. 1979, Nature 276:565; Lewis, E. B. and F. Bacher 1968, DIS 43:193.

Crosses between each of these 16 lethal lesions and the homoeotic lesions known to map to 84B1,2 were performed to further delineate these sites. In this manner, the group showing the reduced sex comb phenotype was shown to be allelic to Multiple Sex Comb (Msc), which also displays this phenotype. The next most distal group was similarly shown to be allelic to Antennapedia (Antp), Extra Sex Comb (Scx) and Humeral (Hu). Further, the members of the most distal site failed to complement the recessive lethality of Antp<sup>73b</sup> but were viable in combination with Antp<sup>B</sup> and Scx. The remaining group to date has not been associated with any known homoeotic lesion.

A clarification of the proximal distal orientation of these four groups has been provided by crossing the 16 lethal lesions to 8 additional revertants of Antp<sup>Ns</sup> (Duncan and Kaufman 1975; Denell 1973). The results of these crosses confirm the proximal-distal array found in the deletion analysis and further

Loverre, A. and R. Cicchetti. Istituto di Genetica, Università di Roma, Italy. A male-specific lethal gene in *D. melanogaster*.

present in equal number, but in the  $Cy^+$  progeny there are no males, independent of the maternal genotype, and the females are present but are fewer than expected. These results suggest

Cross		F <sub>1</sub> Progeny (expected ratio)			
		$\delta$ $Cy^+$	$\delta$ $Cy$	$\delta$ $Cy$	$\delta$ $Cy$
$\frac{SD}{SD}$	$\delta$ X $\frac{SD}{SM5,Cy}$	0 ( 1	180 : 1	272 : 1	276 )
$\frac{SD}{SM5,Cy}$	$\delta$ X $\frac{SD}{SM5,Cy}$	0 ( 1	149 : 1	564 : 2	500 )

Table 2.

A. F<sub>1</sub> progeny of the cross between SD/B1 L females and SD/SM5,Cy males.

Phenotype	$Cy^+$		$Cy$	
	$\delta$	$\delta$	$\delta$	$\delta$
+	0	280	328	371
B1 L	361	331	335	330
B1	50	53	66	56
L	2	50	63	53

B. Presence of the *mll* factor in chromosomes from the above cross.

Chromosome examined	<i>mll</i>	<i>mll</i> <sup>+</sup>
+	55	0
B1 L	0	59
B1	1	44
L	42	0

Table 3. Viability of various developmental stages in experiment and control. Percent values in relation to individuals of the preceding developmental stage are given in parentheses.

Egg	I instar larva	II or III instar larva	Pupa	Adult
413	314 (76%)	286 (91%)	220 (76%)	190 (90%)
145	130 (90%)	124 (95%)	119 (98%)	112 (94%)

and E.L.P. of *mll* are very similar to those of the maleless gene found by Fukunaga et al. in a Japanese population (Genetics 81:135-141, 1975).

In a natural population of *D. melanogaster* from Corato (Apulia, Italy), a second SD (Segregation Distorter) chromosome was found which in the homozygous condition is present only in females.

The results of the crosses in Table 1 show that in the  $Cy$  progeny males and females are present in equal number, but in the  $Cy^+$  progeny there are no males, independent of the maternal genotype, and the females are present but are fewer than expected. These results suggest that the absence of homozygous males is due to the action of a lethal recessive factor(s) acting in males alone. In order to localize this factor on the 2nd chromosome, the progeny of SD/B1 L females crossed with SD/SM5,Cy males was studied (see Table 2A). An examination of the class  $Cy^+$  for the presence or absence of males led to some preliminary conclusions: the lethal factor, which will be termed malelethal (*mll*), is located between B1 (54.8) and L (72.0), closer to B1 than to L, and not coinciding with the SD factor which is to the left of B1. Moreover, a series of parental chromosomes and crossovers from the cross in Table 2A recovered in  $Cy$  males and females were examined for the presence of *mll*. These, crossed with *mll*/SM5,Cy males and females, supplied the data in Table 2B. Pooling the data, we have three recombinant chromosomes between B1 and *mll* (2 *mll*<sup>+</sup> L and 1 B1 *mll*) and 136 between *mll* and L (42 *mll* L and 50 + 44 B1 *mll*<sup>+</sup>), which places *mll* at 3/139 of 17.2 m.u. to the right of B1, that is, at  $55.2 \pm 0.2$  in the centromeric region.

In order to individuate the effective lethal phase of *mll* the development of progeny from crosses between *mll*/*mll* females and *mll*/SM5,Cy males (experiment) was observed, ORE males and females being used as the control (see Table 3). A significant difference in viability in the progeny of the two crosses was observed in the development from egg to first instar larva and a still more marked difference in the development from second and third instar larva to pupa. It is still to be ascertained whether the greater mortality of the eggs in the experiment is related to *mll*/*mll* zygotes or is due to a maternal effect. The major effect of *mll* is to be found, however, in second or third instar larva.

The phenotype, mapping

Majumdar, D. and A.S. Mukherjee. University of Calcutta, India. Morphogenetic expression of a reversible Bar-inversion of *D. melanogaster*.

An analysis of morphogenetic expression of polytene chromosomes in  $\text{In}(1)^{\text{BM}2}$  ( $\text{rv fB}^{15}$  reinversion; mosaic) of *D. melanogaster* is presented here. The inverted region includes the segment 16A 2-5 to 20E of the X chromosome. In homozygous or hemizygous conditions the inversion ap-

pears as a small ring at the base of the X chromosome (Fig. 1a) by pairing of the shifted heterochromatic portion (20A-E) with the chromocenter. In females all three possible configurations (homozygous inversion, homozygous reinversion and heterozygous inversion) have been observed. The last one has been observed in only a few nuclei. In males, specifically, the

post-reinversion conditions are associated with an interesting morphogenetic behavior of the X chromosome in different nuclei. Firstly, the inversion-bearing chromosome appears similar to wild type male X chromosome; secondly, the reinverted chromosome extraordinarily puffs out at one or more sites, namely 1B-2B,

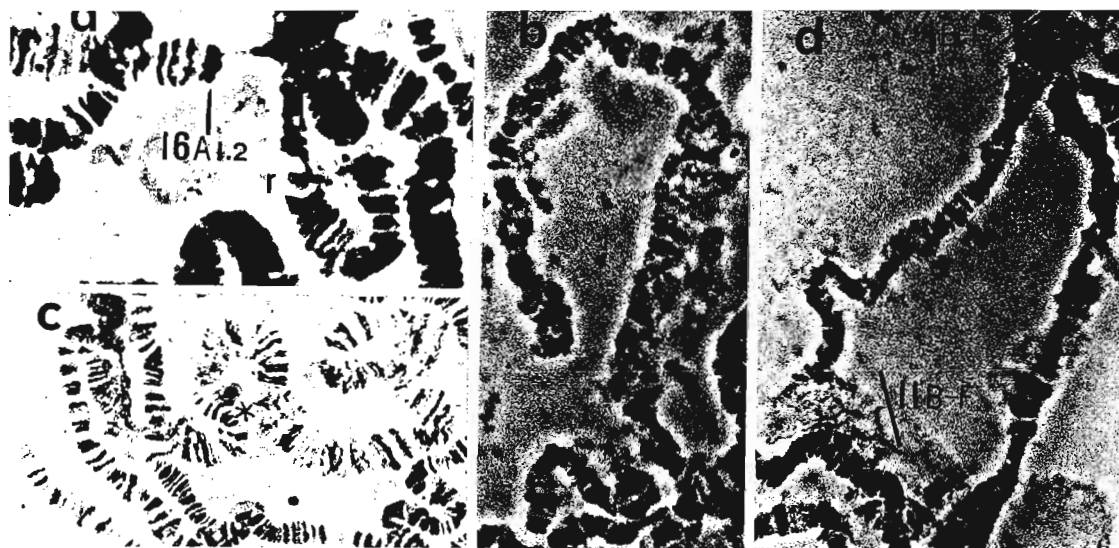


Fig. 1. (a) Homozygous inversion in female showing the ring (r) attached to chromocenter and the distal breakpoint: 16A 1.2 (pointer). (b) RF chromosome decondensed at the proximal half. (c) RF chromosome highly decondensed all through except at the region 17 A-F (\*). (d) RN chromosome excessively and specifically puffed out at 1 B-F and 11 B-F.

10EF and 11B-F, and is significantly less wide than the paired autosomes or wild type male X chromosome (RN); and lastly, some reinverted chromosomes are extraordinarily decondensed at the proximal or distal half or through the whole of the X chromosome (Fig. 1b). Such decondensed chromosome or chromosomal segments are wide, stumpy and much less stained than the normal male-X; sometimes the whole chromosome appears fuzzy (RF) and no thick band is discernible. Other notable properties of reinverted chromosomes are (1) the usual male-X type condensed banding of the region 17 A-F in RF chromosomes (Fig. 1c) despite excess decondensation (2-4 times the width of any pair of autosomes) at other regions; (2) characteristic puffing of 11B-F, in all RN chromosomes, ranging between 2-6 times its activity in wild type males (Fig. 1d); (3) at 24°C the frequency of RN chromosomes is very high (90-95%) and that of RF chromosomes varies between 0 and 10%. At 18°C the frequencies of occurrence of RN, RF and inversion-bearing chromosomes are 42%, 26%, and 32% respectively.

The stock has been obtained from Cal. Tech., Pasadena. The work has been supported by a C.S.I.R. Senior Research Fellowship to D.M.

Malogolowkin-Cohen, Ch. Institute of Evolution, University of Haifa, Israel. Inversion polymorphism in *Drosophila subobscura* in Israel.

Natural populations of *Drosophila subobscura* from seven sites of three different biogeographic regions have been sampled in Israel in 1976 and 1977 (Malogolowkin-Cohen, 1978). Salivary glands of one F<sub>1</sub> larva of wild females were dissected and examined for inversion polymorphism.

Samples were collected during spring from March-May when *D. subobscura* is abundant in the seven sites and from October-February (autumn and winter) at the Mount Carmel site only. No attempts to collect *D. subobscura* during the summer or winter in any site except for Mount Carmel were made. No flies were found at the Mount Carmel site during the summer months.

Twenty-six inversions and 46 heterozygous combinations have been found in Israel. This can be compared with 54 inversions and 64 gene arrangements that are known to occur in natural populations of *D. subobscura* (Jungen, 1968). The highest number of heterozygous combinations per population was found in Oranim (25 combinations). Number of females, average number of inversions per female, number of inversion limits for female with and without considering the A chromosome, number of inversion types, average number of heterozygote chromosomes per female and index of free recombination (IFR) are listed in Table 1.

Table 1. Site and year of sampling as well as number of females, average number of inversions per female, number of inversion limits for female with and without the A chromosome (sex chromosome), number of inversion types, average number of heterozygote chromosomes per female and index of free recombination (IFR) are listed.  
S = Spring, FW = Fall-Winter, FH = Foothills, CP = Coastal Plain

Site	Year	No. of females	Average no. of inversions/♀		No. of inversion limits/♀		No. of inversion types	Average no. heterozygous chromosomes/♀		IFR
			with A	without A	with A	without A		with A	without A	
Zfat	1976 S	8	5.0	4.5	2-8	2-8	14	2.8	2.2	82.78
Zfat	1977 S	62	5.4	5.0	0-12	0-12	23	2.9	2.6	78.05
Zfat	1978 S	34	4.9	4.6	0-10	0-10	15	2.4	2.1	81.30
Carmel	1976 S	22	4.3	4.0	1-9	0-8	19	2.6	1.8	80.48
Carmel	1976-7 FW	14	4.5	4.0	1-14	0-13	21	2.5	2.3	84.10
Carmel	1977 S	40	5.8	5.4	0-13	0-12	19	2.9	2.6	78.61
Carmel	1977-8 FW	9	4.0	3.4	1-13	0-12	10	2.3	1.7	86.48
Carmel	1978 S	18	5.5	5.4	1-10	0-10	14	2.4	2.2	83.90
Q. Anavim	1977 S	14	5.5	5.1	1-13	0-12	14	3.0	2.7	78.57
Oranim (Tivon)	1977 S	28	5.2	4.9	1-13	0-12	24	2.9	2.6	81.02
Maian Zvi (FH)	1977 S	13	5.1	4.7	1-14	0-13	24	1.9	1.8	78.32
Maian Zvi (CP)	1977 S	3	5.2	4.9	2-13	2-12	19	2.6	2.3	80.30
Israel		265	5.0	4.6	0-14	0-13	24	2.6	2.2	81.15

The only previous data of *D. subobscura* in Israel were published in Goldschmidt (1956, 1958) and in Stumm-Zollinger and Goldschmidt (1959). Only three sites were sampled in these studies, Quiriat Anavim, Oranim, and Eilon, with differing results from the present study. Stumm-Zollinger and Goldschmidt found the Israeli populations to be marginal with IFR values of  $88.8 \pm 0.6$ ,  $89.8 \pm 1.3$ ,  $89.6 \pm 0.8$  respectively. Our values of IFR range from 78.61 at Carmel, spring (S) 1977 to 86.48 at the same site in the following fall-winter (F-W) 1977-1978, with the average Israeli value being 81.15. From the values published by Prevosti (1964) and Sperlich (1964), central populations of *D. subobscura* in France, Italy and Iberia had IFR values ranging from 78.2 to 83.8; intermediate populations in Austria, France and Switzerland had values ranging from 84.1 to 87.8; and marginal populations in Israel, Scotland and Norway had values ranging from 88.8 to 95.0. As such our new IFR values for Israeli populations fall in the central and intermediate range. In addition, the average number of heterozygous chromo-

somes per female (average Israeli value 2.36 for all chromosomes) is as high as values reported in Italy (Sperlich 1964) and higher than those in Norway and Austria.

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Malogolowkin-Cohen, Ch. Institute of Evolution, University of Haifa, Israel. The distribution of *Drosophila subobscura* in relation to other species in Israel.

In the course of trapping *Drosophila subobscura* Collin with the purpose of analyzing its inversion polymorphism in a population considered to be marginal in Israel by Goldschmidt (1956, 1958) we made some interesting observations related to its distribution in relation with other species found in artificial baits in four

different biogeographic zones in Israel.

*D. subobscura* has a typical Mediterranean pattern of distribution in Israel penetrating chiefly the more humid areas of the country but does not colonize the hot and dry southern deserts. Traps were distributed according to the four biogeographic longitudinal regions of Israel, Coastal Plain, Foothills, Mountains and Rift Valley in the three mesothermal regions according to Thornthwaite's (1948) classification (Atlas of Israel, 1970, IV/3) avoiding the megathermal or high temperature zone. A total of 4006 individuals of five species of *Drosophila* were collected from March until the end of June in 1976 and 4888 during the same period in 1977. The species attracted to the artificial baits prepared with malted barley according to Lakovaara and col. (1969) were as follows: *D. subobscura* Collin, *D. melanogaster* Meigen, *D. simulans* Sturtevant, *D. hydei* Sturtevant, *D. buskii* Coquillett and *D. immigrans* Sturtevant. For technical reasons *D. melanogaster* and *D. simulans* were scored and analyzed under the heading of *simulans* group.

In general, the frequencies of the species collected changed greatly in time and according to the biogeographical regions where they were collected, as can be seen in Table 1. The most pronounced changes were exhibited by *D. subobscura* which decreases in frequency with the increase of temperature. Significant deviation from the 1:1 normal sex ratio was observed in the collected populations of *D. subobscura* and in a lower degree in *D. hydei*. In contrast to Shorrocks' (1975) observations the predominance of males was a constant trait of *D. subobscura* in the four biogeographic regions during the collecting season of 1976 and continued to be so in 1977 as may be seen in Table 2.

Table 2. Proportion of sexes of *D. subobscura* and of *D. hydei* in four geographic zones during the collecting season of 1976 and 1977 in Israel.  
n = total number of flies

		Biogeographic Zone							
		Coastal Plain		Foothills		Mountains		Rift Valley	
		♀	♂	♀	♂	♀	♂	♀	♂
<i>D. subobscura</i>	1976	n=256 54.70	45.30	n=130 34.0	66.0	n=912 35.0	65.0	n=16 31.25	68.75
	1977	n=115 19.13	80.87	n=130 36.15	63.85	n=380 22.0	78.0	n=38 29.0	71.0
<i>D. hydei</i>	1976	n=190 40.0	60.0	n=523 51.0	49.0	n=61 49.0	51.0	n=168 37.5	62.5
	1977	n=68 79.4	20.6	*	*	*	*	n=11 27.27	72.72

\* only two individuals.

Table 1. Changes in the relative frequencies of the different species of *Drosophila* collected in four biogeographic zones in Israel from March until May 1976 and 1977. The figures show the percentages of each species among the total number of *Drosophila* flies collected during a given period in each zone.

Zone	Month	D. subobscura		Simulans gr.		D. hydei		D. buskii		D. immigrans		no. of flies	
		1976	1977	1976	1977	1976	1977	1976	1977	1976	1977	1976	1977
Coastal Plain	March	44.72	51.39	1.19	19.75	19.77	*	20.22	5.03	8.10	26.46	435	179
	April	9.63	0.94	33.28	54.92	16.90	1.21	19.42	2.02	20.77	40.89	592	741
	May	0.0	0.75	10.20	76.31	4.08*	3.17	26.53	28.02	59.18	17.02	49	1862
Foot-hills	March	20.91	87.24	11.76	11.41	53.59	*	11.11	0.0	2.61*	0.0	152	149
	April	39.74	0.0	20.09	*	17.48	0.0	20.52	76.47*	2.18	0.0	235	17
	May	0.74	0.0	55.40	100.00	42.50	0.0	0.32*	0.0	1.06	0.0	944	60
Moun-tains	March	96.93	100.00	1.31*	0.0	1.31*	0.0	0.0	0.0	0.44*	0.0	228	35
	April	90.43	76.01	1.85	23.23	7.60	*	0.0	0.0	0.13*	*	763	396
	May	*	60.25	0.0	39.33	0.0	0.0	0.0	0.0	0.0	*	1	239
Rift Valley	March	12.70	0.63	30.95	68.74	35.71	1.14	6.35	3.05	14.28	26.43	136	787
	April	0.0	15.94	50.25	73.91	30.44	*	10.15	5.08	9.16	3.38	404	207
	May	-	-	-	-	-	-	-	-	-	-	-	-

\* four or less than four individuals  
 # only 13 individuals  
 (-) no traps laid

It is suggested that temperature may influence the local number of flies: lower temperature may diminish their sexual drive as suggested by Begon (1976) and high summer temperatures may possibly sterilize the females. This last suggestion is supported by the fact that no sperm cells were found in the spermathecae of females collected in late spring. To account for the deviation of the 1:1 sex ratio we suggest the possibility that females look for better habitats in order to oviposit their fertilized eggs. This would explain the higher percentage of males trapped near habitats where no other vegetation but pine trees and dry shrubs were to be found.

References: Begon, M., *Oecol.* (Berl) 23:31-47; Goldschmidt, E. 1956, *J. Genet.* 54:474-496; Goldschmidt, E. 1968, *Proc. Tenth Intern. Cong. Ent.* 2:821-828; Lakovaara, S., W. Hackman and K. Vespäläinen 1969, *DIS* 44:123; Shorrocks, B. 1975, *J. Anim. Ecol.* 44:851-863; Thornthwaite, C.W. 1948, *Geogr. Rev.* 38:55-94.

This work was supported by the Israel Absorption Center and by a grant from the U.S. - Israel BNSF, Jerusalem, Israel.

Marcos, R. and A. Creus. Autonomous University of Barcelona, Bellaterra, Spain. Predictive value of heritability estimates.

In the heritability studies we find two important questions. The first is about the method giving us the best estimate. The second is for how long are the predictions on the changes that result from selective breeding accurate.

According to Falconer (1960) the response to selection is  $R = h^2S$ . In spite of the fact that this prediction is theoretically valid only for one generation, the data reported by Clayton et al. (1957), Sheldon (1963) and other authors suggest its validity for more than one generation. The purpose of this work is to check for how long the expected response according to this formula is in agreement with the observed facts and to study which method of estimating heritability is the best.

We have worked with a natural population (AR) at 19°C. The heritability of interocellar bristles was estimated in the base population before selection began by the methods of parent-offspring regression, half sib correlation and full sib correlation. The results were:

parent-offspring regression:  $0.258 \pm 0.030$  (1)  
half sib correlation:  $0.354 \pm 0.050$  (2)  
full sib correlation:  $0.356 \pm 0.050$  (3)

This population has been submitted to mass selection with an intensity of selection of 20%. Four lines of selection were made: two high lines (A1H and A2H), and two low lines (A1L and A2L). Table 1

shows the response to selection from the first 10 generations and the expected responses according to the three methods mentioned.

Table 1

Line	Gen	$\Sigma S$	$\Sigma R_{Ob}$	$R_e(1)$	$R_e(2)$	$R_e(3)$	Line	Gen	$\Sigma S$	$\Sigma R_{Ob}$	$R_e(1)$	$R_e(2)$	$R_e(3)$
<u>A1H</u>	1	1.226	0.413	0.316	0.434	0.436	<u>A1L</u>	1	1.300	-0.006	0.335	0.460	0.463
	2	2.654	0.577	0.684	0.939	0.944		2	2.325	0.708	0.599	0.823	0.827
	3	3.958	0.708	1.021	1.401	1.409		3	3.508	0.876	0.905	1.241	1.249
	4	5.318	0.980	1.372	1.882	1.893		4	4.772	1.146	1.231	1.689	1.699
	5	7.485	-0.368	1.931	2.649	2.665		5	7.238	-0.040	1.087	2.562	2.576
	6	8.978	0.847	2.316	3.178	3.196		6	8.689	1.743	2.241	3.075	3.093
	7	10.451	1.668	2.696	3.699	3.720		7	10.342	1.906	2.668	3.661	3.681
	8	11.845	1.748	3.056	4.193	4.216		8	12.355	2.580	3.187	4.373	4.398
	9	13.371	1.948	3.449	4.733	4.760		9	14.455	3.626	3.729	5.117	5.146
	10	15.278	2.435	3.941	5.408	5.439		10	15.882	4.466	4.022	5.622	5.655
<u>A2H</u>	1	1.202	0.474	0.315	0.425	0.428	<u>A2L</u>	1	1.240	-0.180	0.319	0.439	0.441
	2	2.608	0.433	0.673	0.923	0.928		2	2.446	0.453	0.631	0.866	0.870
	3	3.922	0.693	1.011	1.388	1.396		3	3.560	0.519	0.918	1.260	1.267
	4	5.208	1.020	1.343	1.843	1.854		4	4.673	0.613	1.205	1.654	1.663
	5	6.955	1.326	1.794	2.462	2.476		5	5.473	0.760	1.468	1.942	1.948
	6	8.629	1.600	2.226	3.054	3.072		6	6.693	0.940	1.726	2.369	2.383
	7	10.982	2.586	2.833	3.887	3.909		7	7.720	1.033	1.991	2.733	2.748
	8	13.162	3.126	3.396	4.659	4.685		8	9.326	1.519	2.406	3.301	3.320
	9	16.665	4.080	4.297	5.896	5.929		9	11.414	2.339	2.945	4.040	4.063
	10	20.688	6.073	5.337	7.323	7.365		10	13.346	2.826	3.443	4.725	4.751



Table 2

Lines	Method		
	(1)	(2)	(3)
AlH	7	4	4
A2H	10	4	4
AlL	10	6	6
A2L	4	2	2

From the two-way anova without replication, we obtain the results summarized in Table 2 that show during how many generations the expected response estimated by the three methods is in agreement with the observed values. It is clear that the parent-offspring regression gives us the best estimate.

References: Clayton, G.A., J.A. Morris and A. Robertson 1957, J. Genet. 55:131-151; Falconer, D.S. 1960, Introduction to Quantitative Genetics, Ronald Press Co., New York; Sheldon, B.L. 1963, Aust. J. Biol. Sci. 16:490-515.

Marengo, N.P. C.W. Post College of Long Island University, Greenvale, New York. The ultrastructure of normal and "rotated" prepupal muscles of *D. melanogaster*.

The mutation abdomen rotatum (*ar*) of *D. melanogaster* was discovered and named by Beliajeff (1931). The effect of this gene on development was described by the writer and Howland (1942). It was then suggested that the symmetrical abnormalities of the mutant puparium as well as

the sudden rotation of the imaginal abdomen at the end of the prepupal period were both due to abnormally strong contractions of the persisting larval muscles, which bring about the shortening of the puparium as well as the movements of the imago as it separates from the puparium.

It appears to be of interest to determine whether or not there might be an ultrastructural difference between the heterozygous normal prepupal muscles and the homozygous "rotated" muscles in individuals in the balanced lethal stock *ar/ey<sup>D</sup>*.

Muscle fibers of normal prepupae and genetically "rotated" prepupae were dissected out in glutaraldehyde, routinely fixed and post-fixed in osmium tetroxide, embedded in epon, sectioned with a diamond knife, stained with uranyl acetate and lead citrate and examined with an Hitachi HU-11A EM. There appear to be consistent differences between the prepupal muscles of the normal and genetically "rotated" individuals. An account of these follows.

The ultrastructure of the normal prepupal muscles appears identical to the intersegmental abdominal muscle of the insect *Rhodnius prolixus* (Toselli and Pepe 1968). These are described as typical "slow-acting" muscles, lacking the "H" zone and the "M" line (Fig. 1).

The "rotated" prepupal muscles show a number of consistent differences from the normal muscles. First, there appears to be an exaggerated irregularity in the "Z" bands (Fig. 1, 2; Z). Second, the "I" bands show a markedly increased width, with the resultant decrease in the width of the "A" bands (Fig. 1, 2, I, A). Third, the myofibrillae of the "rotated" muscles show a disorganization in the regions where they impinge upon the "I" bands (Fig. 2, D). It would seem that the combination of these three apparent muscle pathologies is the ultrastructural basis for the abnormally strong contractions which bring about the phenotypic abnormalities of the "rotated" puparium and imago.

References: Beliajeff, N.K. 1931, Biol. Zbl. 51:701-709; Marengo, N.P. and R.B. Howland 1942, Genetics 27:604-611; Toselli, P.H. and F.A. Pepe 1968, J. Cell Biol. 37:445-461.

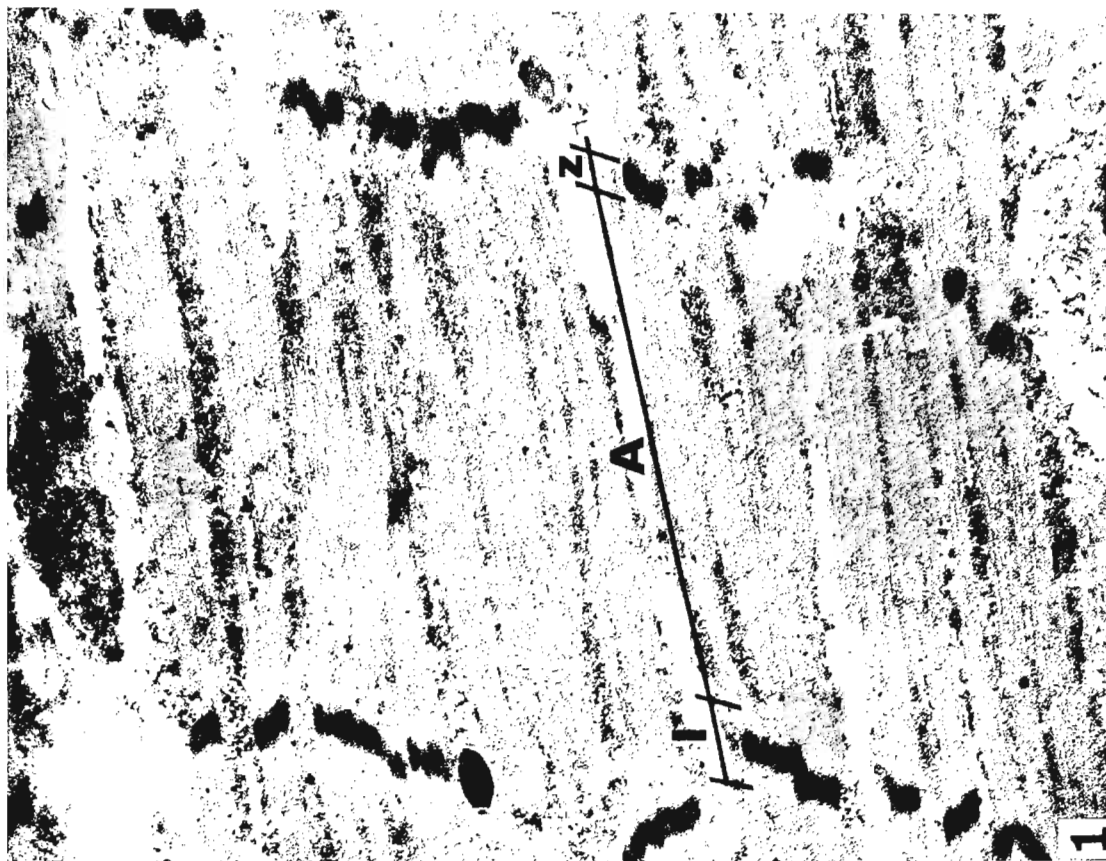


Fig. 1. Electron micrograph of a longitudinal section of pre-pupal muscle from a heterozygous normal prepupa of the ar/ey<sup>D</sup> stock. The "I", "A" and "Z" bands are designated by letter. X2893.

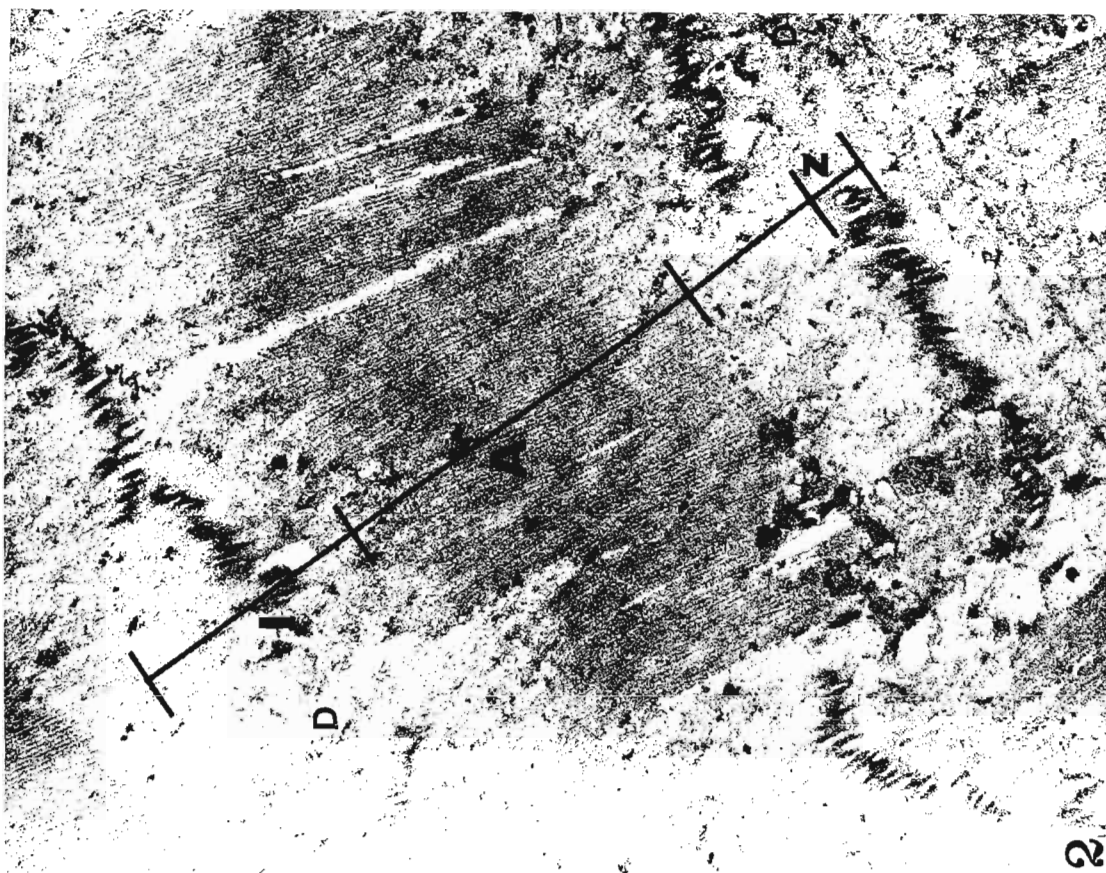


Fig. 2. Electron micrograph of a longitudinal section of pre-pupal muscle from a homozygous "rotated" prepupa of the ar/ey<sup>D</sup> stock. The identified "I", "A" and "Z" bands are to be compared with equivalent bands in the normal muscle. Note the disruption of the "A" bands in the regions indicated (D). X2893.

Maroni, G. University of North Carolina, Chapel Hill, North Carolina. A duplication of Adh in association with Sco.

From the following cross:  $b\ el\ Adh^{n1}/Adh^F Sco$  X  $b\ el\ Adh^{n1}/b\ el\ Adh^{n1}$  two recombinants carrying  $b\ el\ Sco$  among a total progeny of 10,094 flies were obtained. Both were Adh-positive. One of these chromosomes was kept in a stock and further

analyzed by genetic means. As a result of these studies, it is concluded that this recombinant chromosome carries a duplication that involves the Adh locus and that this duplication may have arisen as a consequence of the Sco mutation being associated with a chromosomal rearrangement. The approximate map positions of the loci used are shown below (O'Donnell et al. 1977 and pers. obs.).  $1^2$  is a lethal mutation obtained in our laboratory by treatment with EMS. The remaining markers are described in Lindsley and Grell (1968). Several crosses were performed to select recombinants between  $1^2$  and Sco. Heterozygotes of the type  $1^2 el^+ Sco^+ / 1^2 el Sco$  were

b	$1^2$	el	Adh	Sco
48.5	49.8	49.9	50.1	50.12

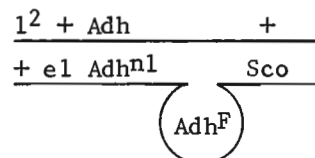
crossed to males carrying the deletion  $Df(2L)64j, pr$  which includes both loci. The specific genotypes used in the different experiments and the results obtained are indicated in Table 1.

Parents			Progeny		
Female	X	Male	Curly	$b1^2 el^+ Sco^+$	$b1^2 el Sco^+$
$b\ 1^2 + Adh^{B7} +$					
$b + el\ Adh^F Sco$			899	1	8
$+ 1^2 + Adh^{A1} +$		$Df(2L)64j, pr$			
$b + el\ Adh^F Sco$		CyO	1287	0	4
$+ 1^2 + Adh^{n5} +$					
$b + el\ Adh^F Sco$			4707	16	33
Total			6893	17	45

Recombinants were crossed to a second-chromosome balancer stock for further tests. Of the 45 recombinant chromosomes between  $el$  and  $Sco$ , three had detectable ADH activity and its electrophoretic mobility corresponded to the chromosome carrying  $1^2$ : in two cases  $Adh^{n5}$  and in the other case  $Adh^{B7}$ . The remaining 42 recombinants had no ADH activity at all. When these ADH-negative

chromosomes were made heterozygous with an ADH-slow allele and extracts run in polyacrylamide gel electrophoresis, staining of the gels for ADH put in evidence a band of activity running slightly ahead of the major ADH-S band. This behavior is typical of the  $Adh^{n1}$  allele and it is due to the fact that the inactive polypeptide made by  $Adh^{n1}$  produces an active heterodimer with the ADH-S polypeptide.

Since this result was obtained with recombinants from three different  $Sco^+$  chromosomes and since neither  $Adh^{B7}$  nor  $Adh^{A1}$ , both with slow mobility, show by themselves the extra band indicating the presence of an inactive polypeptide, the presumed  $Adh^{n1}$  allele must be carried by the  $b\ el\ Sco$  chromosome. Given the way in which this chromosome was obtained (see above), it seems reasonable to suspect that unequal crossing-over occurred leading to the production of a duplication-carrying chromosome:  $b\ el\ Adh^{n1} Adh^F Sco$  such that pairing with it and crossing over would occur as follows:



If this were the case, it should be possible to recover, also, the reciprocal cross-over class with the  $Adh$  allele of the  $Sco^+$  chromosome and  $Adh^F$  from the  $Sco$  chromosome. In the crosses presented above, such flies do not survive because  $1^2$  and  $Sco$  are uncovered by the deficiency  $64j$ .

Crosses were then set up in which recombination between  $el$  and  $Sco$  would be detected by visual inspection, allowing the survival of all classes of progeny. The results obtained are as follows:

Parents			Progeny		
Female	X	Male	Parental	1 <sup>2</sup> el <sup>+</sup> Sco	1 <sup>2</sup> el <sup>+</sup> Sco <sup>+</sup>
+ 1 <sup>2</sup> + Adh <sup>Al</sup>		+			
b + el Adh <sup>nl</sup> Adh <sup>F</sup> Sco			1235	8	7
		b el Adh <sup>nl</sup>			
+ + Adh <sup>S</sup>		b el Adh <sup>nl</sup>	3604	15	13
b el Adh <sup>nl</sup> Adh <sup>F</sup> Sco					
Total			4839	23	20

cases the two alleles are in the same chromosome indicating the existence of a duplication. The remaining five recombinant chromosomes were phenotypically ADH-F. Whether Adh<sup>nl</sup> is present or not in these chromosomes has not been established.

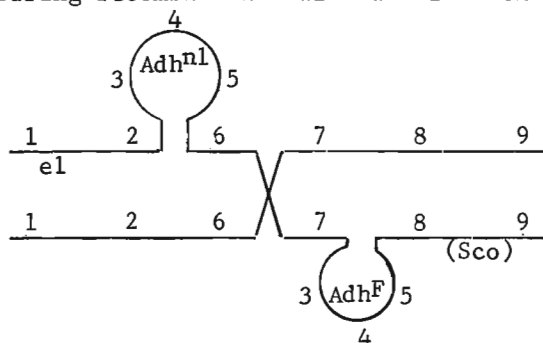
Of the 20 recombinants 1<sup>2</sup>el<sup>+</sup>Sco<sup>+</sup>, as before, the majority (16) were ADH-negatives and upon testing in heterozygotes with ADH-slow they put in evidence the presence of an Adh<sup>nl</sup>-like allele. Of the remaining four, one had Adh<sup>Al</sup> and three Adh<sup>S</sup> without Adh<sup>nl</sup>. These results lend support to the interpretation given in the diagram shown above. That is to say that in b el Sco there is an Adh<sup>F</sup> gene, presumably the one in the original Sco, that is positioned to the right of the normal Adh site and another Adh gene which occupies the standard Adh locus, as determined by the fact that the specific allele present at this second site can be interchanged by crossing over with a standard-sequence chromosome.

One question of interest is to establish whether the occurrence of a duplication in b el Sco has a structural basis in the two chromosomes originally involved: b el Adh<sup>nl</sup> and Sco. It has been suggested by Ashburner, Woodruff and Camfield (pers. comm.) that Sco is a rearrangement involving at least three breakpoints to the right and left of Adh. This suggestion is based on the properties of reversions of Sco and cytological observations. A deletion-insertion type of rearrangement with breakpoints as indicated in the figure below would fit the observations presented here.

el<sup>+</sup> Adh<sup>+</sup> → el<sup>+</sup>(Sco) Adh<sup>+</sup>

1 2 3 4 5 6 7 8 9      1 2' 6' 7' 3 4 5' 8 9

During recombination with a normal chromosome:



To give rise to the b el Sco chromosome, with the Adh<sup>nl</sup>, Adh<sup>F</sup> duplication.

b      Adh<sup>nl</sup>      Adh<sup>F</sup>      (Sco)

1   2   3   4   5   6   7   3   4   5   8   9

Parents			Progeny		
Female	X	Male	Parental	cross-over between b and el	el and Sco
b el Adh <sup>F</sup> rd pr cn		b el Adh <sup>nl</sup>	6711	120	4
+ + Adh <sup>F</sup> + (Sco)		b el Adh <sup>nl</sup>			

The 23 1<sup>2</sup>el<sup>+</sup>Sco chromosomes were made heterozygous with CyO Adh<sup>nl</sup>, a balancer chromosome that produces no detectable ADH polypeptide. Seventeen of them produced the multiple bands characteristic of flies with two active alleles, one slow and one fast, as in heterozygotes. In these

An attempt was made to test this interpretation by generating new duplications and the expected reciprocal deletions. Heterozygotes b el Adh<sup>F</sup>rd pr cn/Adh<sup>F</sup>Sco were crossed to males b el Adh<sup>nl</sup> to detect recombination between el and Sco. Recombinants b el Sco would carry two doses of Adh<sup>F</sup> and show a marked increase in ADH activity and, more conclusively, recombinants b<sup>+</sup>el<sup>+</sup>rd pr cn would be deficient for ADH. The result is shown in the table below.

Of the four recombinants between el and Sco three were b<sup>+</sup>el<sup>+</sup>rd pr cn and all of them had Adh<sup>F</sup>. One was the reciprocal, b el Sco; it could not be kept in a stock but the recombinant itself did not seem to have an increased level of ADH. Thus, these results are inconclusive in demonstrating that Sco is a transposition. It is entirely possible that the size of the segments involved

and constraints on the recombinational process imposed by the rearrangement itself make recombination in the segment 6-7 much less frequent than in the segment 1-2 (see diagram).

The frequency of recombination in this region also supports the idea that *b el Sco* carries a sizeable duplication. The frequency of recombination between *el* and *Sco* in this chromosome is 1.3% in the experiments in Table 1 and 0.9% in those in Table 2. The recombination frequency between these two markers when the original *Sco* chromosome is involved is 0.06% from Table 3, a value which is in agreement with published results. It should be mentioned that in all the crosses described here females also carried a pair of attached X's (*C(1) RM, y*). The results presented here, although they do not close the issue, fit well with the idea that *Sco* is a multiple point rearrangement. Possibly an insertion of a chromosomal segment, which includes *Adh*, to a position slightly to the right of its normal location. It might be noted that if this is correct the left breakpoint of this segment (2-3 in the diagrams) should be to the right of *el* since the recombinant *b el Sco* carries *Adh<sup>F</sup>* in the duplicated piece (the insertion) but not *el<sup>+</sup>*. Finally, the chromosome *b el Sco* is a useful tool to generate duplications involving any allele of *Adh* in combination with *Adh<sup>F</sup>*.

References: Lindsley and Grell 1968, Carnegie Inst. Wash. Publ. 627; O'Donnell, J. et al. 1977, Genetics 86:553-566.

Maróy, P., K. Koczka, É. Fekete and J. Vargha. Biological Research Center, Szeged, Hungary. Molting hormone titer of *D. melanogaster* larvae.

The molting hormone (MH) titer of *D. melanogaster* has been studied during metamorphosis by Borst and O'Connor (1972), de Reggi et al. (1975), and Hodgetts et al. (1977). In this paper we study the changes of MH titer during larval life of *D. melanogaster* using MH specific radio-

immunoassay (Maróy et al. 1977). Eggs were collected for a period of one hour and cultures were synchronized for hatching. Specimens were weighed and homogenized in an all-glass Potter-type homogenizer in 60% methanol, and treated in the standard way according to Maróy and Tarnóy (1978).

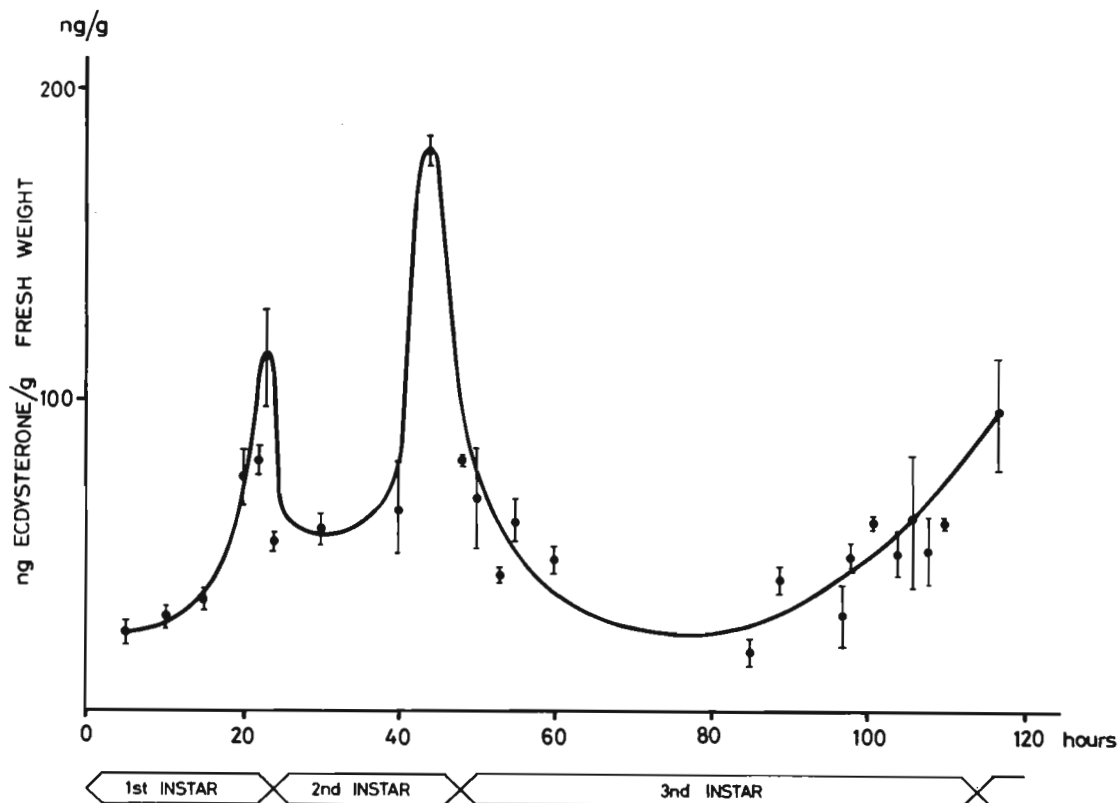


Fig. 1. MH titer of *D. melanogaster* during larval life.

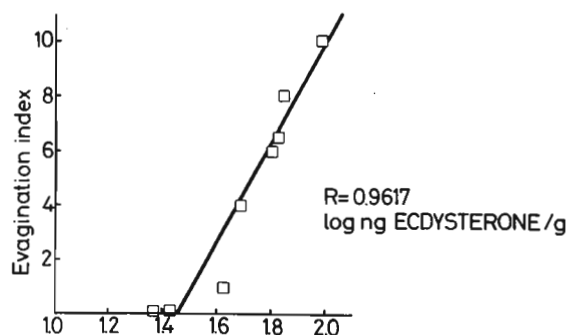


Fig. 2. Effect of in situ titer value on evagination index reached after 16 hours incubation of leg discs in Robb's medium without MH at 25°C.

discs were dissected. The discs were rinsed and incubated in Robb's medium (Robb 1969) at 25°C without MH. The evagination score was determined after 16 hours. The rest of the larvae were used to measure MH titer. Fig. 2 shows the scores reached plotted against RIA activity measured in the rest of the colonies from which discs had been taken. 54.4 ng/g MH titer in situ at the time of dissection is enough to reach a score of 5. Considering the short exposure time, this number fits well with the 34.7 ng/ml necessary for in vitro evagination determined by Fristrom and Yound (1975). For full evagination under in situ conditions 92.7 ng/g titer is necessary.

References: Borst, D. and J.D. O'Connor 1972, *Science* 178:418; de Reggi, M.L., M.H. Hirn and M.A. Delaage 1975, *Biochem. Biophys. Res. Commun.* 66:1307-1315; Hodgetts, R.B., B. Sage and J.D. O'Connor 1977, *Develop. Biol.* 60:310-317; Maróy, P., J. Vargha and K. Horváth 1977, *FEBS Lett.* 81:319-322; Maróy, P. and K. Tarnóy 1978, *J. Insect Physiol.* 24:325-327; S.A. Robb 1969, *J. Cell Biol.* 41:876-884; Fristrom, J.W. and M.A. Yund 1976, in: *Invertebrate Tissue Culture Research Applications* (ed. K. Maramorosch), Acad. Press, NY.

Mather, W.B. and G. Balwin. University of Queensland, Brisbane, Australia. Inversions in two species of *Drosophila* from the River Kwai, Thailand.

From a collection of *Drosophila* from the River Kwai region of Thailand (June 1978) 76 isolines of *D. sulfurigaster albostrigata* and 14 isolines of *D. albomicans* were established.

Table 1. *D.s. albostrigata*

Inversion	Type	Chromosome	Breakpoints	Het. Freq. %	
				June '78	Nov '77
C	Sim.	III		1.3	2.8
E	Sim.	II L		14.5	28.6
W <sub>2</sub>	Sim.	III		3.9	
X <sub>2</sub>	Com.	III		2.6	
A <sub>5</sub>	Sim.	II L		55.3	31.4
B <sub>5</sub>	Sim.	III C		7.9	2.8
C <sub>5</sub>	Sim.	II R		55.3	25.7
D <sub>5</sub>	Com.	II L		34.2	5.7
Q <sub>5</sub>	Sim.	II L	5.0 - 9.3	2.6	

Note: Sim. = simple; Com. = complex

#### (a) *D.s. albostrigata*

Seven simple and two complex inversions were detected. Six of these had previously been detected at the River Kwai (Mather, Knibb and Balwin 1979) and two had been detected elsewhere in South East Asia (Mather, Thongmeearkom, Clyde and Lambert 1974; Thongmeearkom 1977). The remaining inversion Q<sub>5</sub> is new, and a photograph is presented and breakpoints assigned (in relation to the standard photographic map - Thongmeearkom 1977) (see Table 1).



Note: C.E. = centromere end; F.E. = free end



Table 2. *D. albomicans*

Inversion	Type	Chromosome	Breakpoints	Het. Freq.
R <sub>5</sub>	Sim.	I	12.0 - 18.0	14.3
S <sub>5</sub>	Sim.	II L	6.0 - 10.4	14.3
T <sub>5</sub>	Sim.	II L	8.2 - 21.3	7.1
U <sub>5</sub>	Com.	II L	1.1 - 13.2	50.0
C	Sim.	III	6.1 - 11.0	28.7
V <sub>5</sub>	Sim.	III	2.5 - 10.8	7.1
W <sub>5</sub>	Sim.	III	27.0 - 30.2	14.3
X <sub>5</sub>	Com.	III	21.6 - 40.4	74.5

Note: Sim. = simple; Com. = complex

Table 2). The heterozygosity frequency of all inversions detected is given (Table 2).

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

References: Mather, W.B., W.R. Knibb and G. Balwin 1979, DIS 54; Mather, Thongmeearkom, Clyde and Lambert 1974, DIS 51:86; Mather and Thongmeearkom 1979, DIS 54; Thongmeearkom 1977, DIS 52:154; Thongmeearkom 1977, DIS 52:117.

Mather, W.B. and P. Thongmeearkom. University of Queensland, Brisbane, Australia. Chromosome map of *D. albomicans*.

A photographic chromosome map of *D. albomicans* is presented. This map was constructed from an inversion free iseline established from Taiwan in 1972.

Inversion	Locality	Chromosome	Breakpoints	Inversion Photograph Reference
B'	Taiwan	III	36.0 - 43.6	1972b
C	Kuala Lumpur	III	6.1 - 11.3	1972a
E'	Taiwan	II L	6.5 - 20.3	
L <sub>3</sub>	Phuket	III	21.6 - 36.5	
T <sub>4</sub>	Phuket	III	11.1 - 16.4	

Note E' differs from E (Mather and Thongmeearkom 1972a) because it is also homozygous for I<sub>2</sub> (Mather and Thongmeearkom 1973).

40; Mather and Thongmeearkom 1972b, DIS 49:110; Mather and Thongmeearkom 1973, DIS 50:60.

The heterozygosity frequency of all inversions detected is given and compared with November 1977. It will be noted that there are very marked differences in frequency.

#### (b) *D. albomicans*

Six simple and two complex inversions were detected. Only one (C) had previously been detected in South East Asia (Thongmeearkom 1977; Mather and Thongmeearkom 1979). The others are new and photographs are presented and breakpoints assigned (in relation to the standard photographic map - Mather and Thongmeearkom 1979) (see

Photographs of two new inversions from Phuket, Thailand 1975 are presented.

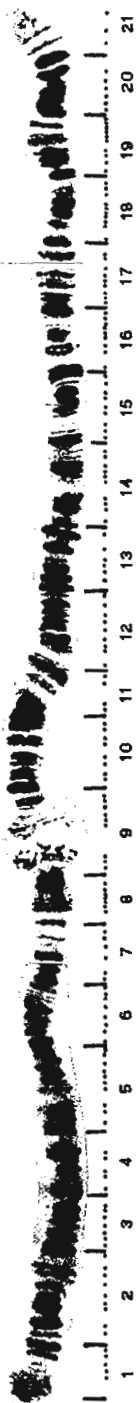
The breakpoints of inversions previously detected in natural populations from Taiwan and Kuala Lumpur as well as those of the new inversions are assigned.

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

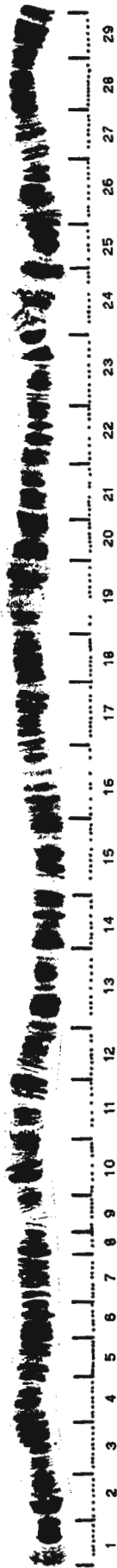
References: Mather and Thongmeearkom 1972a, DIS 48:



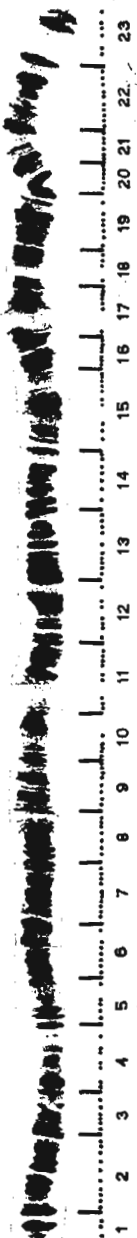
# Chromosome I



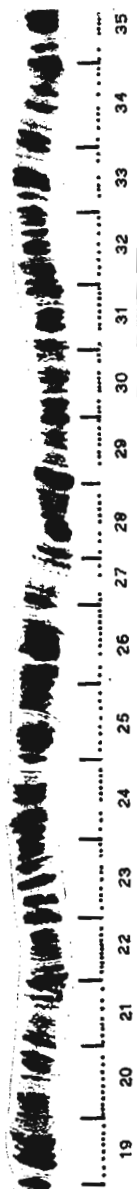
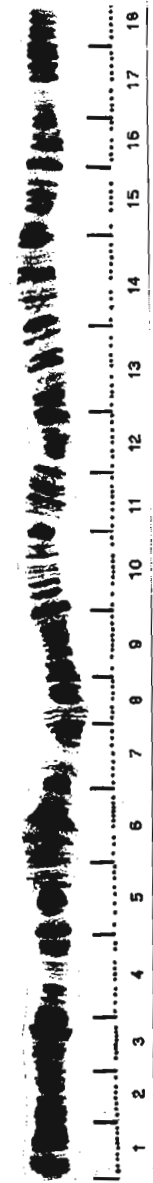
# Chromosome II R



# Chromosome III



# Chromosome III



# Chromosome IV



36 37 38 39 40 41 42 43 44 45 46 47 48 49 50

Mather, W.B., W.R. Knibb and G. Balwin.  
University of Queensland, Brisbane,  
Australia. Inversions in *D. sulfurigaster*  
*albostrigata* from the River Kwai,  
Thailand.

From a collection of *Drosophila* from the River Kwai region of Thailand (November 1977) 35 isolines of *D. sulfurigaster albostrigata* were established.

Sixteen simple and four complex inversions were detected. Three of the simple and one of the complex inversions have previously been detected in South East Asian populations (Mather and Thongmeearkom 1972, DIS 48:40; Mather and Thongmeearkom 1973, DIS 50:60; Mather, Thongmeearkom, Clyde and Lambert 1974, DIS 51:86). The remaining inversions are new and photographs are here presented.

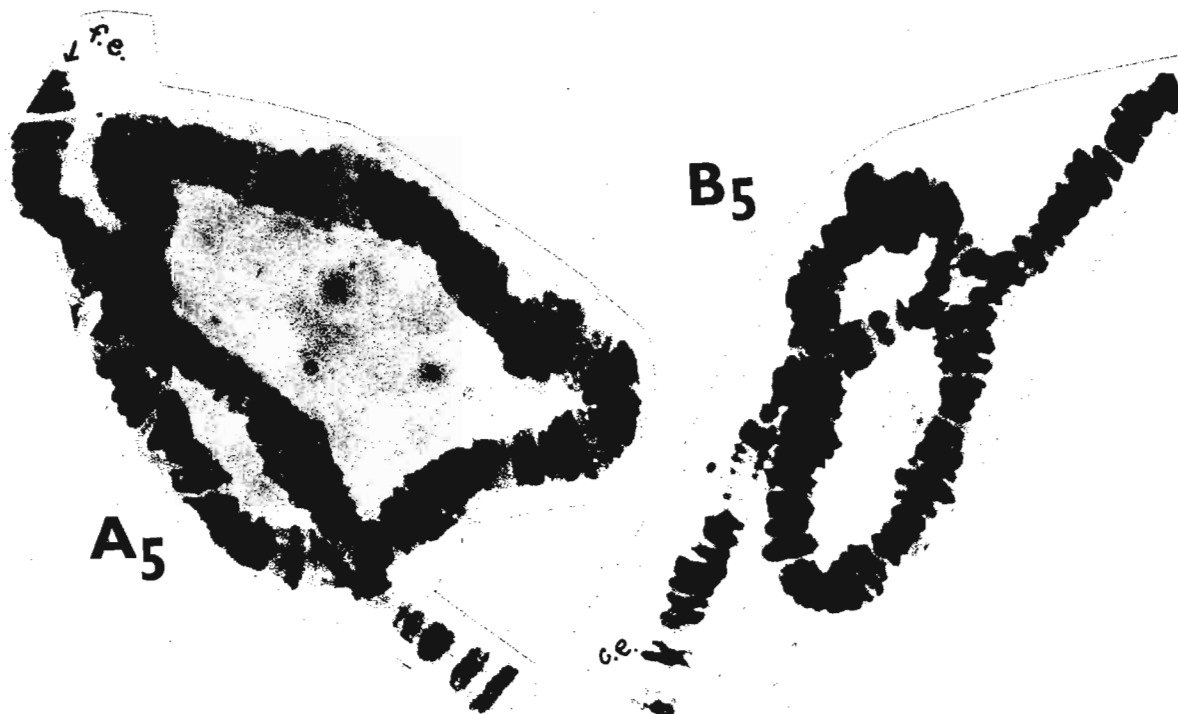
Breakpoints of these inversions (in relation to the standard photographic map - Thongmeearkom 1977, DIS 52:154) are given in the table. Breakpoints of inversions C, E and Y<sub>2</sub> are given in Thongmeearkom 1977, DIS 52:154. Breakpoints of inversion J<sub>2</sub> have not been previously determined and are here presented. The heterozygosity frequency of all inversions detected are given.

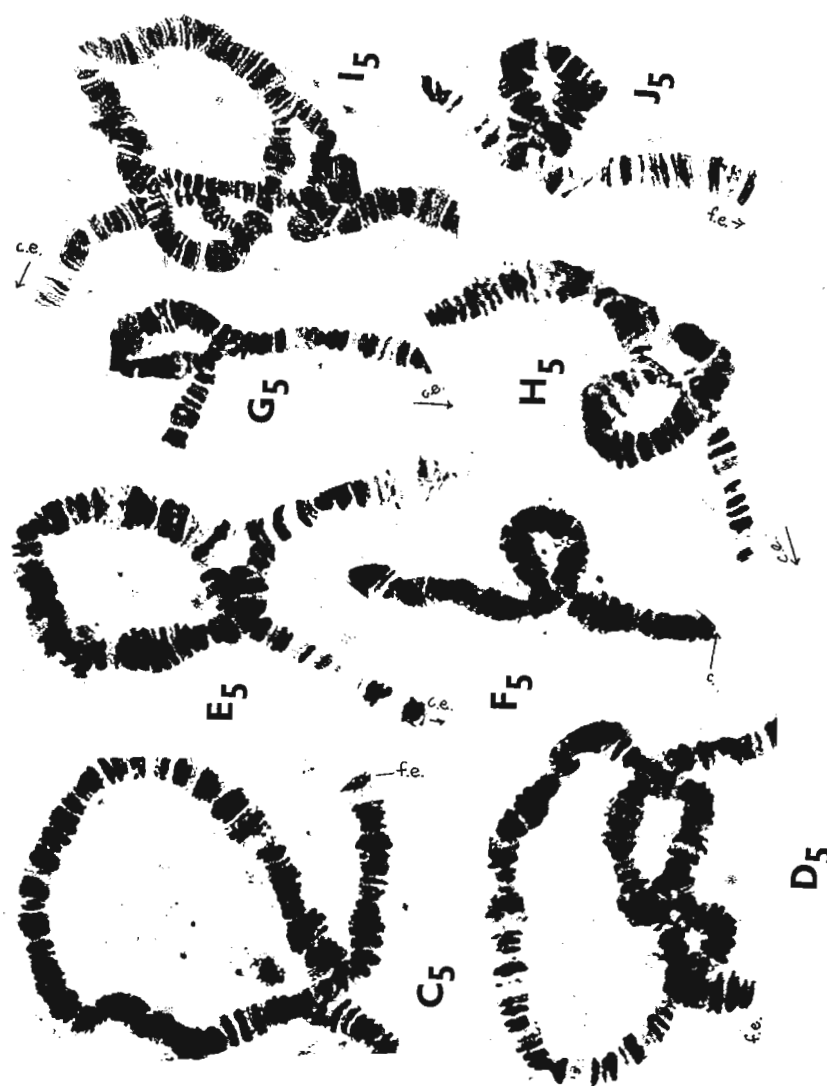
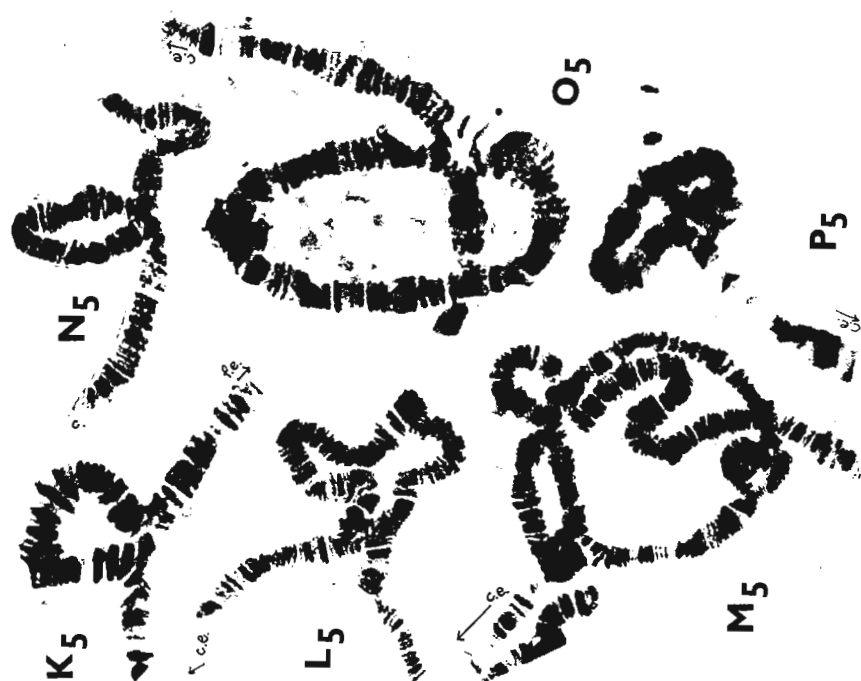
The material was collected and the isolines established by W.B.M. The laboratory work was carried out by W.R.K. and G.B.

