

# ***DROSOPHILA***

*Information Service*

# 42

*January 1967*

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**DEPARTMENT OF BIOLOGY  
UNIVERSITY OF OREGON  
EUGENE, OREGON**



DROSOPHILA INFORMATION SERVICE

Number 42

January 1967  
(Issued in 1,200 Copies)

## DROSOPHILA MELANOGASTER stock lists

United States

Ames, Iowa . . . . .	34: 9
Ames, Iowa (Genetics). . . . .	38: 9
Amherst, Mass. . . . .	42:11
Arlington, Texas . . . . .	41:27
Austin, Texas. . . . .	35: 6
Baltimore, Md. . . . .	41:22
Berkeley, Calif. . . . .	42:15
Buffalo, N. Y. . . . .	35: 8
Carbondale, Ill. . . . .	40:11
Chapel Hill, N.C. . . . .	41:21
Chicago, Ill. . . . .	40:15
Cleveland, Ohio (Fenn Coll.) . . . . .	38:13
Cleveland, Ohio (West. Res.) . . . . .	42:17
Cold Spring Harbor, N. Y. . . . .	41:27
College Park, Md. . . . .	41:22
De Kalb, Ill. . . . .	37:26
Detroit, Mich. . . . .	41:26
Duarte, Calif. . . . .	38:19
East Lansing, Mich. (Agr. Chem.) . . . . .	34:12
East Lansing, Mich. (Zoology). . . . .	42:16
Emporia, Kansas. . . . .	41:25
Gainesville, Fla. . . . .	34:13
Johnson City, Tenn. . . . .	38:13
Knoxville, Tenn. . . . .	42:18
Lafayette, Ind. . . . .	35:11
Lake Forest, Ill. . . . .	42:19
Lawrence, Kansas . . . . .	35:11
Le Mars, Iowa. . . . .	36:12
Lexington, Ky. . . . .	42:18
Lincoln, Neb. . . . .	40:18
Long Beach, Calif. . . . .	38:12
Los Angeles, Calif. . . . .	34:14
Madison, Wis. (Genetics) . . . . .	40:13
Madison, Wis. (Zoology). . . . .	41:23
Minneapolis, Minn. . . . .	35:11
Medford, Mass. . . . .	41:28
New Brunswick. . . . .	37:27
New Haven, Conn. . . . .	41:29
New York, N. Y. . . . .	42:16
Norman, Oklahoma . . . . .	34:15
Oak Ridge, Tenn. . . . .	42: 9
Oxford, Ohio . . . . .	37:27
Pasadena, Calif. . . . .	42:14
Philadelphia, Pa. (Ins. Can. Res) . . . . .	39: 9
Philadelphia, Pa. (Univ. of Pa.) . . . . .	39:35
Philadelphia, Pa. . . . .	42:17
Pittsburgh, Pa. . . . .	42:13
Rochester, N. Y. . . . .	40:16
Salt Lake City, Utah (Genetics). . . . .	36:14
Salt Lake City, Utah (Surgery) . . . . .	34:26
St. Louis, Mo. . . . .	42:13
Stony Brooke, N. Y. . . . .	42:12
Syracuse, N. Y. . . . .	40:17
Tucson, Ariz. . . . .	35:32
University Park, Pa. . . . .	34:27
Urbana, Ill. . . . .	40:11
Waltham, Mass. . . . .	38:10

Foreign

Argentina. . . . .	41:31
Australia	
Adelaide . . . . .	41:48
Brisbane . . . . .	35:34
Hobart . . . . .	40:23
Melbourne. . . . .	34:28
Sydney . . . . .	42:20
Austria. . . . .	42:36
Belgium. . . . .	41:50
Brazil	
Curitiba . . . . .	35:34
Pôrto Alegre . . . . .	41:56
Sao Paulo. . . . .	42:30
Canada	
Toronto, Ont. . . . .	36:16
Vancouver, B. C. . . . .	36:16
Chile . . . . .	42:28
Columbia . . . . .	42:35
Czechoslovakia . . . . .	41:56
Denmark . . . . .	42:35
Finland	
Helsinki . . . . .	41:52
Turku . . . . .	42:23
France	
Clermont-Ferrand . . . . .	42:19
Gif-sur-Yvette . . . . .	37:31
Lyon . . . . .	42:20
Strasbourg . . . . .	42:26
Germany	
Berlin-Buch. . . . .	42:26
Berlin-Dahlem. . . . .	42:31
Göttingen . . . . .	35:35
Freiburg . . . . .	41:36
Hamburg . . . . .	37:32
Heidelberg . . . . .	37:33
Karlsruhe. . . . .	34:33
Marburg Kahn . . . . .	34:33
Mariensee. . . . .	37:33
München. . . . .	42:27
Münster/Westf. . . . .	40:24
Tübingen . . . . .	42:33
Ghana. . . . .	34:35
Great Britain	
Bayfordbury. . . . .	41:50
Birmingham . . . . .	42:20
Brighton . . . . .	42:28
Cambridge. . . . .	41:45
Chalfont St. Giles . . . . .	41:46
Edinburgh. . . . .	37:34
Glasgow. . . . .	35:36
Harwell. . . . .	42:19
Keele. . . . .	37:35
Leicester. . . . .	37:36
Liverpool. . . . .	41:53
London . . . . .	41:55
Manchester . . . . .	41:54
Sheffield. . . . .	42:35

Greece	
Athens . . . . .	37:36
Thessaloniki. . . . .	42:34
India	
Calcutta (I. S. I.) . . . . .	34:37
Calcutta (U. of C.) . . . . .	42:25
Chandigarh . . . . .	41:47
Hyderabad . . . . .	37:37
Kalyani . . . . .	41:55
New Delhi . . . . .	42:34
Varanasi. . . . .	42:32
Vepery. . . . .	42:33
Israel . . . . .	42:21
Italy	
Milano. . . . .	42:29
Naples (U. of N.) . . . . .	41:47
Naples (Int. Lab.) . . . . .	38:21
Pavia . . . . .	34:39
Rome. . . . .	41:49
Japan	
Anzyo . . . . .	41:47
Chiba-Shi . . . . .	41:53
Hiroshima . . . . .	35:38
Kyoto . . . . .	36:28
Misima. . . . .	34:42
Mitako, Tokyo . . . . .	42:24
Osaka . . . . .	41:41
Sapporo . . . . .	41:38
Tokyo . . . . .	41:36
Korea	
Kongju. . . . .	34:45
Kwangju . . . . .	40:21
Seoul (Chungang U.) . . . . .	41:35
Seoul (National U.) . . . . .	42:36
Seoul (Sung Kyun-Kwan U.) . . . . .	36:31
Seoul (Yonsei U.)m. . . . .	41:56
Mexico . . . . .	41:45
Netherlands	
Haren (Gr.) . . . . .	41:30
Leiden. . . . .	41:38
Utrecht . . . . .	41:42
New Zealand . . . . .	41:56
Nigeria . . . . .	38:21
Norway. . . . .	41:54
Rhodesia . . . . .	42:31
Spain . . . . .	35:41
South Africa	
Johannesburg. . . . .	41:51
Pretoria. . . . .	36:34
Sweden	
Stockholm . . . . .	41:37
Uppsala . . . . .	41:34
United Arab Republic. . . . .	34:47
U.S.S.R. . . . .	42:32

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## OTHER DROSOPHILA SPECIES Stock Lists