Inversion	Type	Chromosome	Breakpoints	Het.Freq.
C	Sim.	III	-	2.8
E	Sim.	II L	-	28.6
J <sub>2</sub>	Com.	II L	6.6 - 21.2	11.4
Y <sub>2</sub>	Sim.	II L	-	11.4
A <sub>5</sub>	Sim.	II L	3.2 - 17.2	31.4
B <sub>5</sub>	Sim.	III	26.4 - 35.4	2.8
C <sub>5</sub>	Sim.	II R	4.9 - 20.9	25.7
D <sub>5</sub>	Com.	II L	3.2 - 21.2	5.7
E <sub>5</sub>	Sim.	I	10.5 - 19.3	2.8
F <sub>5</sub>	Sim.	III	38.1 - 41.4	2.8
G <sub>5</sub>	Sim.	III	31.3 - 35.2	2.8
H <sub>5</sub>	Sim.	III	36.5 - 40.9	2.8
I <sub>5</sub>	Com.	III	16.8 - 35.6	2.8
J <sub>5</sub>	Sim.	III	16.1 - 18.2	2.8
K <sub>5</sub>	Sim.	III	31.3 - 35.8	2.8
L <sub>5</sub>	Sim.	III	21.0 - 33.2	2.8
M <sub>5</sub>	Com.	III	18.2 - 35.4	8.6
N <sub>5</sub>	Sim.	III	35.6 - 41.0	2.8
O <sub>5</sub>	Sim.	III	23.9 - 34.6	2.8
P <sub>5</sub>	Sim.	III	35.3 - 40.5	2.8

Note: Sim. = simple, Com. = complex

Photographic note: c = centromere, c.e. = centromere end, f.e. = free end





Mglinetz, V.A. Institute of Medical Genetics, Moscow, USSR. Bristle phenotype of split flies contradicts the hypothesis of quantal mitoses.

ments and only exceptionally more than four elements are involved. These data are in agreement with the suggestion by Lees and Waddington (1942) that the split allele induces one or two additional divisions of tormogenic and trichogenic cells. In all cases the shafts of the

The structure of bristle apparatus in distal parts of the femurs was studied in split mutants of *D. melanogaster*. The main observed types of relations between the shaft, the socket and the bract are shown in Fig. 1. As a rule abnormal bristle apparatus consists of four ele-

ments and only exceptionally more than four elements are involved. These data are in agreement with the suggestion by Lees and Waddington (1942) that the split allele induces one or two additional divisions of tormogenic and trichogenic cells. In all cases the shafts of the bristles are lacking the bracts are also invariably lacking in spite of the presence of extra socket-cells. But if even a short shaft is present - just protruding above the surface - the bract is also present. Bracts are never observed in the presence of the shaft if the socket cell surrounding the shaft is mounted itself, completely or nearly so, into another socket usually consisting of two cells. Thus, the second socket cell is an obstacle preventing the induction of the bract. It may be suggested that the atypically located bracts are induced in the same way: when the inducing stimulus cannot pass in its usual direction it may spread in some other one, free of the second row of socket cells. As alterations in trichome orientation near the atypical chaetal sets are independent of the bract position it seems plausible that the alteration in bract position is not due to changes in bristle apparatus polarity. The changes in orientation of trichomes become more clear on the tergites of the split mutants as trichomes form more or less parallel rows there (Fig. 2). In the vicinity of anomalous bristle apparatus not only the orientation of single trichomes but of the rows of trichomes is altered. As each row has to correspond to a certain level of intrasegmental gradient, the disorientation of the row may be interpreted either as its mechanical deformation due to excessive growth of the bristle cells or as a result of a decrease in the gradient values around the bristle apparatus. If the former were true the trichome rows might be shifted both forwards and backwards as the enlargement of the bristle cells is rather isotropic. If the second interpretation were true, it is to be suggested that the chaetal cells decrease the gradient level around them, perhaps utilizing the gradient-carrier for their growth. Then the proximo-distal gradients

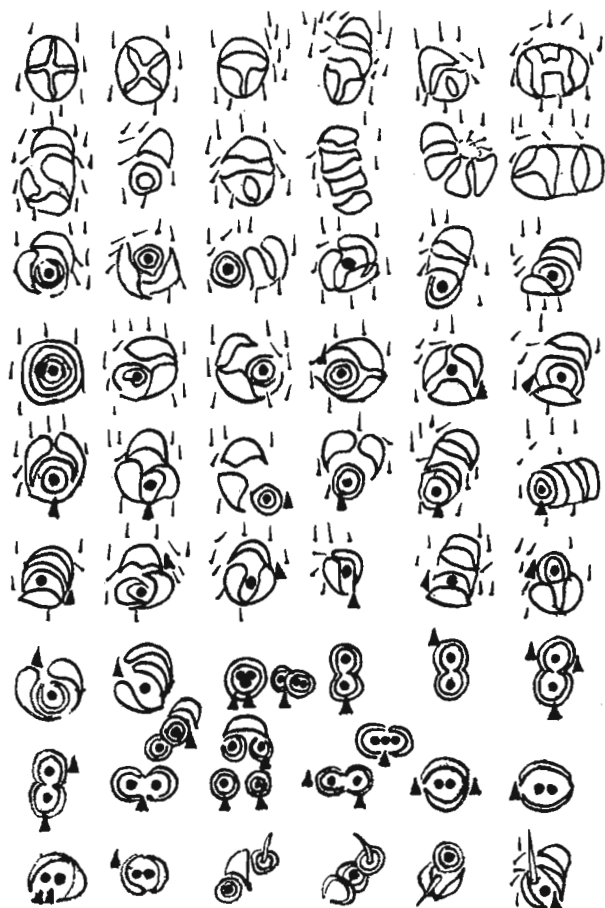


Fig. 1. A scheme of some bristle apparatus types found in the distal part of the femur in *spl/spl* flies. The position of socket cells is shown; the shafts and the bracts are shown as black circles and triangular, respectively.

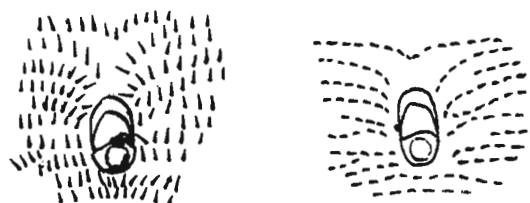


Fig. 2. A scheme of a bristle apparatus having two socket cells and the rows of trichomes on the tergite of *spl/spl* fly (A). B - a supposed position of gradient levels in the bristle apparatus region.

in the legs and the anterior-posterior gradients in the tergites have logically to be described as concentrational.

The observations on the position of socket cells in the bristle apparatus clearly show that the additional divisions in chaetal mother cells may take place in any plane, independently in each cell. A scheme of the chaetal phenotypes which are to arise depending on the division plane and suggesting that the cells of the upper layer are as usually tormogenic, and the underlying ones trichogenic, is shown in Fig. 3. Practically all the actual types

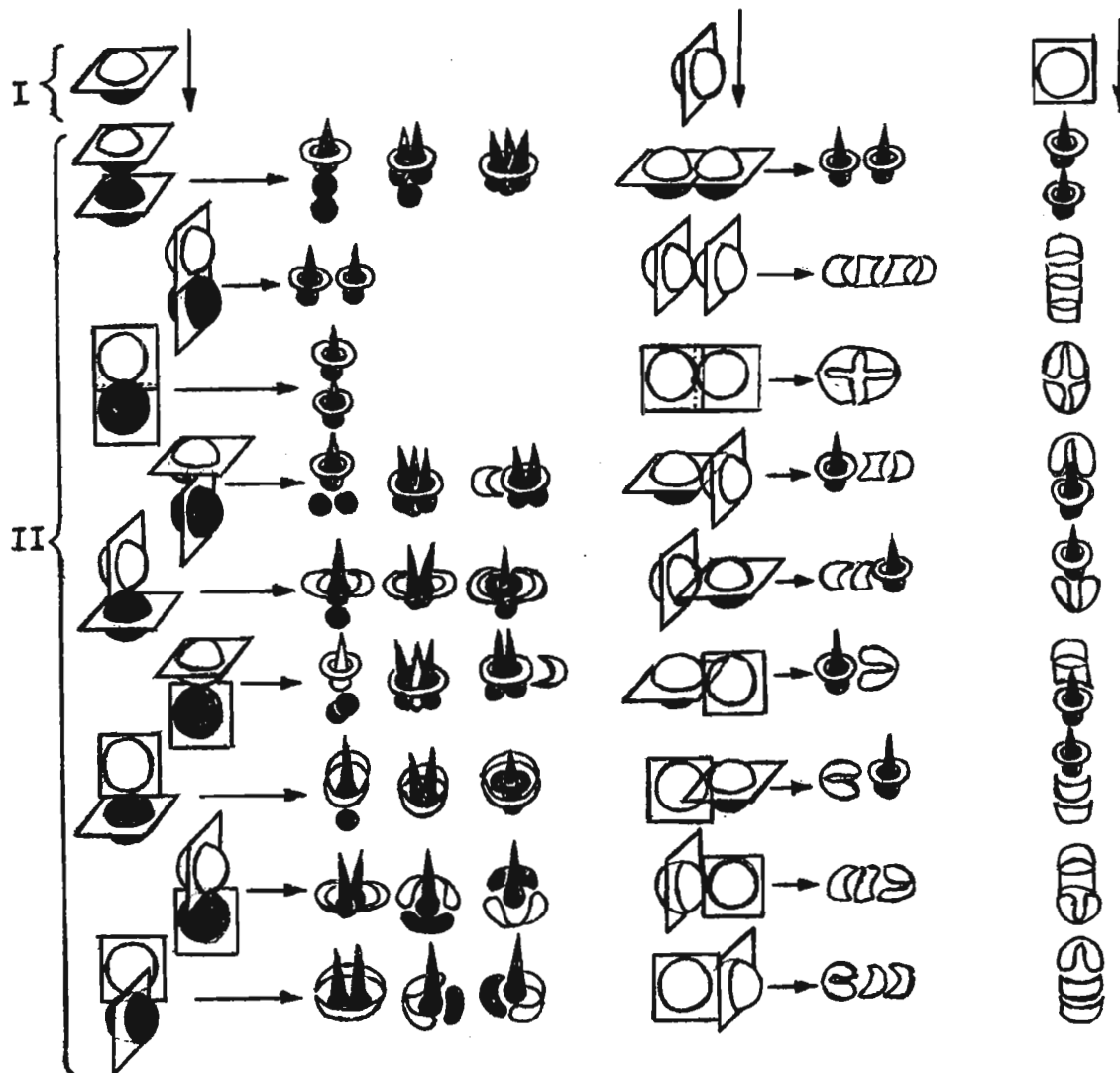


Fig. 3. A scheme of division of the bristle mother cells in *spl/spl* flies. I - three diverse division planes of the bristle mother cells. II - possible division planes during additional divisions and supposed relations between the division descendants (black = trichogenic cells; white = tormogenic ones).

of anomalous *spl* chaete may be foreseen in this way, which proves our above suggestion to be true. All the division descendants occurring in the surface cuticular layer differentiate into socket cells. It seems that the conditions in the upper and lower layer do qualitatively differ, thus causing the differences between the trichogenic and the tormogenic cells. Thus the hypothesis of the quantal mitoses (Holtzer et al. 1972) seems to be inapplicable to chaeta development in *Drosophila* as the type of the cell is determined not by the cell division per se, but rather by the environmental conditions. The data obtained also allow for a suggestion that the split allele changes the division plane.

References: Holtzer et al. 1972, *Cur. Top. Develop. Biol.* 17:229; Lees and Waddington 1942, *Proc. R. Soc. B.* 131:87.

Mglinetz, V.A. Institute of Medical Genetics, USSR Academy of Medical Sciences, Moscow. Interaction of homoeotic mutations  $Pc^2$  and Cbx.

As  $Pc$  mutations affect not only the leg but also the wing development (Denell 1978) it seemed worthwhile to investigate whether or not such mutations show interaction with wing to haltere transforming mutation Cbx. The rate of Cbx induced homoeosis was estimated as the size of

wings and halteres and that of  $Pc^2$  as the number of sex comb teeth in Cbx/TM1, Me ri sbd<sup>1</sup>, Cbx/h  $Pc^2$ red, and h  $Pc^2$ red/TM1, Me ri sbd<sup>1</sup> flies. The estimates per fly rather than body side were preferred. 50 males were examined in each of the strains kept at 25°C. The data obtained are presented in the table.

Strain	area, mm <sup>2</sup>		$\bar{X} \pm s_{\bar{x}}$ no. sex comb teeth on tarsi of legs		
	halteres	wing	I	II	III
Cbx/TM1	1.28±0.11	19.59±4.08	22.44±1.42	-	-
P	<0.001	<0.001	<0.001		
Cbx/ $Pc^2$	1.70±0.14	4.74±2.10	24.70±1.70	5.52±3.46	1.74±2.06
P	<0.001	<0.001	<0.05	<0.001	<0.01
TM1/ $Pc^2$	1.46±0.14	76.83±5.45	23.32±1.73	1.52±2.35	0.44±1.00

Rather small but significant differences were found in haltere size, which could be well due to  $Pc^2$  induced enlargement of the halteres, the latter being better expressed in  $Pc^2$  than in Cbx strain. The wing size was also found to be substantially

strain-dependent: in Cbx over  $Pc^2$  strain a sharp reduction in wing size due to enhancement of wing to haltere transformation was observed.

The number of sex comb teeth in the first leg pair varied only moderately, being somewhat higher in  $Pc^2$  bearing strains. In the second and third leg pairs the sex combs developed only when  $Pc^2$  was present in the genotype and the number of teeth increased when Cbx was present.

Thus, Cbx and  $Pc$  exerted mutual enhancement of their homoeotic effects, which means that  $Pc$  locus is active not only in the leg discs but in the wing and haltere ones, also; similarly the Cbx is active not only in the wing disc, but in the leg ones as well. No second to third leg transformation could be traced in either Cbx/TM1 or Cbx/ $Pc^2$  strains.

Reference: Denell 1978, Genetics 90:277.

Mglinetz, V. A. Institute of Medical Genetics, Moscow, USSR. Temperature-sensitive period (TSP) of lethal action of split (spl) in *D. melanogaster*.

A number of temperature-sensitive alleles are known in a complex locus of Notch. TSP of some of the Notch alleles suggests the activity of the locus from embryonic to prepupal stages (Foster 1973). No temperature sensitivity was found in spl belonging to the same complex locus

(Shellenbarger and Mohler 1975; Portin 1977). However, spl stock from the Institute of Cytology and Genetics in Novosibirsk was found to be heat sensitive; when kept at 29°C the mutant larvae died in the first instar. TSP was estimated in samples of eggs laid within successive 6 h periods. 24 h heat treatment (29°C) was applied to fertilized females, embryos, and larvae of all three instars (taking the time of oviposition as the zero age). In each sample the eggs and the imago were counted. The results obtained are shown in Fig. 1 in which a distinct TSP corresponding to the last 30 h of oogenesis may be seen.



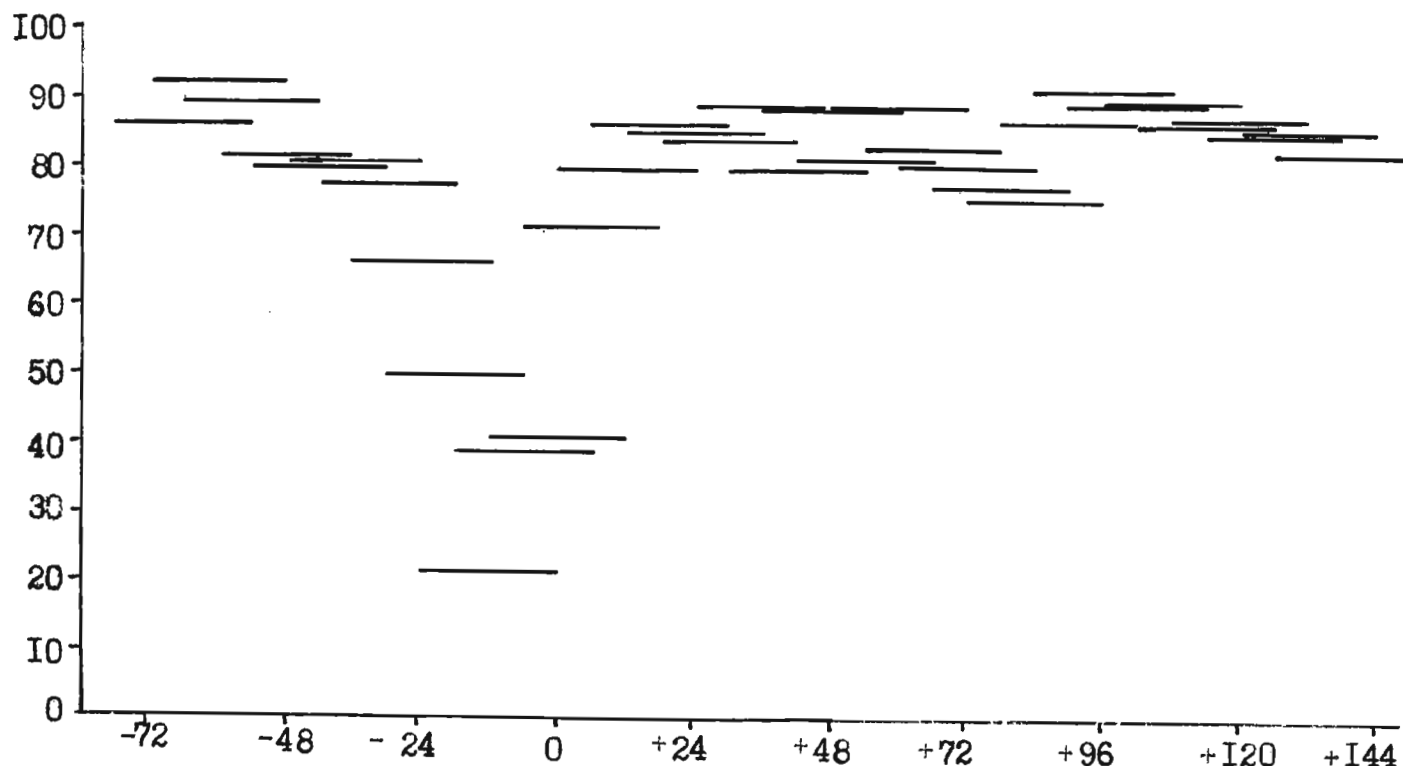


Fig. 1. Egg-to-imago survival of split mutants in *D. melanogaster* following 24 h heat treatment (29°C) in different periods of development. Percent of survivors, time and duration of heat treatment in each sample is shown by respective horizontal stripe.

References: Foster 1973, *Develop. Biol.* 32:282; Portin 1977, *Hereditas* 87:77; Shellenbarger and Mohler 1975, *Genetics* 81:143.

Michinomae, M., S. Shindo and S. Kaji.  
Konan University, Okamoto, Kobe, Japan.  
Patterns of acid phosphatase during the development in *D. melanogaster*.

acid phosphatase but no details are given. Mulherkar et al. (1972) and Michinomae (1976) pointed out that the acid phosphatase activities are changeable during the development of *D. melanogaster*. The maximum activity in whole larvae homogenates was observed in the prepupal to pupal stage. However, the maximum activity in the mutant Bar eye-antennal discs was detected in the larvae of 95 hours after hatching. Such a difference between whole larvae and eye-antennal discs may be resulted from the cell death accompanied by the specific degeneration of the mutant imaginal eye discs (Michinomae 1976).

Many investigations have been made on the acid phosphatase in various *Drosophila* species. MacIntyre (1966) reported that the adult flies of *D. melanogaster* and *D. simulans* contain the acid phosphatase-1, while Esposito and Ulrich (1966) also reported the developmental changes of the

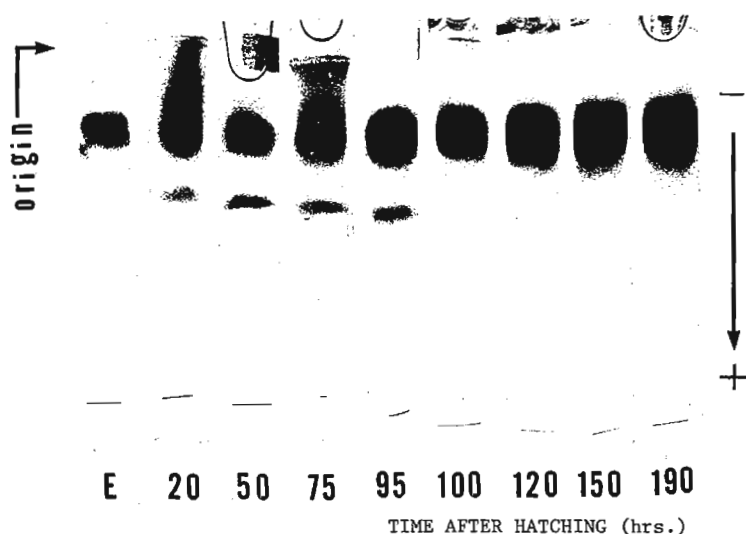


Fig. 1. Zymograms of the relative positions of acid phosphatase of nine developmental stages. E = egg stage, 20-95 = larval stages, 100 = prepupal stage, 120-190 = pupal stages.

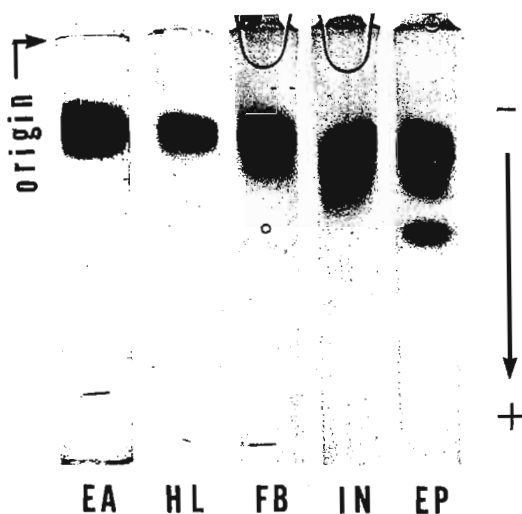


Fig. 2. Zymograms of acid phosphatase in five different larval tissues. IN = intestine, FB = fat body, HA = haemolymph, EA = eye-antennal disc, EP = epithelium.

The properties of acid phosphatase were examined, mainly from the viewpoints of the developmental changes and the cell death, in the wild type (Oregon-R) and Bar eye mutant of *D. melanogaster*, by the use of polyacrylamide disc electrophoresis. Disc electrophoresis was carried out according to the method described by MacIntyre (1971), with the use of 7% polyacrylamide gel and Canalco buffer system. The samples of electrophoresis were obtained by homogenizing of eggs, larvae and pupae, which were applied on each original point of the gel columns. Electrophoresis was conducted in 5 cm gels for 4 hours at 2.5 mA/column. The enzyme activity was detected with  $\alpha$ -naphthyl phosphate and Fast Red-ITR.

The types of acid phosphatase were investigated on nine stages during the development. In the wild type, two types of acid phosphatase were detected, and the slowly moving band (major band) has the highest activity, while the faster moving band (minor band) has lower activity than the highest one. The major band was detected throughout the egg, larval and pupal stages, but the minor band was not detected at the egg and pupal stages. Only at the larval stage could the minor band enzyme be detected (Fig. 1). Therefore, the localization of this enzyme was examined in each of the larval tissues or organs: epithelium, fat body, haemolymph, intestine, eye antennal disc. The major enzyme band was also detected in each of the tissues or organs, while only in the epithelium both the major and minor bands were so detected that the minor band enzyme may be represented in the epithelium of larvae (Fig. 2).

In the Bar eye mutant, these developmental changes of zymograms were as the same as the wild type. Two fundamental types of acid phosphatase were found in the various developmental stages. One enzyme was detected only in the epithelium of the larvae as a minor band. The other, major band enzyme was common to all stages whether it is larvae or not.

From these results, the changes show that the acid phosphatase activity in the Bar eye-antennal discs, with its characteristic degeneration by the cell death, may be due to one enzyme, detected as the major band enzyme.

References: Esposito, V.M. and V. Ulrich 1966, *Genetics* 54:334; MacIntyre, R.J. 1966, *Genetics* 53:461-474; MacIntyre, R.J. 1971, *Biochem. Genet.* 5:45-56; Michinomae, M. 1976, *Japan. J. Genetics* 51:315-326; Mulherkar, L., R.M. Kothari and V.G. Vaidya 1972, *Wilhelm Roux Archiv* 171:195-199.

Michutta, A., J. Krause, and W. Köhler.  
Institut für Genetik der Freien Universität,  
Berlin, Germany. Reproductive fitness in exchange lines of a selected photonegative strain and its control.

During selection, fertility and vitality of the selected strains commonly decrease. One explanation for this is that during artificial selection such genotypes are necessarily considered which under the effect of natural selection would be neglected because of their lower fitness. For instance, Pyle (1976) investigated the reproductive fitness of positive and negative phototactic and geotactic strains of *D.m.*, which he had selected in 15-unit classification mazes for over 40 generations. In the photopositive and photonegative strains he found a significant decrease in reproductive fitness (egg to adult survival). A possible explanation therefore is that genes which influence fitness are linked with those being directly selected.

This hypothesis we tried to investigate for a photonegative selected strain of *D.m.* The selection procedure took place in a 13-unit classification maze (Hadler, 1964). In generation 42 we started a synthesis of chromosome exchange lines out of the negative selected strain (NON) and its photopositive control (KOK), according to the design of Hirsch (1967). These exchange lines carried homozygous combinations of chromosomes from the negative strain (N) and its positive control (K), namely KKK, KNN, KKN, KNK, NNN, NKK, NNK, and NKN. Following a method of Nöthel (1967) the fecundities (eggs/female) of the synthesized lines and their two founder populations NON and KOK were investigated for 9 days (Table 1).

Table 1. Rates of fecundity and standard errors of the base populations NON and KOK and their exchange lines.

day line	1.	2.	3.	4.	5.	6.	7.	8.	9.
NKK	32.38 ±11.45	73.37 ± 8.98	86.56 ± 3.43	77.31 ± 1.31	73.87 ± 7.26	71.56 ± 2.97	74.81 ± 1.68	61.70 ± 1.74	60.27 ± 5.97
NNK	40.83 ± 6.44	64.38 ± 2.07	72.35 ± 3.21	70.18 ± 2.01	67.10 ± 5.31	70.08 ± 3.55	65.07 ± 5.11	55.65 ± 5.40	52.26 ± 5.00
NON	27.65 ± 2.11	51.19 ± 2.89	51.92 ± 7.09	49.50 ± 7.01	42.92 ±10.91	36.50 ± 7.30	36.76 ± 6.70	35.13 ± 9.44	32.94 ± 7.91
KNN	21.28 ± 7.28	50.22 ± 4.29	61.39 ± 5.24	63.91 ± 2.90	60.37 ± 4.41	49.02 ± 8.70	50.10 ± 1.89	51.13 ± 7.06	49.88 ± 9.54
KKN	21.85 ± 9.84	58.46 ± 7.58	65.89 ± 7.90	63.76 ± 4.92	62.07 ± 3.26	58.68 ± 3.89	58.43 ± 6.17	52.89 ± 6.05	50.87 ±11.93
KOK	31.70 ± 7.93	63.18 ± 3.71	68.23 ± 1.99	61.70 ± 4.01	53.65 ±11.08	50.36 ±12.81	45.68 ±10.94	41.18 ± 9.69	37.26 ±11.60
NKN	18.19 ± 5.45	65.61 ± 2.95	78.07 ± 5.83	67.77 ±10.17	71.23 ±12.65	72.23 ± 6.69	67.64 ± 8.13	67.63 ±12.04	57.15 ± 9.71
KNK	29.97 ± 4.54	53.59 ± 6.66	64.90 ± 8.59	64.66 ± 5.78	62.36 ± 8.22	50.79 ± 1.74	47.44 ± 5.30	53.60 ± 9.91	48.05 ± 7.59

There exist significant differences between the investigated lines ( $P < 0.01$ ) according to the Friedman two-way analysis of variance by ranks (Siegel, 1956). We ranked these data in order of increasing size:  $KNN < KNK < KKN < NNK < NKN < NKK$ . The first strain KNN is significantly different from the last three lines (NNK, NKN, NKK); or alternatively the line with the highest fecundity NKK differs significantly from KNN, KNK, and KKN (Wilcoxon-Wilcox test).

But there is no clear-cut evidence in the data for the influence of the chromosomes of different origin. An analysis of variance according to the method of Hirsch (1967) leads to inconsistent results. Following our hypothesis we expected that those lines with unselected control chromosomes should show the highest fecundity. This is not the case. Our results

show that there must exist interactions between all three chromosomes without considering their origin.

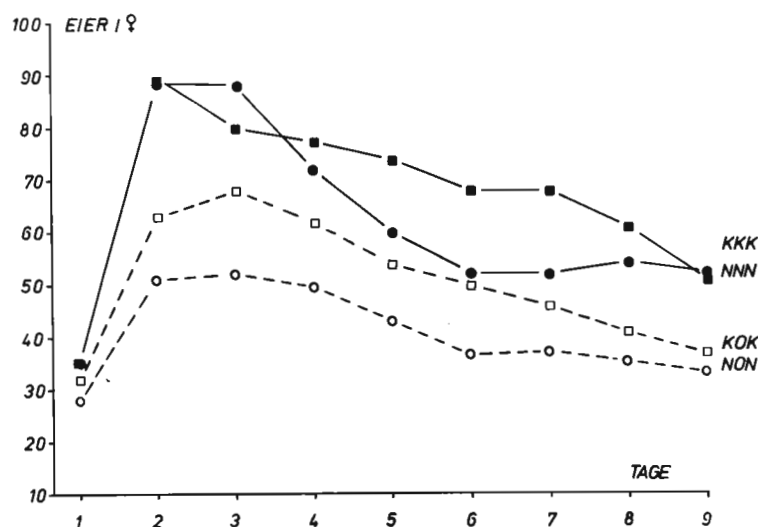


Figure 1. Fecundity (eggs/♀) of the base populations NON and KOK and the re-synthesized lines NNN and KKK until the ninth day.

populations NON and KOK with the strains NNN and KKK, which were again synthesized from the exchange lines. It is obvious (Fig. 1) that the fecundities of both lines (NNN, KKK) are higher than those of the base populations KOK and NON. This may be an effect of the exchange procedure which lasted 15 generations and must have brought a higher degree of heterozygosity.

References: Hadler, N.M. 1964, DIS 39:131; Hirsch, J. 1967, in Behavior-Genetic Analysis, McGraw Hill, New York; Köhler, W. 1977, Genetica 47:93-104; Nöthel, H. 1967, Strahlentherapie 134:609-624; Siegel, S. 1956, Nonparametric Statistics, McGraw Hill, Tokyo.

Nevertheless it is interesting that each line with an X-chromosome from the selected photonegative strain leads to a higher fecundity in comparison with all the other exchange lines. This may support the conclusion that genes which increase fitness are linked with those being directly selected because the effect of the X-chromosome for negative phototactic behavior in *D.m.* is obvious (e.g. Köhler, 1977).

Similar results were obtained from the egg to adult survival rates. In this case the rank order is KNN < KNK < NKK < NNK < KKN < NKN. The first and second lines show significant differences as compared with the last three lines ( $P < 0.01$ ).

Another interesting result came from the comparison of the fecundity of the popu-

Mikasa, K. and T. Narise. Josai Dental University, Sakado, Saitama, Japan. The relation between dispersive behavior and temperature. II. Sex difference.

The experiments were conducted to examine the sex difference in dispersive behavior of *D. melanogaster*. The strains used were two wild strains, MS-1 and Oregon-R, and six mutant strains (cn bw, e, ss, ss<sup>a</sup>, vg and w<sup>a</sup>) of melanogaster. In addition, six wild strains (HD,

AM, KZ, OD, AT and FO) newly collected in Japan were utilized. Fifty pairs of one to three day old flies of a strain were introduced in a Sakai's migration-tube (original tube) and kept for 24 hrs in it. Then, three new tubes having fresh food were connected with the tube

Mglinetz, V.A. Institute of Medical Genetics, USSR Academy of Medical Sciences, Moscow. Interaction of homoeotic mutations  $Pc^2$  and Cbx.

As  $Pc$  mutations affect not only the leg but also the wing development (Denell 1978) it seemed worthwhile to investigate whether or not such mutations show interaction with wing to haltere transforming mutation Cbx. The rate of Cbx induced homoeosis was estimated as the size of

wings and halteres and that of  $Pc^2$  as the number of sex comb teeth in Cbx/TM1, Me ri sbd<sup>1</sup>, Cbx/h  $Pc^2$ red, and h  $Pc^2$ red/TM1, Me ri sbd<sup>1</sup> flies. The estimates per fly rather than body side were preferred. 50 males were examined in each of the strains kept at 25°C. The data obtained are presented in the table.

Strain	area, mm <sup>2</sup>		$\bar{X} \pm s_{\bar{x}}$ no. sex comb teeth on tarsi of legs		
	halteres	wing	I	II	III
Cbx/TM1	1.28±0.11	19.59±4.08	22.44±1.42	-	-
P	<0.001	<0.001	<0.001		
Cbx/ $Pc^2$	1.70±0.14	4.74±2.10	24.70±1.70	5.52±3.46	1.74±2.06
P	<0.001	<0.001	<0.05	<0.001	<0.01
TM1/ $Pc^2$	1.46±0.14	76.83±5.45	23.32±1.73	1.52±2.35	0.44±1.00

Rather small but significant differences were found in haltere size, which could be well due to  $Pc^2$  induced enlargement of the halteres, the latter being better expressed in  $Pc^2$  than in Cbx strain. The wing size was also found to be substantially

strain-dependent: in Cbx over  $Pc^2$  strain a sharp reduction in wing size due to enhancement of wing to haltere transformation was observed.

The number of sex comb teeth in the first leg pair varied only moderately, being somewhat higher in  $Pc^2$  bearing strains. In the second and third leg pairs the sex combs developed only when  $Pc^2$  was present in the genotype and the number of teeth increased when Cbx was present.

Thus, Cbx and  $Pc$  exerted mutual enhancement of their homoeotic effects, which means that  $Pc$  locus is active not only in the leg discs but in the wing and haltere ones, also; similarly the Cbx is active not only in the wing disc, but in the leg ones as well. No second to third leg transformation could be traced in either Cbx/TM1 or Cbx/ $Pc^2$  strains.

Reference: Denell 1978, Genetics 90:277.

Mglinetz, V. A. Institute of Medical Genetics, Moscow, USSR. Temperature-sensitive period (TSP) of lethal action of split (spl) in *D. melanogaster*.

A number of temperature-sensitive alleles are known in a complex locus of Notch. TSP of some of the Notch alleles suggests the activity of the locus from embryonic to prepupal stages (Foster 1973). No temperature sensitivity was found in spl belonging to the same complex locus

(Shellenbarger and Mohler 1975; Portin 1977). However, spl stock from the Institute of Cytology and Genetics in Novosibirsk was found to be heat sensitive; when kept at 29°C the mutant larvae died in the first instar. TSP was estimated in samples of eggs laid within successive 6 h periods. 24 h heat treatment (29°C) was applied to fertilized females, embryos, and larvae of all three instars (taking the time of oviposition as the zero age). In each sample the eggs and the imago were counted. The results obtained are shown in Fig. 1 in which a distinct TSP corresponding to the last 30 h of oogenesis may be seen.

Table 1. Dispersive activities of both sexes at 15, 20, 25 and 30°C in laboratory and wild strains of *D. melanogaster*.

Strain	Temp.	15	20	25	30°C
vg	♀	2.4	4.2	2.2	7.4
	♂	5.6	13.0	9.4	11.4
ssa	♀	3.4	1.8	1.6	10.6
	♂	6.0	21.0	16.2	15.8
cn bw	♀	1.8	4.2	16.4	22.2
	♂	1.8	15.0	29.6	32.8
e	♀	2.0	3.8	2.8	13.4
	♂	0.4	1.6	5.8	9.0
ss	♀	1.6	2.2	3.2	17.0
	♂	3.2	3.8	8.8	17.4
wa	♀	4.0	2.8	4.4	10.2
	♂	2.0	4.0	9.0	17.6
MS-1	♀	0.0	1.6	4.4	1.8
	♂	1.8	11.0	21.8	8.6
Oregon-R	♀	0.8	5.2	9.8	20.8
	♂	2.8	6.6	25.0	16.4
HD	♀	14.2	21.8	23.6	45.0
	♂	17.8	34.2	28.0	45.2
AM	♀	7.2	36.4	28.2	31.4
	♂	12.4	26.0	25.6	32.4
KZ	♀	12.8	33.4	37.6	30.4
	♂	15.8	27.6	31.8	33.8
OD	♀	9.2	35.0	35.0	32.8
	♂	13.0	27.0	32.8	37.2
AT	♀	20.4	31.2	37.8	32.6
	♂	23.2	29.2	37.4	35.6
FO	♀	6.0	10.8	11.6	36.6
	♂	7.4	36.6	30.0	36.6

more often than at unfavorable temperature. Five strains out of eight in laboratory strains belong to the second type (vg, ssa, cn bw, e, and wa), while female flies tend to disperse more frequently with increasing temperature in three other strains.

Strain	Temp.	15	20	25	30°C
vg		.29 ± .18	.24 ± .14	.16 ± .17	.37 ± .17
ssa		.33 ± .30	.08 ± .10	.07 ± .11	.34 ± .23
cn bw		.46 ± .50	.20 ± .13	.32 ± .15	.39 ± .16
e		.88 ± .21	.75 ± .40	.26 ± .25	.60 ± .13
ss		.36 ± .39	.46 ± .34	.44 ± .32	.50 ± .22
wa		.66 ± .41	.40 ± .39	.30 ± .23	.46 ± .27
MS-1		.00 ± .	.10 ± .12	.16 ± .09	.18 ± .21
Oregon-R		.25 ± .27	.53 ± .27	.29 ± .21	.56 ± .11
HD		.43 ± .15	.39 ± .14	.44 ± .10	.51 ± .08
AM		.36 ± .21	.59 ± .07	.52 ± .07	.50 ± .10
KZ		.45 ± .17	.55 ± .08	.55 ± .10	.48 ± .08
OD		.37 ± .23	.56 ± .09	.51 ± .05	.47 ± .07
AT		.45 ± .15	.52 ± .06	.50 ± .06	.47 ± .10
FO		.31 ± .30	.25 ± .13	.26 ± .11	.48 ± .15

and dispersal was allowed for six hours. After that period of time, dispersed male and female flies in the three connected tubes were counted separately. Four kinds of temperature, 15, 20, 25 and 30°C, were used, and the experiments were carried out at each temperature in a dark room. Ten replications were made, and the dispersive activity was calculated in percent of dispersed flies to the number of flies introduced into the original migration tube in each sex.

Table 1 shows the dispersive activities of both sexes for each temperature in each population. From Table 1, it can be said that in general, the activities of both sexes increased with increasing temperature, and the activity is quite different among strains. Table 2 presents the female ratio (number of dispersed female flies/total dispersed flies) for each temperature in each population. As seen in Table 2, male flies move more frequently at each temperature in eight laboratory strains with few exceptions. However, in newly collected wild strains it was observed that female flies dispersed more often than males at optimum temperature for *melanogaster*, 20° and 25°C, in AM, KZ, OD and AT strains, while at unfavorable temperatures like 15° and 30°C female flies move frequently in two other strains. Furthermore, there was a tendency for female flies to move more often at 30°C than at other temperatures in each strain. The correlation between activities of both sexes at each temperature was also studied, and no correlation was detected.

From this experiment, it was found that the response of dispersive activity to temperature was quite different between sexes as well as strains. It was also detected that in wild strains there were two types of response to temperature. One of them is that female flies disperse more frequently at optimum temperature as seen in AM, KZ, OD and AT strains, and the second is that at optimum temperature male flies move

Table 2. The female ratio at 15, 20, 25 and 30°C in laboratory and wild strains of *D. melanogaster*. (Mean and standard deviation)

Milkman, R. University of Iowa, Iowa City, Iowa. Thermostability variation in D. m. allozymes.

Electrophoretic mobility and thermostability were examined in sets of individual flies from wild (Cedar Rapids, IA; Berkeley, CA) and laboratory strains. Mobility studies utilized electrophoresis for 30 min at 400V with 1/4-strength

Gelman HR buffer, or 50 min at 210V with 1/2-strength Gelman HR buffer (or occasionally 100 min when the loaded cellulose acetate strips are coated with oil to reduce evaporation). Thermostability tests involved 10 min electrophoresis at 200 or 400V, after which the strips were enclosed in a cellophane bag and immersed in a water bath for 20-120 sec, then removed and stained. Mobility class frequencies are listed in Table 1, with those of Band (1975) below for comparison. (Extensive ADH and  $\alpha$ -GPDH data are reported by Sampsell 1977.) 88-110 strains were tested for each enzyme.

Table 1

Enzyme	Slow	Commonest	Fast	Faster	Test Temp.
ACPH	0.02	0.98			47°C
	0.05	0.95			
AK		1.00			54
		-			
AO		0.90	0.09	0.01	70
		0.88	0.12		
ME		1.00			57
		0.99	0.01		
IDH		0.97	0.03		53
	0.03	0.94	0.01	0.02	
PGM	0.17	0.78	0.05		65
	0.03	0.95	0.01		
XDH		1.00			72
	0.02	0.98			
Est-6		0.74	0.26*		59
		0.57	0.41 [sic]		
MDH		0.95**	0.05		60
		1.00			

\*Thermostability classes: 0.18, 0.03, 0.04.

\*\*0.94, 0.01. Order of increasing sensitivity. No thermostability variation was detected in the other enzymes.

alter bond energies by far smaller values, thermostability analysis is not a sensitive detector of amino acid substitutions. In any event, the increased genetic variability indicated by the present tests is negligible. On the other hand, the asymmetry evident so far in thermostability variation (most variants are more sensitive than the commonest class) should be noted as potential evidence for the Ohta and Kimura (1975) model of very slightly deleterious mutations. Supported by NSF Grant DEB 76-01903.

References: Band, H.T. 1975, Genetics 80:761-771; Cochrane, B. 1976, Nature 263:131-132; Ohta, T. and M. Kimura 1975, Am. Nat. 109:137-145; Sampsell, B. 1977, Biochem. Genet. 14: 971-988; Trippa, G., A. Loverre and A. Catamo 1976, Nature 260:42-44.

The Est-6 classes likely correspond to Cochrane's (1976) 1.10 classes. Trippa et al. (1976) clearly demonstrated variants in PGM. In MDH, one sensitive slow strain was found. The thermostability difference was mapped to the Mdh locus (or very close) as follows. Progeny of an S<sup>S</sup> (slow sensitive) X F<sup>F</sup> cross were propagated for 10 generations, after which 60 heterozygotes were examined. In all cases, the slow band was sensitive and the fast band was not (heterodimer appeared intermediate). In a similar test of S<sup>F</sup> X F<sup>F</sup> 10th generation heterozygote progeny, both homodimer bands invariably evinced equal thermostability. These tests were made after 60 min at 200V.

The observed stability differences were in the range of four-fold differences in the treatment durations required for criterion response. These differences corresponded to differences in  $\Delta F^\ddagger$  on the order of 800 cal/mol, which is in the range of one van der Waals bond energy. While somewhat subtler differences could have been detected, none were. Since bond substitutions, as well as conformational changes, could

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Drosophila fauna of Dandeli and Ambikanagar.

a rich abode for insect fauna. Hence, the present investigation of Drosophila fauna was made in Dandeli and Ambikanagar during the first week of October, 1978 when Drosophila flies are usually found in abundance. Collections were made both by sweeping and banana trap methods, and the collected data are presented in Table 1.

Table 1. Distribution of different species of Drosophila at Dandeli and Ambikanagar.

Species	Dandeli	Ambikanagar
1. <i>D. malerkotliana</i>	208	198
2. <i>D. bipectinata</i>	112	91
3. <i>D. nasuta</i>	71	64
4. <i>D. anomelani</i>	82	51
5. <i>D. jambulina</i>	36	18
6. <i>D. mysorensis</i>	29	9
7. <i>D. neonasuta</i>	45	22
8. <i>D. eugracilis</i>	4	1
9. <i>D. punjabiensis</i>	29	10
10. <i>D. varietas</i>	3	1
11. <i>D. busckii</i>	-	1
12. <i>D. elegans</i>	-	6
13. <i>D. sahyadrii</i>	18	6
14. <i>D. meijere indicus</i>	8	-
15. <i>D. rajasekari</i>	-	11
16. <i>D. nigra</i>	8	3
Total	653	492

ments and valuable suggestions. The financial aid by U.G.C. is acknowledged.

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

Though some areas of India have been explored in regard to Drosophila fauna and their ecology, several parts of this country need to be extensively surveyed. North-Kanara is one such virgin area where congenial ecological conditions associated with a variety of flora provide

A total of 1145 individuals comprised of 16 species of Drosophila have been collected. Most of the species belong to Sophophora and Drosophila. Among the species trapped, *D. malerkotliana*, *D. bipectinata*, *D. nasuta*, *D. anomelani* and *D. neonasuta* were found in abundance in order of decreasing density. The rare species collected include *D. nigra*, *D. meijere indicus*, *D. busckii*, *D. rajasekari*, *D. eugracilis* and *D. sahyadrii*. It is interesting to note that *D. elegans*, a member of elegans subgroup of melanogaster species group, was collected on flowers of Ipomea species. *D. anomelani*, *D. punjabiensis*, *D. jambulina* and *D. mysorensis* of montium subgroup are the other members captured. Collection records indicate that most of the species captured belong to either melanogaster or immigrans species group and this supports the view of Bock and Wheeler (1972) that both these species groups are always in abundance in southeast Asia.

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Effect of crowding and temperature on the rate of development in *D. nasuta*.

groups (A, B, C and D). Group A consisted of 40 vials each with 25 eggs, group B of 20 vials each with 50 eggs, group C of 10 vials each with 100 eggs and group D of 5 vials each with 200 eggs. Two sets of each group were employed. One set of four groups was permitted to develop at constant temperature of  $21 \pm 1^\circ\text{C}$  and another set of four groups at fluctuating temperatures of  $24^\circ\text{C}$  to  $29^\circ\text{C}$ . The flies were counted from the first day of eclosion to the last day. The patterns of emergence at constant and fluctuating temperatures calculated from each group are shown in Figs. 1 and 2, respectively. The mean developmental times at both constant and fluctuating temperatures are presented in Table 1.

In order to investigate the effect of crowding and temperature on the rate of development in a polymorphic strain of *D. nasuta* from Mavinagundi (North Kanara, India), eggs of the same age were collected following the procedure of Delcours (1969) and were distributed into four



Table 1. Mean developmental time of *D. nasuta* at constant and fluctuating temperatures

Group	Mean developmental times at	
	Constant temperature	Fluctuating temperature
A	$9.90 \pm 0.04$	$8.32 \pm 0.04$
B	$10.97 \pm 0.012$	$9.74 \pm 0.109$
C	$13.48 \pm 0.141$	$12.16 \pm 0.374$
D	$17.17 \pm 0.264$	$15.16 \pm 0.192$

menced on day 9 with a peak on day 10 and terminated on day 13, in contrast to the highest density (200 eggs) where the eclosion commenced on day 13, with a peak on day 16 and terminated on day 23. At fluctuating temperatures in the lowest density (25 eggs) the eclosion commenced on day 7 with a peak on day 8 and terminated on day 15, whereas in the highest density (200 eggs) the eclosion commenced on day 12 with a peak on day 15 and terminated on day 23. Thus, from the above mentioned data it is clear that the mean developmental time increases with the increase of density, and increase in temperature decreases the mean developmental time. Increase in mean developmental time is due to crowding. Hence it is concluded here that there is an effect of crowding and temperature on the rate of development in *D. nasuta*.

At constant temperature the mean developmental times in groups A, B, C and D are  $9.90 \pm 0.04$ ,  $10.97 \pm 0.012$ ,  $13.48 \pm 0.14$  and  $17.17 \pm 0.264$ , respectively, while at fluctuating temperatures the mean developmental times in groups A, B, C and D are  $8.32 \pm 0.04$ ,  $9.74 \pm 0.109$ ,  $12.16 \pm 0.374$  and  $15.16 \pm 0.192$ , respectively. At constant temperature in the lowest density (25 eggs) the eclosion com-

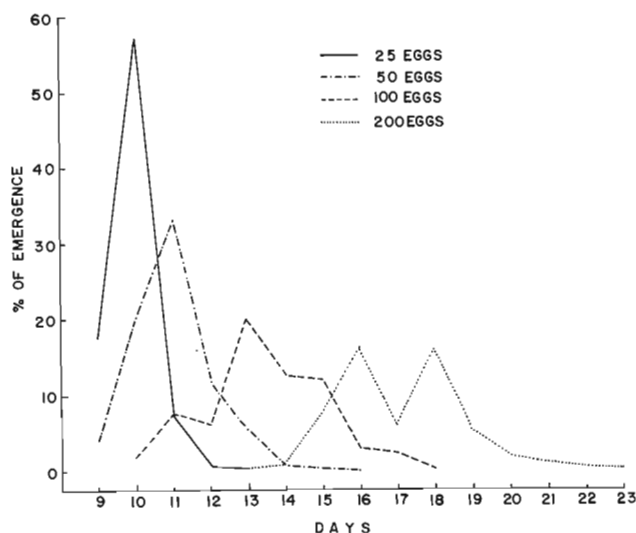


Fig. 1 Pattern of emergence of flies in different densities at constant temperature in *D. nasuta*

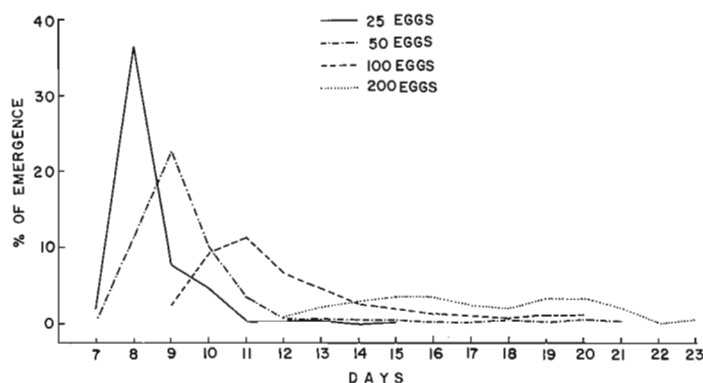


Fig. 2 Pattern of emergence of flies in different densities at fluctuating temperatures in *D. nasuta*

The authors are grateful to Dr. M.R. Rajasekarasetty for his constant encouragement and valuable suggestions. The financial aid by U.G.C. is acknowledged.

Reference: Delcour, J. 1969, DIS 44:133-134.

Narise, S. and M. Sasaki. Josai University, Saitama, Japan. Thermal stability difference among  $\alpha$ Gpdh allozymes from *D. virilis*.

patterns of the  $\alpha$ Gpdh from the three strains and their hybrids are shown in Fig. 1.

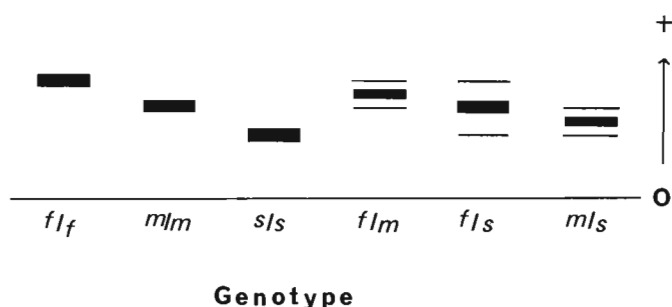


Fig. 1. Electrophoretic patterns of cytoplasmic  $\alpha$ Gpdh allozymes from homogenates of *D. virilis*.

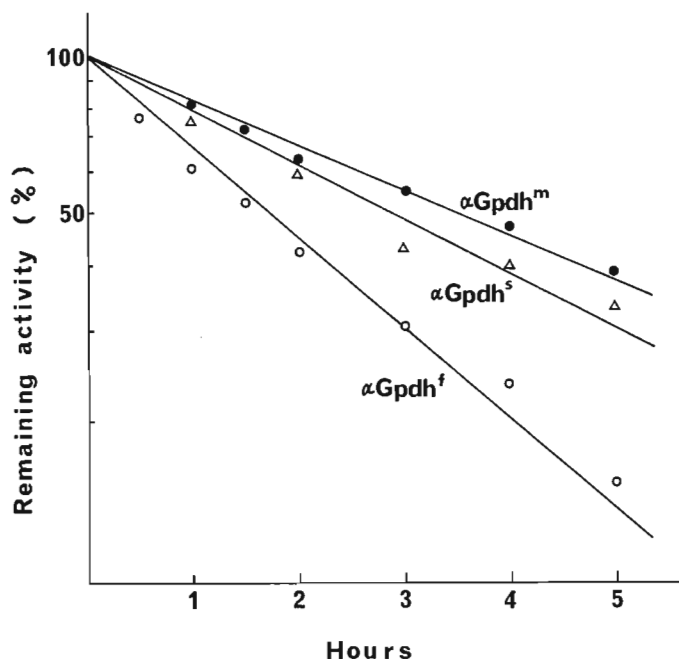


Fig. 2. Thermal inactivation of  $\alpha$ Gpdh allozymes at 35°C.

$\alpha$ Gpdh<sup>f</sup> lost all activity in 2 min incubation, whereas the  $\alpha$ Gpdh<sup>m</sup> maintained about 60% of the original activity and the  $\alpha$ Gpdh<sup>s</sup> about 40%.

These results indicate that  $\alpha$ Gpdh<sup>f</sup> allele at the  $\alpha$ Gpdh locus specifies a thermolabile form of  $\alpha$ Gpdh protein.

Thermal inactivation was compared among three  $\alpha$ Gpdh allozymes. Three homozygous strains ( $\alpha$ Gpdh<sup>f</sup>/ $\alpha$ Gpdh<sup>f</sup>,  $\alpha$ Gpdh<sup>m</sup>/ $\alpha$ Gpdh<sup>m</sup> and  $\alpha$ Gpdh<sup>s</sup>/ $\alpha$ Gpdh<sup>s</sup>) for cytoplasmic  $\alpha$ Gpdh were isolated from single females collected from a natural population in Omaezaki, Japan. Starch gel electrophoretic

The enzyme activity of  $\alpha$ Gpdh<sup>f</sup> homozygote on the gel did not develop, when the gel was incubated at 55°C for one hour after electrophoresis, while the activities of  $\alpha$ Gpdh<sup>m</sup> and  $\alpha$ Gpdh<sup>s</sup> homozygotes were slightly reduced. In both  $\alpha$ Gpdh<sup>f</sup>/ $\alpha$ Gpdh<sup>m</sup> and  $\alpha$ Gpdh<sup>f</sup>/ $\alpha$ Gpdh<sup>s</sup> heterozygotes, fast moving bands disappeared and intermediate hybrid bands appeared more faintly than did their corresponding slow moving bands, after the same treatment. 92 isofemale lines from natural populations in Omaezaki, Nagoya and Toyama were tested for the thermal stability at 55°C and the same result was obtained, i.e.,  $\alpha$ Gpdh<sup>f</sup> allozymes from every population were thermolabile, whereas  $\alpha$ Gpdh<sup>m</sup> and  $\alpha$ Gpdh<sup>s</sup> allozymes were thermostable, and no heterozygote advantage was observed with respect to thermostability of  $\alpha$ Gpdh.

Three allozymes were extracted from their homozygous strains and purified 130-160 fold by fractionation with ammonium sulfate, DEAE cellulose, Sephadex and hydroxylapatite. When each purified enzyme was examined by polyacrylamide-gel disc electrophoresis, a single band stained for protein was coincided with the  $\alpha$ Gpdh activity.

Samples of purified enzyme preparations in 0.05 M Tris acetate buffer pH 7.0 were incubated at 35°C and were assayed for activity immediately after various periods of time as indicated in Fig. 2, with the standard assay mixture (0.1 mM DHAP, 0.1 mM NADH, enzyme and 0.1 M Tris acetate buffer pH 6.75 in a total volume of 3.0 ml). Fig. 2 shows a comparison of the thermal stability at 35°C of three  $\alpha$ Gpdh allozyme activities. As seen in Fig. 2, the activity of  $\alpha$ Gpdh<sup>f</sup> decreases markedly as compared with the other two. With 45°C treatment, the  $\alpha$ Gpdh<sup>m</sup> maintained about 60% of the

Nissani, M. University of California, Irvine. Brain transplantation and behavior.

Whole brains of mature third instar Oregon-R male larvae were dissected and their connections to all associated structures, including the ventral cord, severed. The brains were implanted in mature female larvae near their brain (12 brain-injected females eclosed), or near one of their ovaries (17 eclosed). In addition, 2 surviving females were each injected with 2 whole male brains. All females (including 19 Ringer injected controls) were kept in isolation after eclosion except for 4 one-hour observation periods on the 7th, 15th, 22nd and 30th days after eclosion. At these observation periods 6-8 yellow virgin females (2-6 days old) were placed in a food vial with either a single Oregon-R brain-injected female or a control Ringer-injected female. During an observation period, the flies in each vial were confined to a space of approximately 10 cm<sup>3</sup>. No clear differences in behavioral responses to the presence of females were observed between the experimental and control groups.

I dissected eight 30-day old females which were injected at the larval stage with a whole male brain in the vicinity of one of their ovaries and recovered 5 implants. Thick and long tracheal branches originated from all 5 implants. Also, in all 5 cases a pair of the host's abdominal nerves formed connections with the anteriorly located end of the implant and 4-6 terminal abdominal nerves arose from the implant's posterior end and formed attachments to internal posterior organs of the host.

During the course of these observations, 3 brain-injected and 2 Ringer-injected females (which were at least 20 days old) showed a peculiar behavioral pattern. When chanced to be very close on the food medium to a recently introduced yellow female, they occasionally tapped and followed the decamping female. Although this behavior never lasted more than a few seconds, there is no doubt that it was directed towards other females, because both followed and following females traversed the same, and usually very wiggly, path.

Ogaki, M., H. Nabata, E. Nakashima-Tanaka and S. Gamo. University of Osaka Prefecture, Japan. Ether sensitivity at embryonic stage of *D. melanogaster*.

LT<sub>50</sub> (50% lethal time) of the Eth strain in 24-hour-old adult flies was 5.4 minutes in females and 5 minutes in males; that of the bw;st;sv<sup>n</sup> strain was 2.6 minutes in females and 2 minutes in males. However, 50% non-hatchability time of the Eth strain in 3-hour-old eggs was 10 min-

utes; that of the bw;st;sv<sup>n</sup> strain was 22 minutes. Thus, the Eth strain is resistant to ether at the adult stage, but is sensitive to ether at the egg stage. Reciprocal crosses between the Eth and the bw;st;sv<sup>n</sup> strains showed that maternal effect existed at the egg stage, although maternal or cytoplasmic effects were negligible at the adult stage.

In order to investigate whether chorion had an effect on the strain differences in ether sensitivity, dechorionated 3-hour-old eggs of Eth and bw;st;sv<sup>n</sup> strains were tested for their sensitivity to ether. The 50% non-hatchability time of the Eth strain was 6.5 minutes and that of the bw;st;sv<sup>n</sup> strain was 10.5 minutes. The dechorionated eggs of the Eth strain were more sensitive than those of the bw;st;sv<sup>n</sup> strain. In the dechorionated 3-hour-old eggs of F<sub>1</sub> hybrids of Eth ♀ x bw;st;sv<sup>n</sup> ♂ and bw;st;sv<sup>n</sup> ♀ x Eth ♂, the 50% non-hatchability times were 6.5 minutes and 10.5 minutes, respectively. The results showed that the strain differences in sensitivity to ether were mainly due to the inherent character of embryo rather than chorion, and that maternal effect also existed in the embryonic stage.

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"Masculinizer" is an allele of "doublesex".

male characteristics, very similar to *dsx* (ref. 2) and *dsx<sup>D</sup>* (ref. 3; originally described as *Hr*, ref. 4).

Short description of the mutant - Body size and segmentation of *XX;Mas/+* are female; there are 7 sternites with bristles, and 8 tergites; tergites 5 to 8 show dark (male) pigmentation. The sex combs are rotated, but not completely, and their bristles are heavy, but not

quite typical of males. The derivatives of the genital disc contain an almost complete set of male internal and external genitalia which are, however, abnormal; the female genitalia are very reduced with only the spermathecae being regularly present; the anal plates occupy a lateral position typical of males and have a sexually intermediate bristle pattern. The gonads vary widely; they range from almost normal ovaries with mature eggs (very rare) to rudimentary ovaries, and sometimes no gonads are found; testis-like vesicles with yellow pigment, attached to yellow-colored vasa deferentia are frequent. The main difference between *Mas* and *dsx* or *dsx<sup>D</sup>* concerns the vaginal plates: these are reduced in *dsx* and *dsx<sup>D</sup>* bearing a few bristles whereas they are completely absent in *Mas*; however, an amorphous chitinous mass is always found in the position of the vaginal plates. In summary, *Mas* appears to be a slightly stronger masculinizer than *dsx<sup>D</sup>*.

Genetic tests - The mutant *Hr* has recently been identified as an allele of *dsx* and is now called *dsx<sup>D</sup>* (ref. 3). We have produced three genotypes which provide evidence that *Mas* is another dominant allele of *dsx*:

i) *XX;Mas/dsx* is a phenotypically normal, but sterile male (Fig. 1). The combination *Mas/dsx* acts like *dsx<sup>D</sup>/dsx* (ref. 3) or *tra/tra* (ref. 5).

ii) *XX/Y.dsx<sup>+</sup>;Mas/+* is a phenotypically normal, but sterile female, as is *XX/Y.dsx<sup>+</sup>;dsx<sup>D</sup>/+*. This result and the fact that a deficiency for *dsx* is completely recessive define *Mas* and *dsx<sup>D</sup>* as antimorphs. (The *Y.dsx<sup>+</sup>* was kindly provided by E.B. Lewis, Pasadena, and is described as *T(3;Y)P92* in ref. 3).

iii) *XX;Mas/dsx<sup>D</sup>Sb e* is a phenotypically normal, but sterile male. This combination was constructed by transplanting pole cells of *XX;dsx<sup>D</sup>Sb e/Ki pp* embryos into *XX, fs(1)K10* female embryos (see ref. 6 for description of *fs(1)K10*). The adult hosts were then crossed to *XY;Mas/TM1* males. The mutation *dsx<sup>D</sup>* has apparently no effect on the germ line so that normal *X;dsx<sup>D</sup>Sb e* eggs are produced which may then be fertilized with *X;Mas* sperm.

The phenotypes produced in combinations with *dsx*, *dsx<sup>D</sup>* and *Y.dsx<sup>+</sup>* reveal that *Mas* is another dominant allele of *dsx* and should therefore be renamed *dsx<sup>Mas</sup>*.

Supported by grant 3.741.76 of the Swiss National Science Foundation and the Julius Klaus-Stiftung Zürich.

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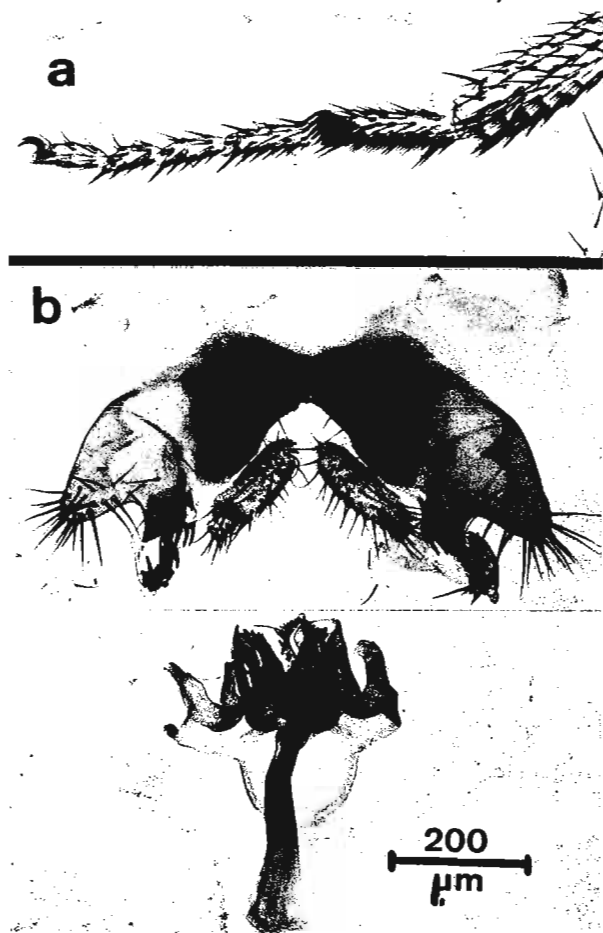


Fig. 1. Tarsus of foreleg with sex comb (a), and external derivatives of the genital disc (b) of *XX;Mas/dsx*. All structures are perfectly male.

Ohnishi, S. and R.A. Voelker. National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina. Genetic mapping of hexokinase-C in *D. simulans*.

In order to map hexokinase-C locus (Hex-C) genetically, a wild type stock with a variant allele (Hex-C<sup>6</sup>) was crossed to a multiple marker stock for the second chromosome [net (net, 0) b (black, 43) py (polychaete, 74) sd (spread, 80) pm (plum, 103)], which carries the common allele (Hex-C<sup>4</sup>).

F<sub>1</sub> females obtained from the cross were backcrossed to males of the marker stock. These offspring were electrophoretically analyzed after scoring for visible markers. The data are summarized in Table 1.

Table 1. The summary for genetic mapping of Hex-C in *D. simulans*.

Visible Phenotype	Hex-C <sup>4</sup> /Hex-C <sup>6</sup>	Hex-C <sup>4</sup> /Hex-C <sup>4</sup>
sd pm	3*	31
+ +	40	0
sd +	3	14
+ pm	26	13

\*These are double crossovers between sd and pm.

Hex-C was located between sd(80) and pm(103) at 86.6 (= 80 + 23 x 16/56) with the 95% binomial confidence interval extending from 83.7 to 89.2 on the second chromosome. The location corresponds to that in *D. melanogaster* (Jelnes 1971, 2-73.5; Mukai and Voelker 1977, 2-74.5).

References: Jelnes, J.E. 1971, *Hereditas* 67:291-293; Mukai, T. and R. A. Voelker 1977, *Genetics* 86:175-185.

O'Tousa, J. and P. Szauter. University of Washington, Seattle, Washington. The initial characterization of non-claret disjunctional (ncd): evidence that ca<sup>nd</sup> is the double mutant, ca ncd.

Females homozygous for ca<sup>nd</sup> show two distinct phenotypes: an abnormal eye color and aberrant meiotic chromosome behavior. Several lines of evidence have suggested that this compound phenotype is actually the result of ca<sup>nd</sup> being defective at two closely linked loci, one governing eye pigment metabolism and the other meiotic

chromosome behavior (see Baker and Hall 1976 for a review on the literature of ca<sup>nd</sup>). We report here on the recovery of a new meiotic mutant, non-claret disjunctional (ncd) that provides strong evidence that ca<sup>nd</sup> is indeed a double mutant.

ncd was isolated in a search for meiotic mutants among EMS-treated 3rd chromosomes in the laboratory of D.L. Lindsley at U.C. San Diego and analyzed by us in Seattle. Females homozygous for this mutant exhibit meiotic abnormalities similar to those of ca<sup>nd</sup> / ca<sup>nd</sup> females, and the mutant fails to complement ca<sup>nd</sup> (Table 1). Furthermore, the ncd mutation has been mapped to the distal region of 3R where ca<sup>nd</sup> is located. These results confirm that ca<sup>nd</sup> and ncd are allelic; however, both ncd/ncd homozygotes and ncd/ca<sup>nd</sup> compound heterozygotes have wild-type pigment. Therefore, ncd must be defective only at the ca<sup>nd</sup> locus governing chromosome behavior. The recovery of ncd, then, is evidence that ca<sup>nd</sup> is a double mutant with the implied constitution: ca ncd.

Table 1. The results of the crosses of the indicated females by  $\widehat{XY}$ , vFB;  $\widehat{44}$ , ci ey males.

maternal genotypes:	progeny classes									exceptions per 10 <sup>3</sup> ova				
	y♂♂ & B/+♀♀				vfB♂♂				y♀♀					
	+	ci	ey	pol	+	ci	ey	pol	+	ci	ey	pol	X	4
y;ncd;pol y;ncd;pol	45	39	1		1	25	0		0	1	2		405	650
y;ca <sup>nd</sup> ;pol y;ncd;pol	13	35	0		7	20	0		3	1	0		564	700
y;ncd;pol y;+;pol	137	0	0		0	0	0		0	0	0		0	0

References: Baker, B.S. and J.C. Hall 1976, in: *Genetics and Biology of Drosophila*, vol. I (ed. by E. Novitski and M. Ashburner), Academic Press, NY.

Paik, Y.K. and K.C. Sung. Hanyang University, Seoul, Korea. Chromosome inversions in Korean populations of *D. melanogaster*.

The natural populations of *D. melanogaster* were sampled at 17 different localities in Korea from 1977 to 1978. The results to be reported below include a portion of the present study, which was done in 1977. Fifty-eight different inver-

sions were detected from nine local populations on examination of 1875 wild-caught females; four inversions were found in Chromosome X; ten in Chromosome 2R and 17 in the 2L; 15 in Chromosome 3R and 12 in the 3L. Of these inversions, six were common cosmopolitan types, four were semicosmopolitan, seven were new and common endemic, and 41 were new and rare endemic. Of this last group there were five overlapping inversions occurring in Chromosome 3. In a population sampled four times, the frequency changes of some of the cosmopolitan inversions followed a seasonal trend; some of the common endemics remained stable in frequency from month to month. Coefficients of similarity obtained based on the types and frequencies of inversions found appear to illustrate distinctiveness of each population rather than similarity between populations tested. In the following list the approximate breakpoints of the present series of inversions are given in terms of Bridges' salivary maps.

Chromosome	Break points	Chromosome	Break points	Chromosome	Break points
X	1D; 3F	2L	31F; 36F	3L	66D; 73B
	8C; 18B	(cont'd)	37A; 40A	(cont'd)	69C; 77C
	10B; 12B		37E; 39E		71E; 75E
	13F; 16E	2R	42A; 60A		72F; 78B
2L	22A; 26B		42D; 60F	3R	83C; 85B
	22A; 33B		42E; 43A		86D; 88E/F**
	22B; 25C		43B; 46E		86F; 96A
	22D/E; 34A*		47C; 54D		87B; 92F
	23B; 25E/F		47E; 55E		87F; 90F
	23E; 33E		51F; 60D		88C; 98F
	24A; 31F		52A; 56F*		88C/D; 93C
	25B; 28C		54B; 59C		88D; 90F
	26A; 31A		56D; 59B		88D; 94A
	26A; 34E	3L	61F; 67E		89C; 96A*
	37A; 40A		62A; 63C		91C; 93B
	30A; 34A**		63C; 72E*		92D/E; 100F*
	31B/C; 34E/F**		65E; 67D		93D; 98F*
	31F; 35D		66D; 71D**		

\* denotes cosmopolitan types; \*\* denotes semicosmopolitans.

Note: Three overlapping inversions on the 3L and two overlappings on the 3R are not included in the list.

Pinsker, W. University of Tübingen, Germany. Relation between effective population size and allozyme polymorphism in *D. subsilvestris* and *D. subobscura*.

Wild flies of the *Drosophila obscura*-group were collected in a forest near Tübingen (West Germany) during September 1975, 1976 and 1978. Using the malt bait method recommended by Prof. Lakovaara (Oulu, Finland), flies of six different species could be trapped. The numbers of specimens are given in Table 1.

Table 1. Number of flies collected in a forest near Tübingen.

	Sept. 75	Sept. 76	Sept. 78	Total
<i>D. subobscura</i>	157	392	201	750
<i>D. obscura</i>	15	44	75	134
<i>D. subsilvestris</i>	5	23	34	62
<i>D. helvetica</i>	-	3	3	6
<i>D. ambigua</i>	1	-	-	1
<i>D. tristis</i>	-	1	-	1

According to these collection data, *D. subobscura* seems to be 12.1 times more frequent than *D. subsilvestris* in this area. Kimura and Crow (Genetics 49: 725-738) have postulated a correlation between the effective population size and the genetic variability for selectively neutral alleles. In the formula

$$H = \frac{1}{4N_e u + 1}$$

the average heterozygosity  $H$  is described as a function of the effective population size  $N_e$  and the mutation rate  $u$ . To prove this theoretical concept the allozyme polymorphism of both species, *D. subobscura* and *D. subsilvestris*, was studied by means of horizontal starch gel electrophoresis. Fourteen loci were investigated: *Adh*, *Ao*, *Aph-3*,  $\alpha$ *Gpdh-3*, *Hk-1*, *Hk-3*, *Idh*, *Mdh-2*, *Me*, *Odh*, *6Pgdh*, *Pgm*, *Phi* and *Tpi*. In *D. subobscura* 78.6% of the loci turned out to be polymorphic; the corresponding percentage in *D. subsilvestris* was 28.6%. The average heterozygosity was determined with 13.1% for *D. subobscura* and 1.5% for *D. subsilvestris*. Thus allozyme variation is actually much higher in the common species than in the rare species which is in accordance with the prediction of Kimura and Crow mentioned above. Using the experimental data for  $H$  and assuming a constant mutation rate of  $10^{-6}$  for both species, the effective population size can be calculated. The result is a number of 37,687 individuals for *D. subobscura* and 3,801 for *D. subsilvestris*. These fictitious population sizes are quite dubious because of the inaccuracy of the parameter  $u$ . The proportion between the two values, however, does not depend on  $u$ . Hence the result that the population of *D. subobscura* is 9.9 times larger than the population of *D. subsilvestris* seems reliable.

In this study information about the proportion between the population sizes of *D. subobscura* and *D. subsilvestris* has been obtained from two completely different sources: from the number of flies trapped in malt baits and from the analysis of allozyme variation. Both methods yield surprisingly similar results of 12.1:1 and 9.9:1 respectively. A  $\chi^2$  test reveals that the deviation is not statistically significant ( $\chi^2=2.3$ ;  $df=1$ ). This conformity of the data leads to the conclusion that the average heterozygosity represents a suitable basis for the estimation of population sizes.

Pinsker, W. University of Tübingen, Germany. Sterility in *D. subobscura* males homozygous in a rare allele at the  $\alpha$ *Gpdh*-locus.

The allozyme variant  $\alpha$ *Gpdh*<sup>90</sup> was detected in a sample of wild flies collected in Tübingen.  $\alpha$ *Gpdh*<sup>90</sup> has an extremely slow electrophoretic mobility in starch gels of pH 7.1 compared to the common variant  $\alpha$ *Gpdh*<sup>100</sup> and was not found again in samples from natural populations, al-

though about 2500 *D. subobscura* flies from several geographic regions had been screened. Since rare alleles were needed for other experiments, single pair crosses were set up in order to obtain a strain homozygous for  $\alpha$ *Gpdh*<sup>90</sup>. This trial, however, failed completely. It turned out that a considerable proportion of the single pair cultures did not yield offspring. Among the fertile pairs, only some females were homozygous for  $\alpha$ *Gpdh*<sup>90</sup> but no males of this genotype could be detected.

To investigate this phenomenon in detail, two different experiments were carried out. In both of them homozygous 90/90-females were crossed with heterozygous 90/100-males. From the  $F_1$  offspring, where the parental genotypes are expected to be present in the ratio of 1:1, single couples were set up in small culture vials. After three weeks the cultures which still contained both partners alive were separated into fertile and sterile pairs and the genotypes of the flies were determined electrophoretically. In the first experiment flies with the original chromosomes were used. For the second experiment the genetic background of the  $\alpha$ *Gpdh*<sup>90</sup>-allele was recombined and substituted to a large extent by means of a marker strain. The results are given in Tables 1 and 2.

Table 1. Number of  $\alpha$ *Gpdh*<sup>90/90</sup> and  $\alpha$ *Gpdh*<sup>90/100</sup> genotypes in sterile and fertile cultures.

	Males		Females	
	90/90	90/100	90/90	90/100
sterile cultures	21	5	4	22
fertile cultures	--	56	14	42
	21	61	18	64
	$\chi^2=60.8$ $p<0.001$	$df=1$	$\chi^2=1.0$ n.s.	$df=1$

In both experiments 90/90-males were only found in the sterile cultures and never among the fertile pairs, whereas the females were randomly distributed. The changing of the genetic background had no influence on the sterility of the males. In this connection it should be noticed that in the first experiment 90/90-homozygotes occurred significantly less frequently in the offspring of 90/90-females and 90/100-males than expected ( $\chi^2=45.1$ ;  $df=1$ ;  $p<0.001$ ),

Table 2. Number of  $\alpha$ Gpdh<sup>90/90</sup> and  $\alpha$ Gpdh<sup>90/100</sup> genotypes in sterile and fertile cultures after substitution of genetic background.

	Males		Females	
	90/90	90/100	90/90	90/100
sterile cultures	41	6	25	22
fertile cultures	--	62	32	30
	41	68	57	52
	$\chi^2=86.7$ df=1 p<0.001		$\chi^2=0.03$ df=1 n.s.	

Table 3. Single pair mating: number of females inseminated by  $\alpha$ Gpdh<sup>90/90</sup> and  $\alpha$ Gpdh<sup>90/100</sup> males after 72 hours.

$\alpha$ Gpdh genotype of male	Females	
	inseminated	not inseminated
90/90	--	21
90/100	18	3
	$\chi^2=31.5$ df=1 p<0.001	

independent of the sex. This lower viability of about 30% compared to the heterozygotes could be due to detrimental alleles of other genes on the same chromosome. In the second experiment viability of the 90/90-homozygotes was reduced only in males ( $\chi^2=6.7$ ; df=1; p<0.01) but not as much as in the first experiment (60% compared to the heterozygotes). Thus the substitution of the genetic background uncoupled the  $\alpha$ Gpdh<sup>90</sup> allele for the most part from the viability-reducing genes but not from the gene (or genes) responsible for sterility. This means that sterility is caused either by a closely linked locus or by the  $\alpha$ Gpdh<sup>90</sup> allele itself. The latter explanation gave rise to the speculation that the sterile males are not able to copulate. The enzyme  $\alpha$ Gpdh is found in high concentration, especially in the flight muscles, and flies mutationally deficient for  $\alpha$ Gpdh are known to be restricted in their ability to fly.

Since males use their wings during courtship, an effect of the  $\alpha$ Gpdh mutant on this behavior seemed at least possible. Direct observation, however, revealed that 90/90-males are not handicapped in copulation. To prove whether these copulations result in insemination, the females were dissected and the receptacula and spermatheca investigated for the presence of sperm. The result of an experiment where single pairs had been set up in small vials for 72 hours is shown in Table 3. It can be seen that none of those females paired with 90/90-males contained sperm. The testes of 90/90-males were therefore also examined. Since sperm were present, it can be concluded that the sterility factor prevents the transfer of sperm to the storage organs of the females. It remains an open question whether this inhibition of insemination is caused by the disturbed action of the mutated  $\alpha$ Gpdh. Assuming that the  $\alpha$ Gpdh locus is directly involved, it seems possible that the mobility of the spermatozoa might be affected in homozygotes for the  $\alpha$ Gpdh<sup>90</sup> allele.

Prakash, H.S. and Sreerama Reddy, G. University of Mysore, India. Distribution of different species of *Drosophila* in Agumbe (Western Ghats), South India.

The Indian subcontinent with its variable geographic features offers a rich abode for the colonization of *Drosophila* species. However, sustained efforts are essential to survey and take census of various species and their densities to get an insight into the taxonomy and

distributional pattern of the genus *Drosophila*. Western Ghats, a mountainous terrain extending along the western border of peninsular India, is one such unexplored territory. The climatic and physiographic features and its luxuriant flora provide a large number of breeding sites for *Drosophila* species. Agumbe, a part of Western Ghats, is one such natural environment situated at an altitude of 826 m, with an average annual rainfall of 8275.7 mm. It is called "Chirapunji" of South India due to its highest annual rainfall. A characteristic feature of rainfall at Agumbe is that all of it is received from a southwest monsoon from July to October. The northwest monsoon has no effect on it. The heavy rainfall has contributed to the growth of thick timber forest with bushy vegetation underneath, and thus provides congenial habitat for *Drosophila* species.

*Drosophila* collections made by conventional fermenting banana bait technique at five sites in this locality during July 1977 yielded a total of 1170 specimens comprising 12 species representing three subgenera (Table 1). Eight of these - *D. eugracilis*, *D. malerkotliana*, *D.*



Table 1. Distribution of different species of *Drosophila* in Agumbe (Western Ghats), South India.

Sites:	1	2	3	4	5	Total
Subgenus: <i>Sophophora</i>						
<i>D. eugracilis</i>	21	17	4	26	12	80
<i>D. malerkotliana</i>	69	27	53	85	105	339
<i>D. pseudoananassae</i>	9	-	-	8	11	28
<i>D. bipectinata</i>	28	15	-	18	21	82
<i>D. anomelani</i>	35	22	29	41	13	140
<i>D. agumbensis*</i>	-	10	8	-	15	33
<i>D. montium</i>	-	3	18	13	22	56
<i>D. rhopaloa**</i>	15	4	-	21	3	43
Subgenus: <i>Drosophila</i>						
<i>D. nasuta</i>	75	65	33	48	57	278
<i>D. neonasuta</i>	18	11	9	14	16	68
<i>D. grandis**</i>	2	-	-	-	-	2
Subgenus: <i>Scaptodrosophila</i>						
<i>D. mundagenesis</i>	5	3	8	1	4	21
Total	277	177	162	275	279	1170
No. of species	10	10	8	10	11	

\*New species described by the authors

\*\*Reporting for the first time from India

in large numbers in almost all the sites, indicating the occurrence of favorable habitats for the colonization of these species, while the other species were found to be present in small numbers only in some sites. Differences in regard to the number and composition of *Drosophila* species between sites can be correlated not only with ecological attributes of the species but also with the colonizing or invastive abilities of the species concerned. It is clear from the collection data that the members of the melanogaster species group in particular are dominant, as evidenced not only by their large numbers but also in the variety of species. The members of the immigrans species group occupy the next place. These findings are in conformity with the earlier reports of Reddy and Krishnamurthy (1974) and Prakash and Reddy (1978b), and corroborate the suggestion of Bock and Wheeler (1972), who regarded the Indian subcontinent as the general area for the origin of melanogaster species group, and southeast Asia in general for the origin and wide speciation of both melanogaster and immigrans species groups.

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Rahman, R. and D.L. Lindsley. University of California, La Jolla, California. Effect of proximal X-chromosome deletions on male fertility.

have tested are male fertile when covered by the long duplication  $y^{+}mal^{+}$ .

*pseudoananassae*, *D. bipectinata*, *D. anomelani*, *D. agumbensis*, *D. montium* and *D. rhopaloa* (melanogaster species group) - belong to subgenus *Sophophora*; four species - *D. nasuta*, *D. neonasuta*, *D. immigrans* (immigrans species group) and *D. grandis* - belong to subgenus *Drosophila*; and only one species - *D. mundagenesis* - belongs to subgenus *Scaptodrosophila*. 33 flies of a new species, *D. agumbensis* (Prakash and Reddy 1978b), 43 flies of *D. rhopaloa* and 2 flies of *D. grandis* were collected for the first time in this locality and are new additions to the Indian *Drosophila* fauna. Among the species collected, only three (*D. malerkotliana*, *D. anomelani* and *D. nasuta*) were found

It was claimed by Lifschyts and Lindsley (1972) that deficiencies that included both  $su(f)$  and  $bb$  are male sterile, whereas deficiency for either  $su(f)$  or  $bb$  alone are male fertile. Later Schalet and Lefevre (1973) observed that many of the deficiencies for both  $su(f)$  and  $bb$  they

Whereas no one disputes that some proximal X-chromosome deficiencies are male sterile, the genetic concomitants remain undefined. We decided to investigate the factors leading to male sterility of such deficiencies. For this purpose a sample of 14 su(f) deficiencies were picked up by means of the characteristic phenotype of su(f)/Df(1)su(f) females. In addition nine deficiencies were obtained from Dr. A. Schalet and two from Dr. G. Lefevre. The extents of all these deficiencies were determined by testing their survival in combination with y<sup>mal+</sup>, y<sup>Ymal126</sup>, Dp(1;f)3, BSYy<sup>+</sup> and 1(1)Q464, 1(1)Q56, 1(1)R10-10, 1(1)Q463, 1(1)X-4, and 1(1)R-9-18.

Of the total of 25 su(f) deficiencies 9 were deficient for both su(f) and bb. Each of these was tested for viability and male fertility in combination with each of 3 different Y chromosomes carrying a duplication for different amounts of the base of the X. The results of these tests are summarized in the accompanying table. The upper left quadrant of the table contains an ordered list of the loci tested at right end of the X. The constitutions of the deficient X's and the duplicated Y's with respect to these loci are indicated in the lower left

[illegible]

and upper right quadrants respectively. The lower right quadrant tabulates the phenotypes of the various combinations (l=lethal, F=fertile, S=sterile and WF= weakly fertile with <3 progeny per male. Briefly these observations suggest that males carrying short X-deficiencies and short duplications or long X-deficiencies and long duplications are fertile, whereas males carrying short deficiencies and long duplications are sterile; it is important to note that all three duplicated Y's are male fertile in combination with normal X chromosome.

The 16 su(f) deficiencies that carried  $bb^+$  were also tested in combination with the duplicated Y chromosomes; all viable combinations were male fertile. In addition three  $bb$  deficiencies were tested with these duplicated Y chromosomes.  $Df(1)bb^{1-158}$ ,  $Df(1)bb^{1-74}$  and  $bb^1$  are long, intermediate and short  $bb$  deficiencies respec-

tively that have not lost su(f). When tested for male fertility in combination  $y^+Ymal^+$ ,  $bb1-158$  was sterile,  $bb1-74$  and  $bb1$  produced 0.6 and 9.6 progeny per male respectively. Males carrying each of these  $bb$  deficiencies in combination with  $y^+Ymal^{126}$  produced 32.8, 68.7 and 95.6 progeny respectively.

Thus male sterility seems to result from an as yet imperfectly defined interaction of a deficiency for bb in the proximal heterochromatin of the X chromosome and a duplication for euchromatic elements at the right end of the X chromosome.

This work was supported by Rotary Foundation Graduate Fellowship awarded to R. Rahman.

References: Lifschytz and Lindsley 1972, PNAS 69:182-186; Schalet and Lefevre 1973, Chromosoma 44:183-202.

Ramshaw, J.A.M. and J.A. Coyne. Museum of Comparative Zoology, Harvard University, Cambridge, Massachusetts. Is secondary modification primarily responsible for observed enzyme polymorphism? No!

The method of using many electrophoretic conditions to study enzyme variation in natural populations has led to a significant increase in the amount of variation when compared to that detected by only a single electrophoretic condition (Singh et al. 1976; Coyne and Felton 1977; Coyne et al. 1979a). This increase in

variation is most dramatic for loci which are already known to be polymorphic under a single electrophoretic condition. Loci which are monomorphic under one electrophoretic condition, however, remain monomorphic even when studied under several conditions.

It is possible that some of the observed enzyme variation may not be due to changes at the structural locus but rather to modifier genes which influence enzyme mobility. It is thus important to investigate the genetic basis of variation whenever possible.

Such genetic studies have been done in our laboratory for the loci xanthine dehydrogenase, esterase-5, and alcohol dehydrogenase-6 in *D. pseudoobscura* and *D. persimilis*. Our investigations have always shown codominant segregation of mobility variation with no evidence of inheritance patterns suggesting modification elsewhere in the genome. However, it is possible that modifiers of enzyme mobility exist in addition to the variation which has been so far discovered. The way to search for such variation would be to hold the structural locus constant while varying the genetic background to investigate possible changes in mobility of the enzyme. We have done this for the xanthine dehydrogenase locus in *D. pseudoobscura* and have found no such modifiers in a sample of 52 X chromosomes from 12 geographic populations (Coyne et al. 1979b).

Esterase loci are generally highly polymorphic in species of *Drosophila*. Cochrane and Richmond (1979) have reported segregation at a locus on the 3rd chromosome of *D. melanogaster* which modifies the mobility of the esterase-6 enzyme produced by a structural locus elsewhere on this chromosome. Although this allele is not common in natural populations of the species (a search of 50 lines from nature failed to reveal it), these authors suggest that such modifier genes may account for esterase polymorphisms in other drosophilids.

We report here the initial results of a search for modifier loci on the major autosomes of *D. pseudoobscura* which might modify electrophoretic mobility of the esterase-5 protein produced by a locus on the X chromosome. In this study we have used a wide variety of isochromosomal lines which were constructed in this laboratory. Such lines homozygous for a given autosome derive their X chromosomes entirely from the marker stock used to construct them (see Coyne et al. 1979b). If the marker stock happens to be homogeneous for one electrophoretic allele at an X-linked locus, then the isochromosomal lines all share the same allelic form of this locus while differing in genes on the isochromosome. These isochromosomal lines can then be examined for possible mobility effects of the X-linked enzyme induced by the autosomes. Our marker stocks for the 2nd, 3rd and 4th autosomes of *D. pseudoobscura* have all proven to be homogeneous for different esterase-5 alleles when investigated under the five electrophoretic conditions of Coyne et al. (1978).

We have studied 51 isochromosomal-2 lines of *D. pseudoobscura* from 11 geographic populations. The Delta-cardinal marker stock used to make these lines was homozygous for the 1.06 allele of the esterase-5 locus, and no variation in mobility of this allele was seen in any of the 51 lines tested at the same five electrophoretic conditions. Initial analysis was also done for six isochromosomal-3 lines constructed with an orange-Blade-scutel stock, and these lines showed no variation of the esterase-5 allele carried by that stock.

We conclude from this and previous studies that while secondary modification of protein structure may exist for certain proteins, it is probably of little importance in accounting for allozyme polymorphisms detected by differences in electrophoretic mobility. Rather, this observed variation is due to either allelic variation at the structural locus or closely linked cis-dominant modifiers of mobility (Coyne et al. 1979b).

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Ransom, R. Open University, Milton Keynes, U.K. Investigation of temperature sensitivity in three eye mutations.

Milani (1946) has previously reported that the *D. melanogaster* mutation *sine oculis* is temperature sensitive, the eyeless effect being enhanced when flies are cultured over 25°C and lethality is reached at 30°C. Because a deeper

analysis of the time span of temperature sensitivity may help to pinpoint the time of gene action, a series of temperature shift experiments were carried out. Prepupal growth was split into three stages, embryonic, 1st-2nd larval instars and 3rd larval instar development. Flies were reared either at 22°C, 29°C or with shifts between these two temperatures at the onset or close of the three stages studied.

Both 22° and 29° growth periods were "corrected" to standard 25° growth periods. This was done because the prepupal growth period took about 1.2X and 0.8X that of 25° growth respectively. Shift times were therefore calculated using these multiplication factors. The results may be seen in Table 1. The effect of embryonic temperature on both the proportion of flies with eyes and lethality is low. The effect of larval temperature shifts is more marked with a 35% increase in eye frequency and 37% drop in lethality when larval culture is at 22°C. The reduction in eyelessness may be totally accounted for by passing the third larval instar at 22°C. It has recently been shown by histology and clonal analysis (Ransom 1979) that cell death occurs in the third larval instar of so, thus suggesting that the temperature reduction may lessen cell death. Lethality cannot be narrowed down in this way, both early and late larval culturing at low temperature being necessary to limit lethality.

Experiments using both *ey*<sup>2</sup> and *ey*<sup>D</sup> failed to show any differences in eye sizes or lethality after culturing at different temperatures. Baron (1935) noted a significant reduction in eye size among *ey*<sup>2</sup> stocks selected for small eye size over a period of some generations and then cultured at high temperature for various periods. The time of greatest change was observed as 36-60 hours, earlier than the so change reported here.

Table 1. Temperature sensitivity in *sine oculis*. "High" and "low" refer to the temperatures (29°C, 22°C) at which the periods in the left hand column were passed. EMB = embryonic; L1, L2, L3 = first, second, third larval instars. L = whole larval period. All = 0-120 hours at given temperature. Proportional differences are calculated as the difference between high and low temperature values divided by the average of the two values.

	temperature at given period	proportion of flies with eyes $\pm$ se	proportional difference in eye freq high/low temperatures	lethality $\pm$ se	proportional difference in lethality high/low temperatures
EMB	high	.16 $\pm$ .03		.64 $\pm$ .16	
	low	.18 $\pm$ .07	0.12	.61 $\pm$ .02	0.05
L1 + L2	high	.25 $\pm$ .03		.70 $\pm$ .20	
	low	.20 $\pm$ .11	0.22	.60 $\pm$ .06	0.15
L3	high	.13 $\pm$ .03		.68 $\pm$ .12	
	low	.20 $\pm$ .04	0.42	.57 $\pm$ .06	0.17
L	high	.14 $\pm$ .03		.73 $\pm$ .13	
	low	.20 $\pm$ .06	0.35	.50 $\pm$ .09	0.37
All	high	.13 $\pm$ .01		.81 $\pm$ .11	
	low	.20 $\pm$ .03	0.42	.50 $\pm$ .08	0.47

This work was performed in the laboratory of Dr. J.A. Campos-Ortega, Institut für Biologie III, Universität Freiburg, West Germany.

References: Baron, A.L. 1935, J. Exp. Zool. 70:461-490; Milani, R. 1946, Boll. Soc. Ital. Biol. Sper 22:112-113; Ransom, R. 1979, J. Emb. Exp. Morph. in press.

Rasmuson, B. and I. Montell. University of Umea, Sweden. Genetic instability and the production of transposing elements in *Drosophila melanogaster*.

In males with the genetic constitution  $z Dp (1:1) (w^{SP}) (w^a)$  kept against attached females, we have on several occasions (6 separate times) found premeiotic eliminations of the duplication with a simultaneous elimination of the  $w^{SP}$  in the left and a  $w^a$  in the right duplication. This

duplication-eliminated chromosome gives a wild type eye color phenotype, characteristic for  $z w^+$  males. But the chromosome region remains genetically unstable, generating deletions (i.e. white mutants) in high frequency, as well as shifts in the white-locus activity, giving  $z w^+$  (zeste) and  $z w^+$  (red) eye color phenotypes (Rasmuson et al.). Recombination experiments have indicated that the unstable DNA possibly is associated with the orientation of the inserted segment, which is localized to the right of the white-locus, and thus regulates the white-locus activity.

The localized unstable DNA is also a part of a transposing element. It can be spontaneously excised from the original position in the X-chromosome and integrated into non-homologous positions. In this transposing process the white-locus can be co-transposed into the new positions, where the locus is completely active.

The following positions have been mapped. The two first mentioned are spontaneous in origin. The first is a transposon into the heterochromatin of the fourth chromosome, in which position it has been shown to retain its instability. The phenotypic expression is associated with the number of Y-chromosomes. The second transposon is inserted into the third chromosome, but its position is still not well known. The last three transposons have appeared after mutagen treatment, and they have all been found to be inserted into the second chromosome. Transposon  $w^{+II}(78c28)$  is mapped to about 74, transposon  $w^{+II}78e01$  to about 57, and transposon  $w^{+II}78h24$  to about 59 in the second chromosome.

They are all very short transposons; no one covers the *rst* or the *vt* loci to the right of white-locus nor one of the closest localized lethals to the left of the locus, i.e., Judd's  $l(1) 63k18$ , localized 0.022 map units to the left of the white-locus. They are all characterized by wild type pigmented males in association with  $z$  in the X-chromosome, except for the transposon  $T w^{+II}78c28$ , the males of which have a halo-pigmented margin of the eye. The  $T w^+ 78h24$  is of particular interest, since simultaneously with the transposon the corresponding deletion of the white-locus was isolated as a premeiotic  $z Df(1) w^- 78h24$  deletion.

Preliminary hybridizing experiments together with Gvozdev show this unstable DNA to be identical with the intercalary heterochromatic DNA, cloned in the Dm 225 plasmid (Ilyin et al.), since the male salivary chromosomes from the  $z w^+$  (zeste) phenotype as well as the  $z w^+$  (red) phenotype show hybridization with this cloned DNA, whereas the  $Df(1) w^-$  deletion, which is a white eyed deletion from this unstable X-chromosome, does not.

References: Rasmuson, B. and M. Green 1974, Genetic instability in *Drosophila melanogaster*. A mutable tandem duplication, *Mol. Gen. Genet.* 133:249-260; Ilyin, Y.V., N.A. Tchurikov, E.V. Ananiev, A.P. Ryskov, G.N. Yenikolopov, S.A. Limborska, N.E. Maleeva, V.A. Gvozdev and G.P. Georgiev 1978, Studies on the DNA fragments of mammals and *Drosophila* containing structural genes and adjacent sequences, Cold Spring Harbor Symposia on Quantitative Biology, Vol. XLII:959-969.

Richmond, R.C. Indiana University, Bloomington, Indiana. Temperature and dessication tolerance in four species of the *affinis* subgroup.

*D. affinis*, *algonquin*, *athabasca* and *narragansett* occur sympatrically in a subset of their ranges. The relative abundance of these species varies within any one locality over time and over a latitudinal gradient between localities (Miller, *Amer. Midl. Natur.* 60:52; Richmond,

unpub. data). Fig. 1 shows the average relative frequency of the four species in a single locality near Bloomington, Indiana for the months of March through September in 1972, 1973 and 1974. We tested the hypothesis that the temperature and dessication tolerance of the four species might account for the pronounced shifts in relative frequency by determining the time required for 50% of a group of flies to die when subjected to combined temperature and dessication stress. A group of 20 flies of one sex was placed into a 71 cc glass vial which was immersed in a water bath. Dry air obtained by passing the stream through a 4:1 mixture of anhydrous calcium chloride and "indicating" Dryrite was routed through each vial at a rate of

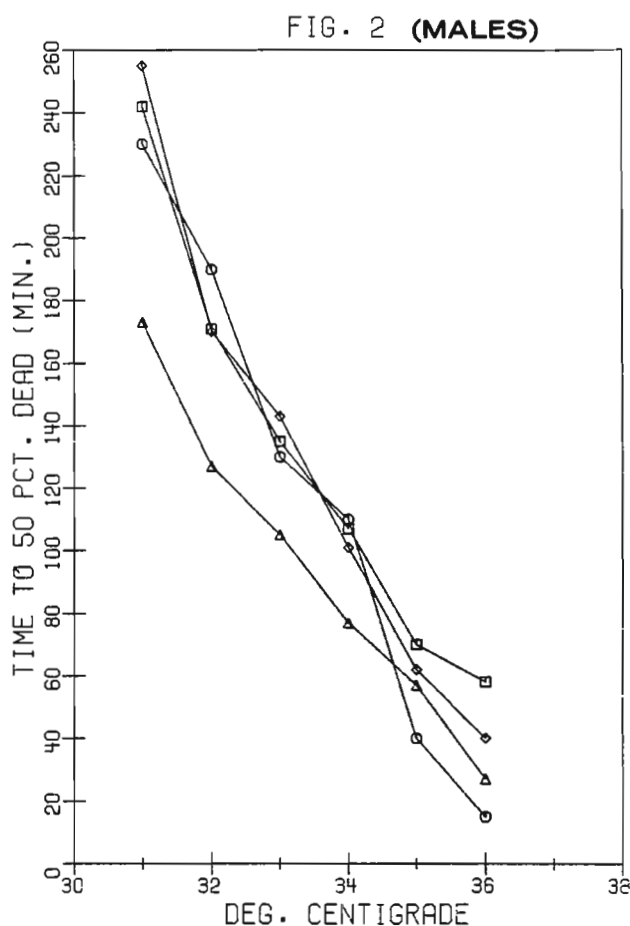
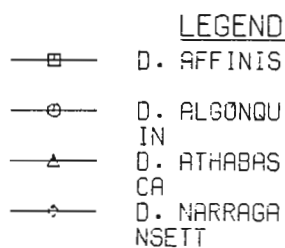
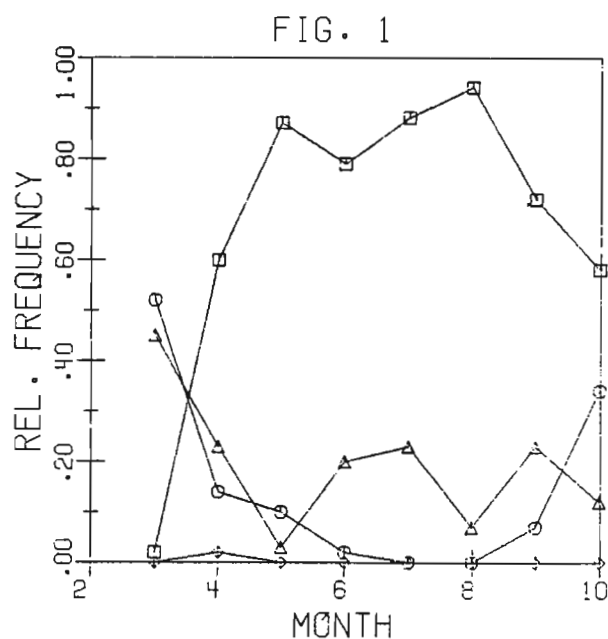
0.2 liters per minute. Air temperature was that of the water bath. Twenty individuals of each sex of the four species were tested at 31, 32, 33, 34, 35 and 36°C. At any one temperature, individuals of one sex of all species were tested simultaneously. The results are summarized in Figs. 2 and 3. *D. athabasca* is the least tolerant of the four species at temperatures below 34°C. At temperatures from 31-34°C, *D. affinis*, *algonquin* and *narragansett* have approximately equal tolerances to the stress conditions. However, at temperatures above 34°C, there are consistent differences between the four species such that the species tolerance to high temperature and dessication can be summarized as *affinis* > *narragansett* > *athabasca* > *algonquin*. A three-way, fixed factor analysis of variance of these data is given below.

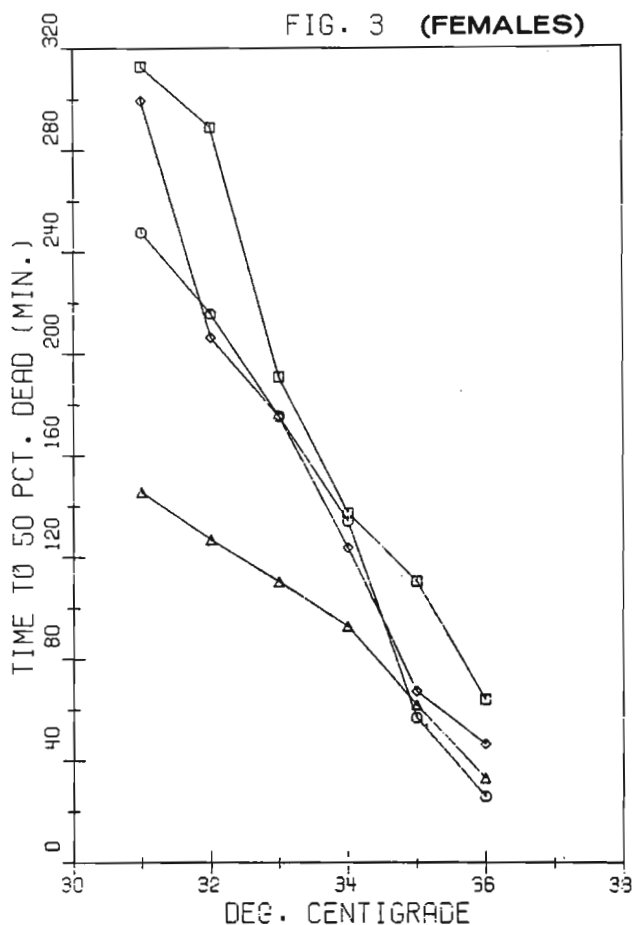
Source	df	Mean Squares	F
Temperatures	5	43416.9	199.5***
Species	3	8049.9	37.0***
Sex	1	5355.2	24.6***
Temperature x Species	15	913.2	4.2**
Temperature x Sex	5	263.4	1.2
Species x Sex	3	1262.0	5.8*
Temperature x Species x Sex	15	217.6	
Total	47		

\*\*\* $p < 0.001$  \*\* $p < 0.005$  \* $p < 0.01$

Each of the principal factors tested had a strongly significant effect on the tolerance of these species. As is suggested by Figs. 2 and 3, there are significant interactions between temperature and species and between species and sex. However, the relative tolerances of the sexes over the temperatures used remain fairly constant, thus the temperature x sex interaction is not significant. The signifi-

cant differences in tolerance to combined dessication and temperature stress especially at higher temperatures suggest that the physiological tolerances of these species may well be a significant factor affecting their distributions and abundances in time and space. Supported by NIH Grant GM23706.





Richmond, R. C. Indiana University,  
Bloomington, Indiana. Effects of tem-  
perature and humidity stress on geno-  
type distribution at six allozyme loci.

Several investigators have studied the effects  
of short term temperature stress on allozyme loci  
in *D. melanogaster* (Johnson and Powell, PNAS 71:  
1783; Milkman, DIS 52:58). Flies collected from  
an established population cage were subjected to  
36°C and 0% humidity until approximately 50%

were dead (about 45 minutes). Both dead and living flies were removed and their genotypes at  
3-6 diallelic, allozyme loci were determined. Since there were no significant differences be-  
tween genotype distributions in the two sexes, the data for both sexes have been combined.

Locus	Dead Flies					Living Flies				
	Genotype numbers					Genotype numbers				
	SS	SF	FF	F	$\chi^2$	SS	SF	FF	F	$\chi^2$
Mdh	184	21	1	-0.054	0.60	133	13	0	-0.047	0.32
Adh	74	74	27	+0.089	1.37	49	40	8	-0.004	0
Odh	4	6	68	+0.529	21.84*	1	18	98	+0.016	0.03
Est 6	38	110	55	-0.091	1.70	32	77	36	-0.063	0.57
$\alpha$ Gpdh	2	25	182	+0.074	1.13	0	22	124	-0.082	0.97
Pgm	99	61	16	+0.109	2.07	36	50	8	-0.167	2.63

\*  $p < 0.005$

In the table above, the genotype distributions at each locus are given as is Wright's inbreeding coefficient,  $F$ , which measures deviations from Hardy-Weinberg expectations (+ = deficiency of heterozygotes; - = excess of heterozygotes). Only the *Odh* locus among dead flies shows a significant deviation from Hardy-Weinberg expectations. However a comparison of the



signs of F indicates a tendency for heterozygote excess among living flies. This hypothesis was tested by computing heterogeneity Chi-squares for each locus as shown below. The Pgm locus clearly shows the effects of selection, and there is a suggestion of an effect at the Odh locus. These two loci are linked (5.8 map units) on chromosome III; however, the Est 6 locus shows no such effect even though it is closely linked (7.4 map units) to the Pgm locus.

	<u>Mdh</u>	<u>Adh</u>	<u>Odh</u>	<u>Est 6</u>	<u><math>\alpha</math>Gpdh</u>	<u>Pgm</u>	<u>Total</u>
Chi-square	0.86	3.45	5.65 <sup>+</sup>	0.66	2.07	9.09 <sup>‡</sup>	21.78*
DF	2	2	2	2	2	2	12
<hr/>							
* p < 0.05	‡ p < 0.025		+ p = 0.059				

Biochemical studies of the in vitro thermal stability of the major alleles at the Adh (Clarke et al., Biochem. Genet. 11:141), Est 6 (Cochrane, Nature 263:131) and  $\alpha$ Gpdh loci (Miller et al., Biochem. Genet. 13:175) show that the S electromorphs at the Adh and Est 6 loci are more stable. Although the shifts in gene frequencies apparent in the above data are not significant, they do agree with predictions from biochemical studies. Supported by NIH grant GM23706.

Robertson, A. Institute of Genetics, Edinburgh University, Scotland. Quantitative variation on the fourth chromosome of *D. melanogaster*.

Following earlier indications (Madalena and Robertson, Genetical Research 24:113), I investigated the effect of different fourth chromosomes from lines selected for high and low sternopleural score (averaging 48 and 8 bristles, respectively) differed in mean score for dif-

ferent bristles as indicated in the table. In the background of the high selected line, there are indications that the difference in sternopleural score between the two selected homozygotes is more than ten bristles. The low chromosome is almost completely recessive in score to the high and is apparently rare in the base population. Fourth chromosomes from four other low sternopleural lines each had a distinct pattern of scores for the three types of bristles. Differences were also found in female abdomen pattern and one chromosome apparently carried the lost mutant "scutenick".

Source of fourth chromosome	<u>Sternopleural</u>	<u>Abdominal (fifth only)</u>	<u>Ocellar</u>
high	13.2	16.2	6.5
low	11.3	13.0	4.1
unselected	13.1	15.7	6.3

Romans, P. Univ. of California, San Diego, La Jolla, California. Gene conversion in mei-9<sup>a</sup>, a crossover defective mutant in *D. melanogaster*.

In *D. melanogaster* females homozygous for mutant alleles at the mei-9 locus, crossing over is reduced uniformly in all genetic intervals studied (to about 8% of the wild type map in mei-9<sup>a</sup>). From these data it has been inferred that the wild type product of the locus functions directly

in the process of exchange (Baker and Carpenter, 1972). Analysis of mutants at this locus has shown that the wild type product is also required for normal mitotic chromosome stability in males and females (Baker et al., 1976, 1978; Gatti, 1979), for repair replication (Nguyen and Boyd, 1977), and for excision repair (Boyd et al., 1976). To probe the function of this gene further, and to investigate the relationship between crossing over and intragenic recombination, I have examined the ability of females homozygous for mei-9<sup>a</sup> to carry out intragenic recombination.

Recombination with the rosy (ry) locus was assessed using the purine selection system (see Chovnik et al., 1977 for review). The crosses were as indicated in the table. When parents were removed from bottles after 3 days of egg laying, the developing zygotes were treated with 0.8 ml 0.185% (w/v) aqueous purine added to the food, or with 0.8 ml deionized distilled water.



Table 1. Production of ry<sup>+</sup> recombinants by wild type and mei-9a females

♀ Parent	♂ Parent	Total Zygotes	X ND per ovum	Total ry <sup>+</sup> recombinants	Rate ry <sup>+</sup> recombinants	Recombination event		
						X-over	conv ry <sup>5(502)</sup>	conv ry <sup>41</sup>
Experiment I:								
$\frac{y}{y} ; \frac{ry^5}{cu\ kar\ ry^{41}\ 126}$	$+\frac{MKRS^1}{\bar{y}} ; \frac{MKRS^1}{In(3R)P_{18}^2}$	--	--	10	~1/22,000 <sup>3</sup>	2	3	4
$\frac{y\ mei-9a}{y\ mei-9a} ; \frac{ry^5}{cu\ kar\ ry^{41}\ 126}$	$+\frac{MKRS}{\bar{y}} ; \frac{MKRS}{In(3R)P_{18}}$	--	--	13	~1/24,000 <sup>3</sup>	0	6	2
Experiment II:								
$\frac{y}{y} ; \frac{ry^{502}\ e^s\ ro}{cu\ kar\ ry^{41}\ 126}$	$+\frac{MKRS}{\bar{y}} ; \frac{MKRS}{kar\ Df(3R)ry^{75}}$	1.47x10 <sup>6</sup>	0.11%	22	1/67,000	10	5	7
$\frac{y\ mei-9a}{y\ mei-9a} ; \frac{ry^{502}\ e^s\ ro}{cu\ kar\ ry^{41}\ 126}$	$+\frac{MKRS}{\bar{y}} ; \frac{MKRS}{kar\ Df(3R)ry^{75}}$	0.51x10 <sup>6</sup>	25.8%	15	1/34,000	1	8	3

1. MKRS = Tp MKRS, M(3)S34 kar ry<sup>2</sup> Sb2. In(3R)P<sub>18</sub> = In(3R)P<sub>18</sub>, Ubx ry<sup>41</sup> kar, e<sup>4</sup>

3. Rates based on complete adult counts from only a portion of the control bottles

Counts of adults emerging from water treated bottles (about 10% of the total) were used to estimate the total population of zygotes yielding  $ry^+$  recombinants. X-chromosome non-disjunction rates were also obtained from these counts, and were close to previously established values for wild type and  $mei-9^a$  females.

The data in the table indicate that intragenic recombination occurs in  $mei-9^a$  females at or above wild type rates. From analysis of flanking marker combinations in the  $ry^+$  recombinant chromosomes it may be inferred (for rationale see Chovnick et al., 1971) that in  $mei-9^a$ , most of the recombination events are gene conversions. Indeed, only one crossover was recovered among the 20  $ry^+$  recombinants tested. Thus, the  $mei-9^a$  defect results in a reduction of intragenic crossing over, but not of gene conversion. Several of the  $ry^+$  recombinant progeny of  $mei-9^a$  females (4 of 8 from  $ry^5/ry^{41}$  crosses, 1 of 12 from  $ry^{502}/ry^{41}$  crosses) transmitted  $ry$  to their offspring. This is inferred to be the result of post-meiotic segregation of  $ry$  and  $ry^+$  in the first mitotic division of the embryo since 2 of the 5 recombinants that transmitted  $ry$  to offspring transmitted  $ry$  and  $ry^+$  maternally derived chromosomes. Since many  $mal^+-mal$   $XDH^+-XDH^-$  mosaic flies do not survive purine treatment although heterozygotes do (see accompanying note), the data presented here are consistent with the hypothesis that gene conversion is actually increased above wild type levels in  $mei-9^a$  females.

Current molecular models of recombination (Meselson and Radding, 1975) suggest that gene conversion and crossing over are alternative fates of a heteroduplex DNA intermediate. The reduction in crossing over and concomitant increase in gene conversion observed in  $mei-9^a$  females are not inconsistent with these models. Since a high level of post-meiotic segregation is not a feature of recombination at the  $ry$  locus or any other locus that has been tested in  $mei-9^+$  flies, these results also suggest that the  $mei-9^+$  excision repair function may be an important agent of excising base pair mismatch from heteroduplex DNA formed during gene conversion in *Drosophila*.

Supported by Public Health Service Grant GM23345 to B. Baker.

References: Baker, B.S. and A.T.C. Carpenter 1972, *Genetics* 71:255-286; Baker, B.S., A. T.C. Carpenter and P. Ripoll 1978, *Genetics* 90:531-578; Baker, B.S. et al. 1976, *PNAS* 73:4140-4144; Boyd, J.B. et al. 1976, *Genetics* 84:527-544; Chovnick, A., W. Gelbart and M. McCarron 1977, *Cell* 11:1-10; Chovnick, A., G.H. Ballantyne and D.G. Holm 1971, *Genetics* 69:179-209; Gatti, M. 1979, *PNAS* 76:in press; Meselson, M.S. and C.M. Radding 1975, *PNAS* 72:358-361; Nguyen, T.D. and J.B. Boyd 1977, *Molec. Gen. Genet.* 158:141-147.

Romans, P. Univ. of California, San Diego, La Jolla, California. Effects of purine selection on survival of *Drosophila* mosaic for Xanthine Dehydrogenase (XDH) activity.

In certain crosses rosy mosaic flies (post meiotic segregants) appear to be produced by recombination (see previous report). Since the recombinants recovered were selected for under conditions of purine treatment which result in death of  $XDH^-$  ( $ry$ ) individuals, and survival of  $XDH^+/XDH^-$  ( $ry^+/ry$ ) heterozygotes, it is of interest

to know how well mosaics which have some tissues heterozygous  $XDH^+/XDH^-$  and the rest  $XDH^-$  survive the treatment to be scored as recombinants. The following experiment was performed to answer this question.

$y f^{36a}$   $mal$  females were crossed to  $R(1)2 w^{vc}/y^+.Y$  males to generate mosaic zygotes of the required kind: maroonlike flies, like rosy flies, lack detectable XDH activity (for review, see Dickinson and Sullivan, 1975). The following breeding protocol was followed in order that numbers of zygotes of the various genotypic classes in purine and water treated (control) cultures should be nearly identical. In order to insure healthy culture conditions and minimize culture dependent effects of purine, large numbers of rosy flies were allowed to lay eggs in bottles used for the crosses for about six hours prior to introduction of the experimental parents. Then a three-day brood was collected from each set of parents and treated with either 0.8 ml deionized distilled water or 0.8 ml of 0.165% (w/v) aqueous purine at the time the parents were removed from the bottles. Three additional broods were obtained in exactly the same way. Half the cultures begun on a particular day received the purine treatments in broods 1 and 3, water in 2 and 4, and the other half, the reverse.

Table 1. Results of crosses of  $y f^{36a} \text{ mal } \text{♀♀} \times R(1)2 w^{vc}/y^+.Y \text{ ♂♂}$ 

Treatment	Genotype					total
	$y f^{36a} \text{ mal}/y \cdot Y \text{ ♂♂}$	$y f^{36a} \text{ mal}/R(1)2 w^{vc} \text{ ♀♀}$	gynandro-morphs	$y f^{36a} \text{ mal}/0 \text{ ♂♂}$	other	
Water	3660	660	198	419	1*	4938
Purine	6	668	117	0	0	791

\*  $y f^{36a} \text{ mal}/y f^{36a} \text{ mal}/y^+.Y \text{ ♀}$

The progeny recovered from the crosses are summarized in Table 1. Equal numbers of  $y f^{36a} \text{ mal}/R(1)2 w^{vc}$  non-mosaic females were recovered following both treatments: because of the protocol employed, it is assumed that close to 100% of the  $XDH^+/XDH^-$  heterozygotes survived at the dose of purine used. However, many fewer (58.3% of control)  $XDH^+-XDH^-$  mosaic gynandromorphs were recovered from the purine treated cultures. This suggests that purine treatment caused pre-adult death of a large proportion of mosaic zygotes. This might be because either a large fraction of their tissue was  $XDH^-$  or each of the lethal mosaics was  $XDH^-$  in some critical tissue or tissues.

In order to examine these possibilities, the distribution of  $y f^{36a} \text{ mal}$  tissue in the recovered mosaic gynandromorphs was determined by scoring  $y$  and  $f^{36a}$  in their cuticle. Forty-six cuticular structures were scored per side. All of the mosaics recovered from water treatments and 115 of the 117 recovered from purine treatments were scored. In these mosaics the average proportion of  $y f^{36a} \text{ mal}$  tissue was 60.8% for water treated zygotes and 45.6% for purine treated ones. The 60.8%  $f^{36a} \text{ mal}$  tissue found in the control is higher than values usually reported for loss of  $R(1)2 w^{vc}$  (values are in the range 49-51% for maternally inherited  $R(1)2 w^{vc}$ , Hall et al., 1976). Since the two groups of mosaics were identified and scored concurrently, it is unlikely that mosaics with a low percentage of  $y f^{36a}$  cuticle were missed in one case but identified in the other. A more reasonable interpretation is that the paternally contributed ring X was very unstable and could be lost at more than one cell division.

Table 2. Distribution of amounts of  $y f^{36a} \text{ (mal)}$  cuticle in recovered mosaics

Treatment	Number of flies with indicated % structures $y f^{36a}$ per mosaic					Total
	1-20	21-40	41-60	61-80	81-100	
Water	33	19	30	48	68	198
Purine	36	17	14	21	27	115

purine treatment (35) is about the number to be expected if all mosaic zygotes produced had survived purine treatment, rather than the observed 58%. Thus it appears that all or most mosaics with a low percentage of  $y f^{36a} \text{ mal}$  tissue survived, and progressively fewer survived in the higher percentage groups.

Table 3. Distribution of  $y f^{36a}$  cuticular structures in recovered mosaics

Region (number of structures scored)	Average fraction of cuticle $y f^{36a}$ per half mosaic		
	Water	Purine	Ratio Purine/water
Head (11)	.593	.548	.93
Thorax (21)	.623	.503	.79
Abdomen (14)	.537	.326	.61
Tergites 1-4, sternites 2-4	.579	.390	.67
Tergites 5-7	.511	.315	.62
Sternites 5-6	.519	.279	.52
Genitalia and analia	.447	.167	.38

The distribution of the amounts of  $y f^{36a}$  cuticular tissue found in the mosaics of the two series is summarized in Table 2. A contingency  $\chi^2$  test shows that the two distributions are different ( $\chi^2_4 = 13.22$ ,  $P = 0.01$ ). More than half the total  $\chi^2$  value (7.00) is contributed by the 1-20%  $y f^{36a} \text{ mal}$  tissue classes. The number of mosaics in this class recovered after

To inquire whether any particular region of the fly must be  $XDH^+$  in order for it to survive purine treatment, I examined the fraction of cases in which any particular structure was  $y f^{36a}$  among mosaics recovered from the same treatment. An obvious feature of this analysis (Table 3) is that abdominal structures, in particular the posterior ventral ones, sternites 5 and 6 and the genital structures and analia, exhibited very low frequencies of  $y f^{36a}$  cuticle

in purine treated as compared to control mosaics. This implicates tissues in the posterior ventral parts of the larva as those responsible for lethality of  $XDH^-$  (mal) and  $XDH^+-XDH^-$  (mal) flies following purine treatment.

The failure of a large fraction of  $XDH^+-XDH^-$  (mal) mosaic zygotes to survive purine selection suggests that recombination values may be underestimated in rosy experiments such as those referred to above in which mosaic recombinants are produced.

Supported by Public Health Service Grant GM23345 to B. Baker.

References: Dickinson, W.J. and D.T. Sullivan 1975, Gene Enzyme Systems in *Drosophila*, 49-69; Hall, J.C., W.M. Gelbart and D.R. Kankel 1976, in: The Genetics and Biology of *Drosophila*, ed. Ashburner and Novitski, Vol. 1a, 265-314.

Schuchman, E., A. Port and E.A. Carlson.  
State Univ. of New York, Stony Brook.  
Mutagenesis of the vestigial region of  
*D. melanogaster*.

Spontaneous mutations of vestigial are well known and include minor (nicked or notched wings), moderate (excised or antlered wings) and intense (strapped and vestigial) reductions of the wings. The vestigial region is located at 67.0 on the second chromosome. The normal

allele is highly mutable with both x-rays and chemical mutagens, but there are many differences in its response to these mutagens than is the case for dumpy, a mutable multiple allelic system with numerous gradations of allelic expression.

Table 1. EMS induced vestigial phenotypes using  $+ \delta \times "12 \text{ pl}" \varphi$  (19°C)

Phenotype	Total	Complete	Mosaic
nick	= 91	6	85
notch	= 102	27	75
excised	= 22	8	14
antler	= 61	5	56
strap	= 11	1	10
vestigial	= 2	1	1

The 289 allelic vestigial phenotypes were obtained among 5380 total progeny (5.37% frequency). Only 33 transmitted (0.61%), mostly from the excised, antler and strap phenotypes.

In the first series (Table 1), wild type males fed EMS (0.0125M in 2% sucrose for 24 hours) were mated to "12 pl"/Cy females. The features of this series included an abundance of nick and notch  $F_1$  phenotypes which are probably heterozygous penetrance effects (more abundant at 19°C than 25°C). Nevertheless, many transmitted mutants demonstrated the high frequency of mutation of  $vg^+$  with both EMS and x-rays.

In the second series (Table 2) the EMS fed  $vg^+$  males were mated to  $cn \text{ } vg \text{ } sf$  or  $cn \text{ } vg^{E7} \text{ } sf$  females, the  $cn$  (57.5) and  $sf$  (71.5) markers reducing the amount of modifiers compared to the "12 pl" chromosome. The  $vg^{E7}$  allele, which is homozygous normal, shows an antlered phenotype in the heteroallelic  $vg^{E7}/vg$  compound. The mutagen tests were also carried out at 25°C to reduce the penetrance of  $vg$  in the  $F_1$  heterozygotes. The EMS induced mutants in this second series consisted of 35 fertile exceptions, 6 phenotypically complete (3 of which were gonadally normal and 3 were gonadal mosaics) and 29 phenotypically mosaic (25 of which did not transmit and 4 of which were gonadal mosaics). The phenotypes of the transmitted alleles included 5 strap alleles, one classical vestigial, and one allele similar to  $vg^{NO2}$  (with charring of the vestigial wings).

Vestigial resembles dumpy in (1) being highly mutable, (2) consisting of a range of allelic types, (3) arising mostly as mosaic phenotypes, and (4) transmitting only about 20% of its  $F_1$  phenotypes. It differs from dumpy (1) in giving rise to more mild than extreme induced alleles, (2) in having the opposite response to temperature (high temperature enhances mutant dumpy expression and diminishes vestigial expression; low temperature enhances vestigial expression and diminishes dumpy expression), and (3) in being more sensitive to modifiers. Successful mutagenesis studies with vestigial demand the use of warmer temperatures (25°C to 28°C) or milder alleles to act as sifters (such as the  $vg^{E7}$  allele).

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Table 2. EMS induced vestigial phenotypes using  $+ \delta \times cn \text{ } vg \text{ } sf \text{ } \varphi$  or  $+ \delta \times cn \text{ } vg^{E7} \text{ } sf \text{ } \varphi$  (25°C)

Series	(vg)	Total	%	Transmitted	%
$cn \text{ } vg \text{ } sf$	18	8,521	0.211	4	0.047
$cn \text{ } vg^{E7} \text{ } sf$	17	13,570	0.125	3	0.022

Most of the 35 allelic phenotypes were excised, antlered, or strapped. The higher temperature eliminating the nicked and notched expressions of the  $F_1$  heterozygotes.

Schärer, E. Freie Universität Berlin, Germany. Brief ecological notes on *Drosophila* collections from Berlin.

skirts of Berlin and one backyard rather located in the city. The flies were trapped in 1-liter jars filled with the four different baits, banana, potato, tomato (each fermented with baker's yeast), and fermented malt (prepared according to Lakovaara 1969). The jars were exposed on the ground about half a meter apart. The flies were collected every day about two hours before sunset.

Table 1. *Drosophila* species and total numbers of individuals per species collected during the months May until October in Berlin.

Species	♀♀	♂♂	Σ
<i>D. melanogaster</i>	3446	2450	5896
<i>D. simulans</i>	*	44*	44*
<i>D. ambigua</i>	17	30	47
<i>D. obscura</i>	56	225	281
<i>D. subobscura</i>	1001	3118	4119
<i>D. busckii</i>	917	1879	2796
<i>D. limbata</i>	1	--	1
<i>D. phalerata</i>	9	36	45
<i>D. transversa</i>	6	18	24
<i>D. littoralis</i>	14	17	31
<i>D. funebris</i>	105	108	213
<i>D. hydei</i>	5	8	13
<i>D. repleta</i>	7	11	18
<i>D. immigrans</i>	28	23	51
Total	5638	7941	13579

\*Only in October the males of *D. simulans* were differentiated from *D. melanogaster* (see text).

Table 2. Ecological distances between six collected *Drosophila* species in their food preferences.

	<u>D.mel.</u>	<u>D.amb.</u>	<u>D.obsc.</u>	<u>D.sub.</u>	<u>D.busck.</u>
<i>D. melanogaster</i>					
<i>D. ambigua</i>	0.16				
<i>D. obscura</i>	0.13	0.15			
<i>D. subobscura</i>	0.17	0.26	0.08		
<i>D. busckii</i>	0.63	0.64	0.60	0.51	
<i>D. funebris</i>	0.33	0.40	0.36	0.17	0.38

showed very little difference. As shown in Figure 3, *D. melanogaster*, *D. subobscura*, and *D. obscura* were caught on banana with a frequency of more than 60%. The fewest individuals were caught on potato and tomato, respectively. The strongest affinity to banana showed *D. ambigua* with a relative frequency of 91%. *D. busckii* and *D. funebris* showed a rather even distribution over the four offered baits.

In the period from May until October 1976 collections of *Drosophilids* were made in Berlin in order to get a survey of the different species occurring in this area. There were five collection sites, four domestic gardens in the outskirts of Berlin and one backyard rather located in the city. The flies were trapped in 1-liter jars filled with the four different baits, banana, potato, tomato (each fermented with baker's yeast), and fermented malt (prepared according to Lakovaara 1969). The jars were exposed on the ground about half a meter apart. The flies were collected every day about two hours before sunset.