United States

Ames, Iowa . . . . .	34:56
Amherst, Mass . . . . .	41:65
Austin, Texas . . . . .	42:42
Baltimore, Md . . . . .	40:28
Berkeley, Calif . . . . .	42:44
Boston, Mass. . . . .	42:44
Chicago, Ill. . . . .	42:47
Cold Spring Harbor, N. Y. . . . .	37:55
Dayton, Ohio. . . . .	42:47
DeKalb, Ill . . . . .	41:64
East Lansing, Mich. . . . .	42:44
Emporia, Kansas . . . . .	41:65
Lexington, Ky . . . . .	42:48
Lincoln, Neb. . . . .	42:47
Los Angeles, Calif. . . . .	36:42
Medford, Mass . . . . .	41:71
New Haven, Conn . . . . .	42:44
New York, N. Y. . . . .	41:63
Oxford, Ohio . . . . .	37:57
Pasadena, Calif . . . . .	34:59
Philadelphia, Pa. . . . .	36:44
Pittsburgh, Pa. . . . .	35:48
Poughkeepsie, N. Y. . . . .	42:48
Raleigh, N. C. . . . .	37:57
Richmond, Va. . . . .	41:66
Rochester, N. Y. . . . .	41:65
Syracuse, N. Y. . . . .	40:28
St. Louis, Mo. . . . .	42:47
Tucson, Ariz. . . . .	41:64
Waltham, Mass. . . . .	38:26

ForeignAustralia

Melbourne . . . . .	34:62
Sydney . . . . .	42:49
Austria . . . . .	42:54
Belgium . . . . .	40:34

Brazil

Pôrto Alegre. . . . .	41:63
São José Do Rio Preto . . . . .	42:45
São Paulo . . . . .	42:46

Canada. . . . .	34:63
Chile . . . . .	42:46
Colombia. . . . .	42:45
Finland . . . . .	42:52

France

Gif-sur-Yvette. . . . .	38:27
Lyon. . . . .	42:51

Germany

Berlin-Buch . . . . .	37:60
Berlin-Dahlem . . . . .	42:52
Freiburg. . . . .	41:73
Marburg . . . . .	34:64
Tübingen . . . . .	42:50

Great Britain

Bayfordbury . . . . .	41:72
Brighton . . . . .	42:52
Edinburgh . . . . .	37:60

London . . . . .	40:30	Sweden . . . . .	41:73
Sheffield . . . . .	34:65	Switzerland . . . . .	42:50
Greece . . . . .	37:61	Venezuela . . . . .	42:52
India			
Calcutta . . . . .	42:51	NEW MUTANTS - Reports of:	
Chandigarh . . . . .	41:69	<u>Melanogaster</u>	
Kalyani . . . . .	41:70	Becker, H. J. . . . .	42:40
Varanasi . . . . .	42:49	Bender, H. A. . . . .	42:38
Israel . . . . .	42:52	Brosseau, G. E. Jr. . . . .	42:38
Istanbul . . . . .	41:63	Clancy, C. W. . . . .	42:37
Italy		Frye, S. H. . . . .	42:37
Milano . . . . .	42:51	Gethmann, R. C. . . . .	42:39
Napoli . . . . .	38:27	Ives, P. T. . . . .	42:39
Japan		Kaplan, W. D. & N. Hayes . . . . .	42:38
Anzyo . . . . .	41:70	Mittler, S. . . . .	42:38
Misima . . . . .	34:66	Nelson, M. K. . . . .	42:37
Osaka . . . . .	41:71	Sturtevant, A. H. . . . .	42:38
Sapporo . . . . .	42:51	Tokunaga, C. . . . .	42:37
Tokyo . . . . .	41:68	Whittinghill, M. & Mother	
Korea		M. Clancy . . . . .	42:37
Seoul, (Chungang U.) . . . . .	41:72	<u>Other Species</u>	
Seoul, (Yonsei U.) . . . . .	41:71	Crumpacker, D. W. . . . .	42:53
Mexico . . . . .	41:72		
Netherlands . . . . .		LINKAGE DATA - <u>Melanogaster</u> - Reports of:	
Haren (Gr.) . . . . .	41:72	Bahn, E. . . . .	42:41
Leiden . . . . .	41:69	Brosseau, G. E. Jr. . . . .	42:41
Utrecht . . . . .	38:27	Ives, P. T. . . . .	42:40
Rhodesia . . . . .	42:52	Thompson, S. R. . . . .	42:41
South Africa . . . . .	36:50	Tokunaga, C. . . . .	42:40
Spain			
Barcelona . . . . .	41:73		
Madrid . . . . .	38:27		

## RESEARCH NOTES

Angus, D. Drosophila collection from British Solomon Islands Protectorate and the Territory of Papua-New Guinea . . . . .	42:96
Cytological evolution in the quadrilineata species group . . . . .	42:112
Arking, Robert & R. Hillman. Analysis of the mode of action of the eyeless-Dominant allele. . . . .	42:69
Bächli, G. & H. Burla. Breeding Drosophila from Mushrooms. . . . .	42:108
Bahn, E. Crossing over within "alleles" determining multiple amylases in D. mel . . . . .	42:84
Barigozzi, C. & Sari Gorla M. Mitotic recombination in Drosophila melanogaster. . . . .	42:74
Beatty, R. A. & N. S. Sidhu. Spermatozoan nucleus length in minutes of D. mel. . . . .	42:93
Berendes, H. D. Amino acid incorporation into giant chromosomes of D. hydei. . . . .	42:102
Bösiger, E., Pentzos, A. & A. Kanellis. Frequency of visible mutants in natural populations of D. subobscura. . . . .	42:88
Bowman, J. T. Another "super-reversion" at the white locus of D. melanogaster. . . . .	42:73
Brosseau, G. E., Jr. The effect of low temperature on the mitotic chromosomes of Drosophila . . . . .	42:61
Burla, H. Induction of mutations in homokaryotypic D. subobscura. . . . .	42:66
Carlson, J. H. A mutant stock exhibiting complete absence of the second longitudinal vein (L <sub>2</sub> ) in D. melanogaster. . . . .	42:75
Clancy, C. W. Identification of ptm (pteridine modifier) as a wild type isoallele of the bw locus. . . . .	42:57
Cole, T. A. Alcohol dehydrogenases in the pupae of D. melanogaster (Oregon R-C). . . . .	42:94
Danieli, G. A. & E. Rodinò. Biochemical estimate of DNA content in D. hydei salivary glands. . . . .	42:63