14 *Drosophila* species were captured during the six months with a total of 13,579 flies (Table 1). The most common species was *D. melanogaster* with a frequency of 44%, followed by *D. subobscura* with 31%, and *D. busckii* with 20%. The various species showed a different abundance during the season. Figure 1 demonstrates that the largest numbers of *D. subobscura* specimens were trapped in May and June, whereas *D. melanogaster* and *D. busckii* individuals were rather rare during these months; *D. busckii* was most abundant in August and September, and most of the *D. melanogaster* individuals (about 65%) were caught in October.

The frequencies of the distinct species as a function of the total capture per month varied during the season. Figure 2 shows that from May until July *D. subobscura* was the most abundant of all captured species. In August most individuals caught were classified as *D. busckii*. In September and October *D. melanogaster* was the dominating species.

Data on *D. simulans* are only available for October because the first male was classified at the end of September. From that time on we differentiated the males of the two sibling species *D. simulans* and *D. melanogaster*. The mistake resulting from this non-differentiation should not be too heavy as the number of *D. simulans* males captured in October was less than 5% of the *D. melanogaster* males and as about 65% of all *D. melanogaster* flies were caught in this month. It also should be mentioned that the males of *D. obscura* could not be differentiated from possibly captured *D. bifasciata* males.

The distinct *Drosophila* species and also the individuals of one species showed different preferences for the various baits. The very most of the total captured individuals were caught on banana. The numbers of flies caught on the other three baits

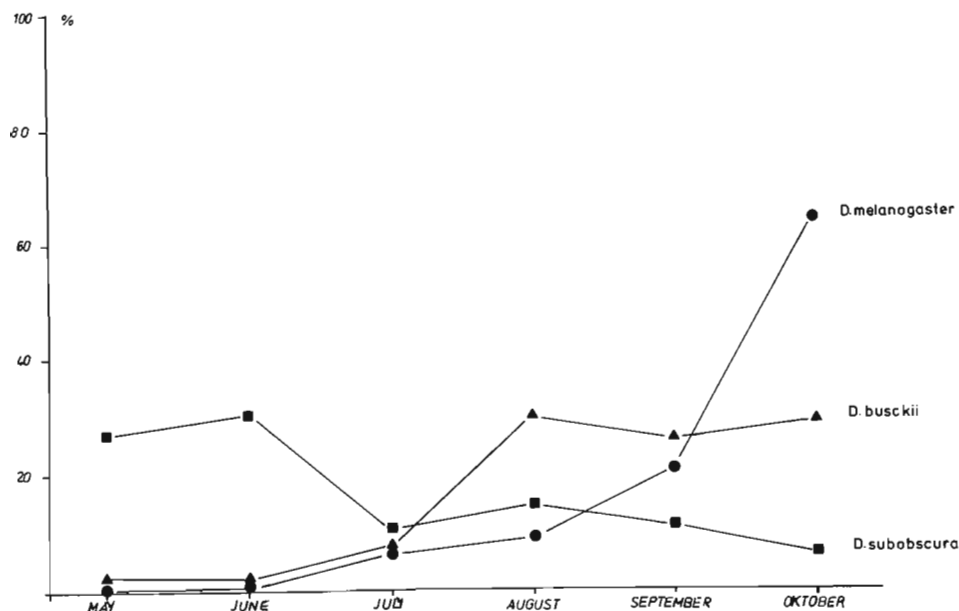


Fig. 1. Distributions of the numbers of captured individuals per *Drosophila* species in the period from May until October. (The illustrated species were the most abundant ones.)

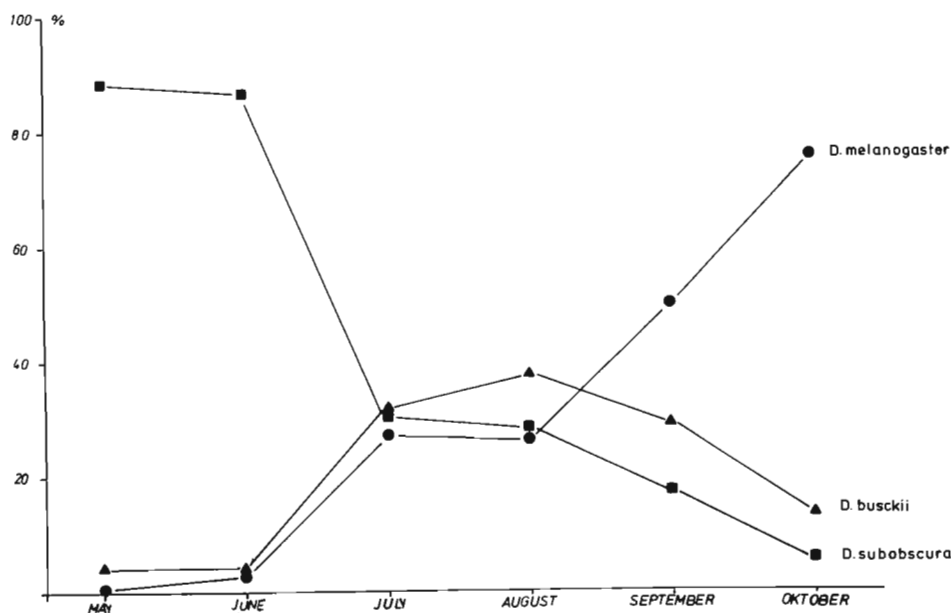


Fig. 2. Frequencies of the most abundant *Drosophila* species as a function of the total capture in the months May until October.

Fig. 3. Food preferences of six collected *Drosophila* species. Ordinate: percentages of individuals caught on banana (B), potato (K), malt (M), and tomato (T).

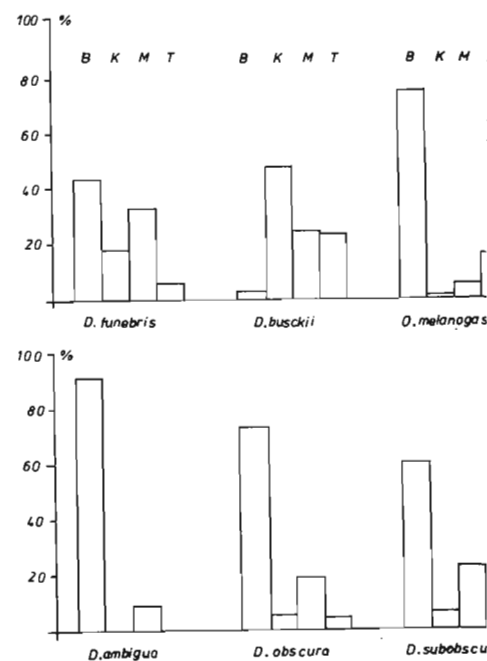
Table 2 shows the "ecological distances" in the food preferences" of the six *Drosophila* species illustrated in Fig. 3. They can be measured by the following geometric distance:

$$d_{i,j} = \sqrt{\frac{1}{2} \sum_h (p_{ih} - p_{jh})^2}$$

where  $p_{ih}$  and  $p_{jh}$  are the relative frequencies of the species  $i$  and  $j$  on the bait  $h$ .

It can be seen that the species *D. melanogaster*, *D. subobscura*, and *D. obscura* of the *sophophora*-group had the smallest distances in their food preferences. Very large distances to other species showed *D. busckii* because of its rather low frequency on banana, which the other species preferred the most.

Reference: Lako-vaara, M. 1969, DIS 44: 128.



Schuppe, N.G., T.V. Syrota and V.T. Kakpakov. Institute of General Genetics, Moscow 117312, USSR. Turnover of ribosomal RNA in the course of *D. melanogaster* development.

The question about structural changes of protein synthesizing apparatus in the course of insect development and metamorphosis remains completely obscure. To study the turnover of ribosomal RNA during the development of *D. melanogaster* larvae, 15 h old larvae were collected and placed on medium containing 50  $\mu$ Ci/ml of 2-[ $^{14}$ C]-uracil and

were grown on labelled medium for 6 h. RNA was isolated from part of these larvae and the remaining larvae were thoroughly washed and transferred to non-radioactive medium. RNA was isolated in every other 12 h after transfer of the larvae up to the white prepupal stages. To determine specific activity of ribosomal RNA, isolated RNA was fractionated by sucrose gradient centrifugation or by polyacrilamide gel electrophoresis. The structural integrity of ribosomal RNA was also studied. It was found that during the development of larvae active synthesis of ribosomal RNA occurs so that specific activity RNA decreased proportionally with increase of ribosomal RNA content per larvae. Total amount of label per larvae practically does not change.

The second stage was the study of the fate of ribosomal RNA in the course of metamorphosis. For that, larvae were labelled as above on the fifth day after oviposition. RNA was isolated just after transfer of larvae to non-radioactive medium (third instar larvae), at 24 h (white prepupal stage), at 48 h (pupal stage) and at 120 h (flies) after transfer. Data obtained show that ribosomal RNA synthesis by third instar larvae was preserved at the late stage of development. During the transition from third instar larvae to white prepupae the specific activity of RNA decreased two times due to increasing RNA content per larva, then the specific activity of RNA remained constant. RNA characteristics obtained by sucrose gradient centrifugation or gel electrophoresis do not change at these stages of development. Our data can be explained by the fact that at the stage of white prepupae active synthesis of RNA and ribosomes occurs. Thereafter synthesis of new ribosomal RNA and ribosomes is virtually absent. This indicated that protein synthesis in pupae and flies is accomplished by ribosomes synthesized at the early stages of *Drosophila* development.

Schuppe, N.G., T.V. Syrota, N.G. Nosova and V.T. Kakpakov. Institute of General Genetics, Moscow 117312, USSR. Protein-synthesizing activity of *Drosophila* ribosomes containing rRNA with hidden breaks.

A mature large subunit of *Drosophila* ribosomal RNA contains a central hidden break when RNA was isolated from whole cells. The small subunit under these conditions remains covalently continuous. At the same time when RNA was isolated from previously isolated ribosomes the breaks become plural and appear on the large

and small subunits of ribosomal RNA although ribosomes remain structurally intact (1). This allows us to study the significance of rRNA integrity within ribosomes for their functions. In this paper we present data about protein synthesizing activity of *Drosophila* ribosomes in a cell-free system. Ribosomes were isolated by the method of Wettstein et al. (2) from flies, larvae at second and third instars, eggs and established cell lines 67j25D (3).

Standard assays (final volume 0.5 ml) were incubated at 30°C and contained the following: 35 mM Tris-HCl (pH 7.6), 60 mM KCl, 5 mM MgCl<sub>2</sub>, 1 mM ATP, 0.6 mM GTP, 20 mM creatine phosphate, 15  $\mu$ g creatine phosphokinase, 4 mM dithiothreitol, 3  $\mu$ Ci of 1-[ $^{14}$ C]-phenylalanine, 0.05 mM of each of the 19 unlabelled L-amino acids, 1 mg ribosomes, 0.2 ml postribosomal supernatant (approximately 0.5 mg protein). This system was a slightly modified cell-free system reported by Ilan for *Tenebrio* pupae (4).

The kinetics of  $^{14}$ C-phenylalanine incorporation in hot acid-insoluble fraction are presented in Table 1. For comparison the ribosomes from rat liver were taken.

Table 1. Protein-synthesizing activity of *Drosophila* ribosomes in a cell-free system.\*

Time of incubation (min)	Incorporation (cpm/mg RNA)	
	- poly U	+ poly U
1.5	1250	1250
3	2900	3100
6	5600	5800
20	15080	15660
Ribosomes from rat liver (min)		
20	16160	17180

\* 100  $\mu$ g poly U were added where indicated.

Data obtained show that although ribosomal RNA within *Drosophila* ribosomes are fragmented these ribosomes not only remain structurally intact but also display sufficient activity in the cell-free system for protein synthesis. Addition of poly U to these ribosomes leads only to a small increase of phenylalanine incorporation suggesting that endogenous templates were present in the ribosomes. This result confirms previous data on the functional activity of ribosomes from animal and bacterial cells which are treated with exogenous ribonucleases (5,6). However, in the case of *Drosophila* ribosomes, we have dealt for the first time with biological activity of ribosomes containing ribosomal RNA whose structural integrity

was damaged by endogenous ribonucleases. It is important to note that results reported above were the same for ribosomes isolated from *Drosophila melanogaster* at various developmental stages as well as for ribosomes from cultured embryonic diploid cells.

References: (1) Schuppe, N.G., T.V. Syrota 1974, Dokl. Akad. Nauk SSSR 217(5):1210-1213; (2) Wettstein, F.O. et al. 1963, Nature 197:430-435; (3) Kakpakov, V.T. et al. 1969, Genetika 5:67-75; (4) Ilan, J. 1968, J. Biol. Chem. 243:5859-5866; (5) Grove, B.K. and T.C. Johnson 1973, Biochem. Biophys. Res. Comm. 55:45-51; (6) Kuechler, E. et. al. 1972, Biochim. Biophys. Acta. 277:615-627.

Smith, C.M., J.N. Thompson, jr., and R.C. Woodruff. University of Oklahoma, Norman, and Bowling Green State University, Bowling Green, Ohio. An attempt to transmit male recombination activity by non-genetic means.

A number of recent studies have suggested that male recombination (MR) and its correlated mutator activity in *Drosophila melanogaster* may be due to an infective factor (Woodruff and Thompson, 1977; Thompson and Woodruff, 1978). The most direct evidence comes from successful transmission of MR activity by injecting homogenates of an MR strain (OK1) into a control

strain (Canton S) that does not show male recombination (Sochacka and Woodruff, 1976). Attempts to transmit MR activity by feeding have given interesting, but ambiguous, results (Hellack et al., 1978). We have repeated the feeding experiments in order to test the efficiency of this hypothetical method of social transfer over a longer period of time.

We established two sets of population cages. The control cages (#1 and #2) were made from styrofoam ice chests and contained Canton-S (a non-MR strain). Each chest had spaces for 6 food tubes in which yeasted Carolina potato-based instant food was introduced into the cages. Two tubes were replaced each week. The experimental cages (Treated #1 and #2) were identical to the controls, except that 1 ml of a concentrated whole-fly homogenate of the MR strain OK1 was added to each food tube. The cages were kept in an incubator at 25°C.

The results are summarized in Table 1. A total of 23,379 progeny were scored from the four cages over a period of 27 weeks of treatment. Seven recombinants were found in the treated cages, but three were found in the control cages. Thus, although some male recombination activity may have been induced, the presence of MR activity in the control cages does not allow us to interpret these results. Indeed, MR activity in control feeding experiments has been the primary source of ambiguity in similar experiments (Slatko and Hiraizumi, unpublished; Hellack et al., 1978). Even the most generous interpretation of these data, however, shows that social transfer through flies dying on the food is not likely to be a significant ecological factor in MR transmission.



Table 1. Summaries of male recombinants in MR assays of treated and control population cages. (All MR assays were carried out by the methods outlined in Woodruff and Thompson, 1977. Total progeny assayed are shown in each column with the number of recombinants in parentheses.)

Week of Sample	Treated Cage #1	Treated Cage #2	Control Cage #1	Control Cage #2
0	1405 (1)	1187 (0)	968 (0)	1911 (0)
8	1173 (0)	1172 (0)	912 (0)	961 (0)
15	954 (3)	1377 (0)	1308 (0)	808 (0)
22	1970 (0)	1954 (0)	--	1503 (0)
27	1148 (1)	925 (2)	667 (3)	1076 (0)
Total	6650 (5)	6615 (2)	3855 (3)	6259 (0)

References: Hellack, J.J., J.N. Thompson, jr., R.C. Woodruff and B.N. Hisey 1978, *Experientia* 34: 447; Sochacka, J.H.M. and R.C. Woodruff 1976, *Nature* 262: 287-289; Thompson, J.N., jr., and R.C. Woodruff 1978, *Nature*: in press; Woodruff, R.C. and J.N. Thompson, jr., 1977, *Heredity* 38: 291-307. This work was supported by DHEW-NIEHS grant ES01439-03.

Stein, S.P. and E.A. Carlson. State Univ. of New York, Stony Brook. Mosaicism of eye color induced in mature sperm of *D. melanogaster*.

Males of  $bw^+;st$  or  $bw;st^+$  genotype were fed EMS (ethyl methane sulfonate) using an 0.0125M concentration for 24 hours. The procedure for preparation of the mutagen was that of Lewis and Bacher (DIS 43:193). These males were mated with virgin  $bw;st$  females.  $F_1$  progeny were examined for eye color mutations reflecting alterations of the  $bw^+$  or  $st^+$  alleles. These appeared in five different patterns whose frequencies are presented in Table 1. The frequency of induced eye color mutation was 0.28% for  $bw^+$  to  $bw$  (31/10,928) and 0.13% for  $st^+$  to  $st$  (9/6739). Of the 40 mutants obtained, 27 were isolated amorphs (white sectors or eyes) and 13 were hypomorphs (lemon-orange sectors or eyes). One of the 27 amorphs, however, turned lemon-orange about a week after being detected.

Table 1. Patterns of eye color mosaicism

Phenotype	Series I		Series II	Total
	$bw^+$ ↓ $bw$	$st^+$ ↓ $st$	$bw^+$ ↓ $bw$	
both eyes full mutant	2	0	4	6
one eye full mutant, one eye normal	2	0	9	11
one eye full mutant, one eye sector	1	0	2	3
both eyes sector	1	5	2	8
one eye sector, one eye normal	7	4	1	12

Each of the 40 eye color mutants was mated to  $bw;st$  flies to test for gonadal transmissibility of the induced mutant.

There were 29 mutants which produced 100 or more  $F_2$  progeny. Of these 8 were transmitted mutations, 4 involving a gonadal complete composition and 4 involving a gonadal mosaic composition. The classification of a gonadal mosaic was based on the presence of 80% or more of mutant (white or lemon) gametes. The transmissibility, 28% is similar to that found for dumpy mutations induced by EMS.

The pattern of mosaicism varied, with one eye sectors and one eye full mutants being the most common forms.

The use of the  $bw^+;st$  or  $bw;st^+$  permitted white or very light eye color sectors to be detected readily. No salt-and-pepper distribution of white and scarlet (or brown) ommatidia were found.

Table 2. Transmissibility of mutations and their relation to parental pattern

Pattern of parental eyes	Non-transmitted	Transmitted
both eyes full mutant	2	2
one eye full mutant, one eye normal	6	1
one eye full mutant, one eye sector	2	0
both eyes sector	5	1
one eye sector, one eye normal	5	4
total	20	8

The induction of single strand lesions in sperm leads to mosaics which tend to remain separated by a left-right symmetry along the anterior-posterior axis. Only a few ( $8/40 = 0.20$ ) of the mosaics represented mixed distributions of mutant and normal cells to each eye. Table 2 presents the transmitted mutations and their relation to the pattern of mosaicism in the initial mutant parent.

Supported by Biomedical Research Support Grant 431-H114G (USPHS) SUNY Stony Brook.

Tsakas, S. and E. Diamantopoulou-Panopoulou. Agricultural College of Athens. Is the "hidden heat sensitive polymorphism" (crude extract) polymorphism of the structural examined locus in all cases? Experiments with *D. subobscura*.

132 isogenic lines for the O chromosome extracted from two natural Greek populations, Parnes (P) and Crete (C), were studied. In our samples composed of the crude extract of 12 flies we tested whether the observed heat-sensitive "alleles" were new hidden alleles of the tested loci, A.O, M.E and Xdh, located on the O chromosome.

The main conclusion from this experimental work is that in our tested cases the new hidden heat-sensitive polymorphism doesn't belong solely to the tested locus but is the result of interaction among the "enzymatic products of different loci". The evidence for this is: (a) non-repeatable heat-sensitive phenotypes within some tested strains; (b) the heat-sensitive phenotypes within  $F_1$  crosses didn't give one pattern in most of the tested cases; (c) in single crosses by using null-strains and single fly analysis (new technique applied by us), we did not observe one locus Mendelian segregation within the offspring.

The report of this work has been submitted to the Biochemical Genetics magazine.

Wakimoto, B.T., R.A. Lewis and T.C. Kaufman. Indiana University, Bloomington, Indiana. Genetic analysis of the Antennapedia gene complex: mutant screen of proximal 3R, bands 84A-84B1.

In order to extend our knowledge of the genetic and functional relationships of the members of the Antennapedia gene complex (ANT-C), we have utilized a proximal 3R deficiency chromosome  $Df(3R)Scr$  in a mutant screen. This chromosome was generously provided for our use by Dr. D. Sinclair. It is deficient for bands 84A1

through 84B1. Like the previously described  $Df(3R)Antp^{Ns+R17}$  (Duncan and Kaufman; Kaufman),  $Df(3R)Scr$  is associated with a dominant reduced sex comb phenotype and fails to complement the recessive lethality of the dominant homoeotics *Msc*, *Antp* and *Antp<sup>Scx</sup>*. However,  $Df(3R)Scr$  extends more proximally than  $Df(3R)Antp^{Ns+R17}$  and exposes the proboscipedia (*pb*) locus. The recovery and characterization of mutants derived from the present screen would establish if the previous screen utilizing  $Df(3R)Antp^{Ns+R17}$  had saturated the 84B1,2 region of the ANT-C (see Lewis, R.A., this volume). Furthermore, we could extend the limit imposed by the  $Df(3R)Antp^{Ns+R17}$  chromosome to include more proximal regions including the *pb* locus.

EMS mutations were induced according to the method of Lewis and Bacher. Using a third chromosome marked with red and ebony, a total of 2,832 chromosomes were screened for visible, lethal and semi-lethal mutations exposed by  $Df(3R)Scr$ . The mutants recovered were designated by the letters Ef followed by an identification symbol. Results of inter se complementations are summarized by Fig. 1.

<i>Df(3R)Antp<sup>Ns+R17</sup></i>									
<i>Df(3R)Scr</i>									
			<i>pb</i>		<i>EbR11</i>		<i>Scr</i>	<i>Antp-Hu-Scr</i>	<i>EdR16</i>
<i>EfW5</i>	<i>EfW36</i>	<i>EfR9</i>	<i>EfW1</i>	<i>EfW4</i>	<i>XaK2</i>	<i>EfR1</i>	<i>EfW17</i>	<i>EfW10</i>	
<i>EfW9</i>			<i>EfW14</i>	<i>EfW19</i>	<i>EfW20</i>	<i>EfR3</i>	<i>EfW22</i>	<i>EfW15</i>	
<i>EfW27</i>						<i>EfW6</i>		<i>EfW24</i>	
						<i>EfW21</i>		<i>EfR4</i>	

Fig. 1. Complementation map of proximal 3R mutants derived from a screen utilizing *Df(3R)Scr*. The mutant sites *EbR11*, *XaK2* and *EdR16* were defined by the previously described *Df(3R)Antp<sup>Ns+R17</sup>* screen (see text).

The *Df(3R)Scr* screen failed to define any new lethal sites within the region that is also exposed by *Df(3R)Antp<sup>Ns+R17</sup>*. However, several new members have been added to the complementation groups originally established in the *Df(3R)Antp<sup>Ns+R17</sup>* screen. Specifically, in the *Antp<sup>Scx</sup>* complementation group, four new lethal mutations have been recovered. One of these, *EfW15*, is a new allele of *Antp<sup>Scx</sup>*. It appears to be more extreme than the original allele in producing extra sex combs on the meso- and metathoracic legs and in removing the sternopleural bristles. Other mutants derived from the screen include two new lesions in the *Scr* group. Both of these new mutations show reduced sex comb phenotypes and are entirely consistent with the earlier interpretation that the function of the *Scr<sup>+</sup>* site is to promote prothoracic leg development.

In addition, four new members of the *EbR11* complementation group and two new *pb* alleles have been recovered. The *pbEfw4* allele transforms the labial palps into distal arista and proximal leg tissue. *pbEfw19* appears to transform labial palps completely into antennal structures.

We have defined four new lethal sites exposed by the *Df(3R)Scr* chromosome. The linear order of these sites and their position with respect to the *pb* locus is arbitrary at this point. We are continuing genetic and developmental studies of these newly induced mutations. Further analyses will investigate the possibility that the new lethal sites are members of the *ANT-C* whose function is in the determination of anterior segmentation in *Drosophila*.

References: Duncan, I.W. and T.C. Kaufman 1975, *Genetics* 80:733-752; Kaufman et al. 1979, *Genetics* (submitted); Lewis and Bacher 1968, *DIS* 43:193.

Watabe, H., E. Momma and M.T. Kimura.  
Hokkaido University, Sapporo, Japan.  
Changes in drosophilid fauna at the University Botanical Garden in Sapporo, Japan.

Through the serial papers "Drosophila Survey of Hokkaido", Momma and his collaborators have revealed the distribution and abundance of Drosophilidae in Hokkaido. The authors have studied the dynamic aspects of drosophilid populations at the University Botanical Garden (UBG) in Sapporo. This preliminary report deals with

changes in the species composition of the drosophilid assemblages in this semi-natural area.

A weekly collection during the active season was made by exposing fermenting banana, using two "retainer" traps in 1975 and six from 1976 to 1978. Momma (1965) had previously surveyed the drosophilid fauna of UBG in the ten successive years from 1953 to 1962, using "open" traps. The constitutions of the relative frequencies of the assemblage are different for "retainer" and "open" traps. For example, *D. immigrans* is more frequent in the former than in the latter, while for *D. auraria* the opposite is true. Therefore it is difficult to make a quantitative comparison between the present data and Momma's. For this reason, the survey was also carried out using "open" traps in 1972 and 1977. It would be possible to compare these data with Momma's for the number of species collected and the constitutions of the populations (Beppu and Toda, 1976). Except for *D. suzukii*, *D. lutescens* and *D. immigrans*, the relative frequencies of the common species did not significantly vary between Momma's collection and 1972. Thereafter, domestic species such as *D. immigrans*, *D. melanogaster*, *D.*

*simulans* and *D. busckii* increased in number, whereas several wild species decreased, belonging mainly to the robusta species group. In and near Sapporo, *D. immigrans* had hitherto been scarce in occurrence. Moreover, *D. suzukii* and *D. lutescens*, which are also known as semi-domestic, have become more abundant. This change may be related to changes in the physical and biotic environments. As the garden is located in the central area of the city, its flora must be affected by man. For the last decade a number of high buildings have been constructed around the garden. The forests in UBG have been getting sparse and this probably necessitates floral changes.

The species *D. simulans* is notable for its distribution and abundance. In Japan it was known only in the Bonin Islands after its discovery in 1936. Recently, however, this species has been found in many localities in Japan (Watanabe and Kawanishi, 1978). In Sapporo it appeared in very small numbers in 1974 and its population size reached a maximum in 1977, making up 15.3% of the total population (in collections by "retainer"). A similar phenomenon has been reported from Colombia (Hoenigsberg, 1968) and from Egypt (Tantawy et al., 1970). In 1978 only two individuals were captured, however. This might be due to comparatively high temperatures during this summer. A further study on its population dynamics is in progress.

Table 1. Collection records of drosophilid flies from UBG.

Species	<sup>a</sup> 1953-1962	<sup>a</sup> 1972	<sup>a</sup> 1977	1975	1976	1977	<sup>b</sup> 1978
<i>D. confusa</i>	394	67	113	179	358	212	156
* <i>D. busckii</i>	9	4	4	11	7	20	22
<i>D. bifasciata</i>	1097	79	141	263	654	292	43
o <i>D. suzukii</i>	823	212	600	706	2408	2270	1011
o <i>D. lutescens</i>	4170	886	350	260	1351	493	230
* <i>D. melanogaster</i>	68	0	50	43	294	162	243
* <i>D. simulans</i>	0	0	399	12	174	2350	2
<i>D. auraria</i>	18065	1160	1672	103	174	283	44
<i>D. brachynephros</i>	3332	344	419	858	1000	1114	1281
<i>D. nigromaculata</i>	11894	1106	718	1310	2106	1266	912
<i>D. unispina</i>	208	25	30	84	122	495	188
<i>D. testacea</i>	1635	523	656	886	526	1284	386
<i>D. histrio</i>	74	59	54	313	71	619	102
* <i>D. funebris</i>	32	2	3	2	3	1	2
* <i>D. immigrans</i>	3303	1609	649	4377	6032	4007	1466
* <i>D. virilis</i>	38	0	1	1	7	6	1
<i>D. ezoana</i>	18	2	2	1	0	2	0
<i>D. sordidula</i>	199	4	39	55	190	133	7
<i>D. lacertosa</i>	950	47	16	4	4	27	33
<i>D. moriwakii</i>	11	5	0	0	3	5	4
<i>D. okadae</i>	2	0	0	0	0	0	0
* <i>D. hydei</i>	10	0	0	3	10	5	3
Others	287	227	305	342	473	345	176
Total	46619	6361	6221	9813	15967	15391	6312
No. of species	43	27	28	31	31	29	29

a: Flies were collected on two or three days near the end of each month, using "open" traps.

b: Collections were carried out from September to November.

\* Domestic

o Semi-domestic

References: Beppu, K. and M.J. Toda 1976, J. Fac. Sci. Hokkaido Univ. VI, Zool. 20:299-312; Hoenigsberg, H.F. 1968, Amer. Nat. 102:389-390; Momma, E. 1965, Jap. J. Genet. 40:275-295; Tantawy, A.O., A.M. Mourad and A.M. Masri 1970, Amer. Nat. 104:105-109; Watanabe, T.K. and M. Kawanishi 1978, Zool. Mag. 87:109-116.

Weiss, R.L. Albright College, Reading, Pennsylvania. The inhibition of fertilization in *D. melanogaster* under the influence of sodium tungstate.

D.S. Aaron (DIS, May 1977) recognized the very rapid marked onset of homosexual behavior in virgin male flies treated with sodium tungstate. The males mounted each other and engaged in pseudo-copulation. This experiment set out to test if the sodium tungstate effect was an

aphrodisiac effect or an inducer of homosexual behavior. Glass vials with impregnated (1% sucrose with  $1 \times 10^{-4}M$  sodium tungstate) glass wool were used, following Aaron's procedure. Eight virgin females and a single virgin male of *D. melanogaster* were placed in the vial overnight. A control using only 1% sucrose in the glass wool was also prepared. The following day the male was terminated and the females were placed in individual vials. After 10 days the presence or absence of larvae in each vial was noted. In the control the average number of fertilized females was 3.33 of 8 females. In the sodium tungstate trials there was no fertilization in all seven trials performed. The sodium tungstate clearly shows an effect of inhibiting fertilization between male and female *Drosophila*.

Welter, R.J. and J.F. McDonald. Iowa State University, Ames, Iowa. A search for third-chromosome ADH active loci.\*

McDonald and Ayala (1979) recently reported the presence of putative "regulatory elements" on chromosome III of *D. melanogaster* that result in the increased activity of alcohol dehydrogenase (ADH; structural locus: II, 50.1). However, at

least two alternatives to the "regulatory" model could account for their observations.

One, a noncontiguous, independent locus codes for the synthesis of a unique, but functionally related, alcohol dehydrogenase. For example, the octanol dehydrogenase (ODH; structural locus: III, 49.2), whose product ordinarily oxidizes only long-chain alcohols (Chambers et al. 1978) has mutated such that it now encodes a product that contributes (though less efficiently) to the oxidation of short-chain alcohols. Or two, the ADH structural locus itself has been transposed to chromosome III, which in combination with *Adh*<sup>+</sup>, elevates total enzyme activity (but is not dosage effected).

To test these hypotheses, we felt that a "second-site" might be uncovered if it were in combination with a functionally inoperative, second-chromosome structural locus. Two third chromosomes (3F and 4F), previously isolated from natural populations and known to increase ADH activity levels (3F, McDonald and Ayala 1978; 4F, personal observation), were combined with a second chromosome bearing an ADH-negative allele (*Adh*<sup>n2</sup>; for a complete description, see Schwartz and Sofer 1976; this chromosome, also marked with *pr*, *cn*, and *b*, was generously provided by W. Sofer). The test strains were constructed by routine manipulations: *n2/n2*; *Sb/+* X *Cy/+*; 3F(4F)/3F(4F) and the progeny intercrossed to yield *n2/n2*; 3F(4F)/3F(4F).

Starch-gel electrophoresis of whole-fly homogenates (5-10 days post eclosion) was performed according to the techniques of Ayala et al. (1972). Total ADH activity was measured according to McDonald and Avise (1976).

No ADH activity was detected in either analysis. We therefore conclude that there are no structural loci coding for a functionally active ADH or ADH-like enzyme on these third chromosomes. These results are consistent with the existence of ADH "regulatory elements" of as yet undetermined function.

References: Ayala, F., J. Pwell, M. Tracey, C. Maurao and S. Perez-Salas 1972, *Genetics* 70:113-139; Chambers, G.K., J. McDonald, M. McElfresh and F.J. Ayala 1978, *Biochem. Genet.* 16: 757-767; McDonald and Avise 1976, *Biochem. Genet.* 14:347-355; McDonald, J. and F. Ayala 1978, *Genetics* 89:371-388; Schwartz, M. and W. Sofer 1976, *Genetics* 83:125-136.

\*Journal Paper No. J-9445 of the Agriculture and Home Economics Experiment Station, Ames, Iowa. Project No. 2272.

Wicker, C. and J.R. David. National Institute of Applied Sciences (INSA) and University Claude Bernard, Villeurbanne, France. Preliminary characterization of a  $\beta$ -N-acetyl-glucosaminidase from *D. melanogaster* adults.

Numerous enzymatic activities have been studied in *Drosophila* (Dickinson and Sullivan, 1975) but, up to now, a single work has been devoted to the analysis of an enzyme capable of using the artificial substrate paranitrophenyl-N- $\beta$ -D-glucosaminide (PNAG). This enzyme was extracted from *D. hydei* larvae and considered as a chitinase (E.C. 3.2.1.29) which, associated with a

chitinase, seems to be involved in apolysis during the moulting cycle (Spindler, 1976). We report here a similar activity found in *D. melanogaster* adults but the responsible enzyme seems better described as a  $\beta$ -N-acetyl-glucosaminidase (E.C. 3.2.1.30).

*Drosophila* adults from a French population (Villeurbanne) aged 4-6 days and fed with a killed yeast medium were homogenized in 0.1 M citrate buffer (pH 5.0) and then centrifuged. The supernatant crude extract was used in this work. 0.4 ml of the enzymatic extract was added to 0.2 ml of 1.5mM PNAG. After incubating 30 minutes at 38°C, the reaction was stopped and the color developed from the released nitrophenol by adding 0.12 ml of a 1 mM solution of NaOH. The optical density was measured at 410 nm. Results are given in Figs. 1, 2 and 3.

Effects of pH were studied in the range 3.5-6.5. The response curve (Fig. 1) was very symmetric with a single optimum at 5.0 and a slow decrease toward higher values. For temperature (Fig. 2) the optimum was found at about 38°C. Inhibition of the enzymatic activity by an excess of substrate was observed (Fig. 3), the effect being more pronounced when a more concentrated enzymatic preparation was used. With low concentrations of the substrate (5 repetitions for each measure) a linear regression between 1/V and 1/S (Lineweaver and Burk plot) was observed and the Michaelis constant ( $K_m$ ) estimated at 0.73 mM.

Finally, it seemed interesting to characterize the enzymatic activity per fly or per mg of fresh weight. Averages of 30 measures gave the following values, expressed in nM of released p-nitrophenol per minute with a 0.5 mM substrate concentration. Two flies were homogenized in 1 ml of buffer, pH 5.0 and incubated 15 minutes at 38°C.

mg of fresh weight:  $2.00 \pm 0.13$  units

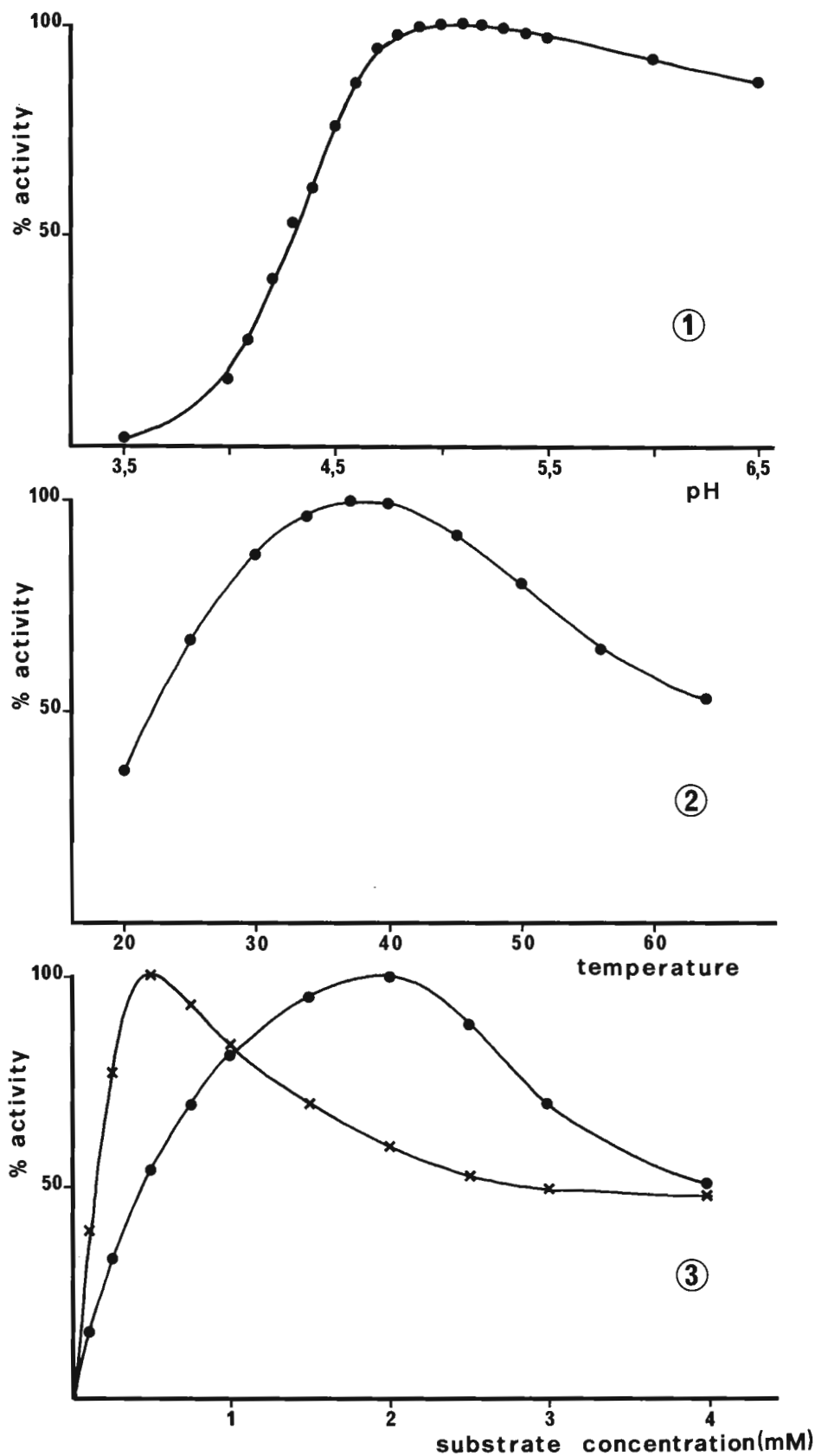
individual fly:  $2.28 \pm 0.18$  units

Acrylamide disc electrophoresis using a tris-glycine buffer pH 8.2 and naphtol-ASB-N-acetyl-glucosaminide for staining the gel showed a single enzymatic band, thus suggesting that one enzyme only was involved in this study.

The main problem arising from the above data is to decide if the enzyme of *D. melanogaster* adults could be homologous to the chitinase described by Spindler from larvae of *D. hydei*. Obviously this question deserves further investigation. Some indications, however, exist that the two enzymes are different. First, the pH optima are not the same: 5.0 versus 5.5-6.2 in *D. hydei*. The temperature optimum found in *D. melanogaster* was 38°C against 50°C in *D. hydei*. Finally, the  $K_m$  values for PNAG are very different: 0.73 versus 5.7 mM.

The existence of two different enzymes having an N-acetyl-glucosaminidase activity has been demonstrated in silkworm larvae by Kimura (1977): one is found in the moulting fluid and seems to be involved in apolysis; the second is most abundant in the hemolymph and its function is unknown. In the case of *Drosophila*, the enzyme here described in adults cannot be involved in apolysis. One possibility is that the larval and imaginal enzymes are not the same, being produced by different genes and having different functions. In that case we have to explain why the *D. hydei* larval enzyme is so much different from the adult *D. melanogaster* enzyme. A study of the larval enzyme of *D. melanogaster* is now in progress to try to answer these questions.

References: Dickinson, W.J. and D.T. Sullivan 1975, Gene enzyme systems in *Drosophila*, Springer, 163 p.; Spindler, K.D. 1976, Insect Biochem. 6:663-667; Kimura, S. 1977, Insect Biochem. 7:237-245.



Figures 1, 2 and 3: Variation of enzymatic activity in relation to pH (1), temperature (2) and substrate concentration (3). In this last case, two enzymatic preparations were used: x: low concentration, 1 fly per ml; o: high concentration, 4 flies per ml.

Zhimulev, I.F. and O.V. Ilyina. Institute of Cytology and Genetics, Novosibirsk, USSR. Localization and some characteristics of sbr in *D. melanogaster*.

sbr (1-33.4) shows "bristles small; one or more missing, particularly the postscutellars" (Lindsey and Grell 1968). Penetrance strongly depends on the temperature (see table, Nos. 1-4). The results of crosses with the various duplication

No.	Genotype	Temperature °C	Flies tested	Without one or more postscutellars (%)
1.	sbr/sbr	18	984	94 (9.6)
2.	sbr/sbr	25	675	282 (42)
3.	sbr/sbr	25	685	269 (39.2)
4.	sbr/sbr	30	168	44 (26)
5.	sbr/Df(1)v <sup>64f29</sup>	25	145	132 (93)
6.	sbr/Df(1)v <sup>L4</sup>	25	136	135 (99)
7.	sbr/Df(1)v <sup>L3</sup>	25	998	1 (0.1)
8.	sbr/Df(1)v <sup>L3</sup>	25	226	0 (0.0)
9.	sbr/Dpv <sup>+</sup> Yy <sup>+</sup>	25	940	10 (1.06)
10.	sbr/Dpv Yy <sup>+</sup>	25	936	9 (0.96)
11.	sbr/Dpv Yy <sup>+</sup>	25	274	0 (0.0)
12.	Batumi wild strain ♀♀ sbr <sup>+</sup>	25	450	5 (1.1)

and deficiency chromosomes (Lefevre 1971) allow estimates of the cytological location of sbr (see table, Nos. 5-12) within the interval 9F5-6 - 9F8-11. Genetical position is between ras (1-32.35) and 1(1)Q54 (1-32.81) (map positions from Lefevre 1971).

In the haplo condition with the "allelic" deficiencies (64f29 and L4) the following peculiarities were found:

(1) Reduced viability: 205 FM-6/sbr and only 145 Df/sbr hatched from FM-6/

Df(1)64f29 x sbr males. Df/sbr heterozygotes hatched two days later. Haplo sbr females had normal fertility. (2) High frequency of postscutellars missing: more than 93% in haplo condition compared with 42% in the homozygotes. Moreover, about half of the heterozygotes had missed all four postscutellars.

References: Lefevre, G., Jr. 1971, Genetics 67:497-513.

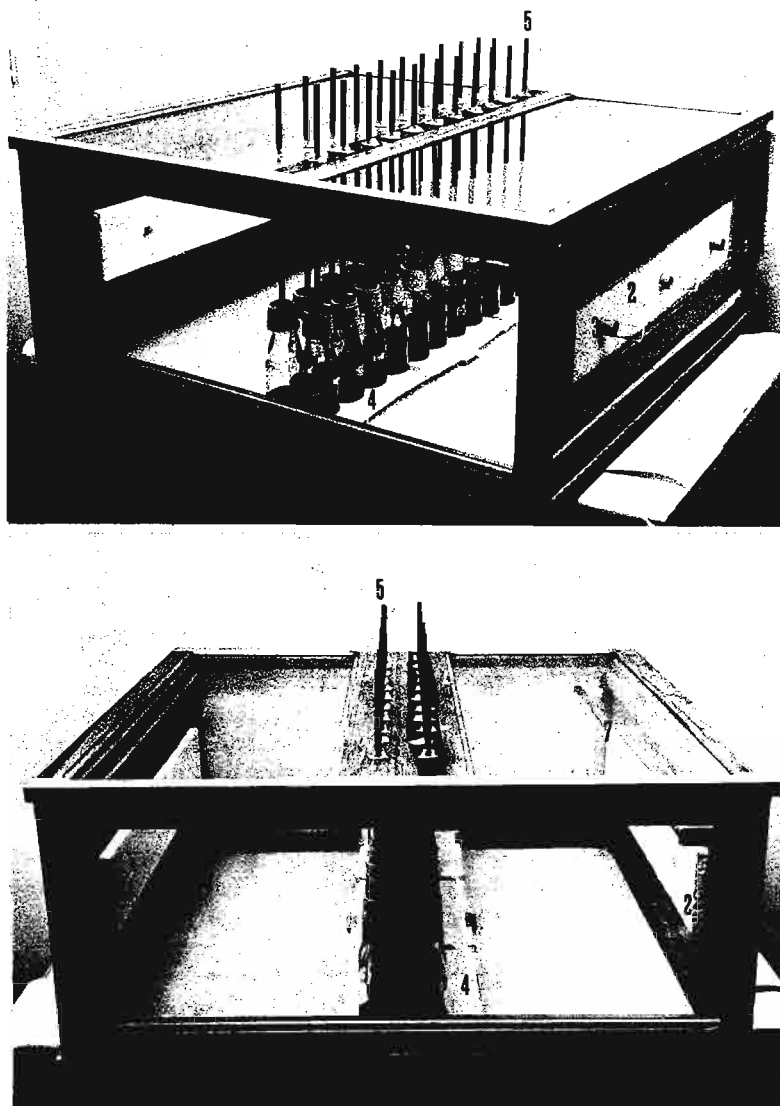
#### TECHNICAL NOTES

Bélo, M. and P.M. Lacava. Universidade Estadual Paulista "Julio Mesquita Filho". Faculdade de Ciências Agrárias e Veterinárias, Jaboticabal, SP, Brasil. Box for testing nutritional preferences (yeasts) in *Drosophila*.

This box has been used for our studies in reference to nutritional preferences in *Drosophila* species, under controlled laboratory conditions. Figures 1 and 2 show the box, which is 96 cm wide, 97 cm long, and 38 cm high, built with glass and wood.

On the sides (1) there are sheets of styrofoam (2), measuring 12 x 70 x 4 cm. In each





Figs. 1 and 2: Two views of the box for nutritional preferences. (1) Lateral wall; (2) styrofoam sheets; (3) holes; (4) map; (5) rods; (6) plastic hoods; (7) ultraviolet light.

Bock, I.R. and P.A. Parsons. La Trobe University, Bundoora, Victoria, Australia. Culture methods for species of the *Drosophila* (*Scaptodrosophila*) *coracina* group.

coarse moist sand in which to pupate; vials containing young larvae are placed without stoppers into a jar containing the sand, and the larvae ultimately leave the food vial and bury into the sand for pupation. Adults of the next generation are aspirated from the sand jar.

styrofoam sheet there are three well-spaced holes (3) to fix the tubes of flies. Inside, on the glass bottom, there is a map (4) used in order to distribute the 22 bottles (two for each kind of yeast) to avoid association of the same kinds of yeasts together. The lateral wall (1) may be removed in order to place and remove the bottles.

The 34 cm rods (5) passing through the holes in the wooden part of the top terminate with slightly flared plastic hoods (6) which are used to close the mouths of the bottles. On the upper part of this plastic hood there are two washers, to give them greater weight in adapting to the bottle's mouth. When the bottles with certain kinds of yeasts are exposed to the flies, the plastic hoods are not totally elevated, but remain partially covering the entrance to the bottles to avoid an indiscriminate rush of flies to the bait.

Within the box there is an ultraviolet light (7) of 30W (germicidae) used to sterilize the environment. In each test the 11 species of yeasts were placed separately in 0.25 liter bottles containing synthetic medium (Mittler 1952), two days before each test.

Reference: Mittler, S. 1952, *Science* 115:271-272.  
(Work supported by CNPq)

Special methods have been in use for some time in several *Drosophila* laboratories for rearing species which cannot be cultured on one of the several standard media. In particular, a number of the Hawaiian endemics can be cultured quite successfully if the larvae are given

The Australian *Drosophila* fauna is dominated by members of the subgenus *Scaptodrosophila*. Most of the latter species cannot be collected by fruit baiting and have to date proved impossible to culture, but species of the coracina group (Bock and Parsons, 1978) are attracted to fruit baits and have been cultured successfully by the method mentioned above. When ready for pupation, larvae of the coracina group species crawl to the top of their vial and "skip", landing either on the surface of the sand into which they immediately burrow, or on the side of the sand jar, in which case the process is repeated.

If a culture of one of the above group of species is maintained in a stoppered vial when the larvae are ready to pupate, most larvae crawl to the top of the vial, attempt to squeeze past or burrow into the stopper, and die; pupal integuments are formed in some cases, but the pupae are generally inviable. A small yield of adults of the next generation may be obtained from the minority of larvae remaining to pupate in the food medium itself. However, if the sand jar technique is used, a very substantial yield of adults of the next generation can be obtained.

Except for *D. coracina* itself which is Japanese, the species of the coracina group are Australian. *D. coracina* has been regarded as difficult to culture (Toda, pers. comm.), but newly-collected specimens arriving from Japan went readily into culture using the sand jar technique. *D. coracina* breeds in tree sap (Toda, 1977). The breeding sites of the Australian species are not yet known, although it seems likely from experience with collection methods that some at least may breed in rain forest fruits or fungi. Separate larval feeding and pupation sites are clearly suggested by the behavior of the final instar larvae at the point of pupation, and it is not inconceivable that the technique described above may be extended to species of other groups in which the larvae normally pupate away from the source of food.

The recipe for the larval medium that we use is 36 gm agar, 72 gm dried yeast, 108 gm raw sugar, and 24 ml of 10% nipagin in 75% ethanol. All ingredients are boiled for 5 min in 1000 ml water, then a further 1000 ml cold water with 10 ml propionic acid is added before the medium is dispensed. This medium is more suitable than one with live yeast.

We have cultured the following species with the sand jar technique: *D. coracina*, *D. lativittata*, *D. enigma*, *D. nitidithorax*, *D. specensis*, and a new species of this group recently discovered on Lord Howe Island. In some cases adding a piece of mushroom appears to facilitate oviposition, a not unexpected finding given that the last two species at least are additionally and preferentially attracted to rotted mushroom baits in the wild when given a choice of baits.

Culture temperature is important, since 18-20°C appears more suitable than 25°C, a common *D. melanogaster* culture temperature. This is predictable since many Australian rain forest species are difficult to culture at temperatures as high as 25°C, as is also true of many Hawaiian endemic species (Carson et al. 1970).

Grateful acknowledgements are due to Drs. H.L. Carson and K.Y. Kaneshiro and to Miss K. Resch for explaining the culture techniques used for Hawaiian flies. Dr. M. Toda kindly sent live specimens of *D. coracina*. Financial support was received from the Australian Research Grants Committee.

References: Bock, I.R. and P.A. Parsons 1978, *Syst. Ent.* 3:91-102; Carson, H.L., D.E. Hardy, H.T. Speith and W.S. Stone 1970, in: *Essays in Evolution and Genetics in Honor of Theodosius Dobzhansky* (M.K. Hecht and W.C. Steere, eds.), pp. 437-543 (Appleton-Century-Crofts, N.Y.); Toda, M.J. 1977, *Jap. J. Ecol.* 17:197-214.

Cobel-Geard, S.R. and H. Gay. University of Michigan, Ann Arbor. A new simplified method for the preparation of neuroblast mitotic chromosomes from *D. melanogaster*.

A number of procedures have been devised for spreading metaphase stage chromosomes of neural ganglia from *Drosophila melanogaster* third instar larvae. We wish to report a technique which has been used in our laboratory with great success to obtain large numbers of neuroblast

slide preparations. These are highly suitable for in situ hybridization experiments because they contain well-spread metaphase stages with longer, less condensed chromosome arms. Our procedure is an adaptation of a technique described by Holmquist (1975) and based on an ear-

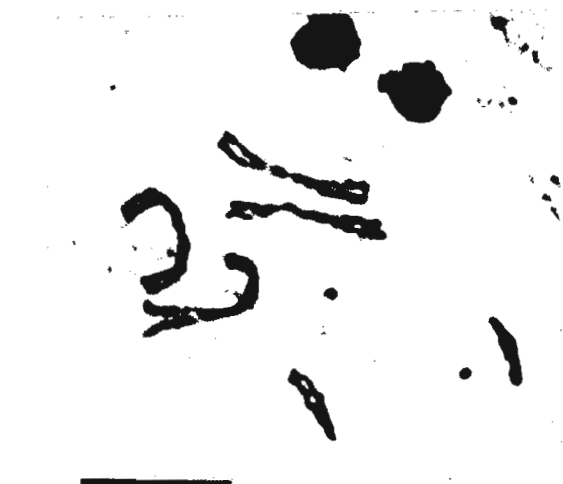


Fig. 1. Mitotic chromosomes of *D. melanogaster* for third instar neuroblast cells. X2160 Bar = 10  $\mu$

0.7  $\mu$ g/ml) and incubate at room temperature for 45 minutes.

4) During the incubation period place 20 dust-free slides (acid washed and subbed with a gelatin solution -- 0.5 g gelatin/500 cc  $H_2O$  + 0.05 g chrome alum,  $KCr(SO_4)_2$ ) on an ethanol-swabbed flat countertop. Subbed slides should be stored with dessicant in the refrigerator during hot and humid months to prevent peeling of the subbing solution. Other equipment and solutions should be assembled at this time: 3 pasteur pipettes and bulbs, 1% sodium citrate ( $Na_3C_6H_5O_7 \cdot 2H_2O$ ), 50% acetic acid, and 18 mm diameter coverslips soaking in 95% EtOH.

5) After the incubation the ganglia are transferred, one per slide, to single drops of 1% sodium citrate by again taking care to avoid damaging the tissue.

6) The neuroblast cells of the first ganglia are hypotonically swollen for 10 minutes, at which time the majority of the hypotonic solution is removed by slow suction with a pasteur pipette. Immediately a new pipette is used to add a drop of 50% acetic acid before the tissue dries out (a second person performing this task is helpful, but not necessary). This step should be carried out rapidly by moving down the line of slides in an assembly line fashion.

7) After the first specimen has been fixed for 5 minutes, an ethanol-rinsed coverslip that has been wiped clean with lint-free lens paper is placed over the drop. To spread the tissue, the coverslip is gently tapped with a small blunt tool, like the wooden handle end of a dissecting probe. Care should be taken to avoid slippage.

8) To remove excess moisture, the slide is then placed between layers of tissue paper and pressure is exerted with a rolling movement of the index finger.

9) The slide is then placed coverslip side down onto a flat surface of dry ice. Continue down the line until all slides are on the dry ice block.

10) After allowing the specimens to freeze for approximately 30 minutes, the coverslips are quickly flipped off with a scalpel blade, and the slides immersed in 100% EtOH and stored overnight at  $-40^{\circ}C$ .

These neuroblast slide preparations are then air dried before using for in situ hybridization. To facilitate microscope scanning for metaphase stages, we stain our slides with 3% Giemsa in 0.01 M sodium phosphate buffer for 20 minutes.

This research was supported by NIH grant GM 10499-15.

References: Holmquist, G. 1975, *Chromosoma* 49:333; Lewis, E.B. and L.S. Riles 1960, *DIS* 34:118; Schneider, I. 1964, *J. Exp. Zool* 156(1):91.

lier method reported by Lewis and Riles (1960). The modifications we have made substantially simplify the above procedures, requiring less individual manipulation of fragile neural ganglia and eliminating the need for a slide warmer. The resulting slides can be stored as air-dried preparations and are suitable for repeated cytochemical treatments.

The procedure for preparing 20 slides is as follows:

1) Third instar larvae should be selected from well-fed and uncrowded bottles.

2) Twenty neural ganglia (dorsal and ventral maintained as a unit) are dissected out in Becker's solution and placed in a watch glass or other small shallow dish containing 5 ml of Schneider's embryonic cell culture medium (Schneider 1964) at room temperature. The ganglia can easily be picked up in a droplet of fluid without crushing the tissue using very fine forceps with the droplet adhering between unclosed tips.

3) After 20 ganglia have been placed in the medium, add 0.04 ml of a 100  $\mu$ g/ml refrigerated stock solution of colcemid (final concentration

Crossley, S. and J. McDonald. Monash University, Clayton, Victoria, Australia. A method for permanently recording courtship song and courtship behavior simultaneously in *Drosophila*.

watching courtship and listening to amplified song at the same time (Bennet-Clark 1972) does not give this precise information because of the speed of interactions between courting flies and more importantly the inadequacy of our perceptual skills in attending to auditory and visual stimuli simultaneously.

To overcome these problems we have developed a method using video tape recording (Fig. 1). One camera (A) records the flies while another (B) records an oscilloscope trace (C) of any sounds produced. Each camera is fitted with a Macro-cosmicar V.T.R. lens ( $f = 25 \text{ mm}$ , 1:1.4). Signals from both cameras are combined using a Special Effects Generator (National Model W1 546N/A) (D) and video taped (E). Digital indications of date (month, day), time (hours, mins, secs, 1/100 secs) are also superimposed on the recorded picture by a video timer (FOR-A Co. Ltd. Model VTG-33) (F). The audio-track of the video tape is used to record the sounds from the flies. The experimenter can also record via a microphone (G) information concerning the kind of pairing and the age of the participants, prior to each recording. A Video Monitor (H) shows the visual display recorded. In addition to recording sounds on video tape we also simultaneously record fly sounds and spoken information using an audio-tape recorder (Revox, Type A77) (I). We included this in the system because we find that it is more convenient to use the audio tape recorder for analyses of, for example, rapid events because playback can be made at half speed. There is no observable difference in the quality of sound recorded on video tapes and on audio tapes.

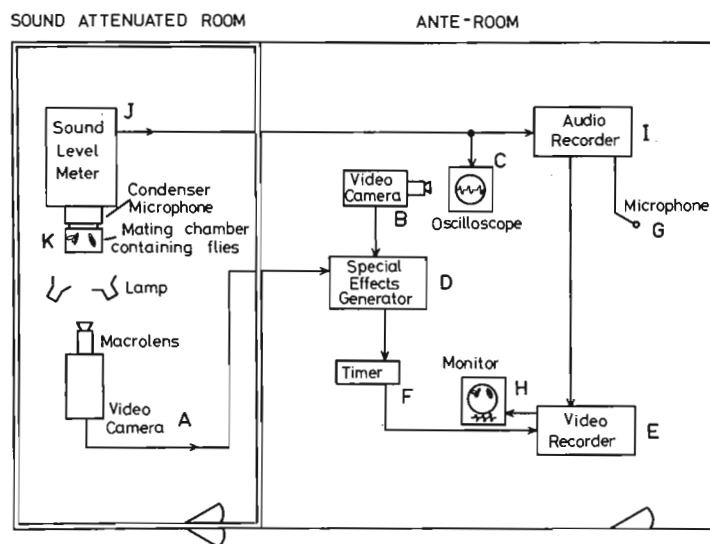


Fig. 1. Experimental set-up for recording courtship song of *Drosophila*.

floor of the mating chamber. Before recording, the original grid is removed from the microphone and the perspex chamber is pushed over the mesh grid. Flies are then introduced through one of two holes in the side of the chamber. The holes are stoppered with perspex plugs and the whole is then screwed onto the microphone base as far as it will go, which results in the mesh being separated from the diaphragm by about 1 mm.

Because of the high sensitivity of condenser microphones, we use a sound attenuated room and ante-room designed for reducing background noise. Fly song is recorded in the sound attenuated room and the experimenters and other apparatus occupy the ante-room. We have also obtained records using a ribbon microphone (Bennet-Clark 1972) which does not require quiet conditions. We prefer to use a condenser microphone, however, because this has known response

Courtship song is an important taxonomic character in *Drosophila* and provides evidence for establishing phylogenies. It is produced during wing displays which vary quantitatively and qualitatively within and between courtships. For comparisons within and between species it is necessary to know which aspects and how much of a total wing display produces sound. Simply

For recording and amplifying courtship song we use a one inch condenser microphone (Brüel and Kjaer Type 4145) attached to a sound level meter (Brüel and Kjaer Type 2203). The auditory signal from the sound level meter is led directly to the oscilloscope as well as to the audio tape recorder (I) and video tape recorder (E). The mating chamber (K) (Fig. 2) is a perspex tube one end of which is covered with glass. This tube slips over the condenser microphone up to an inner flange. This leaves a chamber 7 mm deep and 21 mm diameter between the microphone grid and the glass cover. The protection grid which is supplied with the microphone allows flies to touch the microphone diaphragm. The upper surface of this grid was therefore removed and replaced with another grid of very fine stainless steel wire mesh (48 squares per inch and with wire diameter .004 inches). This mesh grid forms the

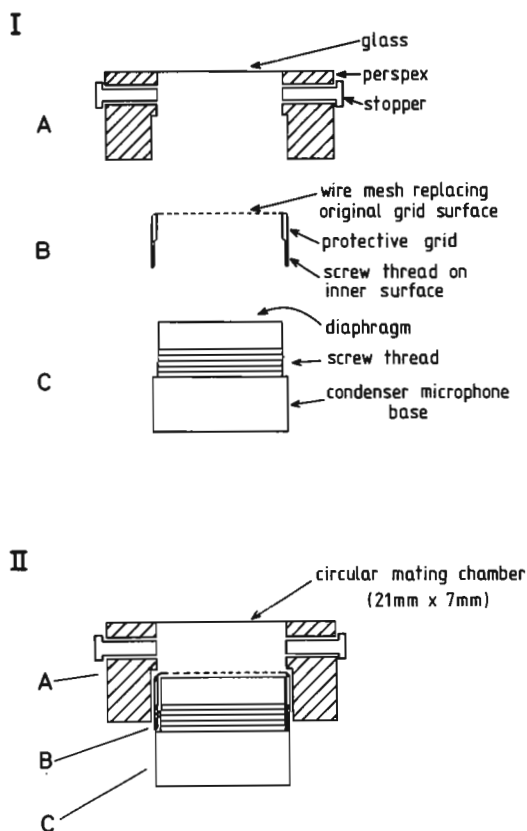


Fig. 2. Mating chamber used for housing pairs of flies during recording of courtship song. I - Mating chamber taken apart to show constituent parts; A cover, B floor, C condenser microphone. II - Mating chamber assembled.

We use photographs for examining the overall pattern of fly song during bouts of courtship and video tape analysis for detailed study, e.g., (1) matching wing angle and courtship component; (2) identifying the source of sounds not produced by wings, (3) seeing how the position of the flies relative to the microphone diaphragm influences the signal recorded. These two methods of analysis complement each other in providing information about fly song.

These techniques were designed for work with *Drosophila*. However, they are also applicable, with little modification, to analyses of the behavioral context of sound emissions in other species and so may be of interest to a wider audience.

Reference: Bennet-Clark, H.C. 1972, DIS 49:127-128.

de Jong, F. and G.E.W. Thörig. University of Utrecht, The Netherlands. A simple test for intra-electromorphic variants of alcohol dehydrogenase in *D. melanogaster*.

centrations of ethanol when little  $\text{NAD}^+$  is present. Under these conditions  $\text{Adh}^{71\text{K}}$ -enzyme still shows high activity.

Considering these differences, the following method was developed to separate  $\text{Adh}^{\text{F/F}}$ ,  $\text{Adh}^{\text{F/71K}}$  and  $\text{Adh}^{71\text{K/71K}}$  individuals, without the use of electrophoresis.

Single flies are homogenized in 0.025 ml 0.5M Tris-HCl buffer, pH 9.0, in grinding holes in ceramic tablets (at  $\pm 20^\circ\text{C}$ ). The tablets are wrapped in aluminum foil to prevent dessication of the homogenates. Then  $\text{Adh}^{\text{F}}$ -enzyme is inactivated in an incubator at  $50^\circ$  during 15-20 minutes. After this 0.2 ml of the following staining solution is added:

characteristics and gives records with improved signal to noise ratio when compared with ribbon microphone recordings.

For recording and playback we use a video tape recorder (SANYO VTR 1100SL) with slow motion (1/5 normal speed) and stop action playback facilities. The latter permits frame by frame analysis of successive still scenes at 1/50 second intervals. Measurements of wing extensions, etc., and associated song patterns are made on these still scenes. Measurements of other behavioral parameters such as courtship duration and copulation length are obtained by reading the superimposed time at the beginning and end of a given behavior. During recording, brief notes are made of audio and video tape position indicator and timer information coincident with behavior under study. This information is necessary for quick access to record segments of interest in later analyses.

A Grass Kymograph Camera (Model C4R) is used for photographing fly song recorded on audio tapes. The camera is attached to a slave oscilloscope (Tetronix Type RM 561A) which is in turn attached to a storage oscilloscope (Tetronix Type RM 564). A 100 cps calibration signal is also filmed.

Fly song is also measured from video tapes by connecting the audio output of the V.T.R. to an oscilloscope (Hewlett Packard 1201B). Bursts of the song trigger the oscilloscope and are stored on the screen. The period of the waveform is measured (in msec) from the calibrated time base of the oscilloscope. Frequencies of the wave forms are then calculated from these measurements.

Thörig et al. (1975) localized a genetic variant of *Adh* at the *Adh* locus, with the same electrophoretic mobility as Grell's  $\text{Adh}^{\text{F}}$ . However, while  $\text{Adh}^{\text{F}}$ -enzyme is rapidly inactivated at  $40^\circ\text{C}$  the new variant,  $\text{Adh}^{71\text{K}}$ , is still active. Moreover,  $\text{Adh}^{\text{F}}$ -enzyme is inhibited by high con-

460 ml 0.05M Tris-HCl buffer, pH 7.1  
 40 ml 96% ethanol  
 25 mg NAD<sup>+</sup>  
 25 mg MTT  
 Δ PMS

The tablets are wrapped again in aluminum foil and put in an incubator at 35-40°C in staining the homogenates.

After 20 minutes, the solution is heavily stained by the Adh<sup>71K</sup> individuals, whereas Adh<sup>F</sup> individuals will not show any activity. Heterozygotes can be distinguished by their intermediate staining.

Reference: Thörig, G.E.W., A.A. Schoone and W. Scharloo 1975, Biochem. Genet. 11:721.

Gupta, A.P. Harvard University, Cambridge, Massachusetts (present address: Cidade Universitaria UFRJ, Rio de Janeiro, Brazil. A new technique for collecting *Drosophila* eggs.

Generally, *Drosophila* eggs are collected by having flies oviposit in bottles on spoons or in petri dishes containing colored food medium. The well fed adults are usually allowed to oviposit 24 to 48 hours to collect an adequate egg sample. It is difficult to collect eggs of sufficient sample size from a number of crosses or

strains simultaneously. To facilitate collecting large egg samples from a number of crosses simultaneously over a short period of time, I modified the prevailing techniques with excellent results. The success of this technique depends upon starving the flies shortly before permitting the flies to oviposit.

Twenty-five to 30 pairs of newly emerged *D. pseudoobscura* were allowed to mate in vials for 5 to 10 days at 24°C under optimal rearing conditions. They were then transferred to empty half-pint milk bottles and allowed to starve for 45 to 90 minutes at room temperature. The time of starvation is determined by noting when the activity of the flies diminishes. At this time, a teaspoon containing Carpenter's medium with food coloring and covered with a thin layer of dead or live Fleischmann's yeast suspension is put into the bottle. If dead yeast is used, prepare the solution 2-4 days before use. The thin layer of yeast suspension is allowed to dry before the spoon is put into the bottle. The back of the spoon must fit firmly against the side of the bottle to prevent females ovipositing between the spoon and the bottle. These bottles are put at 24°C, and the spoons with large numbers of eggs are removed after 6-14 hours.

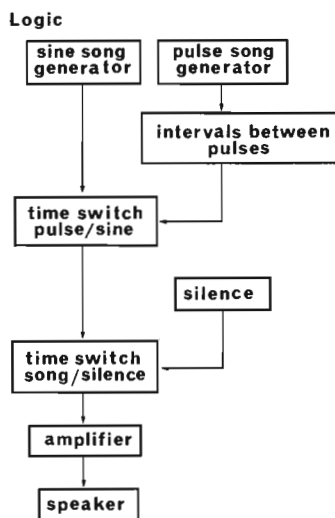
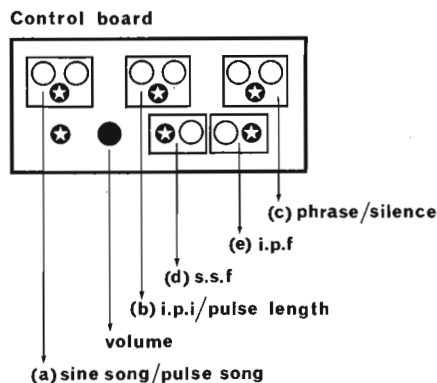
It would appear that the starved females retain their eggs until they once again are able to feed. At that time they lay their eggs in profusion. For a research project I had to collect 1800 fertile eggs for each of two parental and two F<sub>1</sub> classes, for a total of 7200 eggs, to be tested simultaneously. Using this method I had no trouble collecting the required number of eggs in a short period of time. The technique was further tested using 25 to 30 pairs of *D. melanogaster*. Approximately 1000-2000 eggs were collected in 1-3 hours. Thus, this technique is probably generally useful for collecting large numbers of eggs in a number of species in a short time period.

This work was supported by NIH grant GM 21179 to R.C. Lewontin. I thank him for the encouragement during the course of this work.

Johnson, P. and D.E. Cowling. University of Sheffield, England. A courtship song simulator for *Drosophila*.

Auditory stimuli play an important role in the sexual behavior of *D. melanogaster* and a number of other species (Bennet-Clark 1975). These take the form of a song produced by wing vibration by the male during courtship. The male

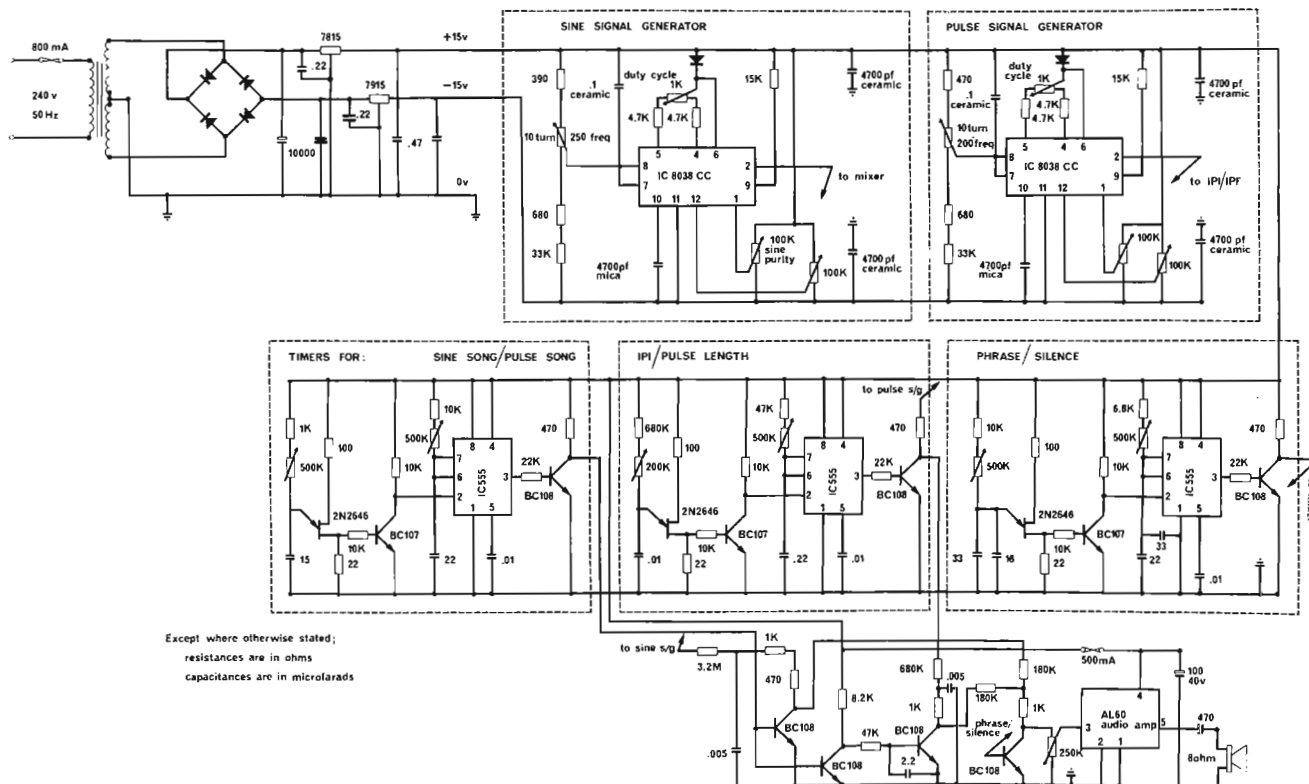
courtship songs are species specific and probably play a role in sexual isolation. The song of *D. melanogaster* consists of phrases which contain two discrete wave form elements known as sine song and pulse song (von Schilcher 1976a, 1976b; Burnet, Eastwood and Connolly 1977). A simulator which allows courtship and mating to be investigated using intact females paired with wingless males in the presence of artificial song of specified characteristics is described here.



The circuit diagram and control panel are illustrated in the figure. The controls are 20-turn potentiometers with modified multi-turn dial mechanisms. There are two signal generator circuits. The sine signal generator specifies the sine song frequency (s.s.f.) which can be varied directly by a single control (d). An adjacent coaxial socket facilitates direct measurement of the frequency. The pulse signal generator specifies the intrapulse frequency (i.p.f.) which can be varied via control (e), and monitored directly through an adjacent coaxial socket. There are three timer circuits. Timer

(i) "ipi/pulse length" controls the interpulse interval and number of cycles per pulse to give monocyclic or polycyclic pulses. These parameters are varied using the pair of controls (b) after prior setting of the ipf (control e), because altering ipf affects the number of cycles per pulse. The ipi/pulse length timer together with the two signal generators specify the basic characteristics of the song.

Timers (ii) "sine/pulse" and (iii) "phrase/silence" specify the overall phrase structure of the song via control pairs (a) and (c), respectively. Timer (ii) sets the maximum required duration of continuous pulse and continuous sine song by having two inputs (sine and pulse) but only one output which alternates between the two. The timing of the alternation is controlled by (a). This forms the input of the phrase/silence timer (iii) which specifies the phrase





length and periods of silence between phrases (interphrase interval). The output of timer (iii) is to an 8", 8 ohm loudspeaker with frequency range 30 Hz - 4 K Hz, and power handling capacity 25W r.m.s., which resonates in free air at 30 Hz. The amplitudes of the sine and pulse song are regulated by a volume control.

The apparatus is switched on for a 30 min. warming up period to stabilize before use. For each trial 10 pairs of virgin flies are placed in a 20 x 45 x 45 mm cage with Perspex sides and cotton gauze roof and floor. The cage is suspended 5 cm above the speaker cone on four wire suspensors rigidly fixed to the speaker casing.

The simulator produces courtship song with any desired combination of characteristics with respect to ssf, ipf and ipi, with phrases of a specified duration and composed of a desired mixture of sine and pulse song and interphrase periods of silence.

References: Bennet-Clark, H. 1975, *Verh. Dtsch. Zool. Ges.* 18-25; Schilcher, F. von 1976a, *Anim. Behav.* 24:18-26; Schilcher, F. von 1976b, *Anim. Behav.* 24:622-625; Burnet, B., L. Eastwood and K. Connolly 1977, *Anim. Behav.* 25:460-464.

Johnston, J.S. Texas A&M University, College Station, Texas. Hawaiian *Drosophila* with colored headlamps: a new mark-recapture technique.<sup>1</sup>

Hawaiian *Drosophila*, which pupate in moist soil, must emerge by repeatedly inflating and deflating the ptilinum. When the soil is covered with a thin layer of fluorescent pigment (5 mg/mm<sup>2</sup>) followed by a 2-4 mm layer of dry sand, the ptilinum picks up a layer of pigment. This layer of pigment gets pulled, upon emergence, into the ptilinal suture. Here, it forms a permanent fluorescent layer which cannot be rubbed off, cleaned, or removed. Under ultraviolet light, the area in and around the ptilinal suture fluoresces brightly, like a "colored headlamp". The headlamp is visible, under a dissecting scope, throughout the life of the fly. In 20 day and older flies, I found the headlamps more visible when the flies were compressed lightly between two pieces of glass.

Twenty-seven quart jars of *D. mimica* pupae, moist sand and pigment were used to test for marking effectiveness using a variety of pigments and colors (Table 1).

Table 1

Total Jars	Pigment Source	Marked Flies	Unmarked Flies
2	Tinopal <sup>2</sup>	42	0
2	Helecon <sup>3</sup>	56	0
6	Poster paint <sup>4</sup>	169	1
17	None (control)	0	410

One fly in 268 emerged without obvious markings. Emergence rate was not significantly different between marked and control jars. ( $\chi^2 = 4.9$ ,  $p > .30$ ). Marked and control flies were next divided into 5 and 10 vials respectively and scored for survival and marking effectiveness. Fifteen survival curves were determined for the flies in the 5 marked and 10 control vials. These survival curves were then compared using an analysis of covariance. The adjusted mean survival for marked and control flies were 64 and 63 days, respectively.

The survival rate for marked flies was not significantly different from that of control flies ( $p = .28$ ).

For field studies, poster paint and sand could be spread directly onto the soil at selected sites. To obtain larger numbers and provide more experimental control, field caught flies could be used to produce a population of pupae in the laboratory. Then, jars, pupae, sand and pigment could be set out at selected sites in the field. Preliminary studies suggest that pigment can be changed daily by scraping away the old sand and color, and replacing with a new color and new sand. This would permit distinct daily marking. After emergence, the numbers marked can be measured by filling the jar (or tray) with water. Empty pupae cases float and can be counted.

(1) Supported in part by NSF Grant DEB-76-19879, to J.S. Johnston; (2) Tinopal SFG, CIBA-GEIGY Corp., Ardsley, NY 10502; (3) Helecon Fluorescent Pigments, U.S. Radium Corp., Hackettstown, NJ 07840; (4) Blacklight Pigments, Barnett Mfg. Co. Inc., Los Angeles, CA 90035.



Keltner, L. University of Oregon, Eugene, Oregon. Low temperature enhancement of fluorescence as an aid in chromosome banding.

to the ground state decreases, resulting in proportionally greater photon emission. Excitation of fluorogenic substances with radiation in the ultraviolet range thus elicits enhanced fluorescence when the temperature is lowered. Photometric studies of two variations of the acridine molecule, quinacrine mustard dihydrochloride and acridine orange, demonstrate significant increases in fluorescent behavior over the lowering temperature range of 0 to  $-190^{\circ}\text{C}$ , with the dyes both in aqueous solutions and in solutions containing calf thymus DNA.

Preliminary results show enhanced resolution of fluorescent banding in chromosomes treated with quite low concentrations of quinacrine mustard dihydrochloride and examined at temperatures in the  $-100$  to  $-190^{\circ}$  region. The stability of the fluorochrome may also be accentuated. Dr. David Wagner of the University of Oregon has also found striking increases in fluorescence at low temperatures of chromatographic preparations of lichen extracts, assisting in identification of difficult materials.

Further studies are underway to determine precise fluorochrome concentrations and optimum temperatures for maximum resolution, and to overcome difficulties encountered with apparatus at the required temperatures.

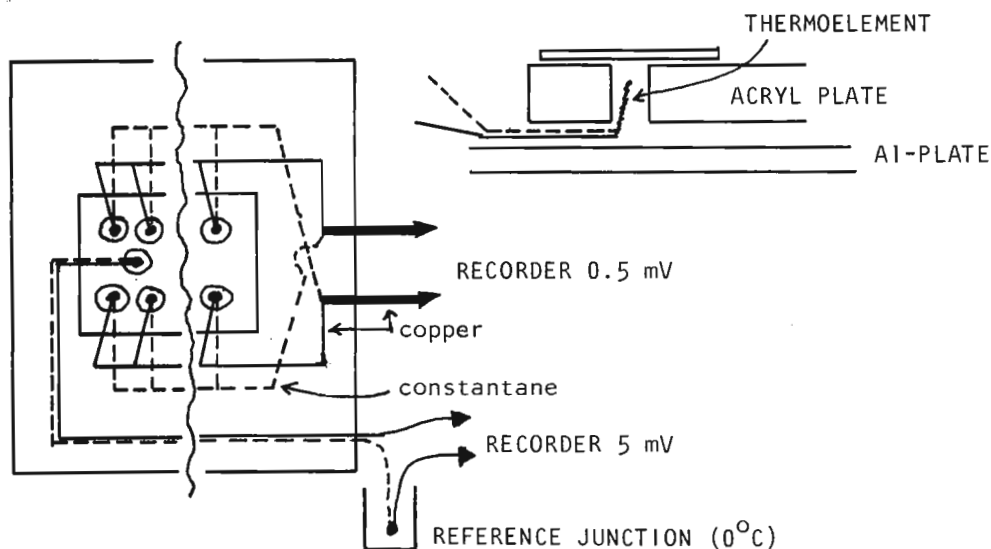
A technique developed by the author for investigation of inorganic crystalline solids is being used to enhance resolution of fluorochrome stained chromosome preparations.

As the relative temperature drops, the probability of non-radiative decay of excited atoms

Lumme, J. University of Oulu, Finland. An efficient instrument to measure freezing points of insects.

A widely used technique to study one aspect of cold resistance (which may play a role in winter resistance) is to determine the freezing point (also called supercooling point) of a biological specimen. Conventionally, the

freezing point of one individual is measured by one channel of a recorder in one run. Gradual cooling of an object is followed by a small thermoelement, and the freezing is seen as a sudden increase in the temperature. Here I present a modification which significantly increases the working capacity of a recorder, an expensive essential part of most methods. My purpose is to point out that our knowledge on the winter resistance of *Drosophila* species is really poor, and it can be improved significantly with easy and cheap methods.



Thermoelements with very small tips are soldered from 2 x 0.1 mm copper-constantane double wire. Ten (or even more) of them are connected in parallel, and two such groups are connected oppositely parallel via a thicker copper cable to a 0.5 mV recording channel. The voltage of this circuit approximates zero in all temperatures, until a fly (or pupa) in contact with one of the thermoelement tips freezes. At

this moment, a short peak is seen in a slowly running (0.5 mm/min) recording paper. The peak is either negative or positive depending on the group, which the frozen fly belongs to. The individuals within groups cannot be identified. This circuit does not measure the temperature, but a separate sensor must be built into the device. This will occupy one channel of a recor-

der, if the temperature is not under independent control. Anyway, the freezing points in two experimental groups, both of them containing 10 or more individuals, can be measured simultaneously by one channel of the recorder.

Some technical solutions in the instrument constructed for *Drosophila* are presented in the figure. I drilled 2 x 10 holes ( $\varnothing$  3 mm) in a 5 mm acryl plate. The small thermoelement tips were inserted into the holes from the bottom, and this system was mounted tightly on an aluminum plate, which is to equalize temperatures in the holes. A separate thermoelement was inserted into a similar hole to measure the actual temperature of the system. The temperature was recorded by the second channel (5 mV) of Kipp & Zonen BD9 two-channel recorder. The whole measuring unit was placed into an insulating styrofoam box, and regular, constantly cold (-35°C) freezer was used to cool this package. Using this primitive temperature regulator the cooling rate is rather repeatable, and it can be varied by modifying the insulation. This instrument has been used for measuring the freezing points of adults, pupae, and 3rd instar larvae of *Drosophila* species. The contact of individuals with the thermoelements is usually good enough without any cementing.

References: Asahina, E. 1969, *Adv. Insect Physiol.* 6:1-49; Crumpacker, D.W., J. Pyati and L. Ehrman 1977, *Evol. Biol.* 10:437-469; Hudson, J.E. 1978, *Can. J. Zool.* 56:1697-1709.

Mahowald, A.P. Indiana University, Bloomington, Indiana. Improved method for dissecting late ovarian stages.

A number of procedures have been devised for isolating sufficient quantities of larval and adult tissues for molecular studies (Zweidler and Cohen 1971; Petri, Wyman and Kafatos 1976). Unfortunately, both adult fat body and ovarian

follicles undergo the "heat shock" response following isolation and culture (Petri, Wyman and Hessikoff 1977; Spradling and Mahowald 1979). In order to avoid this response, it is necessary to carry out labeling *in vivo*. We have recently developed a rapid method for accumulating large numbers of individual ovarian follicles from flies which have received injections of radio-labeled precursors. For the most part this has obviated the need for mass isolation techniques.

The flies are lightly anesthetized and submerged individually in buffered ringer's solution. While holding the fly on his back with one forceps, a small hole is made in the cuticle at the level of the 5th to 6th sternite. The tip of the watchmaker's forceps is pushed into the posterior tip of the exposed ovary. The object is to open the ovarian sheath at a level where the individual ovarioles enter a common chamber.

Immediately, follicles are extruded from individual ovarioles, and these emerge through the opening in the abdominal wall. With the aid of gentle teasing of the original hole in the ovary, it is possible to obtain extrusion of all stage 10 to stage 14 egg chambers. As they are extruded from the ovariole sheath, each egg chamber breaks free of the more anterior chamber so that little dissection is needed.

After the ovarian follicles of one ovary have been exhausted, the same procedure can be carried out on the second ovary. After both ovaries have been exhausted of late ovarian stages, the ovaries can now be removed and any final dissection carried out. With practice, most of the flies can be induced to extrude their eggs. In addition to the greater speed, the follicles are undamaged and clean of any adhering debris.

We have little information concerning the mechanism of this release. Since the ovary must remain *in situ* for success, a reasonable assumption is some neural response is involved. In some insects neurosecretory cells appear to be involved in ovulation and oviposition (Maddrell 1974). We have recently found neurosecretory neuron endings in both the oviduct and the surrounding muscular sheaths. It is possible that these nerve endings are involved in ovulation in *Drosophila*.

References: Maddrell, S.H.P. 1974, in: *Insect Neurobiology* (ed. J.E. Treherne), Amer. Elsevier, NY, pp. 307-558; Petri, W.H., A.R. Wyman and S. Hessikoff 1977, *DIS* 52:80; Petri, W.H., A.R. Wyman and F.C. Kafatos 1976, *Develop. Biol.* 49:185-199; Spradling, A.C. and A.P. Mahowald 1979, *Cell* 16:589-598; Zweidler, A. and L.H. Cohen 1971, *J. Cell Biol.* 51:240-248.

Loukas, M. and C.B. Krimbas. Agricultural College of Athens, Greece. Isozyme techniques in *D. subobscura*.

The electrophoretic techniques for the detection of 26 polymorphic genes in *D. subobscura* are given here. All electrophoretic assays were performed on horizontal starch gel electrophoresis (starch from Connaught Med. Lab., Toronto).

The following table gives the buffer solutions used for gel preparation, for filling the electrode compartments and for developing the gels. Also given are the substrates, dyes, coenzymes, coupling enzymes and inorganics added to the staining solutions.

#### Key to the symbols used:

A:	0.25 M TRIS, pH 8.6
A1:	1/5 dilution of buffer A
A2:	4/5 dilution of buffer A
B:	0.687 M TRIS + 0.157 M citric acid, pH 8.0
C:	0.135 M TRIS + 0.045 M citric acid, pH 7.0
C1:	1/3.3 dilution of buffer C
C2:	1/15 dilution of buffer C
D:	0.18 M TRIS + 0.1 M boric acid + 0.004 M EDTA.2Na, pH 8.6
D1:	1/4 dilution of buffer D
E:	0.1 M TRIS + 0.1 M maleic acid + 0.01 M EDTA.2Na + 0.001 M MgCl <sub>2</sub> , pH 7.4
E1:	1/10 dilution of buffer E
F:	0.05 M TRIS + 0.05 M maleic acid, pH 7.4
F1:	1/5 dilution of buffer F
G:	0.05 M TRIS + 0.007 M citric acid, pH 8.6
H:	0.76 M boric acid + 0.2 M LiOH, pH 8.2
H1:	1/4 dilution of buffer H
I:	0.076 M TRIS + 0.005 M citric acid, pH 8.7
J:	0.3 M boric acid + 0.062 M NaOH, pH 8.4
K:	0.017 M Na <sub>2</sub> HPO <sub>4</sub> + 0.049 M KH <sub>2</sub> PO <sub>4</sub> , pH 6.4
L:	0.023 M citric acid + 0.053 M Na <sub>2</sub> HPO <sub>4</sub> , pH 5.2
M:	0.023 M citric acid + 0.06 M Na <sub>2</sub> HPO <sub>4</sub> , pH 6.1
N:	0.005 M citric acid + 0.076 M TRIS + 0.005 M MgCl <sub>2</sub> , pH 8.7
O:	0.087 M TRIS + 0.0087 M boric acid + 0.001 M EDTA.2Na, pH 9.0
P:	0.1 M TRIS - HCl, pH 8.5
Q:	0.1 M TRIS - HCl, pH 8.0
R:	0.1 M TRIS - HCl, pH 7.5
S:	0.025 M Na <sub>2</sub> HPO <sub>4</sub> , pH 7.6
T:	0.05 M NaOH, pH 5.2 (with acetic acid)
U:	0.1 M TRIS - HCl, pH 7.1

#### Abbreviations used:

Est:	esterase
Lap:	leucine aminopeptidase
Odh:	octanol dehydrogenase
Idh:	isocitric dehydrogenase
Ao:	aldehyde oxidase
Xdh:	xanthine dehydrogenase
G-6-PD:	glucose-6-phosphate dehydrogenase
6-PGD:	6-phosphogluconate dehydrogenase
ME:	malic enzyme
Pgm:	phosphoglucomutase
α-GPD:	α-glycerophosphate dehydrogenase
Mdh:	malate dehydrogenase
Pept:	peptidase
Hk:	hexokinase
Phi:	phosphoexose isomerase
Aph:	alkaline phosphatase
Acph:	acid phosphatase
Adh:	alcohol dehydrogenase
Ald:	aldolase
Fum:	fumarase
GA-3-PD:	glyceraldehyde-3-phosphate dehydrogenase
DCIP:	2,6-dichlorophenolindophenol
NBT:	nitro blue tetrazolium
PMS:	phenazine methosulfate
NAD:	nicotinamide adenine dinucleotide
NADP:	nicotinamide adenine dinucleotide phosphate
ATP:	adenosine triphosphate

Genetical information concerning the above mentioned enzyme polymorphisms, the localization of the corresponding genes and mapping data are given by Loukas et al. (*J. Heredity*, 70:17-26).

## Enzyme Techniques

Allozyme assays, bridge and gel buffers and constituents of enzyme stains are indicated.

Enzyme	Gel buffer	Bridge staining solution	Substrate(s)	Dyes	Coenzymes	Coupling enzymes	Inorganics
Est-3	9G : 1H	H1	K	{ $\alpha$ -naphthyl acetate }{ $\beta$ -naphthyl acetate	Fast blue BB	-	Mg <sup>++</sup> , Mn <sup>++</sup>
Est-5 & Est-7	9G : 1H	H1	K	$\alpha$ -naphthyl acetate	Fast blue RR	-	Mg <sup>++</sup> , Mn <sup>++</sup>
Est-9	9G : 1H	H1	M	{ $\alpha$ -naphthyl acetate }{1-leucyl- $\beta$ -naphthylamide	{Fast blue RR (Black K)	-	Mg <sup>++</sup> , Mn <sup>++</sup>
Lap	9G : 1H	H1	L	1-leucyl- $\beta$ -naphthylamide	Black K	-	Mg <sup>++</sup> , Mn <sup>++</sup>
Odh	I	J	P	n-octanol	NBT, PMS	NAD	-
Diaphorase (*)	I	J	Q	NADH	DCIP	-	-
Idh	I	B	Q	Isocitric acid	NBT, PMS	NADP	Mn <sup>++</sup>
Ao	Al	A	P	Benzaldehyde	NBT, PMS	-	-
Xdh	Al	A	R	Hypoxanthine	NBT, PMS	NAD	-
G-6-PD	Al	A2	R	Glucose-6-phosphate	NBT, PMS	NADP	-
6-PGD	Al	A2	P	6-phosphogluconate	NBT, PMS	NADP	-
ME	El	E	P	Malic acid	NBT, PMS	NADP	-
Pgm	El	E	Q	Glucose-1-phosphate (G-1, 6-DP)	NBT, PMS	NADP	Mg <sup>++</sup>
$\alpha$ -GPD	El	E	P	$\alpha$ -glycerophosphate	NBT, PMS	NAD	-
Mdh	El	E	Q	Malic acid	NBT, PMS	NAD	-
Pept-1 & Pept-3 (*)	D1	D	S	L-leucyl-L-tyrosine	3-amino-9 ethyl carbazole	-	-
Hk-1	D1	D	P	Glucose, ATP	NBT, PMS	NADP	Mg <sup>++</sup>
Phi (*)	D1	D	Q	Fructose-6-phosphate	NBT, PMS	NADP	Mg <sup>++</sup>
Aph	N	J	N	$\alpha$ -naphthyl phosphate	Fast blue RR	-	Mg <sup>++</sup> , Mn <sup>++</sup>
Acph	C1	C	T	$\alpha$ -naphthyl phosphate	{Fast blue RR (Black K)	-	Mg <sup>++</sup> , Mn <sup>++</sup>
Adh	N	J	P	Isopropanol	NBT, PMS	NAD	-
Ald	C2	C	U	Fructose-1, 6-diphosphate	NBT, PMS	NAD	-
Fum	O	O	R	Fumaric acid	NBT, PMS	NAD	-
ATPase	F1	F	F1	ATP	(Ammonium) sulfate	-	Ca <sup>++</sup> , Pb <sup>++</sup> , Mg <sup>++</sup> , K <sup>+</sup> , Na <sup>+</sup>

(\*) Staining is performed by agar overlay.

Majumdar, D. and A.S. Mukherjee. University of Calcutta, India. Culture of *Drosophila* salivary glands.

In an attempt to establish a purely synthetic culture medium for *Drosophila* salivary glands different media have been tested using ingredients in different proportions. Our main object was to increase the polytenization of sal-

ivary gland chromosomes in culture, beyond its limit in the larvae and to study the control of DNA and RNA synthesis in vitro.

Salivary glands (L3) of *D. melanogaster* and *D. hydei* were first cultured in Schneider's medium with a little modification (SM). This medium designed primarily for cell lines did not prove suitable for polytene tissue. On the basis of the composition of different insect media we designed one for salivary glands and modified it further. In the modified medium, M II, we changed the proportions of different salts and added pentose sugars. The concentrations of amino acids, serum and yeast hydrolysate in SM appeared to be too high for salivary cells. Some of these might have hindered the penetrance of labeled precursors into the cells, bursting of membranes and proper spreading of the chromosomes. In M II, proportions of amino acids were minimized; organic acids were deleted; the undefined components, serum and yeast hydrolysate, were replaced by vitamins; and different nucleotide precursors were added. In this medium, glands are maintained in good condition till 100 h. In squash preparations, about 70% of the nuclei are well spread and the bands, interbands and puffs are clearly discernible (Fig. 1). Polytene chromosomes after 72 h of culture were compared with 0 h control chromosomes and

have been found to be significantly increased in width. However, in M II, the nucleolar Fuelgen-positive granules normally dispersed throughout (even at 72 h; Fig. 2) are found to be compact in a central, deeply-stained mass after 96 h of culture (Fig. 3).

Attempts to label the chromosomes

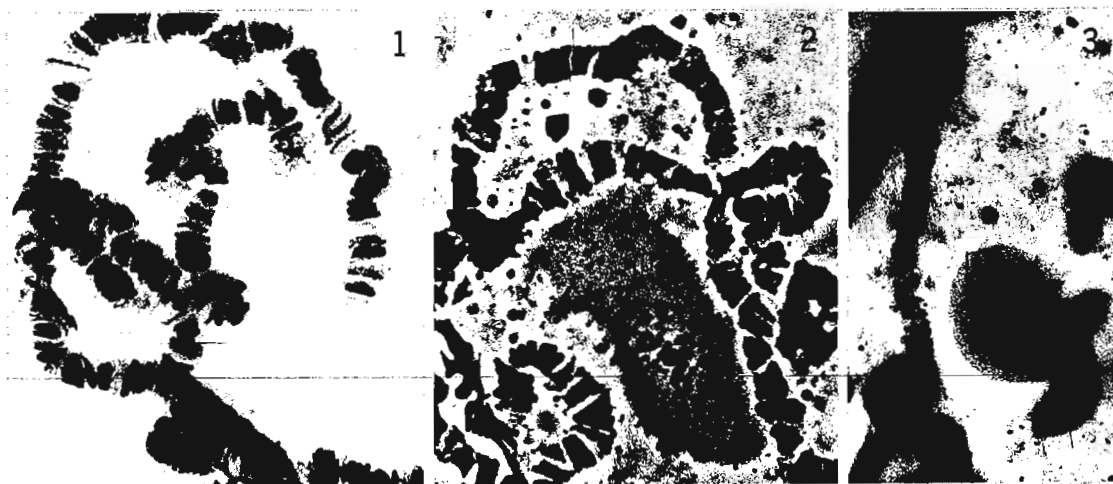


Fig. 1. A nucleus showing part of the chromosome complement after 72 h of salivary gland culture in M II.

Fig. 2. The nucleolus (nu) after 72 h of salivary gland culture in M II.

Fig. 3. The nucleolus (nu) after 96 h of salivary gland culture in M II.

cultured in Medium II resulted in partial success. Low, dispersed  $^3\text{H}$ -TdR labeling of 96 h cultured chromosomes could not be specified as representing any particular replicative phase. It may either be due to penetrance of only a small number of isotopes into the nuclei or due to slowing down of the replicative process in vitro.

We have recently started with a third medium (M III). Different chemicals known to interfere with DNA synthesis have been left out or minimized in amount. Adenine HCl has been reduced to 0.3 mg/l and adenylic acid has been eliminated. Cholesterol, tween and glutathione, and n-pagin (formerly added to avoid fungal infection) have also been left out. Overall concentrations of amino acids have been kept between SM and M II; only cystine has been deleted. Glucose and ATP have been increased to enhance the energy level. Lastly, serum and yeast hydrolysate have been incorporated in low amount in spite of using simple vitamins.

Amount of different components used in different media (mg/l)

Components	SM	M II	M III	Components	SM	M II	M III
NaCl	2100	6800	6800	KCl	1600	1100	1100
NaHCO <sub>3</sub>	400	--	--	CaCl <sub>2</sub>	600	400	400
NaH <sub>2</sub> PO <sub>4</sub> , 2H <sub>2</sub> O	--	150	150	MgSO <sub>4</sub> , 6H <sub>2</sub> O	700	1390	1390
CH <sub>3</sub> COONa	--	84	84	MgCl <sub>2</sub> , 6H <sub>2</sub> O	--	1390	1390
Fe(NO <sub>3</sub> ) <sub>2</sub> , 9H <sub>2</sub> O	--	0.72	0.72	Tryptophan	100	20	100
KH <sub>2</sub> PO <sub>4</sub>	450	--	--	Tyrosine	500	40	200
Na <sub>2</sub> HPO <sub>4</sub>	700	--	--	Valine	300	50	100
Deoxyribose	--	0.5	0.5	Malate	100	--	--
Ribose	--	0.5	0.5	α-ketoglut.	200	--	--
Sucrose	--	16000	--	Succinate	100	--	--
Glucose	2000	1000	3000	Fumarate	100	--	--
Trehalose	2000	--	2000	Vit. A acetate	--	0.1	--
Alanine	500	70	100	Menaphthene	--	0.01	--
Arginine	400	70	100	Ascorb. acid	--	0.05	--
Asp. acid	400	95	100	Riboflavine	--	0.01	--
Cystine	100	20	--	Ca pantothe.	--	0.01	--
Cysteine	60	0.1	60	Pyridox. HCl	--	0.025	--
Glut. acid	800	150	150	P.A.B.A.	--	0.05	--
Glutamine	1800	100	500	Folic acid	--	0.01	--
Glycine	250	50	100	Nicot. acid	--	0.025	--
Histidine	400	245	250	Inositol	--	0.05	--
H. proline	--	10	10	Biotin	--	0.01	--
Isoleucine	150	40	100	Choline Cl	--	0.05	--
Leucine	150	120	120	Aneurine HCl	--	0.01	--
Lysine	1650	70	500	Calciferol	--	0.1	--
Methionine	800	30	300	α-tocoph. P	--	0.01	--
β-alanine	--	50	--	Adenine HCl	--	5.1	0.3
Proline	1700	40	500	Guanine HCl	--	0.3	0.3
Serine	250	135	150	Xanthine	--	0.3	--
Threonine	350	60	100	Hypoxanthine	--	0.3	--
Thymine	--	0.3	0.3	Br. thymol blue	10	--	10
Uracil	--	0.3	0.3	Glutathione	--	0.05	--
Cytosine	--	--	0.3	FBS	10-15%	--	0.5-1%
Adenylic acid	--	0.2	--	Yeast hydrol.	2000	--	10
ATP	--	5	7	Nipagin	5	5	--
Cholesterol	--	2	--	Streptomycin	100	100	100
Tween	--	5	--	Penicillin G	200,000	200,000	200,000
Phenol red	--	10	--		units	units	units

Yeast hydrolysate was obtained as a gift from Dr. Herbert Oberlander.  
The work has been supported by a C.S.I.R. Scheme awarded to A.S.M.

Milanović, M. and W.W. Doane. Arizona State University, Tempe, Arizona. Affinity chromatographic purification of amylase from *Drosophila* species.

We are using an improved method for the purification of α-amylase from crude extracts of *D. melanogaster* and *D. hydei*. This simple, rapid and highly efficient method employs the affinity chromatographic technique of Vretblad (1974) as modified by Silvanovich and Hill (1976). Amy-

lase is retained on a column of cyclohepta-amylose (CHA) epoxy Sepharose 6B. Contaminating proteins are washed through the column in an elution buffer and the amylase is selectively removed from the column by means of a CHA-containing elution buffer.

When crude extracts of larvae or flies are passed through the CHA affinity column, the amylase subsequently eluted from the column appears pure on the basis of disc electrophoresis and staining with Coomassie blue and/or starch-iodine. The CHA column thus represents a con-

siderable improvement over the previously used glycogen precipitation method of Loyter and Schram as modified for *Drosophila* by Doane et al. (1975). Application of the latter method results in the co-precipitation of several contaminating proteins which require an additional electrophoretic step for removal. Another disadvantage of the glycogen precipitation method, which is avoided by use of the CHA column, is the difficulty encountered in removing all of the dextrans bound to the purified amylase by autodigestion.

Steps for the purification of *Drosophila* amylases by CHA affinity chromatography follow. Further details of the method and its application are being prepared for publication (Milanović and Doane).

#### I. Preparation of Crude Amylase Extracts

1. Homogenize larvae or flies in the same buffer used for washing the affinity column (step II, 5, d) at a weight:volume ratio of about 1:3. Add PTU to saturation.
2. Homogenates should be frozen/thawed and autodigested at 25°C for 2-3 hours to release additional amylase from cell debris and native glycogen. Store homogenates at -20°C or chill on ice and proceed.
3. Centrifuge homogenates at 17,000 rpm (34,800 g on Sorvall RC5 refrigerated centrifuge) for 20 minutes. Decant or pipette supernatant through glass wool to remove lipids and repeat centrifugation until the crude extract is clear.

#### II. Coupling of Cyclohepta-amylose (CHA) to Sepharose

1. Place 4.0 g of epoxy-activated Sepharose 6B (Pharmacia; Sigma) in a beaker and add about 25 ml of redistilled, demineralized H<sub>2</sub>O. Stir.
2. Transfer gel slurry to sintered glass filter funnel and wash with redistilled H<sub>2</sub>O (400 ml) for 1 hour by applying suction.
3. Wash gel with 25 ml of 0.1 M NaOH solution to remove excess liquid.
4. Transfer gel to a solution of 300 mg of CHA (Sigma) in 12 ml of 0.1 M NaOH; shake for about 19 hours in a water bath at 45°C.
5. Following incubation, wash gel on a glass filter with:
  - a. 100 ml of redistilled water, 30 minutes;
  - b. 200 ml of glucose solution (25 mg/ml), 30 minutes;
  - c. 100 ml of redistilled water, 30 minutes;
  - d. 400 ml of 0.05 M phosphate buffer, 2 hours. The phosphate buffer, pH 6.9, contains: KH<sub>2</sub>PO<sub>4</sub> (3.03 g/l), Na<sub>2</sub>HPO<sub>4</sub> (3.96 g/l), NaCl (0.41 g/l), and CaCl<sub>2</sub> (0.111 g/l).

#### III. Purification of Amylase

1. Set up column (e.g. Pharmacia column K 16/20). It should be very clean.
2. Rinse column with 0.05 M phosphate buffer.
3. Add CHA-Sepharose slurry and allow it to settle.
4. Wash column several times with starting buffer.
5. Run crude amylase extract through column and wash column immediately with 100 ml of phosphate buffer, adjusting flow rate to 25 ml/hr. (2 ml samples work well.)
6. Elute amylase off column with CHA-buffer (8 mg of CHA/ml phosphate buffer), 100 ml.
7. Collect 5 ml fractions (approximately 50 fractions), monitoring for protein at A<sub>280</sub> on spectrophotometer. This takes about 8 hours.
8. Pool fractions containing amylase (typically fractions #20 or #30 to #45±).
9. Dialyze in redistilled H<sub>2</sub>O 16-20 hours at room temperature.
10. Concentrate and/or lyophilize.

The time required for Steps III, 9 and 10 can be reduced to a few hours by use of Sartorius Membranfilter collodion bags (SM 132 00) to remove or replace the elution buffer and concentrate the purified amylase. For prolonged storage of the pure enzyme at -20°C, 0.05 M Tris-HCl buffer, pH 7.4, containing 0.003 M CaCl<sub>2</sub> proved best for retaining enzyme activity (L.G. Treat, unpubl.). Lyophilization results in some loss of activity.

This work was supported by NIH grant GM-25255 to W.W. Doane. We thank Drs. Peter Hjorth and R.C. Karn for initial advice.

References: Doane, W.W., I. Abraham, M.M. Kolar, R.E. Martenson and G.E. Deibler 1975, in: *Isozymes*, vol. IV (C.L. Markert, ed.) p. 585, Acad. Press, NY; Silvanovich, M.P. and R.D. Hill 1976, *Analyt. Chem.* 73:430; Vretblad, P. 1974, *FEBS Letters* 47:86.

Mittler, S., K. Schroeder and D. Wilson.  
Northern Illinois University, DeKalb,  
Illinois. *Aspergillus flavus* producer of  
mutagen and carcinogen aflatoxin can con-  
tamine laboratory cornmeal-molasses-  
agar media.

An *Aspergillus* mold was isolated from one of our vials that contained cornmeal-molasses-agar media. The *Aspergillus* was suspected to be *A. flavus* for it had colonies from close-textured to rather loose depending on stalk length and was light yellow that darkened to green but did not turn brown. Since we had been conducting a mutagenesis experiment, we worried whether the

*A. flavus* would influence our results. The mold was identified by Northern Regional Laboratory of the U.S. Department of Agriculture at Peoria, Illinois as *Aspergillus flavus*, but was a strain that when tested did not produce aflatoxin, a potent mutagen and carcinogen.

The *Drosophila* media that we used was 1000 ml of H<sub>2</sub>O, 17 g of agar, 40 g cornmeal, 40 g brewers yeast, 11 g rolled oats, 40 ml molasses, and 40 ml dark corn syrup and 5 ml of propionic acid. Was the 5 ml of propionic acid/liter insufficient to prevent the growth of *A. flavus*? We tested the growth of the mold on four small batches of food with different concentrations of propionic acid. One, the control, had no propionic acid and the *A. flavus* did grow on it. However, on the other three batches of food in which the propionic acid of 2.5 ml, 5 ml and 6 ml/liter was thoroughly mixed when added, there was no growth of the mold. Evidently the propionic acid was not thoroughly distributed when the media was prepared in which the mold *A. flavus* was identified. The Department of Agriculture maintains a monitoring service for the detection of aflatoxin in crops, for *A. flavus* is widely spread in the United States and grows on cereals and grains. *A. flavus* can also contaminate *Drosophila* media.

Nöthiger, R. and C. Labhart. University  
of Zurich, Switzerland. A self-amplifying  
system for mass collection of unferti-  
lized eggs.

In order to study oogenesis with biochemical techniques it is necessary to have available large masses of unfertilized eggs. It is very tedious to collect vast numbers of virgins, and furthermore, such uninseminated females lay eggs only at a very low rate. The first obstacle

can be circumvented by using "virginizer" stocks whereby temperature-sensitive lethals are especially useful (ref. 1). We have devised and successfully tested a system which greatly facilitates mass collection of unfertilized eggs. After a single collection of 10 to 20 virgin females, the system will produce, within two generations, some 10<sup>5</sup> females and an almost equal number of XO male sibs. The latter guarantee the excitement of copulation and the transfer of "sex peptide" (ref. 2) which greatly stimulates and enhances the production of unfertilized eggs. The system makes use of three stocks that can be maintained without special care. The crosses are as follows:

1. Select 10 to 20 virgin females from a pn stock, and mate them to a few males from a ca K-pn stock. Due to the lethal interaction between pn and K-pn (ref. 3), only females will survive whereby each of the pn mothers can easily produce some 100 daughters within a few days;

2. The virgin daughters from cross #1 are mated to attached-XY males from any C(1)/XY/0 stock, whereby a C(1) chromosome with a temperature-sensitive lethal can eliminate the need for selecting by hand the XY males. One male per 4-6 females is sufficient. This cross produces large numbers of females and XO males (25% fewer males than females due to the pn K-pn interaction). These are now transferred into population cages which contain a number of petri dishes with standard food. By exchanging these food dishes large masses of unfertilized eggs may be collected at short time intervals. The dishes may be frozen in toto until the desired number of eggs has been accumulated. The eggs are washed off the dishes, collected and rinsed in a narrow-meshed nylon cloth.

The frequency of non-disjunction leading to XY males is negligible, and since the sexual activity of XO and XY males is equal, the contamination by fertilized eggs remains far below 0.1%. Supported by the "Julius Klaus-Stiftung", Zurich.

References: 1) Polan, M.L. et al. 1973, J. Cell Biol. 56:580; 2) Chen, P.S. 1971, in: Biochemical Aspects of Insect Development, S. Karger, p. 124; 3) Sturtevant, A.H. 1956, Genetics 41:118.



Molnár, I. and I. Kiss. Institute of Genetics, Biological Research Center of the Hungarian Academy of Sciences, Szeged, Hungary. An easy method for rearing axenic *Drosophila* lines.

larva to larva appeared in the same culture. Instead, we maintain the lines under constant axenic conditions, and start the cultures with eggs collected from germ-free animals. The following method can produce large numbers of relatively synchronous animals with minimal effort.

The axenic lines were originally initiated from surface-sterilized eggs (7 min in  $\text{CaOCl}_2$  solution saturated at room temperature, followed by rinsing 3 times in sterile distilled water). Once established, the line is maintained by serial transfers of the egg-laying axenic adults.

**Medium:**

$\text{CaCl}_2$	0.54 g
cornmeal	126 g
wet baker's yeast	220 g
agar	8.9 g
water	ad 1000 ml

The agar is cooked in 3/4 l of water until it melts; the rest of the ingredients are mixed up in 1/4 l of water, added to the agar solution and cooked for another 5 min. Then the medium is distributed in 15 ml aliquots into large test tubes (20 mm x 200 mm) closed with tight plugs made of paper-tissue.

Contamination of the test tube wall with medium should be avoided because the flies can stick to it. The media are sterilized by autoclaving at 121°C (2.1 at) for 30 min and then allowed to solidify in a slant agar form. Fresh media are kept at 25°C for a week before use; any contamination by bacteria, yeast or molds can be easily recognized and selected out during this time. Before use, a sterile

strip of rough filter paper is placed onto the surface of the medium which sucks up the condensed water and prevents the flies' sticking to the surface (Fig. 1). The strip should not touch the plug, otherwise the plug becomes wet and contamination can easily get through it. The axenic larvae develop in this medium normally and start to make puparium at about 120 h after egg-laying at 25°C.

**Transfer of flies:** All the operations are made under a laminar-flow sterile hood. After shaking off the flies gently, the plug is removed from the test tube and the opening flamed. The test tube can be left open for minutes after this because the flies cannot escape through the hot opening until it cools down again. The flies are collected from the tube by sucking them up into a sterile Pasteur-pipette attached to a pump (Fig. 2); they cannot escape out of the pipette until the sucking is on. The medium should not be touched with the tip of the pipette because its inner wall will be

Fig. 1

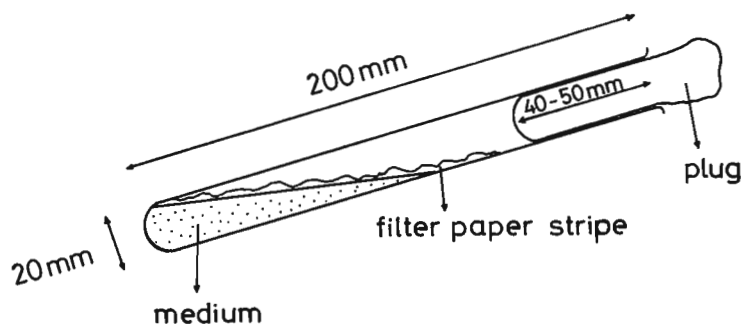
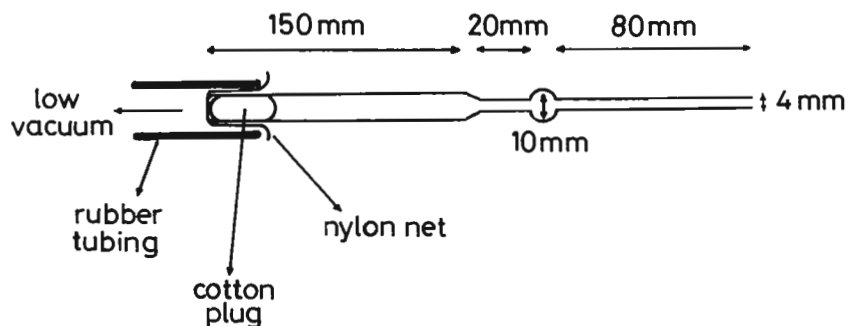


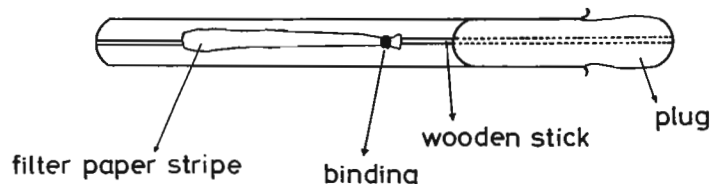
Fig. 2



contaminated and the flies stick to it. Then the sucking is stopped by detaching the rubber tubing and the pipette is put into a new test tube with fresh medium. (The opening of the new

tube has also been flamed.) Holding the tube in a vertical position, the flies are shaken out of the pipette by knocking its tip against the wall.

Fig. 3



Collection of timed eggs: For this purpose, we use an empty test tube, the plug of which has an inserted wooden stick reaching down to the end of the tube. There is a strip of rough filter paper bound to and lying along the stick (Fig. 3). The tube and the insert are sterilized together, then the upper side of the filter paper strip is covered with a few drops of melted, sterile medium. When it has solidified, the plug with the insert is put into another empty, dry test tube into which the egg-laying flies have been trans-

ferred. The flies will lay their eggs onto the strip. The test tube is kept in the dark and the plug with the insert changed for new ones at convenient times, under sterile conditions. The paper strip with the eggs is cut off the stick and placed onto the surface of a sterile slant medium.

Sterility probes are not necessary to make for each subculture. However, it is advisable to avoid mixing the flies from different tubes and to separate, by marking, the tubes with eggs from different fly populations. In the case of any contamination, this helps in identifying and separating the possibly contaminated cultures. For checking yeast contamination, a small sample of the medium or a few flies are suspended in 5 ml of sterile broth (0.5 g yeast extract and 1 g glucose in 100 ml tap water, autoclaved) in a test tube and incubated at 29°C. Yeast growing in the settlements can be seen in 3 days after inoculation.

Parente, A. and R. Arking. Wayne State University, Detroit, Michigan. A convenient method of collecting moderately large numbers of eggs from *D. melanogaster*.

Previous techniques for collecting moderately large numbers of synchronous eggs have been tedious because they usually require large numbers of adults maintained in a population cage. We wish to present an alternative technique for collecting variable numbers (500-5000 eggs/collection) of highly synchronously developing em-

bryos. Peak egg production in *Drosophila* occurs between 5-15 days after eclosion. To maintain this high level of efficiency it is essential to have an adequate supply of matured females available at all times.

In our laboratory this is accomplished by raising *Drosophila* in a rotational sequence under optimal conditions. Newly eclosed adults are placed on food and aged for 4 days at 22°C. On the 5th day, the adults are ready to be used for egg collection. This is done by placing 150 females and 75-100 males in a wide mouth glass bottle, the mouth of which is covered by a scored, yeasted, agar plate. The bottle is then inverted and placed in a totally dark incubator free from disturbances. The females are allowed to lay eggs, and at the end of the desired time periods the old plates are removed and new ones put in their places. The eggs may then be left on the agar plates to incubate for some predetermined time period or may be washed off the agar with a stream of water into a funnel containing an appropriately sized wire mesh. Adult females older than 10 days are discarded or used for stock and replaced by a new supply of mature females.

Table 1.

Collection period	No. females per bottle	No. eggs per plate		No. eggs per female per hr.	Percent synchrony	Percent abnormal*
		Range	Mean			
30 min.	120	116-204	164	2.73	100	0
1 hr.	120	775-915	840	7.00	100	5-6
2 hr.	120	1000-2000	1500	6.25	81	10

\*As measured 1-3 hours after collection.

Table 1 summarizes some of our experiences with this technique, showing the averaged results from three separate samples, each of which involved a 1 hr. pre-collection followed by an egg collection of the indicated time. It is evident that the production of eggs peaks at 1 hr. and appears to slowly decline thereafter. If the pre-collection has been sufficient, then the percentage of synchronously developing embryos stays very high and only decreases after 2 hours. The percentage of abnormally developing embryos also increases as collection time increases but never exceeds 10%. If synchronously developing embryos are not an important consideration, one can use longer egg laying periods (Table 2) and obtain much larger

Table 2.

Hours	Collection	No. females per bottle	No. eggs per plate		No. eggs per female per hr.
			Range	Mean	
0-2	Pre-collection	120	2000-4000	3000	13
2-4	Collection #1	120	1000-2000	1500	7
4-6	Collection #2	120	500-1000	750	4

numbers of eggs per plate. It can be seen, however, that egg production per female decreases linearly to very low levels by 6 hours. There-

fore, females are not pushed for more than 4-6 hours of continuous collections. In our hands, then, the optimal collection time is one hour, which will produce a high level of synchrony, minimal numbers of abnormal embryos and a moderately large number of eggs per plate.

In a 4 hour day it is, theoretically, possible to collect 20,000-40,000 eggs excluding the pre-collection. In practice, this technique has been able to produce a minimum of 28,000 embryos in an 8-day period. This figure is a minimum estimate since it does not include an approximately equal number of eggs which were discarded as surplus. These collections were done using anywhere from 8-15 collection bottles which held the adults obtained from 15-30 food bottles. This gave us an average yield of about 3,500 eggs per day, a figure which is in general agreement with the data contained in Table 1. As a Drosophilist may know, egg production is variable and there have been times when we have obtained little or no eggs; conversely, there have been times when we have obtained 40,000 eggs in one collection. In summary, we have devised a technique which is as productive as a population cage, and which can produce a moderately large number of eggs using a smaller number of adults, and which is more responsive to varying daily needs for embryos.

Pasic, T.R. and H. Nickla. Creighton University, Omaha, Nebraska. A BASIC program for construction of fate maps.

A computer program in BASIC has been developed to aid in the construction of blastula "fate maps". Early mitotic misdivisions in the embryo can be induced by an unstable ring X-chromosome. This results in two clones of nuclei ran-

domly oriented with respect to the blastula cortex and differing in X-chromosome complement. When the nuclei migrate and become fixed to the cortex, a random plane of division divides the blastula. The probability of a plane of division falling between any two imaginal discs increases as the distance between two imaginal discs increases (Hotta and Benzer 1972).

Adult mosaics can be scored for any number of surface landmarks on the basis of tissue type (1 = male, 2 = female, 3 = mixed). Each scoring is entered into a data table at the location (A,B) where A is a unique integer for each fly (1,2,3, . . .) and B represents each landmark in coded form (1 = eye, 2 = wing, etc.). The computer effects a two-by-two pairing of landmarks and counts the number of times that the two scorings are different. This sum represents the number of times a plane of division fell between the imaginal discs. The sum is converted into a percentage and prints it as the distance (in Sturts) between each pair of imaginal discs. (Note: The total number of pairings is  $(N^2 - N)/2$  where N is the total number of landmarks.)

Other subroutines in the program include the distance between homologous landmarks; total percent male tissue, percent male tissue at each landmark and percent male tissue of each fly. The program was tested using data on 196 mosaics having 39 surface landmarks. The program is available upon request.

Reference: Hotta, Y. and S. Benzer 1972, Nature 240:527.

Sawicki, J.A. and W.W. Doane, University of California School of Medicine, San Francisco and Arizona State University, Tempe, Arizona. Preparation of "small" polytene chromosomes for cytological analysis.

The small size of polytene chromosomes in larval tissues such as the midgut and fat body of *D. melanogaster* precludes their cytological analysis for banding and puffing patterns by standard light microscopic techniques. To overcome this difficulty, we used two different approaches to study midgut polytene chromosomes of late third instar larvae: (1) Scanning Electron Microscopy

(SEM), and (2) light microscopy coupled with genetically increased levels of polyteny.

**SEM Approach.** The SEM procedure we adopted is a modification of the procedure described by Mitchell and Lipps (1975). Banding patterns and "puffs" displayed by midgut chromosomes prepared by the following procedure could be satisfactorily compared with those displayed by salivary gland chromosomes which were prepared by either SEM or standard light microscopic methods.

1. Dissect tissue (e.g. three midguts) in *Drosophila* Ringer's solution (Ephrussi and Beadle 1936).
2. Transfer tissue to 50  $\mu$ l of 45% acetic acid and rinse 3 times with the acid.
3. Replace acid with 20  $\mu$ l of a 0.2% solution of orcein (natural) in a 1:1 mixture of lactic acid (85%):glacial acetic acid (v/v). Let tissue stand at room temperature for 1-3 hours in staining mixture.
4. Add 50  $\mu$ l of 45% acetic acid.
5. Suck mixture containing tissue in and out of a small, siliconized glass needle such as a dispopipette flame-drawn to a fine tip.
6. Allow mixture to settle for a few minutes and remove surface layer containing cytoplasmic material with a siliconized dispopipette.
7. Add a small amount of 45% acetic acid to the nuclei remaining and quickly transfer to a very small volume (about 1-2  $\mu$ l) containing 20-30 nuclei to a siliconized slide. Cover immediately with a "subbed" coverslip (10 x 10 mm) on which 5  $\mu$ l of the 0.2% lacto-orcein stain has been dispensed in a circle of small drops. For subsequent identification, mark the side of the coverslip not in contact with the sample by a small dab of Kodak 910 cement colored blue with Azure B Bromide.
8. Spread chromosomes by gently tapping coverslip with a pencil eraser and then pressing with thumb.
9. Study the spread chromosomes with a phase contrast microscope and make an outline diagram of the relative positions of the chromosomes on the coverslip. Chip one corner of the coverslip for reference purposes and photograph particularly good spreads. To prevent distortion of the chromosome preparation due to drying out, limit the time for this entire step to less than one hour.
10. Remove coverslip by supercooling it in liquid nitrogen and flipping it off with a razor blade.
11. Transfer coverslip to 100% ethanol immediately, before ice crystals can form.
12. Let coverslip stand in 100% ethanol for about 20 minutes to dehydrate preparation.
13. Transfer coverslip to amyl acetate and leave it there for 1-3 hours.
14. Critical point dry preparation.
15. Examine coverslip with a light microscope to ensure chromosome spreads are still there. Place a small dab of DAG 154<sup>®</sup> (colloidal graphite in iso-propanol) next to each spread on the sample side; this non-conducting substance acts as a marker in the SEM.
16. Coat with gold/palladium as soon as possible after critical point drying so that samples do not absorb moisture.
17. View in SEM (45 $\times$ ).

**Genetic Approach.** To increase polyteny of midgut chromosomes, two stocks, each carrying a different allele of the giant locus (*gt* 1-0.9), were used to generate third instar larvae heterozygous for the alleles *gt*<sup>1</sup> and *gt*<sup>X11</sup> (Kaufman 1972). One stock, *gt*<sup>1</sup> *wa*, is homozygous viable for the *gt*<sup>1</sup> allele. The other stock, *y*<sup>sc</sup> *gt*<sup>X11</sup>/FM6, is homozygous lethal for the *gt*<sup>X11</sup> allele, which is maintained over the FM6 balancer chromosome.

Under suitable rearing conditions, the polytene chromosomes of the anterior midgut and the posterior midgut from late third instar larvae of the genotype *gt*<sup>1</sup>/*gt*<sup>X11</sup> proved to be large enough to permit accurate banding and puffing designations (Sawicki and Doane, in preparation). *gt*<sup>X11</sup>/FM6 newly emerged females were mated to newly emerged *gt*<sup>1</sup>/Y males. After an initial egg-laying period of two days, the flies were transferred daily to fresh culture bottles for 10

days and reared at 18°C. Larvae that appeared 9-10 days after egg-laying were discarded. The genotype of these larvae was either  $gt^1/FM6$ ,  $F6/F6$ , or  $F6/Y$ . The desired  $gt^1/gt^{X11}$  third instar larvae climb the walls of the food bottle 15-17 days after egg-laying.

A rich food medium was used for rearing flies and larvae which was either sugar-based or starch-based. The sugar medium contained 10% (w/v) anhydrous dextrose (J.T. Baker Chemical Co.), while the starch food contained 5% (w/v) cornstarch (ICN Pharmaceuticals, Inc.). Both media also contained 15% (w/v) Brewer's yeast, 1.2% (w/v) agar, and 1 part buffered propionic acid (Lewis 1960) plus 5,000 Units each of penicillin-streptomycin (Grand Islands) for every 100 parts of food medium.

Chromosome preparations from the anterior and posterior midgut regions of single  $gt^1/gt^{X11}$  third instar larvae were prepared for examination with the light microscope and compared with salivary gland preparations. Larvae were dissected in *Drosophila* Ringer's solution and the tissue was fixed immediately for three minutes in 3:1 ethanol:glacial acetic acid. After transferring the tissue to 45% glacial acetic acid for one minute, midgut preparations were stained for 2-1/2 minutes in 2% lacto-aceto-orcin while the staining time for salivary glands was 1-1/2 minutes. Chromosomes were spread in the usual manner.

While either of the above approaches was satisfactory for banding and puffing analysis of midgut polytene chromosomes, the genetic approach proved the least time consuming. It may be assumed that the combined application of both approaches to "small" polytene chromosomes would improve the resolution of banding patterns beyond that observed by using either approach alone.

This work was performed at Yale University and supported by NIH grant GM-18729 awarded to W.W. Doane.

References: Ephrussi, B. and G.W. Beadle 1936, *Am. Nat.* 70:218; Kaufman, T.C. 1972, *Genetics* 71:s28; Lewis, E.B. 1960, *DIS* 34:117; Mitchell, H.K. and L.S. Lipps 1975, *Biochem. Genet.* 13:585.