Datta, Rajat Kumar. Influence of $y^+$ and $sc$ alleles in suppression of yellow color in the sexcomb of <i>Drosophila melanogaster</i> . . . . .	42:62
David, J. & Clavel, M. F. Variations of the rate of egg chambers production in the ovarioles of <i>Drosophila melanogaster</i> . . . . .	42:101
DeMarinis, F. A comparison of the effects of 5-bromouracil and uracil on the facet number of the Bar eye . . . . .	42:71
Di Pasquale, A. & B. L. Zambruni. Histological study of bsp phenotype. . . . .	42:74
Duffy, J. P. & J. Stiles, Jr. Esterase 6 <sup>S</sup> Isozyme in Alaful-1 and Gluful-1 Mutants of <i>Drosophila melanogaster</i> . . . . .	42:103
Forward, K. J. & B. P. Kaufmann. The bipartite nature of the salivary-gland X chromosome . . . . .	42:90
Frye, S. H. Correction in the report of Sara H. Frye (DIS 41:205) . . . . .	42:80
Gethmann, R. C. A reduced viability effect of a ring duplication, $Dp(1;f)65X^{c2}$ . . . . .	42:70
Giesel, B. J. Ring loss in $X^{c2}In(1)w^{vc}$ , $w^{vc}$ f at two temperatures . . . . .	42:60
Heenigsberg, H. F. & L. A. Granobles. Comparative frequencies of detrimental recessives in European and Neotropical populations of <i>D. mel</i> . . . . .	42:56
Hijikuro, S. Studies on the content of beta-alanine and the body color of <i>Drosophila</i> . . . . .	42:87
Hughes, A. M. & P. E. Hildreth. The production of a new mutant in <i>Drosophila melanogaster</i> by low doses of tritium irradiation . . . . .	42:86
Hochman, B. EMS and ICR-100 induced chromosome 4 lethals in <i>D. melanogaster</i> . . . . .	42:59
Hunter, A. S. Preference in mating time of <i>Drosophila mesophragmatica</i> . . . . .	42:65
Imaizumi, T. A new embryonic lethal strain originated from a tip mutation of the X-chromosome. . . . .	42:78
Ives, P. T. Relocation of the $or$ locus closer to $pd$ . . . . .	42:76
Jones, L. P. Instability of an ebony polymorphism of <i>D. melanogaster</i> . . . . .	42:79
Jost, P. Observation of an unexpectedly high frequency of Bar revertants. . . . .	42:92
Jungen, H. Evidence of spontaneous inversion in <i>D. subobscura</i> . . . . .	42:108
Jungen, H. Abnormal sex ratio, linked with inverted gene sequence, in populations of <i>D. subobscura</i> from Tunisia. . . . .	42:109
Kaneko, A., E. Momma & T. Shima. Frequencies of abundance of robusta species group in Hokkaido . . . . .	42:104
Keller, E. C. Jr.; H. E. Keller; & E. Liner. Heterosis in Xanthine Dehydrogenase activity levels. . . . .	42:98
Koref-Santibañez, S. & M. Lamborot. The effect of temperature on mating activity in <i>D. gaucha</i> & <i>D. pavani</i> . . . . .	42:106
Kuhn, D. T. & G. D. Hanks. Suppression of recovery disrupter effect due to a 4th chromosome carrying Minute . . . . .	42:79
Kumar, Sushil, R. P. Sharma & M. S. Swaminathan. Chromosomal rearrangements result resulting from action of a monofunctional alkylating derivative of acridine in salivary gland chromosomes of <i>Drosophila melanogaster</i> . . . . .	42:93
Lefevre, G. Lethal and visible mutation frequencies . . . . .	42:64
Lefevre, G. & L. Moore. Sperm transfer and storage . . . . .	42:77
Lifschytz, E. Induced X-chromosome lethals covered by $Y.w^+$ . . . . .	42:89
MacIntyre, Ross J. & M. R. Dean. In vitro dissociation and reconstitution of acid phosphatase-1 from <i>D. melanogaster</i> . . . . .	42:94
Malich, C. W. & R. M. Binnard. Variations in the transmission of broken chromosomes of <i>Drosophila melanogaster</i> . . . . .	42:97
Mather, W. B. Inter-yearly fluctuation of <i>D. rubida</i> inversion polymorphism . . . . .	42:85
Mensua, J. L. Some factors affecting pupation height of <i>Drosophila</i> . . . . .	42:76
Miller, D. D., N. J. Westphal, & R. A. Voelker. A preliminary note on gene sequence variation reinvestigation in the C chromosome of <i>Drosophila athabasca</i> . . . . .	42:91
Mittler, Sidney & J. R. Gerdy. The effect of various maternal chromosomes on the spontaneous occurrence of XO males . . . . .	42:83
Momma, E. & A. Kaneko. <i>Drosophilidae</i> from three localities in Hokkaido and in Aomori prefecture, based on collections in summer of 1965 . . . . .	42:58
Moriwaki, D. & Y. N. Tobari. Male crossing over in <i>Drosophila ananassae</i> . . . . .	42:81

Muhammed, A. & J. Trosko. A search for photoreactivating enzymes in <i>Drosophila melanogaster</i> extracts. . . . .	42:82
Nilsson, L. R. On the ontogeny of alkaline phosphatases in <i>D. m.</i> . . . .	42:60
Nöthel, H. An estimation of the number of active oogonia per ovariole in <i>D. melanogaster</i> females . . . . .	42:55
Novitski, E., M. E. Myszewski & H. Goldin. Dicentric chromosomes in <i>Drosophila</i> . . . . .	42:105
Ogaki, M., E. Nakashima-Tanaka & S. Murakami. Ether resistance in <i>D. melanogaster</i> . . . . .	42:73
Ohba, S. & F. Sasaki. Electrophoretic variants of esterase in <i>Drosophila virilis</i> . . . . .	42:75
Ohba, S. & F. Sasaki. Polymorphisms of electrophoretic variants of esterase in a natural population of <i>Drosophila virilis</i> . . . . .	42:77
Okada, T. Character continuities in the caenogenetic organs of <i>Drosophilidae</i> . . . . .	42:60
Palomino, H. & E. del Solar. Density, fertility and gregarian tendency in <i>Drosophila melanogaster</i> . . . . .	42:106
Parkash, Om. On the so-called conditioned (incomplete) and absolute lethals in <i>Drosophila melanogaster</i> . . . . .	42:109
Pasztor, L. M. A tandem metacentric which generates unstable rings . . . . .	42:107
Paterson, H. E. & L. Tsakas. The identification of <i>Drosophila sequyi</i> (smart). . . . .	42:73
Pipkin, S. B. Polymorphism of octonol dehydrogenase in neotropical <i>Drosophila</i> species. . . . .	42:65
Poulson, D. F. Developmental effects of mutants at the Notch locus in <i>D. mel</i> . . . . .	42:81
Poulson, D. F., T. Hyde, & K. Oishi. Interactions between SR spirochetes from different species of <i>Drosophila</i> . . . . .	42:81
Rezzonico Raimondi, G. & A. Gottardi. Growth Behaviour of embryonic cells of <i>Drosophila melanogaster</i> cultured in vitro. . . . .	42:103
Rasmuson, B. Modulation of the puff in the tip of the X-chromosome in <i>D. mel</i> . . . . .	42:72
Shamay, E. Induced spermatogonial crossing over in the centromere regions of chromosomes II and III of <i>D. melanogaster</i> . . . . .	42:68
Shima, T., A. Kaneko & E. Momma. On some aspects on the copulation, insemination reaction and sperm storage in two species of quinnaria group. . . . .	42:100
Tedeschi, M. Valéria & Luiz Edmundo de Magalhães Sex-limited effect of detrimental autosomic chromosome in " <i>D. Melanogaster</i> " . . . . .	42:65
Tsacas, L. <i>Drosophilids</i> of the Ivory Coast . . . . .	42:83
Uda, F. & T. Taira. Cyclic nucleotides and adenylosuccinic acid found in <i>D. mel</i> . . . . .	42:112
Van Delden, W. Adaptation in <i>D. melanogaster</i> populations started from an inbred line.. . . .	42:62
Wagoner, D. E. The linkage group - karyotype relationship in the house fly ( <i>Musca domestica</i> L.) . . . . .	42:78
Waldner-Stiefelmeier, R. & P. S. Chen. Proteolytic digestive enzymes in <i>D. mel</i> . . . . .	42:99
Wasserman, M. Collections of <i>Drosophila</i> from Central Mexico. . . . .	42:67
Whitten, M. J. Quantitative measurement of the effect of temperature on the penetrance of the eye mutant, witty, in <i>D. melanogaster</i> . . . . .	42:110
Whittinghill, M. Somatic spot confirmation of the chromosome arm loci of <i>in ri</i> and <i>p.</i> . . . .	42:80
Yoon, Jong Sik & R. H. Richardson. A study of the relationship between ovariole number and egg production in <i>Drosophila pseudoobscura</i> . . . . .	42:64