Shearn, A. Johns Hopkins University,  
Baltimore, Maryland. Reintroduction of  
 $y^+$  onto a TM3 chromosome.

Some time ago, I planned an experiment which depended on using the  $y^+$  marker which is supposed to be on TM3 balancer chromosomes. However, none of the TM3 chromosomes tested had  $y^+$ .

Therefore, I set out to reintroduce it onto a TM3 which carried Sb and Ser. Females with the genotype  $y;Dp(1:3)sc^{J4},y^+ / y;TM, ri pP sep bx^{34e} Sb e^s Ser$  were treated with 4000r of  $\gamma$ -irradiation and mated to  $y;+/-$  + males. Progeny which were  $y^+$ , Sb and Ser were selected and tested further. In this way we recovered several TM3 chromosomes containing  $y^+$ . They are reasonably stable because the unirradiated recombination frequency of  $y^+$  with TM3 is 0.000. A stock with such a  $y^+$  containing TM3 chromosome is available for distribution (see stocklist, DIS 54).

Takamura, T. Tokyo Metropolitan University, Tokyo, Japan. Multi-roomed, multi-purpose chamber for *Drosophila*.

A simple multi-roomed chamber for observation, or other purposes, was designed using lattice-like frames (Fig. 1). The chamber consists of



Fig. 1. Upper left: the larger lattice with medium; lower left: the smaller one without medium; right: three parts dissolved.

three parts. The bottom plexiglass tray is 5mm deep and can be filled with *Drosophila* medium if it is needed. On the tray (or on the medium) a lattice frame is placed. Plastic lattices of micro tube racks, FC-1 (18 mm x 18mm x 20 mm) and FC-2 (22.5mm x 22.5mm x 20mm) of Nichidenrika Glass Inc., Osaka, were used. The larger one is for an individual fly, a pair,

or a small number of flies, and the larger one for mass assay. A thin transparent plastic cover is attached to the frame with adhesive tape. Flies can be blown into each room easily with a fly aspirator due to the spring-like function of a U-shaped slit on each room. The slit is made too narrow for flies to escape. In our laboratory this chamber is used as an oviposition chamber because it is easy to count silhouetted eggs with the help of light from under the bottom tray containing a thin layer of medium. If a proper mesh is attached to the bottom of the frame, it is easy to survey daily egg production only by replacing the bottom tray with fresh medium, without transferring the flies at all. It is also used for a mating chamber. With the small lattice, it is possible to observe 60 matings at a time. This chamber might be used for other purposes according to the experimental designs.

Valentin, J. University of Göteborg, Sweden. A reasonably priced automatic medium dispenser.

On setting up our new lab, we have tried to scan the market for different brands of dispensing machines and have obtained proposals from a number of manufacturers including

several mentioned in earlier DIS issues. After several trials we chose Jencon's Accuramatic Duo-Speed Mk 3, which was much cheaper than any other brand of similar capacity (\$1,200 retail price). This peristaltic pump is equipped to deliver medium fully automatically with an interval which can be set by the user between 1-5 seconds, or to deliver semi-automatically after manipulation of hand or foot contact. Dispensed volume can be altered instantly within a very wide range. We have now used this machine for more than one year and have not had any kind of problem with it.

Williamson, R.L. City of Hope Medical Center, Duarte, California. A notation for genetic mosaics.

male side carries yellow body and white eyes and whose female side is heterozygous for these mutations.  $y\ w/o;4/o//y\ w/+ +;4/4$  is a more complex mosaic involving loss of the fourth chromosome on the male side. X-irradiation of a  $y\ w/+ +$  fly during development might cause the formation of a  $y\ w/y\ w//+ +/+ +//y\ w/+ +$  individual by somatic recombination.

This notation is simple, immediately understandable and can be found on any typewriter. It has been used previously in the abstracts listed below.

References: Williamson, R.L. and W.D. Kaplan 1976, Genetics 83:s82; Kaplan, W.D., W.E. Trout and C. Parseghian 1978, XIV Int. Cong. of Genetics, Moscow, Contrib. Papers I:552; Trout, W.E., P. Wong and R. Williamson 1978, XIV Ing. Cong. of Genetics, Moscow, Contrib. Papers I:566.

A notation which allows a genetically mosaic fly to be symbolized is long overdue. This can be accomplished simply by separating the genotypes by double oblique strokes. Thus, for instance,  $y\ w/o//y\ w/+ +$  is a gynandromorph whose

#### TEACHING NOTES

Bryant, S.H. Western Illinois University. Salivary preparations from *D. pseudoobscura*.

dents also have a much easier time extracting the glands from *D. pseudoobscura* than they do from *D. melanogaster*. While not as many interesting chromosome aberrations are available in *D. pseudoobscura*, one excellent balanced lethal stock is available which has a very nice single inversion loop in chromosome 2. This stock is  $\Delta/Ba$ : Delta/BareInv.

I have found that the use of *D. pseudoobscura* rather than *D. melanogaster* is much easier for salivary preparations. The larvae are much larger, and so are the salivary glands. Stu-

Klug, W.S., G. Nicholls and T.W. Kottke. Trenton State College, Trenton, New Jersey. *Drosophila* transmission genetics computer package.

General genetics courses often include the performance of some *Drosophila* genetics laboratory experiment. Strickberger (1962) and King (1967) discuss the manner in which the appropriate crosses are executed in order to identify an unknown mutation in this organism. Klug and

Weller (1972) earlier reported the development of a *Drosophila* transmission genetics experiment simulation program for student use. Written in BASIC-PLUS for the Resource Time Sharing System of the PDP-11 family of computers, this program was intended to complement the actual student laboratory exercise rather than replace it. A new, expanded version of this program has now been written for use with an IBM CALL-OS timesharing system. Two additional programs have been written so that a complete computer based learning package for *Drosophila* transmission genetics now exists.

The computer simulation package consists of three separate yet interrelated programs. ILUVFLYS, the first computer program that the student uses, serves two functions. During the first encounter with ILUVFLYS the student is randomly assigned one of 25 unknown recessive mutations. Homozygous mutant females are independently crossed with male flies with a marker gene on either the second or third chromosome respectively. Two generations are carried out for each cross and the student is supplied with number-coded phenotypes of the offspring. From the phenotype ratios of the offspring the student should be able to determine on which chromosome the unknown trait is found. The number of offspring supplied for each generation is between 150 and 250, randomly assigned by the computer at the time of the run.

Once the student has counted the number of flies in each phenotype class the ILUVFLYS program may be recalled for a statistical analysis of the data. This second portion of ILUVFLYS computes the observed class ratios, the chi square statistic for the observed and expected ratios, and provides the probability value that the observed deviation has occurred on the basis of chance.



The second program in this series is a statistical analysis program called GENCHI. Basically, this program performs the same functions as the second portion of ILUVFLYS except that it is oriented toward the analysis of the student's actual laboratory data rather than computer generated data.

To reinforce the students' understanding of basic transmission genetics utilized in ILUVFLYS an informal quiz program called FLYQUIZ forms the third program in the package. In this program the student is asked a series of multiple choice questions which must be answered correctly before the computer will proceed to the next question. The questions are arranged in order of increasing difficulty and include the topics of segregation, independent assortment, sex-linkage and linkage.

All three of the programs in this package are written in the CALL-OS version of FORTRAN IV. ILUVFLYS is 565 lines long (17069 bytes), requires four seconds of CPU time, and usually requires about 10 minutes of student time to run. GENCHI is 280 lines long (8259 bytes), needs two seconds of CPU time, and can be run in about four minutes by the average student. FLYQUIZ has 306 lines in the program (13272 bytes), requires two seconds of CPU time, and requires approximately 15 minutes for the typical student to run. For users with appropriate access, these programs are presently available in the CALL-OS \*\*\*library of New Jersey's Educational Computer Network. For individuals without access to this network, but with a sincere interest in utilizing one or more of the above programs, we will be pleased to supply program listings, sample runs and/or punched paper program tapes.

References: King, R.C. 1967, Genetics, Oxford, New York; Klug, W.S. and D. Weller 1972, DIS 49:134; Strickberger, M. 1962, Experiments with *Drosophila*, John Wiley, New York.

Pye, Q., D. Knipple and R. MacIntyre.  
Cornell University, Ithaca, New York.  
Construction of segmental deficiency  
stocks from Y-autosome translocation  
stocks.

Many *Drosophila* workers have used the Y-autosome translocations of Lindsley and Sandler et al. (1972) to localize autosomal structural genes coding for enzymes based upon the gene-dosage-dependent enzyme activity in segmental trisomics and monosomics. Once a structural

gene is localized, x-ray induced deficiencies spanning the locus can be generated in order to screen for all null activity mutants.

We have obviated the induction of deficiencies with x-rays by constructing strains carrying segmental deficiencies generated from crosses between different T(Y:A) stocks. A basic cross scheme which can be used to construct a strain with a segmental deficiency is shown in Fig. 1.

Deficiency stocks constructed in this manner may exhibit the following phenotypes:  $y$ ,  $y^+$ ,  $y^+y^+$ ,  $y B^S$ ,  $B^S$ , depending upon the location of the breakpoints and the markers present in the Y chromosomes of the T(Y:A) stocks used. In some crosses it is not possible to distinguish the deficiency class from other classes on the basis of phenotypes. It is therefore necessary to make

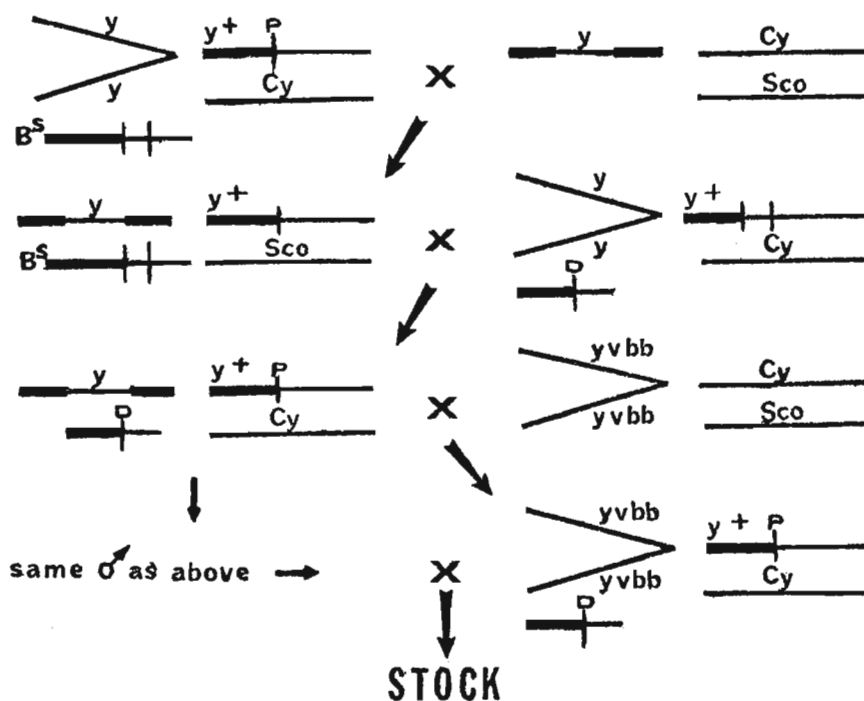


Fig. 1. P = proximal breakpoint; D = distal breakpoint; wide bars = Y chromosome material; attached X chromosomes are C(1) RM; ♂♂ used in generation 1 are  $Y^S X \cdot Y^L$ , In (1) EN,  $y/In$  (2LR) SML/Sco.



many (20-100 depending on the markers present and the extent of the deficiency to be generated) single male matings in step 3 of the cross scheme. These males are saved and subsequently mated to their daughters (we have informally designated these males "Big Daddies" or "Dirty Old Men"). The use of single males is also required because the frequency of 3:1 disjunction occurring in this cross scheme can be as high as 10% resulting in euploid individuals which can be phenotypically indistinguishable from the deficiency class. These lines can be tested for the presence of the deficiency by their ability to uncover biochemical, recessive lethal or visible loci.

We have constructed overlapping deficiency stocks which cover the entire region from 25E to 26A (B137 to D211). We have used the following designation for these deficiencies: T Df(Y:2) followed by the designations according to Lindsley and Sandler et al. (1972) of the two translocations used to generate the deficiency, e.g. T Df(Y:2) H69 D211.

There are a number of possible explanations for the failure to produce some segmental deficiency stocks: (1) triplications of Y chromosome material may result in male sterility; (2) deficiency females may not be produced due to the absence of a  $bb^+$  locus in the translocated Y chromosome elements (this problem can be circumvented by initially using a  $bb^+$  C(1) RM in the series of crosses); and (3) the extent of autosomal material which can be deleted is dependent upon the regions involved or the presence of haplo- insufficient loci in the region.

Pye, Q. Cornell University, Ithaca, New York. New white-eyed *Drosophila* "unknown" stocks for genetics laboratory courses.

In his teaching note MacIntyre (DIS 51:158) discussed the utility of some phenotypically identical (orange eye, dark body and incomplete wing veins) but genotypically unique *Drosophila* "unknown" stocks for genetics laboratory courses.

I have constructed a set of "unknown" stocks that are comparable to his, except that they all have white eyes. White eyes are due to three different situations, (1) the interaction of  $bw$ ,  $bw^D$ , or  $w^{Bwx}$  with  $cn$ ,  $st$  or  $v$ , (2) the epistatic interaction of  $w$  with  $ca$ ,  $bw$ ,  $st$ ,  $bw^D$  or  $v$ , or (3) the allele  $w$ . Another difference between the orange and white-eyed stocks is that some of the white unknowns contain two wing vein mutations (e.g.  $shf^2$ ;  $ve$  or  $ve\ ri$ ) that interact to produce the wing phenotype.

Sved, J.A. Sydney University, Australia. A computer program which saves on cooking and washing up.

Any genetic mapping experiment that can be attempted in a one-term course is usually restricted to one in which students are given stocks already built up, and asked to carry out a limited crossing program. Such an experiment

usually provides more intellectual exercise for the person setting up the program than for the student, since the student has little opportunity to plan crosses, synthesize required stocks, etc. This note describes briefly the use of computer simulation to enable quite complicated "experiments" to be carried out. Most students seem to enjoy the exercise, and hopefully learn a little genetics in the process.

The philosophy of the program is to simulate as closely as possible the problems faced in an actual mapping experiment. Most importantly, the program manipulates genotypes according to Mendelian principles, but displays only phenotypes rather than genotypes to the student. The program generates for each student a different unknown visible mutant, which may lie anywhere in the genome. A set of about 30 markers is provided, mostly recessives but with some dominants and balancers, and the student has to synthesize any stocks required to localize the unknown mutant. It takes 12-15 generations to get to the stage of constructing and carrying out a three-point testcross with markers reasonably close to the mutant. If one generation is run each day, the exercise therefore takes a minimum of 3 weeks. In practice, few students seem to be able to get through in anything like the minimum time. Each student is also provided with a recessive lethal, the mapping of which constitutes a more advanced exercise.

The simulation as described above uses as data the crosses supplied by the students, who have to be taught how to input crosses (instructions are supplied by the program). The program can also be used in a simpler way that is more suitable for larger elementary classes,

in which the data are supplied by the person in charge of the class. In this mode, the program is essentially a means of generating individual problems. Each student is given a different set of genetical data, and asked to infer the parental cross, calculate map distances, etc. The program supplies a list of the correct answers to be used for marking.

Copies of the program are available on request. However, it should be emphasized that a certain commitment may be required to get the program set up. First, the program is too long to be easily typed up from listing or sent on cards, so that it has to be sent on tape, which needs to be interpreted. Secondly, although the program is written in standard FORTRAN, it may be necessary to write a small supplementary program to enable students to submit crosses in the most efficient manner, particularly if a terminal rather than a card punch is being used. Some permanent file space is also needed. Each of these steps requires some computer experience or the assistance of a programmer.

Wright, C.P. Western Carolina University, Cullowhee, North Carolina. A method for transferring etherized flies into a container of active flies.

In working with fruit flies, it is sometimes necessary to transfer etherized flies into a container of flies which are awake and active. It is usually best to make such a transfer without etherizing or disturbing the active flies in the container. One method which is

useful in this situation involves the use of a Pasteur pipette. An etherized fly can be gently brushed or sucked head-first into the small end of a Pasteur pipette. The Pasteur pipette containing the etherized fly can then be carefully inserted along the side of the stopper or cover of the container of active flies. This can be done in such a way that the active flies in the container do not escape and are not disturbed. After the end of the Pasteur pipette which contains the etherized fly has entered the chamber of the fly container, gentle air pressure can be applied with the pipette bulb or by mouth. The etherized fly will be forced out of the pipette into the fly container. The etherized fly should be deposited on a dry surface such as the side of the container and allowed to remain on the dry surface until it wakes up and becomes active.

I have found this to be a useful technique in the situation of introductory genetics labs where beginning genetics students sometimes have difficulty in handling flies. If students try to etherize all the active flies in a container in order to introduce a few etherized flies of another genotype, sometimes all the flies will be killed as a result of accidental over-etherization. This can cause problems, especially in the situation where the active flies are virgin females which might be difficult to replace if they are killed. The use of this method of transfer decreases the amount of ether to which the flies are exposed, and thus increases their chance for survival.

#### Dog Chemical in Man's World

A recent item in the New Scientist pointed out methyl p-hydroxybenzoate as a chemical that makes bitches attractive to dogs. The human world must appear pretty seductive to dogs as this chemical is currently used as a preservative in foods, drink and cosmetics (Merck Index 9, p. 796)!. The lists of ingredients on many shampoos and handcreams show that they contain this chemical under its alternative name of methylparaben. Geneticists and other Drosophila lovers could expect strange happenings should a dog ever enter their labs, for the standard preservative added to Drosophila fly-food is Nipagin. This is yet another alias for methyl p-hydroxybenzoate.

Bryant, S.H. and M. R. Murnik, Western Illinois University, Macomb, Illinois.

The following listing classifies the mutants of *Drosophila melanogaster* according to body parts affected, or other phenotypic characteristics. Only the major effects of each mutant are listed;

effects modified by such terms as "slight" or "tendency toward" are not listed. Rank one mutants available as a singly homozygous stock according to the list in DIS 53 are preceded by \*; rank one mutants available from the list as the only mutant on their chromosome are preceded by #. Only mutants currently available are listed.

This is a revision of The Mutants of *Drosophila melanogaster* Classified According to Body Parts Affected, by Norma B. Braver, Carnegie Institution of Washington Publication 552A, 1956. Our major reference is Genetic Variations of *Drosophila melanogaster*, by Dan L. Lindsley and E. H. Grell, 1967 (Carnegie Institution of Washington Publication 627). In addition, we have listed mutants found in volumes 42-53 of DIS, 1967-1978. Additional references are indicated by letter, and given at the end of this listing.

Mutants are classified according to phenotype, grouped according to chromosome, if known. A hypothetical example follows:

\*Ph: Phlei [69]

\* indicates a rank one mutant available as a singly homozygous stock from the list in DIS 53

# indicates a rank one mutant available as the only mutant on its chromosome from the list in DIS 53

[ ] indicates the volume of DIS in which mutant found, or other reference if not Lindsley and Grell

#### Index to Categories

Abdomen	Duplications	Pupae
Antennae & Aristae	Eclosion	Recombination
Behavior	Eggs	Scutellum
Biochemical & Electrophoretic Variants	Embryos	Sex Combs
Body:	Enhancers	Sex Determination
Color	Eyes:	Sex Ratio
Size or Shape	Bristles & Hairs	Sterility:
Bristles & Hairs:	Color	Complete Sterility:
General increase in size or number	Size or Shape	Both sexes
General reduction in size or number	Texture	Females only
General disturbance of shape or arrangement	Genitalia or Sex Characteristics	Males only
Color	Halteres	Semisterility:
Head:	Head	Both sexes
General	Larvae:	Females only
Ocellars	Malpighian tubules	Males only
Verticals & Postverticals	Other characteristics	Suppressors
Vibrissae	Late Hatching	Temperature Sensitive
Thorax:	Legs & Feet	Thorax
General	Lethals:	Tumors
Dorsocentrals	Complete Lethals:	Viability:
Humeral	Both sexes	Both sexes
Notopleurals	Females only	Females only
Scutellars & Post-scutellars	Males only	Males only
Sternopleurals	Semilethals:	Viruses & Episomes
Supra- & Postalars	Both sexes	Wings:
Abdomen	Females only	Alulae
Legs & Feet	Males only	Blisters or Bubbles
Chemical Resistance or Sensitivity	Progeny Lethals	Color
Deficiencies	Longevity	Curvature
	Maternal Effect	Hairs
	Meiosis or Gametogenesis	Margin Effects
	Mouth	Position held to body
	Musculature	Size or Shape
	Nutrition Sensitive	Veins or Crossveins
	Ocelli	

ABDOMEN

see also Genitalia

## I

A<sup>53G</sup>: Abnormal abdomen  
 Aa: Altered abdomen  
 bb: bobbed  
 bb<sup>1</sup>: bobbed-lethal  
 \*ct: cut  
 mc: microchaetae  
 na: narrow abdomen  
 Qd: Quadroon  
 rea: rearranged tergites  
 sla: slimmer  
 sld: slender  
 slm: slim  
 sts: streaked sterni  
 tw: twisted  
 ws: waisted  
 wy: wavy

## II

abr: abero  
 adp<sup>60</sup>: adipose  
 adp<sup>fs</sup>: adipose-female  
       sterile  
 an<sup>2</sup>: ancon  
 chl: chaetelle  
 #ds: dachsous  
 dw-24: dwarf in 24F  
 \*ft: fat  
 lm: limited  
 mr: morula  
 Ps: Pigmentless  
 #rd<sup>s</sup>: reduced-scraggly  
 rub: rubroad  
 sm: smooth  
 spt: spermatheca  
 tuf: tufted  
 \*wt: welt

## III

a(3)26: abnormal abdomen  
 abd: abdominal  
 bxd: bithoraxoid  
 #Ly: Lyra  
 pic: piccolo  
 pic<sup>21</sup>: piccolo-2 lethal  
 rt<sup>2</sup>: rotated abdomen  
 sk: stuck [45]  
 wk: weak

## IV

ar: abdomen rotatum

ANTENNAE AND ARISTAE

see also Minutes: M()

## I

Apx: Antennapedia [44]  
 \*ct: cut  
 eo: extra organs [49]  
 sa: sparse arista [43]  
 sta: stubarista  
 t: tan

## II

\*al: aristaless  
 ant: antennaless

## III

aa: anarista  
 Antp<sup>49</sup>: Antennapedia  
 ants: antenas [43]  
 art: aristatarsia [45]  
 cmp: crumpled  
 mp: microptera  
 #Ns: Nasobemia  
 Pc: Polycomb  
 \*ss<sup>a</sup>: spineless-aristapedia  
 ss<sup>v</sup>: spineless-variegated  
 \*th: thread

## IV

ey<sup>D</sup>: eyeless-Dominant

BEHAVIOR

## I

arth: arthritic [50]  
 drd: drop dead [A]  
 Eag: Ether a gogo [44]  
 \*Hk: Hyperkinetic  
 iav: inactive [52]  
 Ocd<sup>ts</sup>: Outcold [50]  
 para: paralytic [C]  
 per<sup>0</sup>: arrhythmic [B]  
 per<sup>1</sup>: long period [B]  
 per<sup>2</sup>: short period [B]  
 \*Sh: Shaker [42]  
 sh<sup>ts</sup>: shibire [C]  
 stn<sup>ts</sup>: stoned [D]  
 t: tan  
 unc: uncoordinated [49]  
 uncl: uncoordinated-like [49]

## II

ap<sup>4</sup>: apterous  
 ove: overetherized  
 rw: raised wing  
 sps: spastic

## III

sk: stuck [45]

## U

pyo: pyokori [E]

BIOCHEMICAL AND ELECTROPHORETIC VARIANTS

see also Minutes: M();  
 see also Chemical Resistance  
 or Sensitivity

## I

bb: bobbed  
 #dor: deep orange  
 Fum: Fumarase [53]  
 Hex-A: Hexokinase-A [53]  
 Hex-B: Hexokinase-B [53]  
 lix: little isoxanthopterin  
 mal: maroonlike  
 6-Pgd: 6-Phosphogluconate  
       dehydrogenase  
 pt: platinum  
 Pt-4: Protein-4  
 \*s: sable  
 su(r): suppressor of  
       rudimentary  
 t: tan  
 v: vermillion  
 \*y: yellow  
 Zw: Zwischenferment

## II

Adh: Alcohol dehydrogenase  
 Ak-C: Adenylate kinase-C  
       [53]  
 Amy: Amylase  
 \*cn: cinnabar  
 Dip-A: Dipeptidase-A [53]  
 Got-2: Glutamate-oxaloace-  
       tate transaminase-2  
       [53]  
 Gpd: Glycerol-phosphate  
       dehydrogenase [53]  
 Hex-C: Hexokinase-C [53]  
 lys: lysine  
 Mdh-1: Malate dehydrogenase  
       [44, 53]  
 Mdh2: Malate dehydrogenase-  
       2 [44]  
 Pgi, Phosphoglucose isomer-  
       ase [53]  
 Pkg: 3-Phosphoglycerate  
       kinase [53]  
 Pt-5: Protein-5  
 Rst(2)DDT: DDT resistance  
 tyr-1: tyrosinase-1  
 Tyr-2: Tyrosinase-2

## III

Acph: Acid phosphatase  
 Ald: Aldolase [53]  
 Ali: Aliesterase

BIOCHEMICAL AND  
ELECTROPHORETIC VARIANTS  
continued

III (cont.)

Aph: Alkaline phosphatase  
Ao: Aldehyde oxidase [44, 53]  
\*e: ebony  
Est-6: Esterase-6  
Idh: NADP-Isocitrate dehydrogenase [45, 53]  
Lap-A: Leucine-amino  
peptidase-A  
Lap-D: Leucine-amino  
peptidase-D  
lxd: low xanthine dehydrogenase  
Mdh-2: NAD-Malate dehydrogenase [53]  
NADP-Malate dehydrogenase [53]  
Odh: Octanol dehydrogenase  
Pgm: Phosphoglucosomutase [45, 53]  
Pt-1: Protein-1  
Pt-8: Protein-8  
\*red: red Malpighian tubules  
\*ry: rosy  
\*se: sepia  
To-1: Tetrazolium oxidase-1 [47, 53]  
Tpi: Triose-phosphate isomerase [53]  
Tyr-3: Tyrosinase-3  
Xdh: Xanthine dehydrogenase [53]

U

Pt-9: Protein-9  
Pt-13: Protein-13

BODY COLOR

I

Aa: Altered abdomen  
amb<sup>2</sup>: amber  
amx: almondex  
arth: arthritic [50]  
bb: bobbed  
bb<sup>1</sup>: bobbed-lethal  
dlv: deltoid veins  
eb: ebony  
lac: laquered  
mel: melanized  
mk: murky  
nrs: narrow scoop  
pt: platinum  
ptg: pentagon  
Qd: Quadroon  
rv: raven

\*s: sable  
sts: streaked sterni  
su(f): supressor of forked  
svr: silver  
t: tan  
t<sup>282-1</sup>: tan lethal  
tar: tarry  
\*y: yellow  
wap: wings apart [49]

II

abr: abero  
adp<sup>60</sup>: adipose  
adp<sup>fs</sup>: adipose-female  
sterile

\*b: black  
#Bc: Black cells [44]  
bsp: brown spots  
Frd: Freckled  
rc: red cells  
#rd<sup>s</sup>: reduced-scraggly  
shr: shrunken  
Sk: Streak  
#sp: speck  
std: staroid  
tri: trident  
U: Upturned

III

a(3)26: abnormal abdomen  
in chromosome 3  
abd: abdominal  
\*cu: curled  
db: dark body  
dl: dunkel [44]  
#Dl: Delta  
\*e: ebony  
#Ly: Lyra  
\*sr: stripe  
su(pr)<sup>B</sup>: suppressor of purple  
\*W: Wrinkled

BODY SIZE AND SHAPE

see also Minutes: M()

I

cx<sup>t</sup>g: curlex-twisted genitalia  
dlv: deltoid veins  
#dm: diminutive  
dvr: divers  
dwx: dwarfex  
\*ec: echinus  
fla: flat eye  
gt: giant  
lf: little fly  
lfl: littlefly-like [49]  
lh: late hatching  
mgt: midget  
mk: murky

oc: ocelliless  
pun: puny  
rsi: reduced size  
rst: roughest  
rv: raven  
sla: slimmer  
sld: slender  
slm: slim  
sma: smaller  
smd: smalloid  
sts: streaked sterni  
ty: tiny  
ws: waisted  
wy: wavy

II

ch: chubby  
chl: chaetelle  
chy: chunky  
dp<sup>olv</sup>: dumpy  
dw-24F: dwarf in 24F  
ex: expanded  
\*ft: fat  
gt-4: giant-4  
mi: minus  
rk: rickets  
shr: shrunken  
slt: slight  
std: staroid  
tkd: thickoid

III

\*app: approximated  
bod: bowed  
\*bv: brevis  
Ch<sup>v</sup>: Chubby of Valencia [43]  
dwh: dwarfish  
gm: gleam  
obt: obtuse  
wk: weak

BRISTLES AND HAIRS:

GENERAL INCREASE IN SIZE  
OR NUMBER

I

#Hw: Hairy wing  
lac: laquered  
\*spl: split

II

extra bristles [44]  
pys: polychaetous  
scal: scabrous-like [45]  
#Tft: Tuft  
\*wt: welt

BRISTLES AND HAIRS:  
GENERAL INCREASE IN SIZE  
OR NUMBER  
 continued

## III

dhm: dark hairy margins [45]  
 \*h: hairy  
 mes: messy  
 pyd: polychaetoid

BRISTLES AND HAIRS:  
GENERAL REDUCTION IN SIZE  
OR NUMBER  
 see also Minutes: M()

## I

amb<sup>2</sup>: amber  
 \*Ax: Abruptex  
 bb: bobbed  
 bb<sup>1</sup>: bobbed-lethal  
 #dm: diminutive  
 dow: downy  
 dwg: deformed wings  
 ff: fluff  
 fin: finer  
 fri: frise'  
 kz: kurz  
 mc: microchaetae  
 ot: outheld  
 pub: pubescent  
 sbr: small bristle  
 \*sc: scute  
 shm: short macros  
 slc: slim chaetae  
 sph: sparse hairs [49]  
 sta: stubarista  
 su(f): suppressor of forked [49]  
 supact: suppressor activated  
     effect [48]  
 tc: tiny chaetae  
 trb: thread bristle  
 ty: tiny  
 tyl: tinylike

## II

abb: abbreviated  
 abr: abero  
 #Bl: Bristle  
 dw-24F: dwarf in 24F  
 Fo: Forkoid  
 Fs(2)D: Female-sterile  
     Dominant  
 Go: Gold tip  
 lm: limited  
 mi: minus  
 #rdS: reduced-scraggly  
 rdb: reduced bristle [44]  
 slt: slight  
 std: staroid  
 tb: thin bristles [45]

## III

\*bv: brevis  
 depilated [49]  
 hp: humped  
 #Ly: Lyra  
 mr: morula  
 pic: piccolo  
 pic<sup>21</sup>: piccolo-2 lethal  
 #Pr: Prickly  
 qf: quetas finas [43]  
 ra: rase'  
 #Sb: Stubble  
 \*sbd: stubboid  
 sbd<sup>1</sup>: stubboid-lethal  
 \*ss: spineless  
 \*ss<sup>a</sup>: spineless-aristapedia  
 ss<sup>v</sup>: spineless-variegated  
 wk: weak

## Y

bb<sup>Y</sup>: bobbed

BRISTLES AND HAIRS:  
GENERAL DISTURBANCE OF  
SHAPE OR ARRANGEMENT

## I

clm: clumpy marginals  
 \*f: forked  
 f<sup>257-19</sup>: forked-lethal  
 ff: fluff  
 fin: finer  
 fri: frise'  
 fw: furrowed  
 gg: goggle  
 lac: lacquered  
 rst<sup>2</sup>: roughest  
 \*sn: singed

## II

abr: abero:  
 ck: crinkled  
 #en: engrailed  
 Fo: Forkoid  
 fy<sup>2</sup>: fuzzy [44]  
 hk: hook  
 pys: polychaetous

## III

fz: frizzled  
 jv: javelin  
 jvl: javelinlike  
 \*Ki: Kinked

BRISTLES AND HAIRS: COLOR

## I

pt: platinum  
 svr: silver  
 \*y: yellow

## II

Go: Gold tip  
 stw: straw

## U

Bld: Blond

BRISTLES AND HAIRS:  
HEAD - GENERAL

## I

omm: ommatoreductum

## II

tuf: tufted  
 wt: welt

## III

gro: groucho  
 mes: messy

BRISTLES AND HAIRS:  
HEAD - OCELLARS

## I

ac: achaete  
 dx<sup>st</sup>: deltex-sterile  
 \*fu: fused  
 oc: ocelliless  
 \*Oce: Ocellarless  
 ot: outheld

## II

\*sca: scabrous  
 #Sco: Scutoid

## III

bh: baldhead [50]

## IV

Ce<sup>2</sup>: Cell

BRISTLES AND HAIRS:  
HEAD - VERTICALS AND  
POSTVERTICALS

## I

oc: ocelliless  
 \*Oce: Ocellarless  
 sa: sparse arista [43]  
 vt: verticals

## II

hk: hook

## III

#H: Hairless

BRISTLES AND HAIRS:  
HEAD - VIBRISSAEI  
vb: vibrissaeII  
wi: witty eyeIII  
Dfd: Deformed  
k: kidney  
#Ly: LyraU  
wh: whiskersBRISTLES AND HAIRS:  
THORAX - GENERALI  
dsh: dishevelled  
mc: microchaetae  
#N: Notch  
omm: ommatoreductum  
vt: verticalsII  
bd: bald [49]  
extra bristles [44]  
G: Gull  
hy: humpy  
\*Pin: Pin  
PinTac: Pin-Tack  
#pk: prickle  
sm: smooth  
#Tft: TuftIII  
bxd: bithoraxoid  
#Dl: Delta  
fz: frizzled  
\*gs: gespleten  
Hu: Humeral  
\*in: inturned  
mes: messy  
#R: RoughenedIV  
sv<sup>de</sup>: shaven-depilated  
\*sv<sup>n</sup>: shaven-nakedBRISTLES AND HAIRS:  
THORAX - DORSOCENTRALSI  
ac: achaete  
brc: brachymacrochaetaeII  
\*Pin: Pin  
upt: upturned bristle [42]III  
D: Dichaete  
#Pr: Prickly  
\*ss<sup>a</sup>: spineless-aristapedaBRISTLES AND HAIRS:  
THORAX - HUMERALSII  
#Sco: ScutoidIII  
Hu: Humeral  
Pc: PolycombBRISTLES AND HAIRS:  
THORAX - NOTOPLEURALSI  
fw: furrowedIII  
Pc: PolycombBRISTLES AND HAIRS:  
THORAX - SCUTELLARS AND  
POSTSCUTELLARSI  
brc: brachymacrochaetae  
dvr: divers  
fo: folded  
\*fu: fused  
kz: kurz  
sbr: small bristle  
shf<sup>2</sup>: shifted  
unp: unexpandedII  
ab: abrupt  
abb: abbreviated  
\*al: aristaless  
#Bl: Bristle  
d: dachs  
#ds<sup>W</sup>: dachsous-Wide  
\*ft: fat  
hk: hook  
hv: heavy vein  
\*Pin: Pin  
rk: rickets  
#Sco: Scutoid  
U: Upturned  
upt: upturned bristle [42]  
\*vg: vestigalIII  
\*app: approximated  
\*cu: curled  
#Ly: Lyra  
#Mc: Microcephalus  
#Pr: Prickly  
\*ss: spinelessIV  
Ce<sup>2</sup>: CellBRISTLES AND HAIRS:  
THORAX - STERNOLEURALSII  
\*al: aristaless  
Sp: SternopleuralBRISTLES AND HAIRS:  
THORAX - SUPRA- AND  
POSTALARSII  
Tg: TegulaU  
Din: DintyBRISTLES AND HAIRS:  
ABDOMENII  
an<sup>2</sup>: ancon  
sm: smoothIII  
a(3)26: abnormal abdomen  
in chromosome 3  
#H: HairlessBRISTLES AND HAIRS:  
LEGS AND FEET

see also Sex Combs

II  
sple: spiny legsIII  
bx: bithorax  
fz: frizzledCHEMICAL RESISTANCE OR  
SENSITIVITYsee also Biochemical &  
Electrophoretic VariantsI  
\*f: forked  
f<sup>357-19</sup>: forked-lethal

CHEMICAL RESISTANCE OR  
SENSITIVITY  
continued

II  
es: ether sensitive  
Rst(2)DDT: DDT Resistance

DEFICIENCIES

I  
#N: Notch  
  
Y  
ms(Y)Sl4: deficient for bb<sup>+</sup>:  
bobbied

DEVELOPMENT TIME

see also Minutes: M()

I  
gt: giant  
lh: late hatching

II  
gt-4: giant-4

DUPLICATIONS

I  
\*Ax: Abruptex  
\*B: Bar  
bb: bobbied  
#Co: Confluens  
#Hw: Hairy wing

II  
\*bw<sup>D</sup>: brown-Dominant

III  
#Mc: Microcephalus

ECLOSION

I  
dwg: deformed wings  
ff: fluff  
fin: finer  
\*fu: fused  
lfl: littlefly-like [49]  
mgt: midget  
mk: murky  
pun: puny  
rsi: reduced size  
shm: short macros  
sld: slender  
splw: splaw wing  
sts: streaked sterni  
tc: tiny chaetae  
thv: thick veins  
ty: tiny  
tyl: tinylike

unp: unexpanded

II  
adp<sup>60</sup>: adipose  
adp<sup>fs</sup>: adipose-female  
sterile  
mi: minus  
rdb: reduced bristle [44]

III  
mp: microptera

EGGS

see also Sterility

I  
fs(1)N: female sterile  
oc: ocelliless

II  
adp<sup>60</sup>: adipose  
adp<sup>fs</sup>: adipose - female  
sterile

III  
c(3)G: crossover supressor  
in chromosome 3

EMBRYOS

see also Sterility

I  
\*fu: fused

II  
Kr: Krueppel

ENHANCERS: Listed by abbreviations only as follows -  
enhancer: genes enhanced

I  
\*B: re  
bb<sup>11</sup>: gt  
bi: Bx, cp, sd, vg<sup>np</sup>  
cho: lys  
E(B): B  
e(bx): bx  
e(wg<sup>d</sup>): wg<sup>d</sup> [43]  
f: dvr, Fo  
Hw: h, pyd  
#mw: w  
N: fa, spl  
N<sup>264-39</sup>: nd  
N<sup>264-40</sup>: nd  
N<sup>Co</sup>: nd  
os<sup>S</sup>: tet  
sc: dvr  
spl: H  
su(f): w<sup>a</sup>  
su(r): dp, net [49]

sx: en, ey<sup>D</sup>, tra  
tuh-1: tuh-3  
v: Cor  
y: dvr

II  
abb: shr  
ap<sup>Xa</sup>: NX  
b: Sk  
cg: ds, en  
d: cg, ds, ss<sup>a</sup>  
ds: cg  
E(A): A<sup>53g</sup> [51]  
E(var)7: wm<sup>4</sup> and other  
variegated  
alleles

E(w<sup>a</sup>): w<sup>a</sup>  
en: cg  
fj: cg, ds, ss<sup>a</sup>  
Fo: f  
G: ft  
J: M  
L: M(2)l<sup>2</sup>, M(3)h<sup>33j</sup>, M(3)w  
lt: Rev, stw  
lys: rc  
M(2)l<sup>2</sup>: L  
M(2)S<sup>210</sup>: spa  
px: chl  
rc: lys  
S: ast  
shr: abb  
slt: px  
stw: lt  
vg: Hm

III  
a(3)26: abd  
Antp: Pc<sup>2</sup>, Scx  
Antp<sup>49</sup>: Scx  
Antp<sup>56</sup>: Scx  
Antp<sup>B</sup>: Pc, ss<sup>a</sup>  
Antp<sup>YU</sup>: Pc  
app: ss<sup>a</sup>  
art: Antp<sup>49</sup>, Antp<sup>B</sup>  
bx: Pc  
bx<sup>3</sup>: pbx  
bx<sup>d</sup>: Pc, Ubx  
D: ey<sup>D</sup>, tet  
Dl: M, NX  
dn: se  
s<sup>S</sup>: Sk  
E(bx): bx, Ubx  
e(dp<sup>v</sup>): dp<sup>v</sup>  
E(spl): spl  
e(tu-k): tu-k  
eb: sr  
h: Hw, pyd  
H: Ax, ci, ci<sup>D</sup>, ci<sup>W</sup>, en, ri,  
spl, tt, ve  
Hn: Hn<sup>r</sup>  
K-pn: pn



ENHANCERS

continued

## III cont.

Ly: Dfd, M(3)h<sup>33j</sup>  
 M(3)h<sup>33j</sup>: L, Ly  
 M(3)w: L  
 Me: dp, tet  
 NX: ap<sup>xa</sup>  
 pbx: Ubx  
 Pc: esc, Scx, ss<sup>a</sup>  
 Pdr: pd  
 pyd: h, Hw  
 re: B  
 ry: dor  
 sbd: Sb  
 Scx: esc, Pc  
 ss<sup>a</sup>: Antp<sup>B</sup>, cg, d, ds, f, Pc  
 ss<sup>aB</sup>: d, ds, fj  
 su(Hw)<sup>2</sup>: supact  
 tet: os<sup>S</sup>  
 tuh-3: tuh-1  
 Ubx: bxd, pbx, Pc, Scx  
 wo: w<sup>a</sup>

## IV

ci: cg, en  
 ey: Opt<sup>B</sup>, ss<sup>a</sup>, tet  
 ey<sup>4</sup>: eyr  
 ey<sup>D</sup>: D, gy, tet  
 spae(lz): lz

## Y

Y<sup>L</sup>: tet

## U

E(sd): sd  
 Hm: vg  
 Pinutes: Bd, dl, J, net,  
 px, sc  
 P: w<sup>e</sup>

EYES: BRISTLES AND HAIRS

## I

ac: achaete  
 rm: rimy

## II

#S: Star

## III

gm: gleam  
 ru: roughoid

EYES: COLOR

## I

bis: bistre  
 bo: bordeaux  
 \*car: carnation

\*cho: chocolate  
 \*cm: carmine  
 cop: copper  
 dlw: deltoid veins  
 #dor: deep orange  
 #dor<sup>1</sup>: deep orange-lethal  
 ect: eye color-temperature  
 sensitive [49]  
 fx: frostex [42]  
 \*g: garnet  
 mal: maroonlike  
 mel: melanized  
 mk: murky  
 nrs: narrow scoop  
 \*pn: prune  
 \*ras: raspberry  
 \*rb: ruby  
 rm: rimy  
 rud: ruddle  
 rv: raven  
 sma: smaller  
 smd: smalloid  
 thv: thick veins  
 v: vermillion  
 vao: varied outspread [49]  
 \*w: white  
 z: zeste

## II

bri: bright  
 bs<sup>2</sup>: blistered  
 #bur: burgundy  
 \*bw: brown  
 \*bw<sup>D</sup>: brown-Dominant  
 bw<sup>R3</sup>: brown-rearranged  
 bw<sup>V1</sup>: brown-variegated  
 caramel [43]  
 \*cl: clot  
 \*cn: cinnabar  
 cn<sup>S</sup>: cinnabar-sterile  
 dke: dark eye  
 De: Darkened eye  
 dw-24F: dwarf in 24F  
 Gla: Glazed  
 #lt: light  
 \*ltd: lightoid  
 \*or: orange  
 pd: purpleoid  
 \*pr: purple  
 pr<sup>1M60</sup>: purple-lethal  
 #Pu<sup>2</sup>: Punch  
 pw-c: pink wing-c  
 rl: rolled  
 sf: safranin  
 U: Upturned

## III

\*ca: claret  
 ca<sup>nd</sup>: claret-nondisjunctional  
 ca<sup>V</sup>: claret-variegated  
 cd: cardinal

cmd: carminoid [45]  
 Cor: Corroded eye  
 dn: doughnut  
 #Dr: Drop  
 drb: dark red brown  
 gl: glass  
 Hn: Henna  
 Hn<sup>r</sup>: Henna-recessive  
 \*kar: karmoisin  
 ma: maroon  
 mah: mahogany  
 Me: Moire'  
 \*p: pink  
 Pdr: Purpleoider  
 pers: persimmon  
 \*red: red Malpighian  
 tubules  
 red wine [45]  
 \*rs<sup>2</sup>: rose  
 ru: roughoid  
 \*ry: rosy  
 \*se: sepia  
 snb: sunburst  
 \*st: scarlet  
 st<sup>SP</sup>: scarlet-spotted  
 vi: vin [50]

## U

Mot-K: Mottled of Kriv-  
 shenko  
 rge: rouge [50]

EYES: SIZE OR SHAPE

## I

\*B: Bar  
 Bbd: Bar-baroid  
 Br<sup>601</sup>: Bar-recessive [42]  
 \*ct: cut  
 dsh: dishevelled  
 dwg: deformed wings  
 \*ec: echinus  
 eo: extra organs [49]  
 fla: flateye  
 \*fu: fused  
 fw: furrowed  
 gg: goggle  
 lac: lacquered  
 \*lz: lozenge  
 mo: micro-oculus  
 nrs: narrow scoop  
 oml: ommatidiless [44]  
 omm: ommatoreductum  
 \*os: outstretched small eye  
 rey<sup>3</sup>: rough eye  
 rst: roughest  
 rux: roughex  
 rux<sup>2</sup>: roughex-2  
 sl: small wing  
 \*spl: split  
 splw: splay wing

EYES: SIZE OR SHAPE

continued

## I cont.

spx: split thorax  
sta: stubarista  
supact: suppressor activated  
effect [48]  
sw: short wing  
swb: strawberry  
thv: thick vein  
\*un: uneven  
wa<sup>2</sup>: warty

## II

an<sup>2</sup>: ancon  
ast: asteroid  
awv<sup>2</sup>: augenwulst [47]  
d: dachs  
#ed: echinoid  
el: elbow  
Elp: Ellipse  
fj: four-jointed  
fr: fringed  
Gla: Glazed  
hv: heavy vein  
#If: Irregular facets  
Jag: Jagged  
L: Lobe  
L<sup>r</sup>: Lobe-recessive  
ll: lanceolate  
Mal: Malformed  
msf: misformed  
Opt<sup>B</sup>: Ophthalmoptera [44]  
pi: pied  
rl: rolled  
rub: rubroad  
#S: Star  
\*sca: scabrous  
scal: scabrous-like [45]  
scrp: scarp  
so: sine oculis  
std: staroid  
tkd: thickoid  
\*wt: welt

## III

\*app: approximated  
as<sup>hg</sup>: ascute-haengende  
\*bar-3: bar on chromosome 3  
bul: bulge  
bul<sup>bp</sup>: bulge-bumpy  
cv-c: crossveinless-c  
Dfd: Deformed  
Dfd<sup>r</sup>: Deformed-recessive  
#Dr: Drop  
dwh: dwarfish  
eyg: eye gone  
eyr: eyes reduced

gl: glass  
#Gl: Glued  
gm: gleam  
\*gs: gespleton  
#H: Hariless  
hp: humped  
k: kidney  
ld: loboid  
#Mc: Microcephalus  
#Ns: Nasobemia  
pe<sup>ts</sup>: pupilla eccentrica [45]  
#R: Roughened  
re: reduced eyes  
ru: roughoid  
su(pr)<sup>B</sup>: suppressor of purple

## IV

\*ey: eyeless  
ey<sup>D</sup>: eyeless-Dominant  
spa<sup>Cat</sup>: sparkling-Cataract  
\*spa<sup>pol</sup>: sparkling-poliert

EYES: TEXTURE

## I

\*ec: echinus  
fa: facet  
fx: frostex [42]  
\*lz: lozenge  
mc: microchaetae  
omm: ommatoreductum  
peb: pebbled  
rey<sup>3</sup>: rough eye  
rg: rugose  
rst<sup>2</sup>: roughest  
rux: roughex  
rux<sup>2</sup>: roughex-2  
Sc: Scotched eye  
sph: sparse hairs [49]  
\*spl: split  
supact: supressor activated  
effect [48]  
sw: short wing  
swb: strawberry  
\*un: uneven  
wa<sup>2</sup>: warty

## II

abr: abero  
an<sup>2</sup>: ancon  
ast: asteroid  
d: dachs  
dke: dark eye  
#ed: echinoid  
Elp: Ellipse  
fr: fringed  
Gla: Glazed  
#If: Irregular facets  
Jag: Jagged  
mr: morula

pi: pied  
rdo: reduced ocelli  
rh: roughish  
Roi: Rough eye  
rub: rubroad  
#S: Star  
\*sca: scabrous  
scal: scabrous-like [45]  
std: staroid  
wi: witty eye

## III

\*app: approximated  
as<sup>hg</sup>: ascute-haengende  
bul: bulge  
bul<sup>bp</sup>: bulge-bumpy  
er: erupt  
fz: frizzled  
gl: glass  
#Gl: Glued  
gm: gleam  
Me: Moire'  
#R: Roughened  
\*ro: rough  
\*roe: roughened eye  
ru: roughoid

## IV

spa: sparkling  
spa<sup>Cat</sup>: sparkling-Cataract  
\*spa<sup>pol</sup>: sparkling-poliert

GENITALIA AND SEX  
CHARACTERISTICS

see also Abdomen;  
see also Sterility

## I

\*Ax: Abruptex  
cxt<sup>g</sup>: curlex-twisted  
genitalia  
sx: sexcombless  
\*w: white

## II

\*bw: brown  
chl: chaetelle  
ix: intersex  
pi: pied  
rub: rubroad  
sm: smooth

## III

dn: doughnut  
#R: Roughened  
sk: stuck [45]  
vi: vin [50]

## IV

ar: abdomen rotatum

GENITALIA AND SEX  
CHARACTERISTICS  
continued

U  
ag: atrophie gonadique [53]

HALTERES

I  
fo: folded  
sdt: scalloped [51]

II  
ap<sup>4</sup>: apterous  
Cos: Costal [48]  
el: elbow  
\*vg: vestigial

III  
#Bd: Beaded  
bx: bithorax  
bxd: bithoraxoid  
D: Dichaete  
#Ubx: Ultrabithorax

HEAD

see also Eyes: Shape or Size;  
see also Ocelli

I  
fw: furrowed  
gg: goggle  
omm: ommatoreductum  
tuh-1: tumorous head in  
chromosome 1

II  
fj: four-jointed  
kn: knot  
ll: lanceolate  
rdo: reduced ocelli

III  
D: Dichaete  
eye: eye gone  
eyr: eyes reduced  
gro: groucho  
#Ns: Nasobemia  
tuh-3: tumorous head in  
chromosome 3

IV  
ey<sup>D</sup>: eyeless-Dominant

LARVAE: MALPIGHIAN TUBULES

I  
bo: bordeaux  
\*car: carnation  
\*cho: chocolate

\*cm: carmine  
#dor<sup>1</sup>: deep orange-lethal  
mal: maroonlike  
\*rb: ruby  
v: vermillion  
\*w: white

II  
bri: bright  
\*bw: brown  
\*bw<sup>D</sup>: brown-Dominant  
\*cl: clot  
\*cn: cinnabar  
cru: cream underscored  
Kr: Krueppel  
#lt: light  
\*ltd: lightoid  
sf: safranin  
\*ca: claret  
gl: glass  
Hn<sup>r</sup>: Henna-recessive  
\*kar: karmoisin  
ma: maroon  
Me: Moire'  
\*p: pink  
pers: persimmon  
\*red: red Malpighian  
tubules  
\*rs<sup>2</sup>: rose  
\*st: scarlet  
st<sup>SP</sup>: scarlet-spotted  
vi: vin [50]

LARVAE: OTHER  
CHARACTERISTICS

I  
\*cho: chocolate  
gt: giant  
l(1)bwn: lethal-1 benign  
wing imaginal disc  
neoplasm [50]  
l(1)mbn: lethal-1 malignant  
blood neoplasm [51]  
t: tan  
\*y: yellow

II  
#Bc: Black cells [44]  
ch: chubby  
#ft: fat  
gt-4: giant-4  
l(2)gd: lethal-3 giant discs  
[44, 50]  
l(2)gl<sup>B</sup>: lethal-2 giant  
larvae [43]  
l(2)mbn: lethal-2 malignant  
blood neoplasm [50]  
\*vg: vestigial

III  
Ch<sup>V</sup>: Chubby of Valencia  
[43]  
\*e: ebony  
l(3)gl: lethal-3 giant  
larvae [50]  
tbr: trachae broken  
tu-48j: tumor  
Tubby [50]  
#Ubx: Ultrabithorax

LATE HATCHING

see also Minutes: M()

I  
bb<sup>1</sup>: bobbed-lethal  
kz: kurz  
lh: late hatching  
tu-53: tumor

II  
blo: bloated

III  
\*bv: brevis

LEGS AND FEET

see also Sex Combs

I  
arth: arthritic [50]  
\*Bx<sup>J</sup>: Beadex of Jollos  
eo: extra organs [49]  
kf<sup>2</sup>: kinked femur [49]  
\*lz: lozenge  
pdf: pod foot  
srw: short round wing [49]  
sx: sexcombless  
tar: tarry

II  
an<sup>2</sup>: ancon  
cg: comb gap  
d: dachs  
dp<sup>olv</sup>: dumpy  
#ds: dachsous  
fj: four-jointed  
msf: misformed  
rk: rickets  
th: thick  
tkd: thickoid

III  
\*app: approximated  
cv-c: crossveinless-c  
dwh: dwarfish  
mp: microptera  
Pc: Polycomb  
rn: rotund  
\*sbd<sup>2</sup>: stubbloid  
Scx: Extra sex comb

LEGS AND FEET

continued

## IV

bt: bent  
 ey<sup>D</sup>: eyeless-Dominant  
 gy: gouty legs

## U

chi: chistera [50]

LETHALS: COMPLETE LETHALS- BOTH SEXES

see also Lethals: 1()

## I

bb: bobbed  
 Chp: Chopped [52]  
 E(B): Enhancer of Bar  
 f<sup>257-19</sup>: forked  
 1(1)bwn: lethal-1 behign wing  
           imaginal disc neo-  
           plasm [50]  
 1(1)mbn: lethal-1 malignant  
           blood neoplasm [51]  
 #N: Notch  
 su(f): suppressor of forked

## II

ap<sup>Xa</sup>: apterous-Xasta  
 #Bc: Black cells [44]  
 #Bl: Bristle  
 bw<sup>R3</sup>: brown-rearranged  
 bw<sup>V1</sup>: brown-variegated  
 Cos: Costal [48]  
 #Cy: Curly  
 Dke: Darkened eye  
 dp<sup>D</sup>: dumpy-Dominant  
 dp<sup>olv</sup>: dumpy-oblique lethal  
           vortex  
 Fo: Forkoid  
 Frd: Freckled  
 G: Gull  
 Gla: Glazed  
 Go: Gold tip  
 Kr: Krueppel  
 1(2)gd: lethal-2 giant discs  
           [44, 50]  
 1(2)gl: lethal-2 giant  
           larvae [43]  
 1(2)mbn: lethal-2 malignant  
           blood neoplasm [50]  
 N-2G: Notch-2 from Gallup  
 nw<sup>D</sup>: narrow-Dominant  
 Pin<sup>Tac</sup>: Pin-Tack  
 pr<sup>lm60</sup>: purple-lethal  
 Ps: Pigmentless  
 #Pu<sup>2</sup>: Punch  
 Px: Plexate  
 #S: Star  
 #Sco: Scutoid  
 Sk: Streak  
 Sp: Sternopleural

sps: spastic  
 Srf: Surf wings [46]  
 Su(H): Suppressor of Hairless  
 Tg: Tegula  
 U: Upturned

## III

Antp<sup>49</sup>: Antennapedia  
 #Bd: Beaded  
 ca<sup>V</sup>: claret-variegated  
 Cu-3: Curl in chromosome 3  
 Cyd: Curlyoid  
 D: Dichaete  
 Dfd: Deformed  
 #Dl: Delta  
 #Dr: Drop  
 E(bx): Enhancer of bithorax  
 #Gl: Glued  
 #H: Hairless  
 Hn: Henna  
 K-pn: Killer of prune  
 kar<sup>31</sup>: karmoisin-lethal  
 1(3)gl: lethal-3 giant  
           larvae [50]  
 1(3)mbn: lethal-3 malignant  
           blood neoplasm [52]  
 #Ly: Lyra  
 Mar: Margin [52]  
 Mas: Masculinizer  
 mes<sup>51</sup>: messy-lethal  
 Me: Moire'  
 Msc: Multiple sex comb  
 #Ns: Nasobemia  
 Pc: Polycomb  
 pic<sup>21</sup>: piccolo-lethal  
 #Sb: Stubble  
 sbd<sup>1</sup>: stubbloid-lethal  
 Scx: Extra sex comb  
 #Ser: Serrate  
 #Ubx: Ultrabithorax  
 Vno: Vein off

## IV

Ce<sup>2</sup>: Cell  
 #ci<sup>D</sup>: cubitus interruptus-  
           Dominant  
 ey<sup>D</sup>: eyeless-Dominant  
 spa<sup>Cat</sup>: sparkling-Cataract

## U

Din: Dinty  
 Hm: Haltere mimic  
 Mot-K: Mottled of Krivshenko

LETHALS: COMPLETE LETHALS- FEMALES ONLY

## I

Ap<sup>x</sup>: Antennapedia [44]  
 Fl: Female-lethal

LETHALS: COMPLETE LETHALS- MALES ONLY

## I

Bg: Bag  
 #dor<sup>1</sup>: deep orange-lethal  
 Ext: Extras  
 1(1)ts: lethal-1 tempera-  
           ture sensitive [44]  
 mal: maroonlike [43]  
 Mex 156 [51]  
 Sc: Scotched eye  
 t<sup>282-1</sup>: tan

## II

Km: Killer of male [48]

LETHALS: SEMI-LETHALS- BOTH SEXES

see also Viability

## I

dvr: divers  
 srw: short round wing [49]  
 uncl: uncoordinated-like  
           [49]  
 wap: wings apart [49]

## II

scal: scabrous-like [45]

## III

bul<sup>bp</sup>: bulge-bumpy  
 mes: messy  
 NX: Notch-Xasta  
 #R: Roughened

LETHALS: SEMI-LETHALS- FEMALES ONLY

## I

sld: slender

LETHALS: SEMI-LETHALS- MALES ONLY

## I

Clv<sup>3</sup>: Cloven [43]

## III

db: dark body

LETHALS: TO PROGENY OFHOMOZYGOTES

## I

#dor: deep orange

## II

da: daughterless

LETHALS: TO PROGENY OF  
HOMOZYGOTES

continued

U

grandchildless<sup>87</sup> [49]

LONGEVITY

see also Viability

I

drd: drop dead [A]  
\*fu: fused

II

adp<sup>fs</sup>: adipose-female  
sterile  
ap<sup>4</sup>: apterous  
ap<sup>Xa</sup>: apterous-Xasta  
che: cherub

MATERNAL EFFECT

I

\*fu: fused  
mal: maroonlike  
tuh-1: tumorous head in  
chromosome 1

II

#lt: light  
Rst(2)DDT: DDT resistance

III

Su(var):Suppressor of  
variegation

MEIOSIS AND GAMETOGENESIS  
see also Sterility and  
Semisterility

I

eq: equational producer  
RD(1): Recovery Disrupter

II

adp<sup>fs</sup>: adipose-female  
sterile  
cg: comb gap  
fs(2)B: female sterile  
SD: Segregation Distorter  
St-SD: Stabilizer of Segrega-  
tion Distorter

III

c(3)G: crossover supressor  
in chromosome 3  
ca<sup>nd</sup>: claret-nondisjunctional  
mfs(3)G: male and female  
sterile

MOUTH

I

omm: ommatoreductum

III

pb: proboscipedia

MUSCULATURE

II

ang: angle wing

NUTRITION SENSITIVE

I

\*B: Bar

II

ant: antennaless  
Mal: Malformed

III

\*cu: curled

OCELLI

I

bis: bistre  
\*fu: fused  
v: vermillion  
\*w: white

II

\*cn: cinnabar  
#lt: light  
\*ltd: lightoid  
po: pale ocelli  
rdo: reduced coelli  
so: sine oculis

III

bh: baldhead [50]  
cd: cardinal  
gro: groucho  
\*kar: karmoisin  
pe<sup>ts</sup>: pupilla eccentrica  
[45]  
\*st: scarlet  
wo: white ocelli

IV

Ce<sup>2</sup>: Cell  
ey<sup>D</sup>: eyeless-Dominant

U

rge: rouge [50]

PUPAE

I

gt: giant

II

\*b: black  
#Bc: Black cells [44]  
ch: chubby  
Frd: Freckled  
gt-4: giant-4  
#sp: speck

III

\*e: ebony  
eyg: eye gone  
Ch<sup>V</sup>: Chubby of Valencia  
[43]  
Tubby [50]

IV

ar: abdomen rotatum

RECOMBINATION

I

E(B): Enhancer of Bar

II

#Cy: Curly  
G: Gull

III

c(3)G: crossover supressor  
on chromosome 3

SCUTELLUM  
see also Thorax

I

eb: ebonized  
fu: fused  
fw: furrowed  
shf<sup>2</sup>: shifted

II

ad: arcoid  
\*al: aristaless  
#en: engrailed  
\*ft: fat  
#Tft: Tuft  
tri: trident

IV

\*gvl: grooveless

SEX COMBS

see also Legs &amp; Feet

## I

sa: sparse arista [43]  
sx: sexcombless

## II

cg: comb gap  
#en: engrailed  
esc: extra sex combs

## III

dsx: double sex  
fz: frizzled  
Msc: Multiple sex comb  
Pc: Polycomb  
rn: rotund  
Scx: Extra sex comb

## IV

ey<sup>D</sup>: eyeless-DominantSEX DETERMINATION

## II

ix: intersex

## III

dsx: double sex  
Mas: Masculinizer  
tra: transformer  
tra<sup>D</sup>: transformer-DominantSEX RATIOsee also Lethals: one sex  
only

## I

amx: almondex  
RD(1): Recovery Disrupter  
snl: sonless [45]

## II

da: daughterless  
RD(2): Recovery DisrupterSTERILITY: COMPLETE STERILITY  
- BOTH SEXES

## I

dwg: deformed wings

## II

ap<sup>4</sup>: apterous  
ap<sup>Xa</sup>: apterous-Xasta  
E(w<sup>a</sup>): Enhancer of white-  
apricot  
esc: extra sex combs  
sm: smooth  
sps: spastic

## III

bh: baldhead  
dsx: double sex  
mes: messy  
mfs(3)G: male and female  
sterile  
pic: piccolo  
rn: rotund  
spr: spread wing

## IV

sv<sup>de</sup>: shaven-depilateSTERILITY: COMPLETE STERILITY  
- FEMALES ONLY

## I

amb<sup>2</sup>: amber  
dfw: deflected wing  
#dm: diminutive  
dsh: dishevelled  
eb: ebonized  
Fl<sup>S</sup>: Female lethal-  
sterile  
fs(1)N: female sterile  
fx: frostex [42]  
gg: goggle  
#Hw: Hairy wing  
\*lz: lozenge  
mk: murky  
oc: ocelliless  
rey<sup>3</sup>: rough eye  
rfr: refringent  
sla: slimma  
\*sn: singed  
srw: short round wings  
[44]  
trb: thread bristle  
ty: tiny

## II

adp<sup>fs</sup>: adipose-female  
sterile  
#B1: Bristle  
cg: comb gap  
chl: chaetelle  
cn<sup>S</sup>: cinnabar-sterile  
fs(2)B: female sterile  
Fs(2)D: Female sterile  
lm: limited  
mi: minus  
mr: morula

## III

fs(3)G: female sterile  
pb: proboscipedia  
su(Hw)<sup>2</sup>: suppressor of Hairy  
wing

## IV

fs(4): female sterile [48]

## U

chi: chistera

STERILITY: COMPLETE  
STERILITY - MALES ONLY

## I

Aa: Altered abdomen  
Bbd: Bar-baroid  
Bb: Bubble  
bis: bistre  
Clv<sup>3</sup>: Cloven [43]  
cx<sup>tg</sup>: curlex-twisted  
genitalia  
dow: downy  
dx<sup>st</sup>: deltex-sterile  
fin: finer  
mal: maroonlike [43]  
ms(1): male sterile [52]  
ot: outheld  
pt: platinum  
pub: pubescent  
rux: roughex  
shm: short macros  
splw: splay wing  
sx: sexcombless

## II

che: cherub  
crs: cream underscored-  
sterile  
ms(2): male sterile  
pi: pied  
std: staroid  
ta: tapered

## III

br: broad  
ss<sup>V</sup>: spineless-variegated  
su(pr)<sup>B</sup>: suppressor of pur-  
ple

## IV

ar: abdomen rotatum

## Y

ms(Y)L: male sterile  
ms(Y)S: male sterileSTERILITY: SEMI-STERILES  
- BOTH SEXES

see also Minutes: M()

## I

dvr: divers  
lac: laquered  
lf: little fly

STERILITY: SEMI-STERILES- BOTH SEXES

continued

## I cont.

rea: rearranged tergites  
rst<sup>2</sup>: roughest  
sa: sparse arista [43]  
spx: split thorax  
sts: streaked sterni

## II

d: dachs  
dw-24F: dwarf in 24F  
hy: humpy  
nw<sup>2</sup>: narrow  
#Tft: Tuft

## III

dn: doughnut  
mp: microptera  
qf: quetas finas [43]  
re: reduced eyes

## U

ag: atrophie gonadique [53]

STERILITY: SEMI-STERILES- FEMALES ONLY

## I

amx: almondex  
clm: clumpy marginals  
fla: flateye  
\*fu: fused  
m<sup>D</sup>: miniature-Dominant  
na: narrow-Dominant  
na: narrow abdomen  
nrs: narrow scoop  
obl: oblique wings  
rv: raven  
swb: strawberry  
thv: thick vein  
ww: wider wing

## II

fr: fringed  
scal: scabrous-like [45]  
std: staroid  
ta: tapered

## III

br: broad  
bul<sup>bp</sup>: bulge-bumpy  
ca<sup>nd</sup>: claret-nondisjunctional  
su(pr)<sup>B</sup>: suppressor of purple

## IV

ar: abdomen rotatum

STERILITY: SEMI-STERILES- MALES ONLY

## I

arth: arthritic [50]  
#dor: deep orange  
srw: short round wings [49]

## II

adp<sup>fs</sup>: adipose-female  
sterile  
#bur: burgundy

## III

#R: Roughened

SUPPRESSORS: listed by abbreviations only as follows -  
suppressor: genes suppressed

## I

ac: h  
bi: Bx<sup>J</sup>  
dy: m  
en(w<sup>gd</sup>): f  
f<sup>ih</sup>: f  
In(1)sc<sup>7</sup>: h  
N<sup>8</sup>: Co  
su(Cbx): Cbx  
sc: D  
su(f): f  
su(r): r [49]  
su(s): pr, s, sp, v, v<sup>2</sup>  
Su(SD)X: SD  
su(w<sup>a</sup>): w<sup>a</sup>

## II

ast: net, px  
bw<sup>34e</sup>: bx  
bw<sup>w</sup>: bx  
cru: P, w<sup>e</sup>  
Cy: tet  
ds: G  
Gla: tet  
In(2L+2R)Cy: Hx  
In(2LR)bw<sup>V1</sup>: Hx  
M(2)S<sup>24</sup>: ap<sup>Xa</sup>  
px: ve  
ref: sigma  
Roi: B  
S: net, px  
St-SD: SD  
su(bw<sup>V1</sup>): bw<sup>V1</sup>  
Su(Cy): Cy  
su(dx): dx  
Su(dx): dx  
Su(er): er  
Su(H): H  
Su(S): S  
su(w<sup>coJ</sup>): w<sup>coJ</sup> [51]

## III

Cbx: bx, bxd, pbx  
D: W  
H: Bd, Dl, fa, fa<sup>no</sup>, N, nd,  
NX  
Ns: ss<sup>a</sup>  
su(Hw)<sup>2</sup>: B, Bx<sup>2</sup>, bxd, ct<sup>6</sup>,  
dm, f, Hw, lz,  
sc<sup>1</sup>, sc<sup>D1</sup>, y  
su(lz<sup>34</sup>): lz<sup>34</sup>  
su(pd): pd  
su(pr)<sup>B</sup>: pr  
su(ss)<sup>2</sup>: ss  
su(t): t  
su(tu-bw): tu-bw  
Su(var): dm, fa, nd, rst,  
sc, spl, w, and  
variegated alleles  
at other loci  
su(ve): ve  
su(vg): vg  
su-w<sup>h</sup>: w<sup>a</sup>, w<sup>a4</sup>, w<sup>h</sup> [48]

## IV

ey<sup>D</sup>: B  
gy: ey<sup>D</sup>

## Y

YY: bw<sup>V1</sup>, Rev

## U

P: w<sup>a</sup>

TEMPERATURE SENSITIVE

## I

\*Ax: abruptex  
\*B: Bar  
#bi: bifid  
Bx<sup>r</sup>: Beadex-recessive  
dor: deep orange  
ects: eye color-tempera-  
ture sensitive [49]  
e(w<sup>gd</sup>): enhancer of white-  
garnetoid [43]  
fu: fused  
gg: goggle  
l(1)ts: lethal-1 [44]  
\*nd: notchoid  
Ocdts: Outcold [50]  
oml: ommitidiless [44]  
peb: pebbled  
pl: pleated  
ptg: pentagon  
RD(1): Recovery Disrupter  
\*s: sable  
sdts: scalloped [51]  
shits: shibire  
sph: sparse hairs [49]  
stnts: stoned

TEMPERATURE SENSITIVE  
continued

## I cont.

sw: short wing  
wap: wings apart [49]  
z: zeste

## II

Alu: Alula  
ant: antennaless  
ba: balloon  
bs<sup>2</sup>: blistered  
corr: corrugated wing  
#Cy: Curly  
\*dov: dumpy-oblique vortex  
dsr: disrupted  
fr: fringed  
Go: Gold tip  
hk: hook  
Hv: heavy vein  
Hx: Hexaptera  
j: jaunty  
J: Jammed  
kn: knot  
Lr: Lobe-recessive  
mr: morula  
Pfd: Pufdi  
PinTac: Pin-Tack  
#pk: prickle  
pu: pupal  
Px: Plexate  
psy: polychaetous  
rh: roughish  
rl: rolled  
\*sca: scabrous  
scrp: scarp  
so: sine oculis  
Sp: Sternopleural  
spt: spermatheca  
std: staroid  
tkv: thick veins  
upt: upturned bristle [42]  
\*vg: vestigial  
\*wt: welt

## III

abd: abdominal  
bx: bithorax  
\*cp: clipped  
\*cu: curled  
Dfd: Deformed  
dn: doughnut  
drb: dark red brown  
fl: fluted  
hp: humped  
pb: pebbled  
pets: pupilla eccentrica [45]  
pyd: polychaetoid  
\*ss<sup>a</sup>: spineless-aristapedia  
su(ve): suppressor of veinlet  
su(vg): suppressor of vestigial  
[45]

tbr: trachae broken  
tet: tetraltera

## IV

bt: bent  
\*ci: cubitus interruptus  
#ci<sup>D</sup>: cubitus interruptus-Dominant  
\*ey: eyeless  
spa: sparkling

## U

grandchildless<sup>87</sup> [49]

THORAX

## I

\*Ax: Abruptex  
Clv<sup>3</sup>: Cloven [43]  
mel: melanized  
mk: murky  
ptg: pentagon  
spx: split thorax  
unp: unexpanded  
wap: wings apart [49]

## II

Bsh: Bashed [44]  
D: Dichaete  
dp<sup>olv</sup>: dumpy-oblique lethal vortex  
Fs(2)D: Female Sterile  
\*ft: fat  
Hx: Hexaptera  
hy: humpy  
Kr: Krueppel  
rc: red cells  
Sk: Streak  
std: staroid  
tri: trident

## III

bx: bithorax  
bxd: bithoraxoid  
#by: blistery  
Cbx: Contrabithorax  
\*gs: gespleten  
hp: humped  
pbx: postbithorax  
#R: Roughened  
\*sr: stripe  
tet: tetraltera

## IV

\*gvl: grooveless

TUMORS

see also Lethals: 1();  
see also Tumors: tu()

## I

#dor<sup>1</sup>: deep orange-lethal  
\*fu: fused

1(1)bwn: lethal-1 benign wing imaginal disc neoplasm [50]

1(1)mbn: lethal-1 malignant blood neoplasm [51]

lf: little fly

tu-53: tumor

tuh-1: tumorous head in chromosome 1

## II

1(2)mbn: lethal-2 malignant blood neoplasm [50]

nw<sup>2</sup>: narrow

tu-36a: tumor

tu-bw: tumor with brown

## III

1(3)gl: lethal-3 giant larvae [50]

1(3)mbn: lethal-3 malignant blood neoplasm [52]

tu-48j: tumor

tuh-3: tumorous head in chromosome 3

VIABILITY: BOTH SEXES  
AFFECTED

see also Semilethals

## I

Apx: Antennapedix [44]

bix: bistre

cx<sup>tg</sup>: curlex-twisted genitalia

Hw: Hairy wing

lac: lacquered

lf: little fly

m<sup>D</sup>: miniature-Dominant

mgt: midget

ma: narrow abdomen

ny: notchy

pun: puny

Qd: Quadroon

rea: rearranged tergites

rsi: reduced size

rst<sup>2</sup>: roughest

rv: raven

sa: sparse arista [43]

shm: short macros

sla: slimmer

sld: slender

splw: splay wing

spx: split thorax

sts: streaked sterni

tar: tarry

tu-53: tumor

tuh-1: tumorous head in chromosome 1



VIABILITY: BOTH SEXESAFFECTED

continued

## I cont.

tw: twisted  
unc: uncoordinated [49]  
uncl: uncoordinated-like [49]  
unp: unexpanded  
ws: waisted

## II

abr: abero  
blo: bloated  
d: dachs  
#ds<sup>W</sup>: dachsous-Wide  
dsr: disrupted  
dw-24F: dwarf in 24F  
Elp: Ellipse  
es: ether sensitive  
fr: fringed  
gt-4: giant-4  
J: Jammed  
mi: minus  
nw<sup>D</sup>: narrow-Dominant  
pi: pied  
rdb: reduced bristle [44]  
rk: rickets  
rw: raised wing  
sm: smooth  
std: staroid  
#Tft: Tuft  
tkd: thickoid  
\*vg: vestigial

## III

bod: bowed  
cur: curvoid  
dn: doughnut  
\*e: ebony  
eyr: eyes reduced  
gm: gleam  
hp: humped  
#Mc: Microcephalus  
mfs(3): male and female  
sterile  
mp: microptera  
pb: proboscipedia  
#Pr: Prickly  
#R: Roughened  
rn: rotund  
rt<sup>2</sup>: rotated abdomen  
\*sbd: stubbloid  
su(pr)<sup>B</sup>: suppressor of purple  
su(vg): suppressor of vestigial  
[45]  
wk: weak

## U

chi: chistera

VIABILITY: FEMALES ONLY

## I

clm: clumpy marginals  
dfw: deflected wing  
obl: oblique  
rfr: refringent  
ww: wider wing

VIABILITY: MALES ONLY

## I

mk: murky  
pt: platinum

## III

mes: messy

VIRUSES AND EPISOMES:  
POSSESSION, REFRACTION,  
KILLER OF

## I

Ref(1)H: Refractaire [48]

## II

Frd: Freckled  
ref: suppressor of sigma  
Ref(2)P: Refractaire [48]

## III

ref(3)O: refractaire [48]

WINGS: ALULAE

## II

Alu: Alula  
che: cherub  
el: elbow  
J: Jammed  
\*spd<sup>f</sup>g: spade-flag

## III

D: Dichaete

## IV

#ci<sup>D</sup>: cubitus interruptus-DominantWINGS: BLISTERS OR BUBBLES

## I

At: Attenuated  
Bb: Bubble  
Bg: Bag  
Bx<sup>r</sup>: Beadex-recessive  
dsh: dishevelled  
grotle [44]  
if<sup>3</sup>: inflated  
vs: vesiculated

## II

ap<sup>blt</sup>: apterous-blot  
ba: balloon  
blo: bloated  
bs<sup>2</sup>: blistered  
\*bw<sup>D</sup>: brown-Dominant  
mwg: microwing [46]  
\*nub: nubbin  
Pfd: Pufdi  
puf: puff  
#Tft: Tuft

## III

#by: blistery  
cmp: crumpled  
mes: messy  
\*W: Wrinkled

## IV

#ci<sup>D</sup>: cubitus interruptus-Dominant

## U

chi: chistera [50]

WINGS: COLOR

## I

dvr: divers  
\*dy: dusky  
\*m: miniature  
rfr: refringent  
svr: silver  
vs: vesiculated

## II

ap<sup>blt</sup>: apterous-blot  
\*b: black  
\*nub: nubbin  
Px: Plexate  
rl: rolled  
#sp: speck  
std: staroid  
stw: straw  
U: Upturned

## III

#by: blistery  
dhm: dark hairy margins [45]  
#Dl: Delta  
fl: fluted

## IV

#ci<sup>D</sup>: cubitus interruptus-Dominant

WINGS: CURVATURE

## I

abw: abnormal wings  
 \*Ax: Abruptex  
 ccw: concave wing  
 crt: crumpled tips  
 #cs<sup>53</sup>: creased  
 cu-X: curled on X chromosome  
 cx: curlex  
 cx<sup>tg</sup>: curlex-twisted genitalia  
 mel: melanized  
 nrs: narrow scoop  
 ptd: parted [44]  
 srw: short wound wings [49]  
 #Tu: Turned-up wing  
 Tul: Turneduplike  
 wy: wavy

## II

a: arc  
 ad: arcoid  
 Alu: Alula  
 arch: arch  
 bat: bat  
 \*c: curved  
 che<sup>2</sup>: cherub  
 ck: crinkled  
 \*Coi: Coiled  
 corr: corrugated wing  
 cui: curvi  
 #Cy: Curly  
 el: elbow  
 G: Gull  
 hv: heavy vein  
 j: jaunty  
 mwg: microwing [46]  
 \*nub: nubbin  
 Srf: Surf wings [46]  
 twl: twirl  
 U: Upturned  
 wx<sup>wt</sup>x: waxy-waxtex

## III

bod: bowed  
 \*cu: curled  
 Cu-3: Curl in chromosome 3  
 cur: curvoid  
 Cyd: Curlyoid  
 fl: fluted  
 mp: microptera  
 tt: tilt  
 tx: taxi

## IV

bt: bent  
 #ci<sup>D</sup>: cubitus interruptus-Dominant

## U

chi: chistera [50]  
 ic: incurve' [50]

WINGS: HAIRS

## I

\*Ax: Abruptex  
 clm: clumpy marginals  
 #Hw: Hairy wing  
 mc: microchaete  
 mo: micro-oculus

## II

ap<sup>4</sup>: apterous  
 #pk: prickle

## III

cmp: crumpled  
 fz: frizzled  
 \*h: hairy  
 \*in: inturned  
 \*mwh: multiple wing hairs

WINGS: MARGIN EFFECTS

## I

At: Attenuated  
 Bb: Bubble  
 #bi: bifid  
 \*Bx: Beadex  
 \*Bx<sup>J</sup>: Beadex of Jollos  
 Chp: Chopped [52]  
 clm: clumpy marginals  
 crt: crumpled tips  
 \*ct: cut  
 cx: curlex  
 dfw: deflected wing  
 fa<sup>n</sup>: facet-notched  
 #N: Notch  
 \*nd: notchoid  
 ny: notchy  
 \*Oce: Ocellarless  
 ot: outheld  
 #r: rudimentary  
 rg: rugose  
 #sd: scalloped  
 sd<sup>D</sup>: scalloped-Dominant  
 sd<sup>ts</sup>: scalloped  
 sw: short wing  
 swb: strawberry  
 thv: thick veins  
 trb: thread bristle

## II

abr: abero  
 \*dp<sup>OV</sup>: dumpy-oblique vortex  
 dp<sup>D</sup>: dumpy-Dominant  
 dp<sup>olv</sup>: dumpy-oblique lethal vortex

fr: fringed  
 Jag: Jagged  
 N-2G: Notch-2 from Gallup  
 \*nub: nubbin  
 Rev: Revolute  
 rl: rolled  
 spd<sup>fg</sup>: spade-flag

## III

#Bd: Beaded  
 bul: bulge  
 \*cp: clipped  
 dhm: dark hairy margins [45]  
 #Ly: Lyra  
 Mar: Margin [52]  
 NX: Notch-Xasta  
 #Ser: Serrate  
 #Ser<sup>r</sup>: Serrate-recessive [48]

WINGS: POSITION HELD TO BODY

## I

acc: acclinal wing  
 \*Ax: Abruptex  
 #bi: bifid  
 cx<sup>tg</sup>: curlex-twisted genitalia  
 dfw: deflected wing  
 dlw: deltoid veins  
 dsh: dishevelled  
 \*fu: fused  
 hdp: heldup  
 obl: oblique wings  
 \*Oce: Ocellarless  
 \*os: outstretched small eye  
 ot: outheld  
 ptd: parted [44]  
 rv: raven  
 scp: scooped  
 sdx: spreadex  
 sph: sparse hairs [49]  
 spx: split thorax  
 sw: short wing  
 #Tu: Turned-up wing  
 up: upheld  
 vao: varied outspread [49]  
 fs: vesiculated  
 wap: wings apart [49]  
 ws: waisted  
 ww: wider wing

## II

ang: angle wing  
 ba: balloon  
 bat: bat  
 blo: bloated  
 Bsh: Bashed [44]  
 \*c: curved  
 De: Dented  
 el: elbow

WINGS: POSITION HELD TO BODY  
continued

## II cont.

fr: fringed  
G: Gull  
hk: hook  
\*ho: heldout  
ove: overetherized  
pa: patulous  
Pfd: Pufdi  
Rev: Revolute  
rf<sup>2</sup>: roof wings  
rw: raised wing  
\*spd<sup>f</sup>g: spade-flag  
Tg: Tegula  
Uf: Unfolded  
\*vg: vestigal

## III

\*app: approximated  
as<sup>hg</sup>: ascute-haengende  
bx: bithorax  
cur: curvoid  
D: Dichaete  
\*eg: eagle  
mes: messy  
Pc: Polycomb  
#R: Roughened  
Rf: Roof  
\*rsd: raised  
spr: spread wings  
tt: tilt  
tx: taxi

## IV

bt: bent

U

U

Din: Dinty  
ect: écartées [50]  
spy: spready [43]

WINGS: SIZE, SHAPE OR TEXTURE

## I

abw: abnormal wings  
At: Attenuated  
\*Ax: Abruptex  
Bb: Bubble  
Bg: Bag  
#bi: bifid  
bis: bistre  
\*br: broad  
\*Bx: Beadex  
\*Bx<sup>J</sup>: Beadex of Jollos  
Bx<sup>r</sup>: Beadex-recessive  
ccw: concave wing  
\*ct: cut  
dlv: deltoid veins  
dvr: divers

dwg: deflected wing  
dwx: dwarfex  
\*dy: dusky  
eo: extra organs [49]  
fla: flateye  
fo: folded  
grotle [44]  
kf<sup>2</sup>: kinked femur [49]  
lac: lacquered  
\*m: miniature  
m<sup>D</sup>: miniature-Dominant  
mc: microchaete  
mo: morula  
nrs: narrow scoop  
omm: ommatoreductum  
pl: pleated  
ptd: parted [44]  
#r: rudimentary  
rg: rugose  
rm: rimy  
rv: raven  
sdts: scalloped  
shf<sup>2</sup>: shifted  
sl: small wing  
srw: short round wing [49]  
svr: silver  
sw: short wing  
thv: thick veins  
trb: trachae broken  
#Tu: Turned-up wing  
Tul: Turneduplike  
unp: unexpanded  
vs: vesiculated  
wgo: wing-out [43]  
ws: waisted  
ww: wider wing  
wy: wavy

## II

a: arc  
ad: arcoid  
\*al: aristaleless  
Alu: Alula  
an<sup>2</sup>: ancon  
ap<sup>4</sup>: apterous  
ap<sup>Xa</sup>: apterous-Xasta  
ba: balloon  
che: cherub  
chy: chunky  
corr: corrugated wings  
Cos: Costal [48]  
d: dachs  
#ds: dachsous  
dw-24F: dwarf in 24F  
#en: engrailed  
ex: expanded  
fj: four-jointed  
\*ft: fat  
G: Gull  
hy: humpy  
J: Jammed

kn: knot  
ll: lanceolate  
msf: misformed  
mwg: microwing [46]  
\*nub: nubbin  
nw<sup>2</sup>: narrow  
nw<sup>D</sup>: narrow-Dominant  
pi: pied  
pu: pupal  
puf: puff  
pw-c: pink wing-c  
Px: Plexate  
rk: rickets  
rub: rubroad  
scal: scabrous-like [45]  
sm: smooth  
\*spd<sup>f</sup>g: spade-flag  
std: staroid  
ta: tapered  
tkd: thickoid  
tuf: tufted  
\*vg: vestigal  
whd: withered  
wx<sup>wt</sup>x: waxy-waxtex

## III

bh: baldhead [50]  
Cbx: Contrabithorax  
cmp: crumpled  
Cu-3: Curl in chromosome 3  
#Dl: Delta  
dwh: dwarfish  
mes: messy  
mp: microptera  
obt: obtuse  
rn: rotund  
\*sbd<sup>2</sup>: stubbloid  
#Ser<sup>r</sup>: Serrate-recessive [48]  
tet: tetraltera  
\*W: Wrinkled  
wk: weak

## IV

#ci<sup>D</sup>: cubitus interruptus-Dominant

## U

chi: chistera [50]  
Hm: Haltere mimic

WINGS: VEINS OR CROSSVEINS

## I

abw: abnormal wings  
\*Ax: Abruptex  
Bg: Bag  
#bi: bifid  
\*br: broad  
\*Bx: Beadex  
Bx<sup>r</sup>: Beadex-recessive  
ccw: concave wing

WINGS: VEINS OR CROSSVEINS

continued

## I cont.

Chp: Chopped [52]  
 #Co: Confluens  
 \*cv: crossveinless  
 dl<sub>v</sub>: deltoid veins  
 dx: deltex  
 dx<sup>st</sup>: deltex-sterile  
 Ext: Extras  
 \*fu: fused  
 if<sup>3</sup>: inflated  
 #N: Notch  
 \*nd: notchoid  
 #sd: scalloped  
 sd<sup>D</sup>: scalloped-Dominant  
 shf<sup>2</sup>: shifted  
 srw: short round wings [49]  
 sw: short wing  
 thv: thick veins  
 tu-53: tumor  
 wap: wings apart [49]  
 wy: wavy

## II

a: arc  
 ab: abrupt  
 ad: arcoid  
 \*al: aristaless  
 ap<sup>4</sup>: apterous  
 ap<sup>Xa</sup>: apterous-Xasta  
 ast: asteroid  
 ba: balloon  
 blo: bloated  
 bs<sup>2</sup>: blistered  
 cg: comb gap  
 \*cv-2: crossveinless-2  
 d: dachs  
 #ds: dachsous  
 #ds<sup>W</sup>: dachsous-Wide  
 dsr: disrupted  
 dw-24F: dwarf in 24F  
 el: elbow  
 #en: engrailed  
 fj: four-jointed  
 G: Gull  
 gp: gap  
 Hia: Hiatus  
 hv: heavy vein  
 kn: knot  
 mwg: microwing [46]  
 \*net: net  
 \*nub: nubbin  
 \*px: plexus  
 Px: Plexate  
 rl: rolled  
 scal: scabrous-like [45]  
 \*shv: short vein  
 \*spd<sup>fg</sup>: spade-flag  
 std: staroid

ta: tapered  
 th: thick  
 thv: thick vein

## III

\*app: approximated  
 Cbx: Contrabithorax  
 cv-b: crossveinless-b  
 cv-c: crossveinless-c  
 det: detached  
 dhm: dark hairy margins [45]  
 #Dl: Delta  
 #H: Hairless  
 #Ly: Lyra  
 Mar: Margin 52  
 mes: messy  
 mp: micorptera  
 \*ri: radius incompletus  
 sep: separated  
 su(pr)<sup>B</sup>: supressor of purple  
 tt: tilt  
 \*ve: veinlet  
 Vno: Vein off

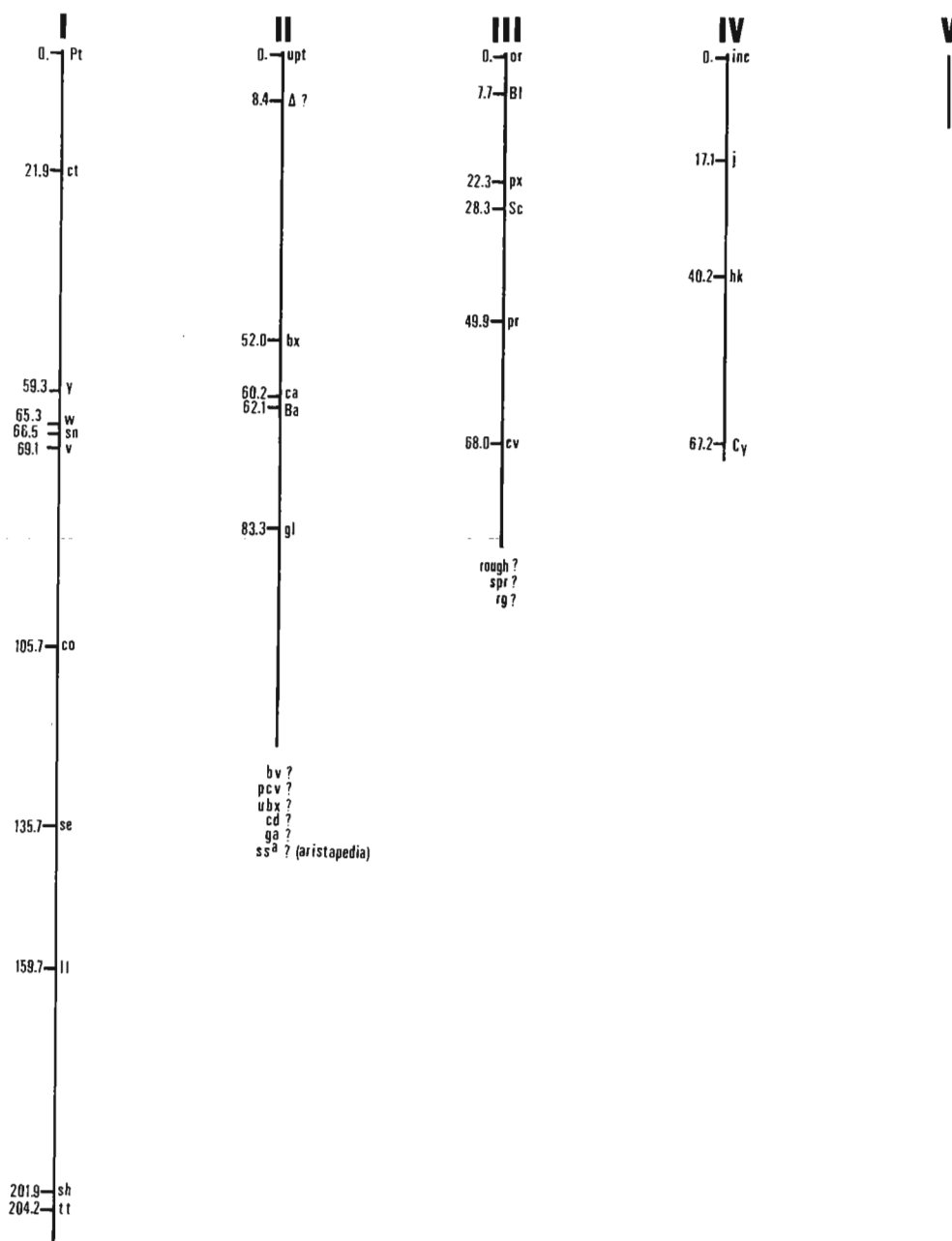
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 (abstract)

Report of W. W. Anderson and R. A. Norman  
University of Georgia, Athens, Georgia

A linkage map for currently available mutants of *D. pseudoobscura* (described in DIS 52: 11-12) is given below. Note that the map position of the Blade mutant on chromosome 3 is 7.7, rather than 77 as given in DIS 52:11. These mutants are now available from the National *Drosophila* Species Resource Center at the University of Texas in Austin.

### Linkage Map for Currently-Available Mutants of *DROSOPHILA PSEUDOBSCURA*



Report of R. Öller

University of Tübingen, Germany

According to Lakovaara and Saura (1971) a common terminology for allozymes and the alleles coding for them should be used in the obscura group of *Drosophila*. They proposed to relate all allozyme bands on gels to the most common allozyme band of *D. obscura*. Lankinen and Pinski (1977) have analyzed the two standard strains of *D. subobscura* "Küsnacht" and "ch-cu-Athens" and published a list showing the allozyme constitution of the strains for 18 different enzyme loci. Three more enzyme loci have now been investigated:

**Fum (Fumarase):** The gene could be located on chromosome A (=X). It is almost monomorphic in all populations including the Küsnacht-strain for the allele Fum<sup>103</sup> but a very rare allele Fum<sup>101</sup> could be detected in a wild population from Tunisia. Two banded heterozygotes could only be found among females, all males being one banded.

**G-6-pdh (Glucose-6-phosphate dehydrogenase):** The Küsnacht strain is monomorphic for G-6-pdh<sup>100</sup>. G-6-pdh is also located on chromosome A (=X). A polymorphism seems to exist in natural populations but the electrophoretic mobility of the different allozymes is very much the same. Heterozygous males, however, could never be observed.

**Tpi (Triose phosphate isomerase):** Mostly monomorphic for Tpi<sup>100</sup> which is also present in the Küsnacht strain. A rare allele, Tpi<sup>92</sup>, was found in Sicily forming three-banded heterozygotes with Tpi<sup>100</sup>. Linkage analysis with visible markers revealed that the Tpi gene is located on chromosome O.

Report of D. G. Yardley and C. A. Yardley

Clemson University, Clemson, South Carolina

Linkage analysis of the Amylase, Acid phosphatase-3 and Peptidase-3 loci in *D. pseudoobscura*.

Previous reports placed the Amylase (Amy), Acid phosphatase-3 (Ap-3) and Peptidase (Pep-3) loci on the third chromosome of *D. pseudoobscura* (Prakash and Lewontin, 1968; Prakash, 1973; and Prakash, 1976). Yardley (1974) later mapped Amy on the Standard (ST) gene arrangement 33.0 map units from or and 18.2 units from pr. Amy, along with Pep-3 and Ap-3 have recently been mapped on the Chiricahua (CH) gene arrangement and these results are reported here.

Two stocks, one homozygous for Amy<sup>F</sup>, Pep-3<sup>.97</sup> and Ap-3<sup>.98</sup> and another homozygous for Amy<sup>S</sup>, Pep-3<sup>1.0</sup> and Ap-3<sup>1.0</sup>, were used. Six replicate crosses and their reciprocals were made of the two homozygous lines. F<sub>1</sub> heterozygous females were then backcrossed to Amy<sup>F</sup> Pep-3<sup>.97</sup> Ap-3<sup>.98</sup> males and the F<sub>2</sub> progeny analyzed for amylase, peptidase and acid phosphatase.

The following results were obtained: non-crossovers, 112; single crossovers between Amy and Pep-3, 59; single crossovers between Amy and Ap-3, 71; and double crossovers, 48. This gives the following linkage order Pep-3 - Amy - Ap-3 on the CH gene arrangement. Amy lies 36.9 ± 4.1 map units from Pep-3 and 41.0 ± 4.1 units from Ap-3, indicating Amy is located near the center of the chromosome. These results are consistent with Yardley (1974) in placing Amy somewhere within bands 73-75. These bands also lie near the center of the chromosome in the CH gene arrangement.

This work was supported by a Faculty Research Grant from Clemson University.

References: Prakash, S. 1973, Genetics 75:347-369; Prakash, S. 1976, Genetics 84:787-790; Prakash, S. and R. C. Lewontin 1978, Proc. Natl. Acad. Sci. USA 59:398-405; Yardley, D. G. 1974, DIS 51:25.

Report of G. Alvarez  
University of Santiago, Spain

sn<sup>qr</sup>: singed-quetas recortadas. Discoverer: J. L. Meñsua. Allelic tests with sn<sup>2</sup> and sn<sup>3</sup> show this mutant belongs to the singed locus (1-21.0). Phenotype: gnarled bristles, weakly kinky hairs and female fertile; therefore according to the classification of several singed alleles by Bender (1960, Genetics 45:867-883), it is possible to include sn<sup>qr</sup> in the third class next to sn<sup>3</sup>. Nevertheless, sn<sup>qr</sup> shows a phenotype less disturbed than sn<sup>3</sup>, so that both alleles are easily distinguishable at the phenotypical level.

F<sub>2</sub> progenies from crosses of w sn<sup>3</sup>/w sn<sup>3</sup> females and w<sup>+</sup>sn<sup>qr</sup>/Y males were scored to compute the linkage distance between sn<sup>qr</sup> and sn<sup>3</sup> alleles. Four individuals of w<sup>+</sup>sn<sup>+</sup> phenotype were detected among a progeny of 10,604 flies, which indicates that sn<sup>qr</sup> is located to the right of sn<sup>3</sup> at a distance of 0.075%.

Report of M. Ashburner, J. Faithfull, T. Littlewood, G. Richards, S. Smith, V. Velissariou and R. Woodruff  
Department of Genetics, Cambridge, England

(Mutants marked with an asterisk are no longer in stock.)

b<sup>75f</sup> EMS (sperm) on Adhuf<sup>3</sup> rds pr cn

b<sup>77.1</sup> X-ray (3.5kR, sperm) on In(2L)t In(2R)Cy, Cy Roi

b<sup>77.2</sup> X-ray (3.5kR, sperm) on In(2L)t In(2R)Cy, Cy Roi

b<sup>77.3e</sup> EMS (sperm) on Sco

b<sup>77.4e</sup> EMS (sperm) on Sco

b<sup>77.5</sup> X-ray (3.5kR, sperm) on Coi

cn<sup>78</sup> Spontaneous on In(2LR)Gla, Gla

Coi<sup>75</sup> X-ray (3.5kR, sperm) on Sp

cpSS305 X-ray (3.5kR, sperm) on h ri es

cpSS307 X-ray (3.5kR, sperm) on h ri es

el<sup>3</sup> EMS (sperm) on Adhuf<sup>3</sup>cn. Temperature sensitive allele. el<sup>1</sup>/el<sup>3</sup> wild type at 25°C, elbow at 29°C.

inSS306 X-ray (3.5kR, sperm) on h ri es

jSF7 EMS (sperm) on Adhuf<sup>3</sup>rds pr cn

osp<sup>76e</sup> EMS (sperm) on bAdh<sup>n2</sup> pr cn

osp<sup>77e</sup> EMS (sperm) on b Adh<sup>n2</sup> pr cn

pu<sup>76</sup> EMS (sperm) on b rds pr cn

rk<sup>76</sup> EMS (sperm) on rds pr cn

stSS34 X-ray (3kR, sperm) on h ri es

thSS107 X-ray (3kR, sperm) on h ri es

wb: wing blister (2:50, just left of el). EMS (sperm) on b Adh<sup>n2</sup> pr cn. A roughly circular area round the crossveins of the wings forms a blister, which often collapses. Viable, but many lethal alleles known (= 1(2)brl of Woodruff and Ashburner, Genetics May 1979, and lethal group Al of O'Donell et al., 1977 Genetics 86:553).

whd<sup>76</sup> EMS (sperm) on b pr

Df(2L)A80:R15-R1 X-ray (3kR, oocytes) induced reattachment of distal element of T(Y;2)A80 and proximal element of T(Y;2)R15 = Df(2L)35A3-4;35B8-C1. Break on T(Y;2)R15 distal to y<sup>+</sup>, the deficiency being, therefore, y<sup>+</sup>act<sup>+</sup>.

- Df(2L)b<sup>75</sup> X-ray (3.5kR, sperm) = Df(2L)34D4-6;34E5-6
- Df(2L)C75RL = In(2L)75c<sup>L</sup>In(2L)C158.1<sup>R</sup> = Df(2L)35A1.2;35B3 + Dp(2;2) 26D1.2;27D1-2 + In(2L)26D1.2;35B3
- Df(2L)c1.1 X-ray (3.5kR, sperm) = Df(2L)25D7;25F1.2
- Df(2L)c1.2 X-ray (3.5kR, sperm) = Df(2L)25E1;25E2
- Df(2L)c1.7 X-ray (3.5kR, sperm) = Df(2L)25D7;26A7
- Df(2L)e1<sup>77</sup> X-ray (3.5kR, sperm) = Df(2L)35A1;35B3 + T(Y;3)64C
- Df(2L)pr<sup>76</sup> X-ray (3.5kR, sperm) = Df(2L)37D;38E
- Df(2L)ScoR<sup>+4</sup> X-ray (3.5kR, sperm) induced revertant of Sco; complex aberration, probable new order: 21-29B/35B2-33F/29F-33F/36F-39E/39E-60; = Df(2L)35B1;35D5.7
- Df(2L)ScoR<sup>+10</sup> X-ray (3.5kR, sperm) induced revertant of Sco. Not cytologically deficient but Adh<sup>-</sup>osp<sup>-</sup>.
- Df(2L)ScoR<sup>+14</sup> X-ray (3.5kR, sperm) induced revertant of Sco. Not cytologically deficient but Adh<sup>-</sup>osp<sup>-</sup>.
- Df(2L)ScoR<sup>+18</sup> X-ray (3.5kR, sperm) induced revertant of Sco. Not cytologically deficient but Adh<sup>-</sup>osp<sup>-</sup>.
- Df(2L)ScoR<sup>+17</sup>LIn(2L)C158.1<sup>R</sup> Crossover of In(2L)ScoR<sup>+17</sup> and In(2L)C158.1 = Df(2L)25D6-E1;26D1.2 + Dp(2;2)35B3;35B8.9 + In(2L)26D1.2;35B8.9.
- Df(2L)W From a wild population (Greece) = Df(2L)35A2-3;35B3-5
- Df(2L)75c X-ray (4kR, sperm) = Df(2L)35A1.2;35D4-7 + In(2L)75c = In(2L)27D1-5;35A1.2
- Df(2L)VV5 X-ray (3.5kR, sperm) = Df(2L)22A3;22E4
- Df(2R)VV4L X-ray (3.4kR, sperm) = Df(2R)43B1;43B2
- Df(2R)VV4R X-ray (3.5kR, sperm) = Df(2R)44F1;45A1.2
- (Df(2R)VV4L and Df(2R)VV4R induced on same chromosome)
- Df(3L)stSS103 X-ray (3kR, sperm) = Df(3L)73E3;74A6
- Df(3L)stSS106 X-ray (3kR, sperm) = Df(3L)72E5;73A4
- \*Df(3L)thSS45 X-ray (3kR, sperm) = Df(3L)72A5;72D6
- Df(3L)thSS102 X-ray (3kR, sperm) = Df(3L)72B1;72D12
- \*Df(3L)thSS112 X-ray (3kR, sperm) = Df(3L)71F3.5;72D12
- Df(3L)thSS113 X-ray (3kR, sperm) = Df(3L)72A2;72D1.2
- Df(3L)thSS117 X-ray (3kR, sperm) = Df(3L)72A1;72D5
- \*Df(3L)W<sup>R+7</sup> X-ray (3.5kR, sperm) induced revertant of W = Df(3L)74D3.5;75C3-7.
- Dp(2;2)C163.41<sup>L</sup>C158.1<sup>R</sup> Crossover of In(2L)C163.41 and In(2L)C158.1 = Dp(2;2)35B3.5;35E1.2 + Dp(2;2)26D1.2;27D1.2 + In(2L)26D1.2;35E1.2.
- Dp(2;2)C75RL = Dp(2;2)26D1.2;27D1.2: see Df(2L)C75RL
- Dp(2;2)ScoR<sup>+17</sup>LIn(2L)C158.1<sup>R</sup> = Dp(2;2)35B3;35B8.9: see Df(2L)ScoR<sup>+17</sup>LIn(2L)C158.1<sup>R</sup>
- In(2L)C158.1 Wild population = In(2L)26D1.2;35B3.5
- In(2L)C163.41 Wild population = In(2L)27D1.2;35E1.2
- In(2L)dp.1 X-ray 3.5kR, sperm) = In(2L)25A2-3;28C7-D3
- In(2L)dp.2 X-ray (3.5kR, sperm) = In(2L)22B;25A2-8
- In(2L)Cy<sup>R+Cl</sup> X-ray (3.5kR, sperm) induced revertant of Cy of In(2L)t, In(2R)Cy, Cy Ro1 chromosome = In(2L)23B;24B on In(2L)5.
- In(2L)ScoR<sup>+2</sup> X-ray (3.5kR, sperm) induced Sco revertant = In(2L)35B2;36C



- In(2L)Sco<sup>R+5</sup> X-ray (3.5kR, sperm) induced revertant of Sco = In(2L)35B2;38A + T(Y;2)40
- In(2L)Sco<sup>R+8</sup> X-ray (3.5kR, sperm) induced revertant of Sco = In(2L)34B6;35D1.2
- In(2L)Sco<sup>R+11</sup> X-ray (3.5kR, sperm) induced revertant of Sco = In(2L)24D1.2;35D1.4
- In(2L)Sco<sup>R+17</sup> X-ray (3.5kR, sperm) induced revertant of Sco = In(2L)25D6-E1;35B8.9
- In(2L)Sco<sup>R+21</sup> X-ray (3.5kR, sperm) induced revertant of Sco = In(2L)35B8;36D
- In(2L)Sco<sup>R+24</sup> X-ray (3.5kR, sperm) induced Sco revertant = In(2L)34A7-B1;35B8.9
- In(2L)VV2 X-ray (3.5kR, sperm) = In(2L)22B;27A
- In(2L)75c = In(2L)27E1-5;35A1.2, see Df(2L)75c
- In(2LR)Sco<sup>R+1</sup> X-ray (3.5kR, sperm) induced Sco revertant = In(2LR)35B2;44
- In(2LR)Sco<sup>R+9</sup> X-ray (3.5kR, sperm) induced Sco revertant = In(2LR)35B;41
- In(2LR)vsh X-ray (3.5kR, sperm) = In(2LR)21B;58A + In(2R)52;56
- In(3L)CA1 Spontaneous in Oregon stock = In(3L)68C15;79D4
- In(3L)th<sup>SS108</sup> X-ray (3kR, sperm) = In(3L)63F3.5;72A3.4 + In(3L)68F5;73F
- In(3L)W<sup>R+16</sup> X-ray (3.5kR, sperm) induced W revertant = In(3L)70F1.2;75C3.4
- In(3LR)W<sup>R+8</sup> X-ray (3.5kR, sperm) induced W revertant = In(3LR)75C3-D2;86D1.2
- T(1;2)c1.1 X-ray (3.5kR, sperm) = T(1;2)1DE;25D7-E4
- \*T(1;2;3)b<sup>75.1</sup> X-ray (3.5kR, sperm) = T(1;3)1B7.8;62B9 + T(1;2)1B7.8;42E + T(2;3)42E;62B9
- T(1;3)W<sup>R+19</sup> X-ray (3.5kR, sperm) induced W revertant = T(1;3)18F3.5;75C3.7
- T(2;3)Cy<sup>R+C3</sup> X-ray (3.5kR, sperm) induced Cy revertant of In(2L)Cy + In(2R)Cy, Cy = T(2;3)23B3.8;72F3.4 on In(2L)Cy
- T(2;3)dp.5 X-ray (3.5kR, sperm) = T(2;3)25A2-3;95B3-5
- T(2;3)dp.6 X-ray (3.5kR, sperm) = T(2;3)25A2-3;81F
- T(2;3)ho.5 X-ray (3.5kR, sperm) = T(2;3)22E4-F1;85B3-5
- T(2;3)Sco<sup>R+7</sup> X-ray (3.5kR, sperm) induced revertant of Sco = T(2;3)35B3.4;93F11-14.  
Also Df(2L) Adh-osp-.
- T(2;3)Sco<sup>R+13</sup> X-ray (3.5kR, sperm) induced Sco revertant = T(2;3)35A1.2;70F4.7
- T(2;3)SS301 X-ray (3.5kR, sperm) = T(2;3)21D1.2;80F
- T(2;3)VV1 X-ray (3.5kR, sperm) = T(2;3)27A1.2;85A2-6
- T(2;3)VV6 X-ray (3.5kR, sperm) = T(2;3)57B;65D
- T(3;4)SS406' X-ray (3.5kR, sperm) = T(3;4)65B;101. Homozygous viable.
- \*T(Y;2)Elp X-ray (3.5kR, sperm) = T(Y;2)57C9-D5. Ellipse like phenotype but allelism not tested before lost.
- T(Y;2)Sco<sup>R+5</sup> = T(Y;2)40, see In(2L)Sco<sup>R+5</sup>
- T(Y;2;3)Coi<sup>R+1</sup> X-ray (3.5kR, sperm) induced Coi revertant = T(Y;2)22B8 + T(2;3)32F;89EF + Tp(3)64C;71A;75C. New orders: 21-22B8/YP;61-64C/71A-75C/64C-71A/75C-89EF/32F-22B8/YD; 60-32F/89EF-100.
- T(Y;3)el<sup>77</sup> = T(Y;3)64C, see Df(2L)el<sup>77</sup>
- Tp(2)Sco<sup>R+12</sup> X-ray (3.5kR, sperm) induced Sco revertant = Tp(2)34E1.2;34B1.2;35D. Region 34E1.2 to 35D inserted in inverted order at 34B1.2.

Report of M. Ashburner, G. Richards and V. Velissariou  
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New or revised cytological locations of miscellaneous mutants. This is a summary of data collected in various ways using the new aberrations reported in this issue of DIS and other stocks. The information concerning the mutants in region 67-69 of chromosome arm 3L has been published by Akam et al. (1978, Cell 13:215).

Adh	Alcohol dehydrogenase	35B2.3	j	jaunty	34E3-34E6
app	approximated	69A1-69A5	M(3)h	Minute(3)h	69B4-69F(?)
as	ascute	72E4-73A6	M(3)S31	Minute(3)S31	=Df(3R)86D1;86D4
b	black	34D4-34D8	osp	outspread	35B2.3
bul	bulge	72E4-73A1.4	pu	pupal	34F5-35A4
cl	clot	25E1.2	rd	reduced	35C1-35C5
cn	cinnabar	43A4;43C5.6	rk	rickets	34E3-34E6
cp	clipped	74BF?	rs	rose	68A1-68A2
cu	curled	86D1.2-86D8	rt	rotated	68C9-68C12
dp	dumpy	25A1.2	Sco	Scutoid	35A4-35B2
DTS-3	DTS-3 of Holden	73A1-74A6	Su(H)	Suppressor of Hairless	35B8.9-36C1
el	elbow	35A4-35B2	st	scarlet	73A1-73A4
Est-6	Esterase-6	69A1-69A5	th	thread	72B1
eyg	eyegone	69C1-69C5	tra	transformer	73BD
gsp	gespleten	69C1-69C5	vin	vin	68C12-68D3
ho	heldout	22E4-22F1	W	Wrinkled	75C5-75D3
			wb	wing blister	34F5-35A4

Report of M. Ashburner and V. Velissariou  
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Lindsley-Sandler Y : autosome translocations; revised breakpoints. We list below those translocations between chromosomes 2 or 3 and the Y from the collection of Y:autosome translocations described by Lindsley, Sandler et al. (1972 Genetics and DIS 47, Supplement) which we have examined cytologically giving either revised or accurate autosomal breakpoints. The precise autosomal breakpoints are often difficult to determine due to the propensity of the translocated element to "pair" ectopically to the chromocenter and due to "adventitious" bands presumably derived from the capped Y's.

<u>T(Y;2)'s:</u>	J122	23E1.2	D6	25D2-3 + In(2L)24C4-6;25D2-3 + Df(2L)24C3;24E1.2
	G146	23E1.2	G74	34C3
	G120	23F6	A80	35A3.4
	H116	24F5-6	R15	35B8.9-C1
	J96	25A2-3	J165	35C4.5
	P51	25D6-7	P58	35D5-7
	B137	25D6-7		
<u>T(Y;3)'s:</u>	D228	74A4.5	J141	87A9-B3 (distal to 87A heat shock puff)
	L131	75D4.5	A78	87B5
	G8	85F9-13	D226	87E5-F2
	L17	86A6 + In(3L)68B;73F		

Report of B. Baker

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The following Y-Autosome translocations were induced by the La Jolla-Seattle *Drosophila* groups (Lindsley and Sandler, et al., 1972 Genetics 71:157-184) but not analyzed at that time.

<u>T(Y:2)</u>	<u>Autosomal breakpoint</u>	<u>T(Y:3)</u>	<u>Autosomal breakpoint</u>
A151	Insertion 41; 42E-F	A142	87D-88A
A183	48D-E	B64	100-85A/61A-72; 61A(?) / 72-85A
D203	Insertion 47A-B; 59	G63	Insertions 83C-85A and 70A-77BC; inserted pieces not visibly connected in chromocenter
L64	21-25A/33A-26; 60-33/25-26	H167	66F-67A
R70	Insertion 21C3; 21C5	H175	64E-F
R131	27E-F	J82	96A; the pieces 100-97F and 96A-97F are not visibly connected in chromocenter
		L20	64C-D plus In(64C-D;66E) plus In(66E;79)
		L58	Insertion 88C-D; 93D

Report of B. Baker

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Bsb: Blunt-short bristle (3-100.6) EMS-induced. Dominant. Chromosome homozygous lethal. Located 0.1 map unit proximal to ca based on 120 ro Bsb +, 263 + + ca and 4 ro + + recombinants among 3872 progeny of ro ca/Bsb females. All bristles markedly shortened. Under a compound microscope bristles are sharply tapered at tip; in profile look like a sharpened pencil. Viability and fertility excellent. Good cell marker. Spontaneous Bsb<sup>+</sup> clones occur at a frequency of about 0.5/abdomen in Bsb/+ females. Salivaries normal. In Pr Bsb heterozygotes bristles reduced to very short stubs.

Report of L. Craymer

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Ach: Achaetous 4 - unlocalized. Craymer, 1978. Spontaneous homozygous viable 4th-chromosomal dominant. Ach/+ lacks one or both postvertical bristles and often lacks other bristles, especially ocellars and scutellars; tends to have L5 incomplete. Ach/Ach has excellent viability and has a more extreme phenotype, with both postverticals absent and with ocellars absent or strongly reduced; patches of microchaetae and other bristles often missing as well. Ach may be a Ce allele: Ach/Ce<sup>2</sup> lacks ocelli and shows a greater than additive effect insofar as bristle loss is concerned, and modifiers in the Ce<sup>2</sup>/spaCat stock act to suppress the Ach phenotype. RK1.

Cma: Comma 3-57.5 (between jv1 and sbd2). E. B. Lewis, 1971. X-ray induced. Cma/+ has a pair of comma-like depressions at the anterior edge of the dorsal metathorax similar to those of some dp alleles, has inturred dorsocentral bristles, a sr-like phenotype, and the cuticle appears soft. Cma/+ imparts dominance to the vortex effects of hy and of dp<sup>v</sup>, dp<sup>lv</sup>, or dp<sup>olv</sup> alleles. Homozygous lethal. Viable when heterozygous with Df(3)sbd105; from this and the map location, a cytogenetic localization of 88C to 88F may be inferred. RK1.

cn<sup>2P</sup>: cn<sup>2</sup> derivative of Preston C. Preston, 1976. EMS induced on In(2LR)0, Cy dp<sup>lv</sup>I Roi cn<sup>2</sup> bw. cn<sup>2P</sup> bw/cn bw gives white eyes. RK1.

D<sup>4</sup>: Dichaete-4 Sigmund, 1978. X-ray induced; associated with T(2;3)D<sup>4</sup>. RK2A.

dsD<sup>4</sup>: dachsous with D<sup>4</sup> Craymer, 1978. X-ray induced; associated with T(2;3)D<sup>4</sup>. RK2A.

T(2;3)D<sup>4</sup>: T(2;3) with Dichaete-4 Sigmund, 1978. X-ray induced reciprocal translocation with breaks at 21D (ds) and at 70-71 (D) as determined by Lewis. T(2;3)D<sup>4</sup>/+ has the D phenotype of missing alulae and the ds<sup>-</sup> phenotype of shortened wings with close crossveins and widely spaced

posterior scutellar bristles; also has small body and smaller wings than might be expected. These last characteristics are presumably an interaction phenotype. Homozygous lethal, as is  $T(2;3)D4/D$ ;  $T(2;3)D4/dsW$  shows all the characteristics of  $dsW/dsW$ .

dp<sup>ol</sup>4: dumpy-oblique lethal 4 Craymer. Discovered in an (ex) (ds)  $SX^{(astX)}$  (dp<sup>ol</sup>4)  $Coi^{Pin/In(2L)t+(2R)Cy}$ ,  $Roi^{bw^{45a}}$  or  $45a^{sp^2}$  stock obtained from Amherst. dp<sup>ol</sup>4/dp shows weak oblique effects; dp<sup>ol</sup>4/dp<sup>lvi</sup> lethal. RK2.

ds<sup>55</sup>: dachsous-55 Craymer. Discovered in an SM5 chromosome; appears to be present in all stocks examined which carry SM5 and may be presumed to be associated with the 21D break. Phenotype like that of ds<sup>33k</sup>. RK2A.

flphd: flipper-hood 2-27. M. Simmons, 1972. X-ray induced. Phenotype like that described for flp except that flphd is fertile in both sexes; allelism inferred from phenotype and location. flphd/+ tends to have dusky, corrugated wings. RK2.

Hn<sup>P</sup>: Henna-Pasadena Craymer. Present in most, if not all, TM6 chromosomes. Phenotype like that described for Hn<sup>53k</sup>. RK3 as dominant; RK1 as recessive.

Lrm: Lobe-rm Origin uncertain, but likely to have been spontaneous in a  $S\ Sp\ Bl\ bw^D/In(2L+2R)Cy$ ,  $Cy\ cn^2$  stock maintained by Muller since it first appears in a  $S\ Sp\ Bl\ Lrm\ bw^D/In(2L+2R)Cy$ ,  $Cy\ cn^2$  stock in the DIS 28 Bloomington stock list and this and earlier stock lists show the  $S\ Sp\ Bl\ bw^D/In(2L+2R)Cy$ ,  $Cy\ cn^2$  stock. Strong L allele often with outgrowths at anterior edge of eye. Homozygote not known. RK1.

lw: long wing 2-unmapped. Probably discovered by Curry about 1940. Wings of homozygote noticeably longer than wild type; body also long. RK3.

nw<sup>B</sup>: narrow-Blade (formerly Bla: Blade) 2-75 (between L and Bc). Origin uncertain, but probably X-ray induced and discovered by P. H. Lewis, 1947. Wings of heterozygote long and narrow; somewhat variable, but does not overlap wild type. Abdomen of female fails to expand with eggs and female fertility may be reduced. Homozygote lethal, as is  $nw^B/nw^2$ . RK2.

Pin<sup>B</sup>: Pin-Bacher Bacher, 1966. EMS induced. Thoracic bristles pale yellow, thin, and twisted; bristle phenotype similar to that of  $Pin^{Yt}/+$  but more extreme, and  $Pin^B/+$  lacks the soft cuticular appearance of  $Pin^{Yt}/+$ .  $Pin^B/Pin$  has only vestiges of chaetae on thorax and has soft, watery-appearing cuticle. Homozygous lethal. RK1.

r<sup>sP</sup>: rudimentary-spontaneous from Pasadena Craymer, 77k. Has good r phenotype. Found by M. Crosby to be suppressed by  $su(Hw)^2$  in a temperature-sensitive fashion: r<sup>sP</sup> partially suppressed by  $su(Hw)^2$  at 25°C and almost completely suppressed at 18°C, although r<sup>sP</sup> is not suppressed by cold in the absence of  $su(Hw)^2$ . RK2.

r<sup>tsB</sup>: rudimentary-temperature sensitive allele of Baker B. S. Baker, 1973. EMS induced. Phenotypically wild type and female fertile at 18°C; at 25°C and higher, wings slightly reduced in size with the characteristic effect of r alleles on marginal bristles. A few offspring are produced when r<sup>ts</sup> females which have been raised at 25°C are mated to r<sup>ts</sup> males and the cross carried out at 25°C. RK2 at 25°C and higher.

T(3;4)Ubx<sup>A</sup> Originally described as  $In(3LR)Ubx^A$ . Carries cu, kar,  $Ubx^A$ , and  $spa^A$ . Cytological examination by Lewis shows an extremely complex translocation. Breaks in 67, 69, 70, 84, 85, 87F, 88F, 89E, 100B and 102E-F have tentatively been identified; others may also be present.

spa<sup>A</sup>: sparkling-A This is the dominant rough eye mutant associated with  $Ubx^A$ . More extreme than  $spa^{Cat}$  and does not overlap wild type.  $spa^A/spa^{Pol}$  shows extreme poliart phenotype;  $spa^A/spa^{Cat}$  lethal. RK1A.

Sc<sup>2</sup>: Scotch-2 1-3 or 4. Craymer, 1971. X-ray induced.  $Sc^2/+$  has glazed eyes which are slightly reduced in size. Abdomen of female fails to expand with eggs and female seems to have impaired fertility. Hemizygous lethal. Allelism inferred from location and phenotype. RK2.

Sc<sup>2rv</sup>: Scotch-2 reverted Craymer, 1977. Spontaneous revertant of Sc<sup>2</sup>. Hemizygous lethal. RK3.

v<sup>71P</sup>: vermillion-71 from Pasadena Craymer, 1971. Spontaneous; not suppressed by su(s)<sup>2</sup> or su(Hw)<sup>2</sup>. RK1.

m<sup>74f</sup>: miniature-74f Craymer, 1974. EMS induced long-winged m allele. RK1.

w<sup>74g</sup>: white-74g Craymer, 74g7. EMS induced; phenotypically like w<sup>1</sup>. RK1.

wy<sup>74i</sup>: wavy-74i Craymer, 74i3. EMS induced extreme wy allele. RK1.

y<sup>74i</sup>: yellow-74i Craymer, 74i3. EMS induced y<sup>1</sup> type of y allele. RK1.

y<sup>cts</sup>: yellow-complementing, temperature sensitive Craymer, 74f. EMS induced. Phenotypically wild type at 18°C. At 25°C and higher, resembles y<sup>2</sup>, but cuticle, bristles, and wings somewhat lighter in color. y<sup>2</sup>/y<sup>cts</sup> is phenotypically wild type at all temperatures. RK1 at 25°C and higher.

The following is supplemental information on three valuable dominant larval markers which have been described elsewhere. Tb is described in detail, since the original reference is usually no longer readily available.

Bc: Black cells (original description given in DIS 44:46). Dissection of a few eggs indicates that Bc becomes scorable at some point during embryogenesis. Often difficult to score in adult and undissected late pupa. RK1 first instar to pupa; RK2 as adult.

ChV: Chubby-Valencia (original description DIS 43:60). 3-47.7 (extremely close to, and slightly to the left of, Ki. Even in the presence of C(1)M4, which drastically increases recombination in this region via the interchromosomal effect, the ChV to Ki recombination frequency is only about 0.1%). Similar to Tb (see below), but heterozygote less extreme and possibly distinguishable from homozygote. Probably an allele of the lost Kg, but could also be allelic to Lamy's Ch mutant (DIS 17:51) which has been lost. RK1 first instar through pupa; RK2 as adult.

Tb: Tubby Originally described in DIS 17:49 as Tu. 3-90. Auerbach. Chemically induced, presumably by a nitrogen mustard. Larvae, pupae, and adults short and thickset in both heterozygote and homozygote; heterozygote not separable from homozygote by phenotype. Larval tracheae are apparently of normal length, which results in their having a meandering appearance. Always separable from wild type, but classification of Tb adults may be slow in some genotypes. RK1 from first instar through pupa; RK2 as adult.

Additions and corrections to Lindsley and Grell:

ca<sup>V</sup>: claret-variegated Cytology by Lewis showed ca<sup>V</sup> to be a four break translocation between the 2nd and 3rd chromosomes with reciprocal breaks between 59D and 94 and between 81F and 99C-E; unfortunately, the 59D; 94 breaks were inadvertently dropped from the description sent for inclusion into Lindsley and Grell. An attempt to isolate the "inversion" resulted in the recovery of the T(2;3)59D; 94 instead; therefore, the new order is 21 to 59D/94 to 99C/81F to 94/59D to 60; 61 to 81F/99E to 100. Thus ca<sup>V</sup> should be described as T(2;3)ca<sup>V</sup>.

cn<sup>3</sup>: cinnabar-3 Like cn, but slightly more extreme. See Bridges and Brehme. RK1.

cn<sup>35k</sup>: cinnabar-35k Weak cn allele; ocelli pale, but not colorless. See Bridges and Brehme. RK2.

Dl<sup>12</sup>, Dl<sup>13</sup>, Dl<sup>14</sup>: Delta Slight alleles. See Bridges and Brehme. RK2.

Dl<sup>9</sup>, Dl<sup>11</sup>: Delta Moderate alleles. See Bridges and Brehme. RK2.

Dl<sup>X</sup>: Delta-extreme Extreme allele. See Bridges and Brehme. RK2.

e: ebony-1 Arose on In(3R)C and has never been separated from the inversion. The vast majority of stocks labeled as carrying "e" without inversions can be traced back to Muller and actually carry e<sup>4</sup>; this includes the C(1)DX, y f; bw; e; spa<sup>01</sup> and C(1)DX; bw; e; ci ey<sup>R</sup> stocks and probably all stocks of this nature in the Bowling Green collection.

fa<sup>fx</sup>: facet-frostex The mutant fx reported by Kaplan and Hayes (DIS 42:38) is a facet allele of the fa<sup>swb</sup> type.

Gla: Glazed Abdomen of heterozygous female fails to expand with eggs; fertility impaired.

In(2LR)px<sup>52g</sup>: Inversion(2LR)plexus-52g Cytology: In(2LR)30A; 58F-59B (Lewis).

In(2LR)U: Inversion(2LR)Upturned Cytology: In(2LR)40; 53A (Bridges, Carnegie Institute of Washington Year Book 35:293).

In(3LR)DCxF: Inversion(3LR)Dichaete Crossover suppressor of Federova Carries ru and h.

In(3LR)P35: Inversion(3LR) from Pasadena 35 Described as In(3LR)65; 83 in Lindsley and Grell. In(3LR)65E; 83D-E (Lewis).

LVM: Balancer of L. V. Morgan Misdescription of LVM in Bridges and Brehme perpetuated by Lindsley and Grell. LVM = In(3L+3R)P, pe 1(3)LVML 1(3)LVMR.

Payne: Balancer of Fernandus Payne In(3L+3R)P, 1(3)PL 1(3)PR.

sple: spiny legs 2-53 (about 0.1 cm from Tft; relative order not determined) Bristles on legs and abdomen directed abnormally. Sternital bristles point in a more anterior direction than in wild type, and tergital bristles directed toward midline of abdomen. Trichomes also directed abnormally. Easily classified by striking "spiny" appearance of tarsi. RK1.

sr: stripe sr is slightly misdescribed in Lindsley and Grell. The longitudinal stripe is dark at 18°C and in the presence of heterozygous or homozygous b or e alleles. In a b<sup>+</sup>/b<sup>+</sup>; e<sup>+</sup>/e<sup>+</sup> background, the longitudinal stripe is light gray at 25°C.

TM3: Third Multiple 3 Carries e, not e<sup>s</sup>.

Ubx<sup>P15</sup>: Ultrabithorax-Pasadena 15 Described in Lindsley and Grell as Ubx<sup>67b</sup>.

vb<sup>2</sup>: vibrissae-2 Associated with In(1)sx.