#### TECHNICAL NOTES

Blaylock, B. G. A population cage for counting adult <i>Drosophila</i> populations . . . . .	42:113
Carlson, J. H. A source of paper for <i>Drosophila</i> cultures . . . . .	42:122
Danieli, G. A. & F. Rodinò. A homogeneous medium for studies of labelled precursors incorporation in growing larvae . . . . .	42:119
Erk, F. C. An expressivity scale for melanotic tumors. . . . .	42:122
Forbes, C. Plastic planchets as Radiation Exposure Holders for <i>Drosophila</i> . . . . .	42:112
Hess, O. New "one way" <i>Drosophila</i> culture containers made of plastics . . . . .	42:114
Hoch, F. Cover Glass removal . . . . .	42:116
Hooper, G. B. An automatic trapping apparatus for <i>Drosophila</i> . . . . .	42:115
Johnson, W. W. A simple <i>Drosophila</i> activity maze . . . . .	42:117
Jost, P. & W. Sistrom. Effects of humidity on increasing the yield from vial pair matings. . . . .	42:123

MacMahon, J. A. & D. Taylor. Another method for removing <i>Drosophila</i> from traps . . .	42:118
Petit, C. An isotherm chamber for behavioral observations. . . . .	42:114
Seecof, R. L. An apparatus for drawing micropipettes . . . . .	42:121
Shelton, E. E. A density method of collecting and cleaning eggs from <i>D. mel.</i> . . . .	42:118
Stalker, H. E. Techniques for improving salivary chromosome preparations . . . . .	42:119
Teaching Note . . . . .	42:123
Bibliography. . . . .	42:124
Personal and Laboratory News. . . . .	42:160
Availability of Materials . . . . .	42:161
Announcements . . . . .	42:162
Directory, Geographical . . . . .	42:164
Directory, Alphabetical . . . . .	42:185
Poem . . . . .	42:188

#### EDITOR'S COMMENTS

#### Reprints of DIS Notes

By arrangement with the University of Oregon Press, it is possible to order reprints of DIS notes. If the order accompanies the note at the time of its submission, the costs will be:

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Furthermore, notes, or reprints of pages, from past DIS's, starting with 34 can also be reprinted (but only at the time that DIS is being printed). Because more time and effort is required to reproduce them than those currently being printed, however, it is necessary to add an additional charge of \$2.00 per order.

Please keep in mind that these will be simply reprints of the pages on which the note or notes are to be found, and that other notes, or sections thereof on the same page may precede and follow yours. It will be the responsibility of the author to mark out the unwanted notes, add a title cover, or otherwise focus attention on the pertinent sections.

In the future, your editor will make an effort to arrange those notes, of which reprints have been requested, in such a position that they will head, or be close to the top of, the pages on which they appear. If one worker or group of workers submits several notes, it will be necessary to indicate whether these should follow successively after each other, to appear together in one reprint, or whether they should be separated, to facilitate their arrangement in individual reprints.

For previous "Quotability of Notes" see DIS #38

Barker, J. S. F. 34:113a; 34:113b; 40:66; 41:195	Ménsua, J. L. 41:105; 41:108-109
Bennett, Jack 30:159; 33:126; 33:178; 36:58; 36:131; 37:138; 38:43	Ogita, Z. 32:142; 33:151a; 33:151b; 34:97; 36:103; 37:142
DeMarinis, F. 38:68; 41:119; 41:147	Patterson, H. E. 34:100
Gersh, E. S. 20:86; 30:115; 41:89a	Prevosti, A. 27:110; 33:154
Gregg, Thomas G. 37:83; 40:85	Seki, T. 36:115
Hess, O. 36:74; 40:41	Sokoloff, A. 32:171; 33:161; 40:90
Hiroyoshi, T. 37:90	Strömnaes, Ö. 40:77
Kikkawa, H. 3:49; 5:25; 13:72; 14:51; 22:71a; 22:72c; 23:90; 24:82; 25:108; 27:96a; 27:96b; 31:125; 32:131; 33:142; 34:89a; 34:89b; 36:83; 38:88	Thompson, P. E. 28:163; 30:155; 36:123; 38:60
Lee, W. R. 38:87; 40:63	Tsukamoto, M. 28:164; 30:79; 32:87; 32:162; 33:172; 34:109; 36:124
Lefevre, G. 30:129; 36:85; 36:86; 37:98a; 37:98b; 39:120	Ulrich, H. 25:130; 25:131; 27:116; 27:117; 27:124; 28:164; 29:170; 30:155; 31:168; 33:172; 33:173
	Wedvik, H. 36:127
	Würgler, F. E. 35:102

Some misunderstanding still exists about the restrictions on the "quotability" of DIS notes. The procedure we have followed, and will continue to follow, is based on the presumption that contributions to DIS are of a preliminary or fragmentary nature, are often casually speculative, are ordinarily not exposed to critical and helpful review before printing and are subject to errors, typographical and otherwise, during preparation. We therefore will not give blanket permission for quotation of "all my notes, past, present and future"; even if a worker were willing to assume this risk, the Editor of DIS cannot. He respectfully requests, therefore, that permission to quote a note not be given until after the worker has examined it carefully as it finally appeared in DIS.

Many colleagues have commented on the very low frequency of typographical errors in DIS. Full credit for this goes to the secretarial staff (at various times Mrs. Gail McKerrick, Mrs. Wendy Greer, Mrs. Dorothy Parker, Mrs. E. Novitski, Miss Diana Childress, Mrs. Frances Stuart) who have shown extraordinary competence and patience in transferring the submitted copy to the printed page. Unhappily, we cannot be equally enthusiastic about a certain fraction of the submitted copy. In fact, if an error appears in DIS, it is more likely than not the result of a direct transcription of an error found in the original copy, and not discovered during proofreading. We would be helped if all copy, especially stock lists, were carefully proofread by the originator, and retyped when necessary.

OAK RIDGE, TENNESSEE: OAK RIDGE NATIONAL LABORATORYWild Stocks

- a-1 Canton-S  
a-2 Oregon-R  
a-3 Oregon-R-C  
a-4 Samarkand  
a-5 Swedish-c

Chromosome 1

- b-1 B/C(1)DX, y f  
b-2 car bb  
b-3 dow/FM6, y<sup>31d</sup> sc<sup>8</sup> dm B  
b-4 f  
b-5 f BB/C(1)DX, y f  
b-6 fa<sup>g</sup>  
b-7 fu<sup>59</sup>/C(1)DX, y f  
b-8 l(1)J1 sc<sup>J1</sup>/Dp(1;f)24  
b-9 m f car/C(1)DX, y w f  
b-10 mal  
b-11 nd rb  
b-12 pn  
b-13 ptg<sup>3</sup> v m g<sup>2</sup> sd f/C(1)DX, y f  
b-14 rst<sup>2</sup>/FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> B  
b-15 sc cv v f B/C(1)DX, y f  
b-16 sc ec cv ct<sup>6</sup> v g/In(1)dl-49, y Hw m<sup>2</sup> g<sup>4</sup>  
b-17 sc ec cv ptg<sup>3</sup> v/C(1)RM, y v f car  
b-18 sc z ec ct  
b-19 spl  
b-20 sw  
b-21 v  
b-22 v f su(f)  
b-23 v m g<sup>2</sup> sd f  
b-24 w  
b-25 w<sup>a</sup>  
b-26 w<sup>a</sup> fa fa<sup>no</sup> rb/C(1)DX, y f  
b-27 w<sup>a</sup> fa spl  
b-28 w<sup>a</sup> fa<sup>g</sup> fa<sup>no</sup> rb/C(1)DX, y f  
b-29 w<sup>a</sup> fa<sup>g</sup> spl sn<sup>3</sup>  
b-30 w<sup>a</sup> fa<sup>no</sup> spl rb/C(1)DX, y f  
b-31 w<sup>a</sup> nd<sup>2</sup> rb/C(1)DX, y f  
b-32 w<sup>a</sup> pdf  
b-33 w<sup>a</sup> spl rb  
b-34 w<sup>e</sup> bb<sup>1</sup>/C(1)DX, y f/B<sup>S</sup>Y  
b-35 w<sup>sp</sup>  
b-36 y  
b-37 y ac sc pn/C(1)DX, y f  
b-38 y ac sc pn w rb cm ct<sup>6</sup> sn<sup>3</sup> ras<sup>2</sup> v dy g<sup>2</sup>  
f car/C(1)RM, In(1)sc<sup>S1</sup> + dl-49, sc<sup>S1</sup>  
v f car