Report of L. Craymer and E. Roy  
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Through the efforts of Terry Johnson, a number of X-chromosomal duplications and deficiencies have been obtained from George Lefevre and added to the Caltech stock collection; these are included in the DIS 53 computerized stock listing. We have carried out an extensive genetic testing of most of the deficiency stocks; these results are summarized below. Several of the chromosomal aberrations in the list below have been published elsewhere by Lefevre, Schalet, and others; however, we list only the results which we have obtained since the major purpose of this testing was to identify stocks which had suffered mislabeling in transit, although a strong secondary justification was to find cytogenetic localizations of the various X-linked visible mutants in the Caltech collection. Some of the deficiencies have been lost, both here and at Northridge; if they do exist elsewhere, we would greatly appreciate being informed of this. Also listed are the cytological determinations supplied by Dr. Lefevre. Very little testing of the duplications has been performed and genetic results of duplication tests are given only where these results are of value for purposes of cytogenetic localization.

## Duplications and insertional translocations

	<u>Cytology</u>	<u>Genetics of Dp</u>
y <sup>2</sup> Y611	1A1 to 1B14; Y	
y <sup>2</sup> Y67g	1A1 to 2B17-18 and 20A3 to base; Y	
Dp(1:3) <sub>w</sub> <sup>+67k27</sup>	3A5 to 3E8; 86E	
Dp(1:2)51b	3C2 to 3D5-6; 52E-F	
(Dp(1:2) <sub>w</sub> <sup>+51b7</sup> )		
T(1:2) <sub>w</sub> <sup>64d</sup>	3C2 to 3E8; 37D	
(T(1:2) <sub>w</sub> -ec; T(1:2)N <sup>St</sup> )		
Dp(1:3) <sub>w</sub> <sup>m49a</sup>	3A10-B1 to 3E2-3; 81	
T(1:2) <sub>w</sub> <sup>64b13</sup>	3C2 to 5A1-2; 26D (also In(1) <sub>w</sub> <sup>m4</sup> )	
T(1:2) <sub>rb</sub> <sup>+71g</sup>	3F3 to 5E8; 23A15	vs <sup>+</sup>
(T(1:2)A1125)		
Dp(1:3) <sub>sn</sub> <sup>13a1</sup>	6C11 to 7C9; 79E	shf <sup>+</sup>
T(1:2) <sub>sn</sub> <sup>+72d</sup>	7A8 to 8A5; 58E	
(T(1:2)FN107)	(also In(2LR)32C; 58E)	
Dp(1:2) <sub>v</sub> <sup>+75d</sup>	9A2 to 10C2; 40-41	
Dp(1:2) <sub>v</sub> <sup>+63i</sup>	9E1 to 10A11; 56A	ras <sup>+</sup>
T(1:3) <sub>v</sub> <sup>+74c</sup>	9E3-4 to 11B12; 80-81	
v <sup>+</sup> Yy <sup>+</sup>	9F3 to 10C1-2, 20B to base, and 1A1 to 1B2; Y	sbr <sup>+</sup> , tyl <sup>-</sup>
v <sup>+</sup> Yy <sup>+</sup> 3	9F3 to 10E3-4; 20B to base, and 1A1 to 1B2; Y	
(v <sup>+</sup> B <sup>S-</sup> Y)		
T(1:2) <sub>v</sub> <sup>65b</sup>	10A1 to 11A7; 40-41	v <sup>m</sup> , fw <sup>+</sup>
Dp(1:4) <sub>r</sub> <sup>+</sup>	13F to 16A2; 102F2-3	
T(1:2) <sub>r</sub> <sup>+75c</sup>	14B13 <sub>+</sub> to 15A9; 35D-E	"pebbly" eye
T(1:3) <sub>f</sub> <sup>+71b</sup>	15A4 to 16C2-3; 80-81	
y <sup>+</sup> Ymal <sup>106</sup>	18F to base and 1A1 to 1B2; Y	

y <sup>74k24.1</sup>	tip; 1B9-10	y <sup>-</sup> , ac <sup>-</sup> , sc <sup>-</sup>
A94	1E3; 2B15	dor <sup>-</sup> , br <sup>-</sup> , sta <sup>-</sup> , tw <sup>+</sup>
*C15	1B14-C1; 2A3	Lost
S39	1E4; 2B11-12	dor <sup>+</sup> , br <sup>-</sup> , sta <sup>-</sup> , tw <sup>+</sup>
Pgd <sup>35</sup>	2D3; 2F5	Lost (in US)
JC19	2F3; 3C5	pn <sup>+</sup> , gt <sup>-</sup> , w <sup>-</sup>
64c18	2E1-2; 3C2	pn <sup>-</sup> , gt <sup>-</sup> , w <sup>-</sup>
w-N <sup>71a</sup>	3A6; 3C10	w <sup>-</sup> , N <sup>-</sup>
N <sup>71h</sup>	3C4; 3D5	w <sup>+</sup> , N <sup>-</sup> , dm <sup>+</sup>
N <sup>69h</sup>	3C6; 3D3-4	w <sup>+</sup> , N <sup>-</sup> , dm <sup>+</sup>
dm <sup>75e</sup>	3C11; 3E4 <sup>+</sup>	dm <sup>-</sup>
	(also In(1)3E; 5E)	
*A82	3D5-6; 4E	Lost
	(also In(1)12C6; 14A8)	
A113	3D6-E1; 4F7-8	
HC244	3E8; 4F11-12	ec <sup>-</sup> , cho <sup>-</sup> , bi <sup>-</sup> , rb <sup>-</sup> , rg <sup>-</sup> , cx <sup>+</sup>
HF366	3E8; 5A7	ec <sup>-</sup> , cho <sup>-</sup> , bi <sup>-</sup> , peb <sup>-</sup> , rb <sup>-</sup> , rg <sup>-</sup> , bo <sup>+</sup> , cx <sup>+</sup>
*HC163	4A1-2; 4D1-2	Lost
RC40	4B1; 4F1	ec <sup>+</sup> , cho <sup>+</sup> , bi <sup>-</sup> , rb <sup>-</sup> , rg <sup>-</sup> , bo <sup>+</sup> , cx <sup>+</sup>
JC70	4C15-16; 5A1-2	rb <sup>+</sup> , rg <sup>-</sup> , cx <sup>+</sup>
C149	5A8-9; 5C5-6	cv <sup>-</sup> , rux <sup>+</sup>
N73	5C2; 5D5-6	cv <sup>+</sup> , rux <sup>-</sup> , vs <sup>+</sup>
ct <sup>J6</sup>	6E1; 7C1	
HA32	6E4-5; 7A6	shf <sup>+</sup> , cm <sup>-</sup> , scp <sup>+</sup>
ct <sup>J4</sup>	7A2; 7C1	shf <sup>+</sup>
C128	7D1-2	sn <sup>-</sup>
RA2	7D10; 8A4-5	gg <sup>-</sup> , oc <sup>-</sup> , ptg <sup>-</sup>
KA14	7F1-2; 8C6	gg <sup>-</sup> , oc <sup>-</sup> , ptg <sup>-</sup>
C52	8E; 9C-D	flw <sup>+</sup>
N110	9B3-4; 9D1-2	flw <sup>-</sup> , ras <sup>+</sup>
v <sup>L15</sup>	9B1; 10A1	flw <sup>-</sup> , ras <sup>-</sup> , sbr <sup>-</sup> , v <sup>-</sup>



HC163	9B9-10; 9E-F	flw <sup>-</sup> , ras <sup>-</sup> , sbr <sup>+</sup>
v <sup>64f</sup>	9E5-6; 10A2	ras <sup>+</sup> , sbr <sup>-</sup> , v <sup>-</sup>
v <sup>L3</sup>	9F6-7; 10A6-7	
v <sup>L2</sup>	10A1	
RA37	10A7; 10B17	tyl <sup>+</sup>
KA7	10A9; 10F10	tyl <sup>-</sup> , dy <sup>-</sup>
N71	10B5; 10D4	tyl <sup>-</sup> , dy <sup>+</sup>
m <sup>259-4</sup>		tyl <sup>-</sup>
HA85	10B-C; 10F	tyl <sup>+</sup> , dy <sup>-</sup>
KA6	10E1; 11A7	dy <sup>-</sup> , fw <sup>-</sup>
RA47	10F1; 10F11	dy <sup>+</sup> , fw <sup>+</sup>
N105	10F7-8; 11C4-D1	fw <sup>-</sup>
KA10	11A1; 11A7	fw <sup>-</sup>
JA26	11A1; 11D-E	fw <sup>-</sup> , wy <sup>+</sup>
HF368	11A2; 11B9	fw <sup>-</sup>
N12	11D1-2; 11F1-2	wy <sup>-</sup> , s <sup>+</sup>
C246	11D; 12A1-2	wy <sup>-</sup> , s <sup>-</sup>
HA92	12A6-7; 12D3	s <sup>+</sup> , g <sup>-</sup> , ty <sup>-</sup> , na <sup>+</sup>
KA9	12E1; 13A5	ty <sup>+</sup> , na <sup>-</sup>
*HF349	13B5; 13E1	Lost
sd <sup>72b</sup>	13F1; 14B1	
N19	17A; 18A2	os <sup>+</sup> , fu <sup>-</sup>
JA27	18A5; 18D1-2	car <sup>+</sup>
HF396	18E1-2; 20	sw <sup>-</sup> , mal <sup>-</sup> , su(f) <sup>-</sup>
mal <sup>12</sup>	19A1-2; 20	mal <sup>-</sup>
mal <sup>10</sup>	19A5-6; 19E1-2	mal <sup>-</sup>
mal <sup>22</sup>	19C4; 19E1	mal <sup>-</sup>
16-3-22	19D1; 20A1-2	mal <sup>+</sup> , su(f) <sup>+</sup>
B57	19E1; 19E8	
Q539	19E7; 19F6	
54	19F1; 20B3	
DCB1-35b	19F1; 20F	su(f) <sup>-</sup>
JC4	19F4-5; 20	su(f) <sup>-</sup>

Several additional comments need to be made. Deficiencies which should contain M(1)3E or M(1)4BC may have very slight Minute phenotypes, but the Minute designation is somewhat of an overstatement. Df(1)C246 shows a moderate Minute phenotype which might be M(1)k, although Bridges' recombination data suggest that M(1)k should be much closer to m (1/746 recombinants m to M(1)k in one experiment while a second experiment showed little, if any, crossover reduction in the v to g region). Three loci could be identified as having haplo-abnormal phenotypes, in addition to those previously described, and a fourth locus identified as likely to be haplo-abnormal: br, oc, m, and probably tw. br deficiencies (A94 and S39) show a br<sup>D</sup> phenotype; oc and oc deficiencies when heterozygous with an oc<sup>+</sup> chromosome have ocelli placed somewhat far back on the head and there appears to be a slight indentation in the cuticle between the postvertical bristles; m<sup>-</sup>/m<sup>+</sup> animals have wings which are slightly reduced in size and pointed; and tw shows a slight dominance. None of these are striking phenotypes. Also, br, bi, flw and s are evidently hypomorphic mutants: br/br<sup>-</sup> is extreme br, bi/bi<sup>-</sup> has outspread, crumpled, and very flimsy wings with an extreme bifid fusion of the basal region of the longitudinal wing veins; flw/flw<sup>-</sup> has bubbles beneath the thoracic cuticle and held-up wings; and s/s<sup>-</sup> has a very dark body color and is female sterile. From this last, it seems likely that the eb (ebonized) mutant is a sable allele. Lastly, Df(1)m259-4/Df(1)KA6 survives and has an extreme miniature phenotype with flimsy wings.

Report of J. Crist, T. Fontaine and D.J. Merrell  
University of Minnesota, Minneapolis, Minnesota.

trw: torn wing. 2-55.2. A spontaneous, completely recessive mutation. Homozygous individuals characteristically show a sawtooth pattern on the distal part of the wing as if the wing tip had been torn away. The mutation exhibits uniform, consistent expression in both sexes. Viability and fertility are good. RK1.

Report of M. Crosby  
California Institute of Technology, Pasadena, California

Mcp: Mischadestral pigmentation. M. Crosby, 1977. Pub. Nature 276:565-570. Causes conversion of fourth abdominal segment to a fifth abdominal state of development. Most striking in homozygous males, in which fourth tergite is completely pigmented; expression in heterozygotes is variable but does not overlap wild type. As pointed out by A.T.C. Carpenter, bristles and trichomes of fourth tergite in both sexes exhibit a pattern which is typical of the wild type fifth tergite. RK1 in males; RK3 in females.

Report of R. Frankham and R.K. Nurthen  
Macquarie University, Sydney, Australia

sca<sup>dc</sup>: scabrousdecanalizing. This new recessive allele at the scabrous locus arose in an abdominal bristle selection line. It exhibits the rough eyes typical of that mutant but results in an increased scutellar bristle mean, a decanalization of scutellar bristle pattern and reductions in sternopleural and third coxal bristle numbers (all effects at 25°C).

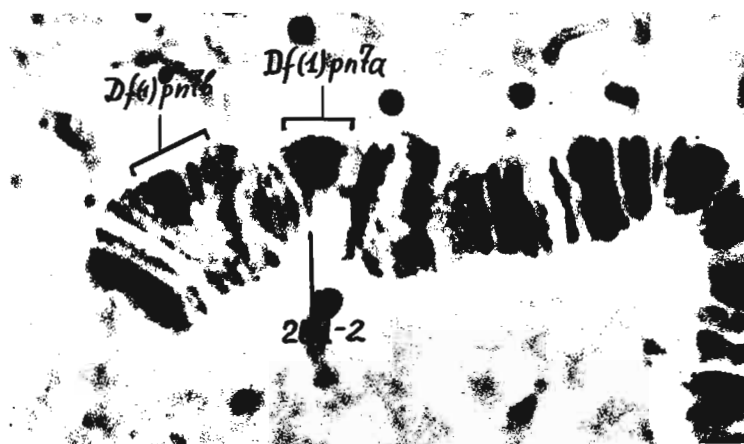
sm<sup>lab</sup>: smooth<sup>low</sup> abdominal bristle number. A new fertile allele at the smooth locus, characterized by low abdominal bristle number, alteration of abdominal bristle pattern and a reversal of the sexual dimorphism for abdominal bristle number. The allele is temperature sensitive with a TSP in the pupal period.

Report of M.A. Hughes and M. Shelton  
University of Newcastle upon Tyne, England

Mpe: Monoplane. T (2:3). X-ray induced in a wild type Oregon-R stock. Mpe is dominant, homozygous sterile and partially inviable. The heterozygote also has reduced overall viability and is partially sterile. A balanced Mpe/Pm stock has been established. The flies'

wings are held out horizontally and at 90° to the body, in a position very similar to that of taxi (tx 3-91.0) (Collins, J.L. 1928, Am. Nat. 62:127-136). In all other respects the wings appear normal, although Mpe flies are unable to perform certain courtship wing movements and are flightless. Recombination has shown Mpe to be a 2-3 translocation. Using the markers st, ca and vg the breakpoint on 3 has been placed at  $54.3 \pm 0.9$ . The breakpoint on 2 has not been placed, other than being in 2L, since recombination in 2L is reduced by 37.0%. This new mutant affects the expression of a number of other mutations including eyeless (ey 4-2.0). Mpe ey flies have an eye size intermediate between that of ey and Mpe flies. Dichaeete (D 3-40.4) is epistatic to Mpe such that Mpe/D flies show the Dichaeete phenotype.

Report of O.V. Ilyina, A.V. Sorokin, E.S. Belyaeva, I.F. Zhimulev  
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The chromosome aberrations having prune phenotype in heterozygotes with pn, recovered from F<sub>1</sub> between y sc ac pn sn females and Batumi-L wild males following X-ray (4000r) mutagenesis of the latter. Among 70,482 F<sub>1</sub> females 12 such chromosomes were selected. All of them are viable in heterozygous condition, homozygous lethal.

Fig. 1. Localization of breakpoints of deficiencies pn 7a and pn 7b.

In(1)pn 1	In(1)2E1-2; 20A-12 + Tp(1-2R)pn1 Tp(1;2R)2E1-2; 20D; 43F
New orders	1A-2D6/20D-F
	21A-43F/20A3-D/2E1-2 - 20A1-2/44A1-2 - 60F
T(1;4)pn2	T(1;4)2D5-6; 101F
Tp(1-2)pn3	Tp(1-2)12E1-2; 20A; 41A + Tp(1-3)pn3
	Tp(1-3)2E1-2; 12E1-2; 80C
New orders	1A-2E1-2/ 20A1-2 - F
	21A-41A/ 12E1-2 - 20A1-2/41A- 60F
	61A - 80C/2E1-2 -12E1-2/ 80D-100F
Df(1)pn7a	Df(1)2E3; 3A5-8 + Df(1)pn7b Df(1)1E1-2; 2B4-5 (Fig. 1)
T(1;3R)pn12	T(1;3R)2E1-2; 98A1-2
Df(1)pn24	Df(1)2D5; 2F5
Tp(1-3L)pn25	Tp(1-3L)2E1-2; 20A1-2; 70A5-6
New orders	1A - 2E1-2/20A-F
	61A -70A5-6/ 2E1-2 - 20A1-2/70A5-6 - 100F
Tp(1-3L)pn26	Tp(1-3L)2E1-2; 20A1-2; 70C1-2
New orders	1A -2E1-2/ 20A-F
	61A - 70C1-2/ 2E1-2 - 20A/ 70C1-2 - 80C
In(1)pn36	In(1)1A; 2E1-2 + Tp(3L-1)pn36 Tp(3L-1)61A; 62C1-2; 2E1-2
New orders	61A 62C1-2/ 2E1-2 -1A/ 2E1-2 - 20F
	62C3-4 -100F
Df(1)pn36	Df(1)2D3-4; 2E3
T(1;2)pn40	T(1;2)2E1-2; 41A
New orders	1A - 2E1-2/ 40F - 21A
	60F - 41A/ 2E1-2 - 20F
In(1)pn45	In(1)2D1-2; 20A

These data place prune in 2E2-3.

## Report of Y. Inoue

National Institute of Genetics, Mishima, Japan

eym: eye missing. 3-67.9. Discovered in a mass culture stock originated from a mixture of Oregon-R males and wild caught females of the Katsunuma (Japan). Eyes are almost completely absent in the homozygote. Heterozygotes with Oregon-R show normal eyes but the heterozygotes with eyg (eye gone, 3-35.5) show various grades of eye sizes (Table 1). Considerable pupal mortality is observed in both eym and eyg homozygotes, but the larval variability of eym is in the range of normal (control), while that of eyg is significantly lower than eym and Oregon-R (Table 2). Fertile, RK1.

Table 1. The number of individuals having various eye sizes.

Genotype	Relative size of eye				
	0	1/4	2/4	3/4	1 (normal)
<u>eym</u>	238	6	0	0	0
<u>eyg</u>	0	5	53	217	3
<u>eym</u> ♀ X <u>eyg</u> ♂	22	80	232	164	0
<u>eyg</u> ♀ X <u>eym</u> ♂	20	35	198	190	0
<u>eym</u> ♀ X Oregon-R ♂	0	0	0	0	100
<u>eyg</u> ♀ X Oregon-R ♂	0	0	0	0	100
Oregon-R (control)	0	0	0	0	100

Table 2. Egg-to-pupa and pupa-to-adult viability (percent).

Genotype	Egg-to-pupa viability	Pupa-to-adult viability	Egg-to-adult viability
<u>eym</u>	87.6 ± 2.2	69.3 ± 2.1	60.6 ± 2.1
<u>eyg</u>	55.8 ± 1.9	71.1 ± 3.7	39.8 ± 2.9
<u>eym</u> ♀ X <u>eyg</u> ♂	73.2 ± 3.2	99.8 ± 0.3	73.0 ± 3.2
<u>eyg</u> ♀ X <u>eym</u> ♂	66.0 ± 1.7	99.7 ± 0.3	65.8 ± 1.6
Oregon-R (control)	92.7 ± 1.4	96.0 ± 0.8	88.9 ± 1.5

## Report of W.-E. Kalisch

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Fig. 1

ter<sup>U</sup>: terraced of Umea<sup>0</sup> 2-36. Spontaneous as a cluster in a mutable sc z w<sup>zmz</sup> stock (Kalisch 1970, Molec. Gen. Genetics 107:321-335). In location, phenotype and variability similar with the lost mutation terraced (Lindsley and Grell 1968). Eye phenotype varies from a tiny facet-less nick at the anterior rim of the eye combined with a horizontal seam of irregularly ordered facets to the phenotype shown in Fig. 1. Position of the seam corresponds with the sectors I of the upper or the lower half of the eye (from the pattern of eye sectors formed by cells from first larval instar eye anlage: Becker 1957, Z. indukt. Abstamm.- und Vererb.-L. 88:333-373). In the most expressive form (Fig. 1) the facet-less nick effects sectors I and II of the upper half and sectors I - IV of the lower half of the eye. A large tuft of bristles is mostly to be found in the facet-less area. In 50% of the flies at least one eye shows this phenotype. Penetrance about 94% in the range of 16-25°C. Viability good, but hatching rate of ter<sup>U</sup>/ter<sup>U</sup> reduced against ter<sup>U</sup>/+. RK3.

Dp(1;1)Gr: Duplication of Green. Spontaneous cluster of 12 females with reddish-yellow eyes among the progeny of y<sup>2</sup> w<sup>-</sup> spl sn<sup>3</sup> females crossed to w<sup>c</sup> sn<sup>3</sup> males. Tandem repeat: Dp(1;1) 3A2-3;8B4-C1. The tandem repeat itself is phenotypically wild-type. Marker genes within the duplicated sections are: (w<sup>-</sup> spl sn<sup>3</sup>) (w<sup>c</sup> sn<sup>3</sup>). Homo- and hemizygous lethal. Hatching rate of heterozygote females reduced, viability of imagoes ± normal. Crossover in region y<sup>2</sup>-v re-

duced to 7.9%. Intrachromosomal effect at the proximal part of the X and interchromosomal effect at the distal parts of autosomes -2 and -3. Intrachromosomal exchange after double loop formation of the duplicated section. 0.07% patroclinous males among 15,269 F<sub>1</sub> males in 365 single cultures of Dp(1;1)Gr/+ and wild-type X-chromosomes. RK3.

References: Kalisch 1973, *Chromosoma* 41:237-242 (fig.); Kalisch 1975, *Theoretical and Applied Genetics* 46:169-180; Kalisch 1976, *Genet. Res.* 26:275-282; Kalisch and Hägele 1973, *Chromosoma* 44:265-283.

w<sup>zmz</sup>: white - zeste mottled z. Spontaneous is a mutable z w<sup>zm</sup> strain. Phenotypically wild-type. Eyes of z w<sup>zmz</sup> females are zeste (lemon yellow); z w<sup>zmz</sup> males raised at 25-30°C are zeste, at 14-17°C lemon background with red salt-and-pepper mottling. Unstable and reverts to w<sup>zm</sup>. Postulated to be an intragenic duplication (of the zeste-locus interacting part) of the white-locus (Becker 1965, *Nat. Cancer Inst. Monogr.* 18:293-307; Kalisch and Becker 1970, *Molec. Gen. Genetics* 107:321-335). RK3.

w<sup>zmzz</sup>: white - zeste mottled zz. Spontaneous in a mutable z w<sup>zmz</sup> strain. Phenotypically wild-type. Eyes of z w<sup>zmzz</sup> females at 14-30°C light zeste; z w<sup>zmzz</sup> males at 25-30°C light zeste, at 14-17°C zeste. Postulated to be an intragenic duplication of w<sup>zmz</sup> (Kalisch and Becker 1970, *Molec. Gen. Genetics* 107:321-335). w<sup>zmzz</sup> is unstable and reverts to w<sup>zmz</sup>. RK3.

In(1)w<sup>w</sup>: white-white. Spontaneous in a mutable z w<sup>zmz</sup> strain. Phenotypically white. Inversion extends from 3B2-C1 to 4B4-C1. Reverts to w<sup>zmz</sup> inseparably together with re-inversion of In(1)w<sup>w</sup> (Kalisch 1970, *Molec. Gen. Genetics* 107:336-350). RK2.

eyh: eyelisch 3-(0.47% crossover with Sb in 1276 chromosomes). Spontaneous as a cluster in the mutable stock In(1)w<sup>w</sup>, sc z. Eye size reduced, varies from flies with no eyes (7.5% in both sexes) to wild-type overlapping of both eyes (0.3% in females and 5.6% in males). Variability in eye size affected by the genetic background. Eye size reduction stronger in homozygous eyh eyg females (flies with no eyes: 29.2%; wild-type overlapping of both eyes: 0%) and males (34.8%; 0%) and even stronger in homozygous eyh;ey females (53.4%; 0%) and males (42.8%; 0%). RK3.

#### Report of D.T. Kuhn and F.C. Walker

Florida Technological University, Orlando, Florida

w<sup>tuh</sup>: white from tumorous-head (1-1.5) Spontaneous mutant discovered in a sample of flies in a tumorous-head stock that has been maintained at Florida Technological University. Phenotypically identical to the white eyed mutant as described in *D. melanogaster* by Lindsley and Grell (1968). Viability excellent. Position in the white locus not determined.

ct<sup>tuh</sup>: cut from tumorous-head (1-20.0) Spontaneous mutant discovered in several males from a tumorous-head stock that has been maintained at Florida Technological University. Wings are cut to points with edges scalloped as described for *D. melanogaster* by Lindsley and Grell (1968). The eyes and antennae are quite deformed in this stock because of the additive effect of tuh-1, tuh-3 and ct. Viability fair.

#### Report of E.B. Lewis

California Institute of Technology, Pasadena, California

bx<sup>4</sup>: bithorax-4. Lewis, isoallele found in ruPrica chromosome stock. bx<sup>4</sup>/bx<sup>4</sup> is +; bx<sup>4</sup>/Df-bxd100 or bx<sup>4</sup>/Df-P9 shows very weak bx-like effects. RK3.

bx<sup>7</sup>: bithorax-7. Lewis, 59i. From X-rayed Canton-S female. Locus based on recovery of bx<sup>7</sup> Ubx. Variable from nearly overlapping + to more extreme than bx<sup>3</sup>/bx<sup>3</sup>. bx<sup>7</sup>/Ubx has halteres like bx<sup>34e</sup>/Ubx and variable transformation of metanotum to mesonotum; rarely shows a humerus-like structure arising between mesopostnotum and metanotum. Not suppressed by su<sup>2</sup>-Hw. Partially complements bx, bx<sup>34e</sup> and bx<sup>3</sup>. Shows a very slight pbx-like effect over pbx comparable to that seen in e.g., bx<sup>3</sup>/bxd100. RK2.

bx<sup>8</sup>: bithorax-8. Lewis, 1965. From EMS-treated Oregon-R male. Intermediate between bx<sup>34e</sup> and bx<sup>3</sup> in its interactions with other Ubx and bx mutants except that it is not suppressed by su<sup>2</sup>-Hw. RK2.

Ubx<sup>6ld</sup>: Ultrabithorax-6ld. Gloor, 1961. Locus based on recovery of bx<sup>3</sup> Ubx<sup>6ld</sup> and Ubx<sup>6ld</sup> bxd double mutants. Ubx<sup>6ld</sup>/+ resembles Ubx/+ except for very slight wing-like development of the posterior portion of the distal segment of the haltere, similar to that found in bx<sup>3</sup>/bx<sup>100</sup>. Ubx<sup>6ld</sup>/Ubx and Ubx<sup>6ld</sup> homozygotes survive with low viability and with weak bx and pbx effects especially on the halteres and missing first abdominal segment as in bxd. bx<sup>3</sup>/Ubx<sup>6ld</sup> has halteres partially wing-like but lack conversion of metanotum towards mesonotum. RK2.

Pc<sup>3</sup>: Polycomb-3. Lewis, 1965. EMS-treated Oregon-R male. Pub. Denell, 1978, Genetics 90: 277-289; Lewis, 1978, Nature 276:565-570. More extreme allele than Pc. Induced simultaneously with In(3R) (85 C-E to 88 E-F) which is separable from and not required for expression of Pc<sup>3</sup>. Pc<sup>3</sup>/+ (and Pc/+ ) have weak Cbx effects. Cis or trans heterozygotes for Pc<sup>3</sup> and Cbx show strong enhancement of the Cbx phenotype with wings usually strongly haltere-like and mesonotum more reduced than in Cbx/+. Pleiotropic effects of Pc<sup>3</sup> are more intense the greater the number of doses of the bithorax gene complex; this dosage effect is greater if the extra doses are maternally as opposed to paternally derived. RK2.

Df(3R)P2: Deficiency(3R)Pasadena 2. Lewis, 1959. X-rayed Canton-S male. Genetic properties similar to those of Df(3R)Ubx<sup>109</sup>. Deficient for 89E1-2.

Df(3R)P9: Deficiency(3R)Pasadena 9. Lewis, 1973. X-rayed pP male. Pub. 1978, Nature 276: 565-570. All bithorax complex genes deleted. Deficient for 89E1-4 and possibly 89E5.

Df(3R)Ubx<sup>109</sup>: Deficiency(3R)Ultrabithorax-109. Lewis, 1956. X-rayed gl e<sup>s</sup> (Canton-S for the bithorax region) female. Pub. 1978, Nature 276:565-570. 89D1-2 through 89E1-2 deleted.

T(2;3)P10: Translocation(2;3)Pasadena 10. Shaw, 1974. From X-rayed Canton-S male. Pub. 1978, Nature 276:565-570. ss<sup>+</sup> through pbx<sup>+</sup> inserted into 2L. Homozygous lethal in late embryonic stage. Dp(3;2)P10 is homozygous viable; Df(3R)P10 survives as a heterozygote and has a Ubx phenotype. Region from must left of 89C1-2 through 89E1-2 inserted into the 29A-C region.

Tp(3)P20, Ubx<sup>P20</sup>: Transposition(3)Pasadena 20. Lewis, 1974. X-rayed Oregon-R male. 60A<sup>+</sup> to 68E<sup>+</sup> is inserted into 89E; Ubx<sup>P20</sup> is associated with the latter break. Both Dp(3;3)P20 and Df(3L)P20 are derivable from the transposition; Df(3L)P20/+ shows a slight Minute phenotype and appears to have reduced female fertility.

Tp(3)sbd<sup>104</sup>: Transposition(3)stubbloid-104. Lewis, 1947. X-rayed pP bx sr e<sup>s</sup> male. sbd<sup>104</sup> is an extreme sbd<sup>1</sup> type of sbd allele and sbd<sup>104</sup>/Sb is lethal. Both Dp(3;3)sbd<sup>104</sup> and Df(3)sbd<sup>104</sup> are derivable; the latter is deficient for ss. Region from 89B5<sup>+</sup> to end of 89C is inserted into 91B.

Tp(3)S462: Transposition(3) of Shaw 462. Shaw, 1973. X-rayed Canton-S male. Homozygous lethal, but Dp(3;3)S462 is homozygous viable; Df(3R)S462/+ survives and ecloses one or two days later than wild type. Inserts 89D1-2 through 90D1 into 64C-E.

Report of H. Nöthel  
Freie Universität Berlin, Germany

Three radioresistant mutants (rar) were isolated which contribute additively and independently to the relative radioresistance of a laboratory wild population with a long history of acute X-irradiations: 255 generations at 2100 R/generation, subsequently >55 generations at 4000 R/g (ROI<sub>4</sub>). They are characterized by Dose-Enhancement-Factors (DEFs) with respect to the X-ray induction, in immature oocytes, of dominant lethals (A), numerical aberrations of chromosomes X, 2, and 3(B), and sex-linked recessive lethals (C). The rar-factors are effective only in specific germ cell stages.

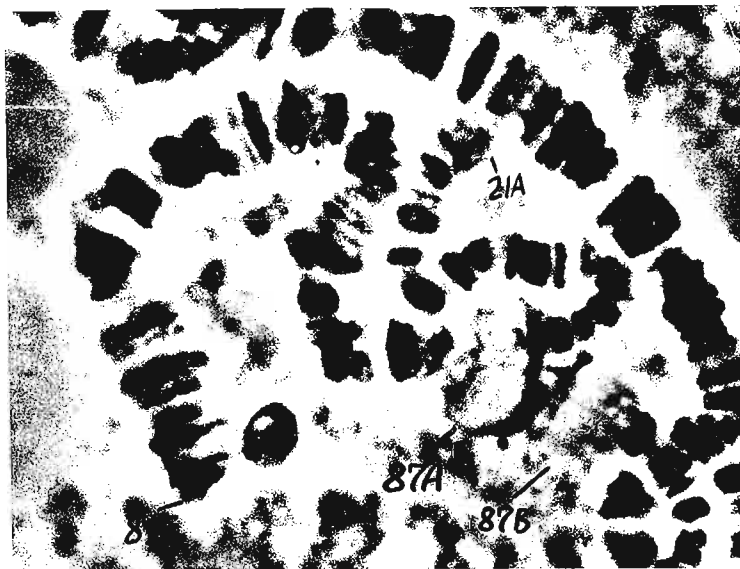
rar-1. 1-?, in RÖI and RÖI<sub>4</sub>, semidominant; DEF 1.3 (A, C); stage: oocytes subsequent to recombination and except 14; effects inhibited by caffeine.

rar-2. 2-? (near centromere), in RÖI and RÖI<sub>4</sub>, semidominant; DEF 1.31 (A, C) and 1.72 (B); stage: as rar-1.

rar-3. 3-49.8, in RÖI<sub>4</sub> only, recessive; DEF 1.58 (A, B) and 1.87 (C); stage: adult oogenesis except 14.

Report of L.I. Mukhina and I.F. Zhimulev

Institute of Cytology and Genetics, Novosibirsk 630090, U.S.S.R.



The following chromosome aberrations were induced in Canton-S males by 5000r X-rays. Irradiated males were mated with females carrying the visible mutations w/w;net/net;e/e. The F<sub>1</sub> males were individually mated with w/w,net/net,3/3 females and the F<sub>2</sub> progenies were tested. The pseudolinkage manifestation of marked loci indicated the presence of translocations between the nonhomologous chromosomes. Salivary breakpoints are given and the new order given only in multi-break cases. Viability of the homozygote translocations is indicated in parentheses (V = viable; L = lethal) and any visible changes of the heterozygote translocations are also indicated.

Fig. 1. Heat-shocked (37°C, 30 min) puffs 87A and 07C1-2 in heterozygote T(2;3)ML443.

T(2;3)ML2 53A1;66C1(L)  
 T(2;3)ML3 34D;70A(L)  
 T(2;3)ML4 40-41;80-81(L)  
 T(2;3)ML5 41A;62B(L)  
 T(2;3)ML8 40-41;80-81(L)  
 T(2;3)ML9 44C/D;64C(L)  
 T(2;3)ML14 40-41;80-81(L)  
 T(2;3)ML76 32E;70A(L)  
 T(2;3)ML122 21F;82E(L)  
 T(1;2;3)ML140 20;40-41;80-81(L)  
 T(2;3)ML192 41;52B;86D;97A(L)  
 New order: 21-41/97A-86D/52B-60;  
 61-86D/52B-41;97A-100  
 T(2;3)ML193 52E1;80-81. Combined  
 with In(3R)ML193 93E-100F(L)  
 T(2;3)ML225 53C;96C(V)  
 T(2;3)ML270 40-41;80-81(L)  
 T(2;3)ML307 50D1;79A(L),  
 L4 interrupted  
 Tp(2-3)ML328 44D;57E;83A1-2(L)  
 New order: 21-44D/57E-60;  
 61-83A1/57E-44C/83A1-100

T(2;3)ML348 35C;70A(L)  
 T(2;3)ML393 40B;63A(L)  
 T(2;3)ML405 57A;92E(L)  
 T(2;3)ML436 40-41;80-81(L)  
 T(2;3)ML443 24D;87C1 (Fig. 1) (L),  
 extra veins  
 T(2;3)ML457 40-41;80-81. Combined  
 with Df(3R)ML457 87A-C(L)  
 T(2;3)ML460 39E;75C(V)  
 T(2;3)ML464 39E;61D(L)  
 T(2;3)ML466 40-41;80-81(L)  
 T(2;3)ML472 32E;96F(L)  
 Tp(2-3)ML473 43A;52B1;89A(L)  
 New order: 21-43A/52B1-60;  
 61-89A/43A-52B1/89A-100  
 T(2;3)ML474 35B;94D(L)  
 T(2;3)ML478 40-41;80-81(L)  
 T(2;3)ML484 40-41;80-81(L)  
 T(2;3)ML486 39E;90D(V)

T(2;3)ML488 40-41;80-81(L)  
 T(2;3)ML490 27E;92B1(L)  
 T(2;3)ML491 40-41;80-81(V)  
 T(2;3)ML494 60E6;99C1(L)  
 T(2;3)ML495 22B1;81F(L)  
 T(2;3)ML498 35A;87F(L)  
 T(2;3)ML499 40-41;80-81(L)  
 T(2;3)ML501 50A;64E(V)  
 T(2;3)ML502 40-41;80-81(L)  
 T(2;3)ML505 21B;97C(L)  
 T(2;3)ML506 55C;85C(L),  
 thin veins L2,L4  
 T(2;3)ML507 40-41;80-81(L)  
 T(2;3)ML508 40-41;80-81(L)  
 T(2;3)ML509 40-41;80-81(L)  
 T(2;3)ML510 40-41;80-81(L)  
 T(2;3)ML511 40-41;80-81(L)  
 T(2;3)ML512 40-41;80-81(L)  
 T(2;3)ML514 40-41;80-81(L)

Report of C.A. Poodry

Thimann Laboratories, University of California, Santa Cruz

Generation of a  $shi^+Y$  chromosome. We have recovered three different  $shi^+Y$  strains by the following method which is based on Brosseau et al. (1961, Genetics 76:339). A  $T(1;Y)P-12, B^{sy+}$  strain with a breakpoint to the left of the shibire locus was obtained from John Merriman (UCLA) and mated to an attached  $XYS.Y^{LBS}$  strain from Dan Lindsley (UCLA). Recombinants were selected which combined the  $T(1;Y)$  and the  $X-Y$ . The double Bar-stone,  $y^+$  phenotype in the male distinguished the recombinant from parental types. Cytological examination confirmed the recombinant chromosome. We mated these males after irradiating them with 4500 rads from a  $Co^{60}$  source to  $y\ cv\ v\ shi^{ts1}\ f\ car$  females. 281 bottles each containing 15 irradiated males and 50-65 females yielded 8 putative  $shi^+Y$  male progeny of which 3 proved fertile.

All three  $shi^+Y$ 's cover tiny chaetae, scalloped and shi. They also carry  $y^+$  and  $B^s$ . The left breakpoints are presumably the same as the parent  $T(1;Y)P-12$  strain, 13 F 1-4. The right breakpoints are unknown, but one, designated  $shi^+Y$  #1, is very small. We have never seen it synapsed with the X in males. It does not cover the  $df(1)sd^{72a}$ , but it does cover the region left deficient by the combination of  $df(1)sd^{72a}$  and  $Dp(1;4)r^+Green$ . Thus the right breakpoint is somewhere in 14A. The  $shi^+Y$  #3 does cover the  $df(1)sd^{72a}$  but does not extend as far as rudimentary. The  $shi^+Y$  #2 has been lost.

The small size of  $shi^+Y$  #1 has been very useful to us in covering a small segment of the genome to demonstrate that all of the bewildering array of temperature-sensitive phenomena in the shibire strains are related specifically to that region of the chromosome and are most certainly the pleiotrophic effects of mutations at a single locus.

Shukla, P.T. Institute of Animal Genetics,  
Edinburgh.  $vg^W$ : Vestigial wingless: A  
dominant allele of vestigial in *D.*  
*melanogaster*.



A dominant allele of vestigial (2-67.0) induced by X-rays. Homozygous lethal. In heterozygotes, wings are absent (see figure) or may be present as blistered wing buds filled with sap. Notopleurals and supraalars absent or reduced in number. Halteres are completely absent or only one is present. Occasionally the halteres are modified into wings or hairy structures. The expression varies in different genetic backgrounds. Fertility and viability excellent.

In combination with vestigial the gene has a very pronounced effect on wings, halteres and major bristles. In the hybrid wings and halteres are completely absent; the scutellum is deformed; the thorax twisted; major bristles are reduced in number; post-scutellars are usually absent. Development of the hybrid is very slow. 20-30% of hybrids eclose. Hybrid eclosion depends on culture conditions. Crowded cultures yield fewer hybrid flies. Viability and fertility of hybrid much reduced. The recombination analysis carried out with vestigial yielded no recombinants in a progeny of 2000 flies. Salivary analysis not carried out.

Report of V. Vasudev and N.B. Krishnamurthy  
University of Mysore, India

$wg^S$ : Single wing. Spontaneous mutant in  $sc\ w^{bl}\ ec\ cv$ . Right or left wing is absent in both males and females. Halteres normal. Besides single wing condition, flies also showed various abnormalities of the mesothorax. The prominent abnormalities included irregular arrangement of hairs, partial to complete absence of mesothoracic bristles and absence or deformation of scutellum.



Report of V. Vasudev, N.B. Krishnamurthy and M.V. Gayathri  
University of Mysore, India

up<sup>WG</sup>: Upheld wing. Mutant induced by 10 mg of 1-amino-2-naphthol-4-sulfonic acid per 100 ml of food medium. The wings are held up at an angle of 60° to the body. Halteres are normal.

Report of J.R.S. Whittle  
Brighton, England

tuf<sup>2</sup>: tufted<sup>2</sup>. 2-55.5. A more extreme and recessive lethal allele which arose following EMS mutagenesis. Wings of tuf<sup>1</sup>/tuf<sup>2</sup> are grossly foreshortened and shaped like a table-tennis bat. They show duplications and triplications of anterior wing structures (veins 2 and 3 and double and triple row marginal bristles) and lack costal bristles. Head bristles and compound eyes are more distorted than tuf<sup>1</sup> homozygote.

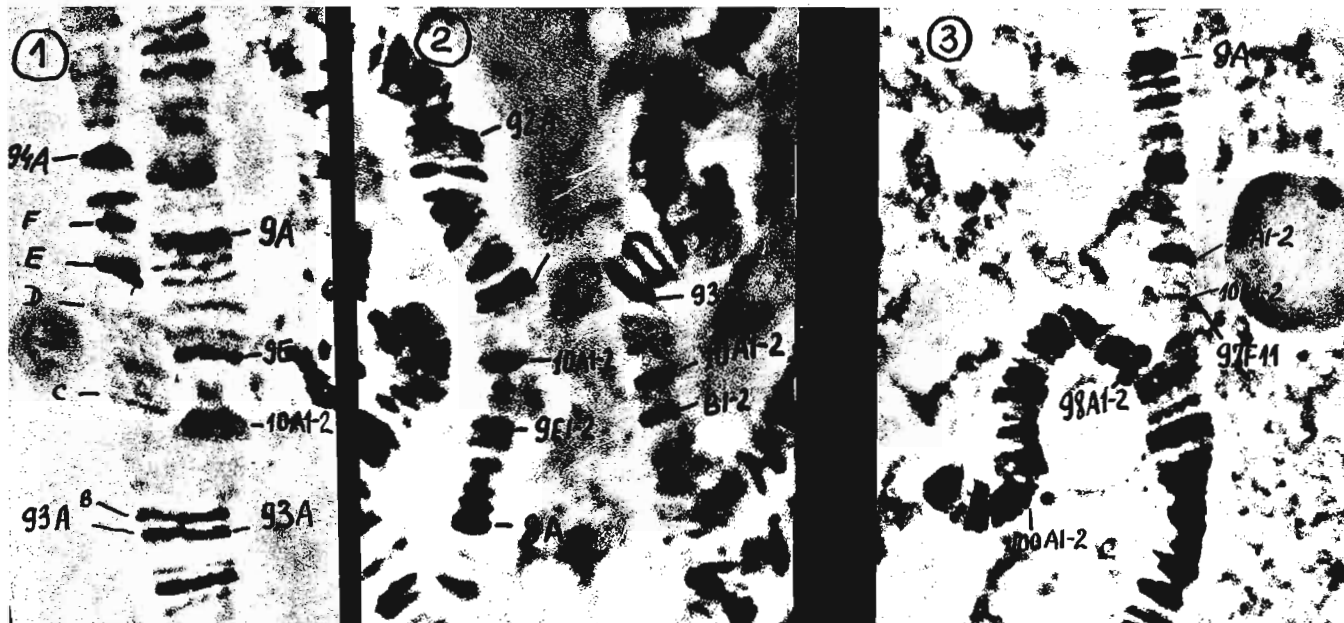
Report of I.F. Zhimulev, E.S. Belyaeva, Yu.E. Khudyakov and G.V. Pokholkova  
Institute of Cytology and Genetics, Novosibirsk, U.S.S.R.

w<sup>Rr</sup>: white Russian red, 1-1.5. Pokholkova, 1976. EMS-induced in yellow strain simultaneously with vGl<sup>18</sup>. Eyes vermillion at hatching, but darken greatly with age and resemble w<sup>+</sup>. w/w<sup>Rr</sup> has light prune at eclosion darkening to garnet-like with age. Eyes of w<sup>Rr</sup>g resemble w<sup>a</sup>, w<sup>Rr</sup> bw have slight lemon yellow color darkening to similar to w<sup>a</sup>. Ocelli in all cases are colorless. Salivary chromosomes normal.

T(1;3)v. New information: one of the breaks is located in the middle part of 10A1-2 band (Figs. 1 and 2). Females Df(1)v<sup>L3</sup>/T(1;3)v are viable and fertile. Males T(1;3)v/Dpv<sup>+</sup>Yy<sup>+</sup> are viable but sterile. Cytologically Dp v<sup>+</sup>Yy<sup>+</sup> overlaps and Df(1)v<sup>L3</sup> removes whole band 10A1-2 (Lefevre 1971, Genetics 67:497-513). Taken together, these data argue that female lethality in homozygotes is related to the second break in 93B7-10 (Lefevre 1970, DIS 45:39). Male sterility depends on dominant effect of either of these breaks.

T(1;Y;3) 154. Corrected information: localization of euchromatin breaks: 10B1-2; 97F11 (Fig. 3).

T(1;3)v and T(1;Y;3)154 were received from Bowling Green Stock Center.



Report of S. H. Bryant - D. pseudoobscura  
Western Illinois University, Macomb, Illinois

The following mutations were discovered in various years in the D. pseudoobscura population in Death Valley, California (Bryant, 1976). The mutants are unlocated, but believed all on chromosome 2, since they appeared in stocks made homozygous by extraction for that chromosome. Linkage data and data on allelism with similar D. pseudoobscura mutants will appear in a future issue of DIS. Stocks will be deposited at the Austin Stock Center in the summer of 1979.

ta: thick arista The aristal shaft is thicker than normal, and appears flattened. The character is fully penetrant at 15-25°C, and a homozygous stock has been maintained since its appearance from a 1975 Death Valley collection. Also appeared in 1977 Death Valley collection. RK1.

dy: dusky Body color blacker than usual; wing color very dark. Wing veins black. Pupal color much lighter than normal. Fully penetrant at 15-25°C; homozygous stock maintained since its appearance from a 1975 Death Valley collection. Also appeared in 1977 Death Valley collection. RK1.

br: brite Eye color bright orange, shows very slight darkening with age. Fully penetrant at 15-25°C; homozygous stock maintained since its appearance from a 1974 Death Valley collection. Also appeared in Death Valley collections from 1975 and 1977. RK1.

wtu: wing tip up Distal 1/4 of wing curled up from 0 to 90°. Wings spread in flies with very curled wing tips, since curling prevents proper wing overlap. Trait not fully penetrant; stock kept for 6 months at 15°C showed only about 50% penetrance. Stock maintained since appearance in 1975 Death Valley collection.

ey: eyeless One or both eyes missing or greatly reduced in size. Often a tuft of bristles in place of missing eye. Not fully penetrant; stock kept for 6 months at 15°C showed about 75% penetrance. Expressivity variable. Stock maintained since appearance in 1977 Death Valley collection.

yh: yellowish Body color yellowish, wing color yellow. Bristle color seems black as normal. In stock with Bare, a balancer chromosome. Fully penetrant at 15-25°C. Stock maintained since appearance in 1977 Death Valley collection. RK1.

oh: outheld Wings held 45-90° from longitudinal axis of body. Effect perhaps enhanced by normal etherization. Not fully penetrant: stock kept at 15°C for 6 months showed about 40% penetrance. Expressivity variable. Stock maintained since appearance in 1975 Death Valley collection.

vg: vestigial Wings reduced to small stubs. Halteres apparently normal. Fully penetrant at 15-25°C. Appeared in 1977 Death Valley collection. Stock lost.

Report of D. DeJianne - D. pseudoobscura  
Rutgers University, New Brunswick, New Jersey

w<sup>1a</sup>: white laetrl. Spontaneous mutant in a laboratory stock of orange Standard (or/or; ST). Sex limited. Females are white eyed. Completely recessive in females. Males are light apricot eyed and variable in expressivity. Complete lack of testes sheath coloration. Viability inferior. Chromosome I, appears allelic to white locus. Phenotypic expression dependent on presence of orange mutation on chromosome III.

Report of C.W. Hinton - D. ananassae  
The College of Wooster, Wooster, Ohio

bb<sup>74</sup>: bobbed-74, 4. Segregated in y ty w<sup>65</sup> stock (1975). Patchy effect on thoracic bristles, weak effect on abdominal sclerites. Allelic to bb<sup>69</sup> (lost).

Bb<sup>2</sup>: Barb-2, 3L. Recovered as one female from pair mating of px female to stw male (1977). Thoracic bristles lengthened and expanded at tips. Good marker in heterozygotes; recessive lethal. Allelism to Kikkawa's Bb inferred from phenotypic similarity and linkage group; mapped 9.2 units left of stw.

bt: bent, 4. Segregated in stw stock (1978). Wings extended laterally and arched downward, often with bubbles of gas or hemolymph. Penetrance strong but viability and fertility depressed.

Bx<sup>2</sup>: Beadex-2, 1R. Found as one male in ca stock (1966). Wings scalloped along both margins in homozygotes; reduced penetrance in heterozygotes. Viability and fertility good. Allelism to Spencer's Bx inferred from phenotypic similarity and linkage group.

c<sup>3</sup>: curved-3, 3R. Found in bb<sup>69</sup> stock (1978). Wings slightly but consistently bent downward and reduced in size. Viability and fertility good. Allelism inferred from 3 linkage and phenotypic similarity.

cop: copper, 1L. Segregated from 3ple testcross (1970). Brownish red eye, distinguishable in combinations with v<sup>2</sup> and g<sup>3</sup>. Good marker.

ct<sup>5</sup>: cut-5, 1L. Segregated in "Bn" stock from Austin (1966). Wings pointed by marginal excisions; antennae thickened and aristae shortened. Good marker.

dc: dorsocentrals, 1L. Segregated from se<sup>A</sup> female (1975). One or more dorsocentral bristles missing in both sexes; wings divergent in females only. Located 3 map units proximal to y.

Dl<sup>2</sup>: Delta-2, 2L. Found as a cluster in progeny of multiply marked male (1968). Longitudinal veins thickened at junctions with margins, sometimes with blisters; eyes roughened slightly. Good marker in heterozygotes; recessive lethal.

Dl<sup>110</sup>: Delta-110, 2L. Recovered as one female from 3ple testcross (1970). Typical Delta phenotype. Included in In(2L)A.

e<sup>76</sup>: ebony-76, 2L. Spontaneous cluster from px female mated to ca; stw male (1976). Good marker, detectable in heterozygotes.

fw: furrowed, 1L. One male found in cd cu ca stock (1968). Oblique wrinkle in eye and malformed thoracic bristles. Penetrance complete but viability and fertility reduced. May be allelic to scar.

g<sup>3</sup>: garnet-3, 1. Found as one male from testcrossed v<sup>2</sup> f<sup>49</sup>/m<sup>2</sup> female (1967). Brownish eye color, darkening with age; distinguishable in combinations with v<sup>2</sup>. Good marker.

gl: glass, 2L. Segregated in e<sup>65</sup> se gv stock (1970). Eyes roughened and reduced in size and pigmentation; ocelli colorless. Satisfactory marker. Mapped 4.3 units to the right of cu in In(2L)A.

gv: groove, 2R. Found in se stock from Tokyo (1969). Eyes reduced or absent, often asymmetrically; consistent anterior midline cleft in dorsal thorax. Previously referred to as eyeless (Hinton 1970) and as reduced (Moriwaki and Tobar 1975). Penetrance complete but viability and fertility reduced. Mapped 9.8 units distal to ma.

Ins(2L+2R)NG2: Inversions of 2L (C,E,B) and 2R (D,A,C) extracted from Futch's New Guinea-2 wild stock and used as a balancer.

lb: little bristle, 1L. Segregated from bri female (1974). Bristles short and fine as in Minutes. Females sterile. Located 11 map units to the left of m. Not tested for allelism with dm or ty.

lw: little wing, 1L. Segregated from px female (1975). Wing somewhat misshapen and reduced to 2/3 normal size; eyes larger than normal. Penetrance reduced in some crosses, otherwise satisfactory marker. Located 15 units to the left of g. Not allelic to m<sup>2</sup>.

m<sup>2</sup>: miniature-2, 1. Segregated in ca stock (1966). Wing reduced to half size and slightly darkened. Good marker.

m<sup>62</sup>: miniature-62, 1. Segregated from px female (1972). Somewhat smaller wings than m<sup>2</sup>. Good marker.

M(2)C: Minute (2) from Cristobal. Found as one male in Cristobal stock (1967). Moderate Minute, sometimes with mosaic expression. Mapped 22 units to the left of cu in In(2L)A.

M(2)53: Minute (2)53. Recovered as one male from px female by bri male mating (1976). Strong Minute with normal cytology; located about 30 map units to the left of e.

M(2)91: Minute (2)91. Recovered as one female from px female mated to bri pe stw px ru male (1971). Slight Minute phenotype. Located 14.4 units to the left of e in In(2L)A.

M(2)108: Minute (2)108. Found as one fly in progeny of ca; stw female mated to bri male (1976). Typical Minute. Normal cytology. Located 18.3 map units to the left of e.

M(2)127: Minute (2)127. Recovered as one fly from bri female crossed to px male (1976). Typical Minute, normal polytenes. Located in distal 2R (44% recombination with pea).

M(2)665: Minute (2)665. Found as one fly from mating of px female to ca; stw male (1976). Typical Minute. Normal polytenes. Mapped 4.5 units to the right of e.

M(3)9: Minute (3)9. Found as one fly from mating of px female to ca male (1977). Typical Minute phenotype. Normal polytenes. Mapped 27 units from stw in 3L.

M(3)172: Minute (3)172. Found as one fly from mating of px female to bri male (1976). Typical Minute. Normal polytenes. Located about 2 map units to the right of pe in 3L.

M(3)281: Minute (3)281. Found as one fly from mating of px female to bri male (1976). Typical Minute. Normal polytenes. Located 13.1 map units proximal to pe in 3L.

M(4)7: Minute (4)7. Recovered as one male from mating of stw female to px male (1978). Good Minute.

pr: purple, 3R. Segregated in wild type stock from Truk (1978). Eye color dull brownish red. Good marker, located 21 map units distal to pc in 3R.

Pr: Prickly, 2R. Recovered as one female from mating of sn<sup>67</sup> female to Tex-3 male (1967). Bristles of head and thorax short, thin and twisted, occasionally missing. Penetrance good in heterozygotes, overlapping homozygotes which are viable and fertile.

Pu<sup>3</sup>: Puffed-3, 2L. Recovered as one female from pc pair mating (1971). Eyes roughened and reduced in size. Good marker in heterozygotes; recessive lethal. Allelism inferred from phenotype and linkage group. Mapped in In(2L)A 7.7 units to the right of cu.

rb: ruby, 1L. Segregated in Texas-3 wild stock (1970). Eye color reddish brown in young flies, becoming more like wild type with age. Good marker. Location suggests allelism with ph.

rd: reduced, 3. Segregated in stw stock (1971). Dorsal thoracic bristles irregularly reduced in size. Satisfactory penetrance, viability and fertility, but difficult to score.

ro: rough eye, 2L. Segregated from multipoint X-linked backcross (1974). Anterior third of eye roughened and reduced. Good marker. Located 3.3 map units distal to cu within In(2L)A.

rst: roughest, 1L. Segregated in cu ca e<sup>65</sup> se stock (1970). Roughened eyes. Good marker.

sc<sup>24</sup>: scute-24, 1L. Recovered as one male from pc pair mating (1971). All scutellars, postalars, postverticals and most orbitals missing. Good marker.

sc<sup>33</sup>: scute-33, 1L. Recovered as one male from pc pair mating (1971). Scutellars, postalars, anterior and median orbitals missing. Good marker.

sn<sup>67</sup>: singed-67, 1L. Segregated in Cristobal stock (1967). Bristles and hairs twisted and shortened. Good marker.

sn<sup>73</sup>: singed-73, 1L. Record of source lost (1973). Phenotype somewhat stronger than sn<sup>67</sup>. Females sterile.

sn<sup>k</sup>: singed-kinky, 1L. Published (Moriwaki and Tobari, 1975) as kk, but shown to be allelic with sn<sup>67</sup>. Phenotype less pronounced than in sn<sup>67</sup>.

T(Y;2)C: translocation (Y;2)C. Arose from +/ca; px/stw female backcrossed to ca; stw male (1976). Marked with ca. Polytene break in 2L at 34B2/3.

T(Y;2;3)A: translocation (Y;2;3)A. Arose from bri/px female mated to ca; stw male (1976). In addition to ca and stw markers, the translocation carries a new Minute mutation. Polytene analysis shows at least 8 euchromatic breaks, but new order not established.

T(Y;3)A: translocation (Y;3)A. Arose in the stock of T(2;3)O (1976). Marked with stw. Polytene analysis suggests chromocentral break in 3.

T(2;3)E: translocation (2;3)E. Arose from backcross of +/ca; bri/stw female to ca; stw male (1975). Marked with ca and stw. Homozygous lethal. Polytene breaks in 20C and 85A.

T(2;3)H: translocation (2;3)H. From mating of bri female to ca; stw male (1975). Marked with ca and stw. Homozygotes lethal. Polytene breaks in 21B and 93C.

T(2;3)J: translocation (2;3)J. From mating of +/ca; px/stw female to ca/ stw male (1976). Associated with new Xasta (Xa, wings reduced to anterior half) mutant. Homozygotes lethal. Polytene analysis showed breaks in 55A and 94B and also in In(3LR) with breaks in 73C and 88B.

T(2;3)K: translocation (2;3)K. From backcross of +/ca; px/stw female to ca; stw male (1976). Coupled with ca and stw markers. Homozygotes lethal. Polytene breaks in 27A and 77A.

T(2;3)L: translocation (2;3)L. From backcross of +/ca; px/stw female to ca; stw male (1976). Coupled with ca and stw markers. Homozygotes lethal. Polytene breaks in 57A and 62A.

T(2;3)M: translocation (2;3)M. From backcross of +/ca; px/stw female to ca; stw male (1976). Coupled with ca and stw markers. Homozygotes lethal. Polytene breaks in 36C and 70C.

T(2;3)N: translocation (2;3)N. From backcross of +/ca; px/stw female to ca; stw male (1976). Coupled with ca and stw markers. Homozygotes viable and fertile. Polytene breaks in 19B and 96A.

T(2;3)O: translocation (2;3)O. From backcross of +/ca; px/stw female to ca; stw male (1976). Coupled with ca and stw markers. Homozygotes lethal. Polytene breaks at 53A and 78A.

T(2;3)P: translocation (2;3)P. From backcross of +/ca; px/stw female to ca; stw male (1976). Coupled with ca and stw markers. Homozygotes lethal. Polytene breaks in 18A and 93B.

T(2;3)Q: translocation (2;3)Q. From backcross of +/ca; px/stw female to ca; stw male (1976). Coupled with ca and stw markers. Homozygotes lethal. Polytene breaks in 55B and 95A.

Tp(2L;2R)Sb: transposition (2L;2R) with Stubble. From backcross of px/bri female to ca; stw male (1976). Bristles of head and thorax half-length with blunt tips. Coupled with ca marker. Heterozygotes viable and fertile; homozygotes lethal. Dp weak but fertile; Df is lethal. Segment 34C7-39B1 transferred from 2L into 2R at 57A1/2.

uk: unkempt, 2R. Segregated in e<sup>65</sup> stock (1974). Bristles of head and thorax irregular in length, curvature and pigmentation in homozygotes; slight expression in heterozygotes. Mapped 18 units to the right of pea.

v<sup>2</sup>: vermilion-2, 1. Segregated in "delta" stock from Austin (1966). Bright red eye color, darkening with age. Good marker.

vg<sup>N</sup>: vestigial-Nicked, 3L. Record of origin (1973) lost. Heterozygotes often show excision of distal wing margin. Semi-lethal in homozygotes; rare survivors show extreme vestigial wing phenotype. Probable allele of Snp.

w<sup>g</sup>: white-golden, 1R. Recovered as one male in e<sup>65</sup> se ma stock (1969). Apricot eye color, distinguishable in combinations with cop, v<sup>2</sup> and g<sup>3</sup>. Good marker.

w<sup>t</sup>: white-tawny, 1R. Record of origin (1973) lost. Somewhat lighter pigmentation than w<sup>g</sup>. Good marker.

y<sup>69</sup>: yellow-69, 1L. Found as one son of a px female (1969). Yellow body, wings and bristles. Good marker.

Report of H.S. Prakash and G. Sreerama Reddy - D. anomelani Reddy and Krishnamurthy  
University of Mysore, Manasa Gangotri, Mysore, India

rp: rotated penis. Spontaneous mutant discovered in a sample of flies collected in Nagarhole (Western Ghats), South India. Male genitalia rotated through 180°; male sterile. Similar to rotated penis (rp) mutant described for D. melanogaster (Morgan et al. 1929) and D. simulans (Agnew 1974).

Report of R.C. Woodruff - D. mauritiana  
Bowling Green State University, Bowling Green, Ohio

cu: curved wing. Woodruff 78h, spontaneous in wild stock. Wings have a phenotype like Curly of *D. melanogaster*. Autosomal recessive mutant.

bg: burgundy eye color. Woodruff 78i, spontaneous in wild stock. Eye is burgundy in color and has no pseudopupil. Sex-linked recessive mutant.

COURTSHIP IN DROSOPHILA MELANOGASTER: A SONG FOR DROSOPHILISTS

D.F. Sewell and J.P. Baggaley  
University of Hull and University of Liverpool, England

It is suggested that this be sung to the approximate tune of "Ain't she sweet?"

Male song

Ain't she swell?  
See her walking round the cell.  
And I ask you very confidentially,  
Ain't she swell?  
  
Ain't she spry?  
She's my ideal kind of fly.  
And I ask you very confidentially,  
Ain't she spry?  
  
Now if she'd care  
For some vibration;  
We're halfway there  
To copulation.  
(Yes sir.)  
  
She's so chic,  
That I think I'll have a lick.  
Yes, I tell you very confidentially,  
She's so chic.  
  
Her body's grown,  
And gives the right smell.  
As has been shown  
By Shorey and Bartell.  
(And others in the literature.)  
  
She's the kind,  
You so very rarely find.  
And I wonder if she realises  
What's on my mind.

Female song

Ain't he fine?  
He's the species same as mine.  
And I ask you very confidentially,  
Ain't he fine?  
  
Ain't he great?  
Hear him coming to vibrate.  
And I ask you very confidentially,  
Ain't he great?  
  
My maiden charms  
Are in confusion;  
Should I accept,  
Or give extrusion?  
(No sir.)  
  
He's so keen,  
That I think we'll make a team,  
And I tell you very confidentially,  
He's so keen.  
  
Just hear that wing,  
Vibration doing.  
It sings the song  
Described by Ewing.  
(With inter-pulse interval.)  
  
Courtship serves,  
To assuage our troubled nerves.  
But how bestial  
To be forced to do it  
For all these perves.

## DROSOPHILA BIBLIOGRAPHY

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D. = DrosophilaD. m. = Drosophila melanogaster

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D. = DrosophilaD.m. = Drosophila melanogaster

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