- b-39 y B/C(1)DX, y f  
b-40 y bb<sup>1-3a</sup>/C(1)RM, y w/y<sup>+</sup>Y  
b-41 y bb<sup>1-74</sup>/C(1)RM, y w/y<sup>+</sup>Y  
b-42 y bb<sup>1-158</sup>/C(1)RM, y w/y<sup>+</sup>Y  
b-43 y bb<sup>1-452</sup>/C(1)RM, y<sup>2</sup> su(w<sup>a</sup>) w<sup>a</sup> bb/y<sup>+</sup>Y  
b-44 y cv v f  
b-45 y cv v f car  
b-46 y fa<sup>n</sup> sn<sup>3</sup>  
b-47 y Hw/C(1)RM, In(1)sc<sup>S1</sup>L sc<sup>8R</sup> + S, sc<sup>S1</sup>  
sc<sup>8</sup> w<sup>a</sup> B  
b-48 y l(1)451/FM6, y<sup>31d</sup> sc<sup>8</sup> dm B  
b-49 y w bb<sup>ds</sup>  
b-50 y w fa<sup>no</sup>  
b-51 y w spl sn<sup>3</sup>  
b-52 y w<sup>a</sup>  
b-53 y w<sup>a</sup> m f car  
b-54 y w<sup>a</sup> spl rb  
b-55 y<sup>2</sup> cv v f  
b-56 Dp(1;1)sc<sup>V1</sup>, y<sup>2</sup> sc cv v f car·y<sup>+</sup>/B<sup>S</sup>Y  
b-57 y<sup>2</sup> v mal<sup>bz</sup>  
b-58 y<sup>2</sup> w<sup>cf</sup>

Chromosome 2

- c-1 a px or  
c-2 al b c sp  
c-3 al dp b pr c px sp  
c-4 al dp b pr Bl c px sp/In(2LR)O, dp<sup>lvI</sup>  
Cy pr cn<sup>2</sup>  
c-5 b cn c bw  
c-6 Bl L<sup>2</sup>/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>  
c-7 bw  
c-8 bw<sup>D</sup>  
c-9 cn bw  
c-10 d  
c-11 da/In(2L)Cy + In(2R)Cy, Cy  
c-12 Fo/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
c-13 lt stw<sup>3</sup>  
c-14 lys  
c-15 lys rc  
c-16 lys rc<sup>2</sup>  
c-17 lys<sup>2</sup> rc<sup>2</sup>/In(2LR)O, dp<sup>lvI</sup> Cy pr cn<sup>2</sup>  
c-18 M(2)S2<sup>10</sup>/In(2L)Cy + In(2R)Cy + Dp(2;2)  
41<sup>2</sup>, Cy pr  
c-19 net al ex ds S ast shv ho rub/SM1, al<sup>2</sup>  
Cy sp<sup>2</sup>  
c-20 nw<sup>D</sup> Pu<sup>2</sup> Pin<sup>Yt</sup>/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
c-21 Pin  
c-22 Pin<sup>Yt</sup>/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
c-23 Pu<sup>2</sup>/SM1, al<sup>2</sup> Cy sp<sup>2</sup>

c-24 px sp Pin<sup>2</sup>/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 c-25 rc pr  
 c-26 sp<sup>2</sup> bs<sup>2</sup>  
 c-27 Sp J (L<sup>2</sup>) Pin/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>  
 c-28 Sp lys d/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 c-29 Sp lys rc J/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 c-30 Sp rc<sup>2</sup>/In(2L)Cy + In(2R)Cy, Cy Bl L  
 c-31 spd<sup>fg</sup>  
 c-32 stw<sup>3</sup> c  
 c-33 Tft/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 c-34 vg  
 c-35 vg<sup>U</sup>/In(2L)t + In(2R)Cy, Roi bw<sup>45a</sup> sp<sup>2</sup>  
                     or<sup>45a</sup>

Chromosome 3

d-1 Bd<sup>G</sup>/In(3R)C, l(3)e  
 d-2 bx<sup>34e</sup>  
 d-3 ca  
 d-4 ca K-pn  
 d-5 ca<sup>nd</sup>/TM3, Sb Ser  
 d-6 cu kar  
 d-7 cv-c sbd<sup>2</sup>  
 d-8 D<sup>3</sup> H/In(3L)P, Mé  
 d-9 e  
 d-10 e<sup>s</sup>  
 d-11 Gl Sb/LWM  
 d-12 H<sup>2</sup>/Tp(3)Vno, Vno  
 d-13 jvl  
 d-14 p<sup>p</sup> bx sr e<sup>s</sup>  
 d-15 p<sup>p</sup> Ki  
 d-16 Pr/In(3R)C, e  
 d-17 Pr<sup>K</sup> Dr/In(3L)P + In(3R)P  
 d-18 R Ly/In(3L)P, gm  
 d-19 red  
 d-20 ru  
 d-21 ru h th st cu sr e<sup>s</sup> ca  
 d-22 ru h th st cu sr e<sup>s</sup> Pr ca/TM1, Mé ri  
 d-23 ry<sup>2</sup>  
 d-24 se  
 d-25 se ss k e<sup>s</sup> ro  
 d-26 sr gl  
 d-27 ss<sup>a</sup>  
 d-28 st  
 d-29 st c(3)G ca/In(3LR)Ubx<sup>130</sup>, Ubx<sup>130</sup> e<sup>s</sup>  
 d-30 st in ri p<sup>p</sup>  
 d-31 st ry<sup>2</sup> sr e<sup>s</sup>  
 d-32 st sbd e<sup>s</sup> ro ca  
 d-33 su(Hw)<sup>2</sup> bx bxd/TM1, Mé ri  
 d-34 ve h th

Chromosome 4

e-1 Ce<sup>2</sup>/spa<sup>Cat</sup>

e-2 ci ey<sup>R</sup>  
 e-3 ci gvl ey<sup>R</sup> sv<sup>n</sup>  
 e-4 ci gvl spa<sup>Cat</sup>/ci<sup>D</sup>  
 e-5 ci<sup>D</sup>/ey<sup>D</sup>  
 e-6 sv<sup>n</sup>  
 e-7 spa<sup>pol</sup>

Multichromosomal Stocks

f-1 v/Y<sup>bb</sup>  
 f-2 br<sup>3</sup> dx<sup>st</sup>; ed Su(dx)<sup>2</sup>  
 f-3 In(1)w<sup>m4</sup>; E(var)7/In(2L)Cy + In(2R)Cy  
 f-4 v; In(2R)bw<sup>VDe1</sup>/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 f-5 y/C(1)DX, y f; dp lys rc pr  
 f-6 FMA3, y<sup>2</sup>/+; b pr Elp/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 f-7 y; In(2L)Cy + In(2R)Cy/In(2LR)bw<sup>V1</sup>; bw<sup>V1</sup>  
                     Sb/In(3L)D, D  
 f-8 FM6, y<sup>31d</sup> sc<sup>8</sup> dm B/In(1)AM, y<sup>2</sup>; pr Bl/  
                     SM1, al<sup>2</sup> Cy sp<sup>2</sup>; Tp(3)Vno, Vno/  
                     In(3LR)Ubx<sup>130</sup>, Ubx<sup>130</sup> e<sup>s</sup>; pol  
 f-9 +/C(1)DX, y f; bw; st; pol  
 f-10 spl; E(spl)  
 f-11 v; In(2R)bw<sup>VDe1</sup>/SM1, al<sup>2</sup> Cy sp<sup>2</sup>; M(4)/ey<sup>D</sup>  
 f-12 In(1)sc<sup>8L</sup> sc<sup>S1R</sup> + dl-49, y<sup>31d</sup> sc<sup>-</sup> v f  
                     (B)/y w<sup>a</sup> N<sup>Nic</sup>/y<sup>+</sup> Y w<sup>+</sup>; ca K-pn  
 f-13 b pr Bl/SM1, al<sup>2</sup> Cy sp<sup>2</sup>; Tp(3)Vno, Vno/  
                     In(3LR)Ubx<sup>130</sup>, Ubx<sup>130</sup> e<sup>s</sup>  
 f-14 bw; st  
 f-15 SM1, al<sup>2</sup> Cy sp<sup>2</sup>/In(2LR)bw<sup>V1</sup>, bw<sup>V1</sup>;  
                     Sb/In(3LR)Ubx<sup>130</sup>, Ubx<sup>130</sup> e<sup>s</sup>  
 f-16 bw; sim-4/ci<sup>D</sup>

Inverted Chromosomes

g-1 In(1)65, y f/B<sup>Sy</sup>  
 g-2 In(1)481, y bb<sup>1-481</sup>/FM6/y<sup>+</sup>Y  
 g-3 In(1)AB, y f  
 g-4 In(1)AM/T(1;3)65, y  
 g-5 In(1)B<sup>M1</sup>, cm B<sup>M1</sup>/C(1)DX, y f  
 g-6 In(1)dl-49, y Hw m<sup>2</sup> g<sup>4</sup> (see b-16)  
 g-7 In(1)dl-49, y v f car bb<sup>amg</sup>/C(1)DX, y f  
 g-8 In(1)dl-49, y w lz<sup>s</sup> bb/In(1)sc<sup>L8</sup>, sc<sup>L8</sup>  
                     car m w<sup>a</sup>  
 g-9 In(1)dl-49, y<sup>2</sup> sc w<sup>a</sup>/B<sup>Sy</sup>  
 g-10 In(1)dl-49 + B<sup>M1</sup>, sc v B<sup>M1</sup>  
 g-11 In(1)EN, y bb/y<sup>+</sup>Y  
 g-12 In(1)l-v59, y l(1)v59/C(1)RM, y w/y<sup>+</sup>Y  
 g-13 In(1)l-v132, y l(1)v132/C(1)RM, y w/y<sup>+</sup>Y  
 g-14 In(1)l-v231, y l(1)v231/C(1)RM, y w/y<sup>+</sup>Y  
 g-15 In(1)rst<sup>3</sup>, rst<sup>3</sup>  
 g-16 In(1)rst<sup>3</sup>, y rst<sup>3</sup> car bb?/C(1)RM, In(1)  
                     sc<sup>S1L</sup> sc<sup>8R</sup> + S, y<sup>31d</sup> sc<sup>S1</sup> sc<sup>8</sup> w<sup>a</sup> B  
 g-17 In(1)rst<sup>3</sup>, rst<sup>3</sup> ras v f/C(1)DX, y f; bw  
 g-18 In(1)sc<sup>4</sup>, y sc<sup>4</sup>

- g-19 In(1)sc<sup>4</sup>, y sc<sup>4</sup> cv v B/C(1)DX, y f/B<sup>S</sup>y  
 g-20 In(1)sc<sup>4</sup>L sc<sup>8</sup>R, y sc<sup>4</sup> sc<sup>8</sup> cv v B/C(1)  
 DX, y f/B<sup>S</sup>y  
 g-21 In(1)sc<sup>4</sup>L sc<sup>8</sup>R + S, y sc<sup>4</sup> sc<sup>8</sup> w<sup>a</sup> B/C(1)  
 RM, y<sup>2</sup> su(w<sup>a</sup>) w<sup>a</sup> bb/y<sup>+</sup>y  
 g-22 In(1)sc<sup>7</sup>, sc<sup>7</sup>  
 g-23 In(1)sc<sup>8</sup>, sc<sup>8</sup> v f car  
 g-24 In(1)sc<sup>8</sup>, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup>  
 g-25 In(1)sc<sup>8</sup> + dl-49, y<sup>31d</sup> sc<sup>8</sup> v f B (see  
 g-39)  
 g-26 In(1)sc<sup>8</sup> + S, y<sup>-</sup> ac<sup>-</sup> sc<sup>8</sup> w<sup>a</sup>/y<sup>+</sup>y  
 g-27 In(1)sc<sup>9</sup>, sc<sup>9</sup> w<sup>a</sup> t Bx  
 g-28 In(1)sc<sup>8</sup>L sc<sup>8</sup>R, sc<sup>8</sup>L sc<sup>8</sup>S1 w<sup>a</sup> m car/  
 In(1)dl-49, y w lz<sup>s</sup> bb  
 g-29 In(1)sc<sup>8</sup>S1 + dl-49, y<sup>62k</sup> sc<sup>8</sup>S1 v f car/  
 C(1)DX, y f  
 g-30 In(1)sc<sup>8</sup>S1L sc<sup>4</sup>R, sc<sup>8</sup>S1 sc<sup>4</sup> cv v B/C(1)  
 RM, y w/y<sup>+</sup>y  
 g-31 In(1)sc<sup>8</sup>S1L sc<sup>4</sup>R, sc<sup>8</sup>S1 sc<sup>4</sup> cv v B/C(1)  
 RM, y f/B<sup>S</sup>y  
 g-32 In(1)sc<sup>8</sup>S1L sc<sup>4</sup>R + AB, sc<sup>8</sup>S1 sc<sup>4</sup> w<sup>a</sup> car/  
 C(1)DX, y f  
 g-33 In(1)sc<sup>8</sup>S1L sc<sup>8</sup>R + dl-49, sc<sup>8</sup>S1 sc<sup>8</sup> v B  
 car/C(1)DX, y f  
 g-34 In(1)w<sup>m4</sup>, w<sup>m4</sup> (see n-12)  
 g-35 In(1)w<sup>m4</sup>L N<sup>264-84</sup>R, y w<sup>m4</sup> N<sup>264-84</sup> sn/  
 FM3, y<sup>31d</sup> sc<sup>8</sup> dm B l/B<sup>S</sup>y<sup>+</sup>  
 g-36 In(1)y<sup>3P</sup>, y<sup>3P</sup> B  
 g-37 In(1)y<sup>3PL</sup> sc<sup>8</sup>S1R + S, y<sup>-</sup> ac<sup>-</sup> sc<sup>-</sup>; Cy/sc<sup>19i</sup>  
 g-38 In(1)y<sup>4</sup>, y<sup>4</sup>  
 g-39 In(1LR)sc<sup>8</sup>V1, v.sc<sup>8</sup>V1 y<sup>+</sup>/In(1)sc<sup>8</sup> + dl-49,  
 y<sup>31d</sup> sc<sup>8</sup> v f B  
 g-40 In(1LR)l-v139, w<sup>m139</sup> l(1)v139/C(1)DX,  
 y w f/Y/Y  
 g-41 In(2LR)lt<sup>m3</sup>/SM5  
 g-42 In(2LR)lt<sup>m12</sup>/SM5  
 g-43 Tp(3)Vno, Vno/H<sup>2</sup> (see d-12)  
 g-44 Basc, In(1)sc<sup>8</sup>S1L sc<sup>8</sup>R + S, sc<sup>8</sup>S1 sc<sup>8</sup> w<sup>a</sup> B  
 g-45 Biny, In(1)sc<sup>8</sup>L sc<sup>8</sup>S1R + dl-49, y sc<sup>-</sup> v  
 f B/y l(1)J1259 w m f/y<sup>+</sup>y  
 g-46 FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> B (see b-14)  
 g-47 FM3 (see g-35)  
 g-48 FM4, w dm f/C(1)DX, y f  
 g-49 FM6, y<sup>31d</sup> sc<sup>8</sup> dm B (see b-3)  
 g-50 FM6, y<sup>31d</sup> sc<sup>8</sup> w dm B/C(1)DX, y f/Y/y<sup>+</sup>y

AMHERST, MASSACHUSETTS: AMHERST COLLEGE

Changes in and additions to the DIS 40:9-11 list.

Wild Stocks

- 1 Oregon-R Inbreeding: generation 485  
 on 66g5.  
 6 Discarded  
 7 Discarded  
 9 Samarkand 204 inbreeding; generation  
 172 on 66g5.

Chromosome 1

- 15 Bx<sup>3</sup>  
 50a m g<sup>53d</sup>  
 99 Discarded  
 99a y sp-w  
 102 Discarded  
 103a z<sup>11G3</sup>  
 103b z<sup>11G3</sup>

Chromosome 2

- 104 a px or If  
 104a al cl b c sp  
 105a b If

- 105b b nub pr  
 107 b Bl vg bw/Cy, bw<sup>45a</sup> or<sup>45a</sup> sp<sup>2</sup>  
 108 b Bl vg bw/Roi, bw<sup>45a</sup> or<sup>45a</sup> sp<sup>2</sup>  
 118a sca<sup>65l31</sup>

Chromosome 3

- 146 se<sup>50k</sup>  
 149 sr Dl<sup>61l5</sup> e/DCxF  
 150a ss<sup>ax</sup>

Multiple Chromosomes

- 163a b vg<sup>51h25</sup>; se<sup>50k</sup>  
 163b bw; h  
 166a Cy, bw<sup>45a</sup> or<sup>45a</sup> sp<sup>2</sup>; se<sup>50k</sup>/T(2;3),  
 Bl vg<sup>51h25</sup>; se<sup>50k</sup>  
 166b Cy L<sup>4</sup> sp; se<sup>50k</sup>/T(2;3), Bl vg<sup>51h25</sup>;  
 se<sup>50k</sup>  
 166c Cy SM5; TM1/T(2;3)g<sup>63d29</sup>  
 169 vg<sup>51h25</sup> bw; se<sup>50k</sup> e<sup>60k</sup>

STONY BROOK, NEW YORK: STATE UNIVERSITY OF NEW YORK  
Department of Biological Sciences

Wild Stocks

001 Oregon-R  
 002 Swedish-B  
 003 Amherst-34  
 004 Canton-S  
 005 Crimea  
 006 Florida (inbred)  
 007 Formosa  
 008 Kyoto, Japan  
 009 Lausanne-S  
 010 Salta, Argentina  
 011 Seto, Japan  
 012 St. Louis-7  
 013 Stephenville  
 014 Tuscaloosa, Alabama  
 016 Varese, Italy

Chromosome 1

101 w  
 102 w<sup>a</sup>  
 103 w m f  
 104 y ct<sup>6</sup> ras<sup>2</sup> f  
 105 f/y  
 106 ec ct<sup>6</sup> (s) car/CLB  
 107 Ins(1)sc<sup>S1</sup> sc<sup>8</sup>, B w<sup>a</sup>  
 108 f  
 111 lz/y  
 114 v<sup>1</sup> (suppressable)  
 115 y<sup>2</sup> cho<sup>2</sup>  
 116 y sn<sup>3</sup> v<sup>36f</sup> (v<sup>36f</sup>-unsuppressed)  
 117 y<sup>2</sup> su<sup>51c15</sup> ras<sup>2</sup> v<sup>1</sup> f (v<sup>1</sup>-suppressed)  
 141 Dp(1)sc<sup>S1</sup>, y w f  
 151 In<sup>49</sup>, y fa<sup>n</sup>  
 161 car l(C1+1)/Ins(1)sc<sup>S1</sup> sc<sup>8</sup>, B w<sup>a</sup>  
 162 car l(B2+6)/Ins(1)sc<sup>S1</sup> sc<sup>8</sup>, B w<sup>a</sup>  
 163 car l(A3+3)/Ins(1)sc<sup>S1</sup> sc<sup>8</sup>, B w<sup>a</sup>  
 191 Y/y<sup>1</sup> ac In<sup>49</sup> B<sup>M1</sup> ♂ and Y/y f:= ♀

Chromosome 2

201 al dp b pr cn c px sp/Cy sp  
 202 al dp b pr cn c px sp/Cy pr cn sp  
 203 ap<sup>(49j)</sup>/Cy  
 204 b vg  
 205 bw  
 206 dp<sup>02</sup>  
 207 al b c sp<sup>2</sup>  
 208 al Sp b L<sup>34</sup>/Cy

209 b pr cn  
 211 Ins(2L, 2R)Cy bw<sup>V2</sup>/al dp b pr cn c px sp  
 212 L<sup>2</sup>  
 213 Frd/Cy L<sup>4</sup>  
 214 vg  
 215 b Tft vg/b vg  
 216 cn Su-Pm/Cy cn vg Pm  
 217 cn Su-Pm Tac/Pm (dp b c?)  
 218 net S ho/Cy E-S  
 219 rn/Cy  
 220 stw<sup>2</sup>/Cy  
 221 Tac sp/Cy sp  
 222 Tft/Cy sp  
 223 Tft/Cy<sup>+</sup> sp  
 224 Df(2)bw<sup>5</sup>/Cy sp  
 225 Df(2)dp<sup>V51</sup>/Cy  
 226 In(2R)bw<sup>A</sup>/Cy  
 227 In(2LR)bw<sup>V29</sup>/Cy  
 228 In(2LR)bw<sup>V30k1</sup>/Cy  
 229 Ins(2L, 2R)Cy bw<sup>V34</sup>/b vg  
 230 In(2)b bw<sup>VDe1</sup>/b lt l cn mi sp  
 231 In(2)bw<sup>VDe2</sup>/Rev l  
 232 In(2)bw<sup>V13</sup>/Cy  
 233 In(2R)bw<sup>V30k10</sup>/Cy

Chromosome 3

301 red  
 302 se  
 303 ru h th st cu sr e<sup>s</sup> ca  
 304 bar-3  
 305 Gl Sb/LVM  
 306 Ly Sb H/Inv LVM  
 308 se ss  
 309 ss  
 310 st<sup>brk</sup>  
 311 st  
 312 Ly Sb/Inv LVM  
 313 M(3)y Sb/Inv LVM  
 314 M(3)y Gl/Inv LVM

Chromosome 4

402 spa  
 403 sv<sup>n</sup>

Multichromosomal

502 Cy sp/al dp b pr cn c px sp; ci ey<sup>R</sup>  
 503 dp; e

504 pr cn; by  
 505 b(Su-er)<sup>+</sup> bw; st er  
 509 Cy/Pm ds<sup>33k</sup>; H/Sb-C  
 511 Cy/tu bw; st su-tu  
 514 SM1, al<sup>2</sup> Cy sp<sup>2</sup>/Pm; Ubx/Sb  
 515 Su-er tu bw; st er su-tu  
 516 tu bw; er<sup>+</sup>(su-tu)<sup>+</sup>  
 517 tu bw; Sb bx<sup>D</sup>/T(2;3)Xa  
 518 Frd/Cy L<sup>4</sup>; Sb/TM5 (Ser)  
 522 v; bw; e  
 523 y<sup>2</sup> v f; bw  
 524 v; In(2R)bw<sup>V2</sup>/v; +

602 T(2;3;4)bw<sup>V30k18</sup> Ins(2LR)/Cy  
 603 T(2;3)bw<sup>V5</sup> st/st  
 604 T(2;3)bw<sup>V5</sup> st/T(2;3)p<sup>Gr</sup> st  
 605 T(2;3)bw<sup>VDe4</sup>/Cy  
 606 T(2;3)p<sup>Gr</sup>/Cy  
 607 T(2;3)rn/Cy  
 609 T(2;4)pr cn; ci ey<sup>R</sup>

Tumor Stocks (also see 515-517)

902 tu A<sub>2</sub>  
 903 tu B<sub>3</sub>  
 904 tu-55G<sup>Jacobs</sup>

Translocations

601 T(2;3)Mé/ru h th st cu sr e<sup>S</sup> Pr ca

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Department of Biology

The stock list remains essentially the same as that appearing in DIS 41:28 with the following changes:

Stocks Removed from List

200 al  
 202 bw sp  
 212 dp cn bw  
 213 L<sup>4</sup>  
 217 net  
 223 stw<sup>2</sup>  
 226 d/SM5, al<sup>2</sup> Cy lt<sup>V</sup> sp<sup>2</sup>  
 227 d-b/Cy  
 228 d  
 310 ru h th st cu sr e<sup>S</sup> Pr cn/TM1, Mé ri  
 314 Ubx<sup>130</sup>, e<sup>S</sup>/Sb (Ubx/Sb)

400 ey<sup>2</sup>  
 500 al<sup>2</sup> Cy lt<sup>3</sup> L<sup>4</sup> sp/Pm, ds<sup>33k</sup>; Ubx<sup>130</sup>, e<sup>S</sup>/Sb (Cy L/Pm; Ubx/Sb)  
 503 l(2)55i/SM1, al Cy sp; pol  
 504 sc<sup>S1</sup> B Ins w<sup>a</sup> sc<sup>8</sup>; Ins SM1, al<sup>2</sup> Cy sp<sup>2</sup>/dp b Pm ds<sup>33k</sup>; C Sb/Ubx<sup>130</sup> e<sup>S</sup>; pol  
 600 M(3)S34/T(2;3)Mé

Additions to List

125 y Hw/Ins(1)sc<sup>S11</sup>, S, sc<sup>8R</sup>, sc<sup>S1+8</sup> w<sup>a</sup>  
 B (RM)  
 315 h<sup>1</sup> gs th

ST. LOUIS, MISSOURI: WASHINGTON UNIVERSITY  
Department of Biology

Wild Stocks

Canton  
 Oregon

Chromosome 2

px  
 cn bw sp

Multichromosomal

bw st  
 Pm dp b/Cy sp<sup>2</sup>; Sb/D CXF

Chromosome 1

w m f  
 y ct<sup>6</sup> f car/y f

Chromosome 3

se ss ro

PASADENA, CALIFORNIA: CALIFORNIA INSTITUTE OF TECHNOLOGY

Note: The following is a list of additions, corrections, and losses to the list of stocks from this laboratory in DIS 41. The convention for listing new stocks can be illustrated by an example: the new stock, dor/y pn, is given the number 28b and should be inserted after 28 in the Pasadena DIS 41 stock list.

Stock Additions to DIS 41 list:Chromosome 1

16b bri  
28b dor/y pn  
49b gg<sup>3</sup>  
91b sc cho t (replaces sc cho)  
137b v bo

Chromosome 2

205b al dp b pr Hx  
371b pym/Cy

Chromosome 3

552b ru lxd by

601b We/Mé, In(3R)C, e l(3)e

Chromosome 4

634b spa<sup>p65</sup>

Attached-XY

672b Y<sup>S</sup> X·Y<sup>L</sup> Ins(1)EN<sup>R</sup> dl-49, Y<sup>S</sup> y·Y<sup>L</sup>/y X·Y;  
bw; e; ci ey

Translocations-2;3

825b T(2;3)dp<sup>D</sup>, dp<sup>D</sup>/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
833b T(2;3)Sb<sup>V</sup>, Sb<sup>V</sup> In(3R)Mo, In(3LR)P-35/  
SM1, al<sup>2</sup> Cy sp<sup>2</sup>; Ubx<sup>130</sup>

Corrections to DIS 41 list:

## For:

38 f BB<sup>36b</sup>/y f:=  
63 lz<sup>BS</sup> lz<sup>46g</sup> ras<sup>4</sup> v/y f:=  
68 M(1)Sp/In(1)AM  
402 spd gt-2/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>  
640 en<sup>X-S</sup>; S/Cy (1;2)  
677 In(X<sup>c2</sup>)w<sup>vc</sup>/y Hw dl-49 m<sup>2</sup> g<sup>4</sup> f<sup>5</sup>

715 Df(3)sbd<sup>105</sup>/Xa  
747 In(1)rst<sup>3</sup> car bb

## Read:

f BB<sup>36f</sup>/y f:=  
lz<sup>BS</sup> lz<sup>46f</sup> lz<sup>g</sup> ras<sup>4</sup> v/y f:=  
M(1)Sp/FM6, y<sup>31d</sup> sc<sup>8</sup> dm B  
spd gt-4/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>  
en<sup>X-S/FMA3</sup>, y<sup>2</sup>; al S ast ho/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
In(X<sup>c2</sup>)w<sup>vc</sup>/dl-49, y w lz<sup>s</sup> female; In(1)dl-49,  
y w lz<sup>s</sup>/sc<sup>8</sup>·Y male (unstable ring-X stock  
of Catcheside)  
Df(3)sbd<sup>105</sup>, p<sup>p</sup> sbd<sup>105</sup> bx sr e<sup>s</sup>/LWM  
In(1)rst<sup>3</sup>, y rst<sup>3</sup> car bb

Stock losses:Chromosome 1

91 sc cho (see 91b in above list of  
additions)  
92 sc cv v dwx/FM6, y<sup>31d</sup> sc<sup>8</sup> dm B  
180 y<sup>2</sup> dvr<sup>2</sup> v

Closed-X

675 X<sup>c</sup>, y/y f:=

Duplications

728 Qn(1)w, w<sub>5</sub>/y f:=

Translocations-1;3

801 T(1;3)v, sc cv v f/In(1)y<sup>4</sup>, y<sup>4</sup> w<sup>a</sup> sn

Translocations-3;4

840 T(3;4)a, D/Mé

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA  
Department of Zoology

Wild Stocks

1 Canton-S  
 3 Samarkand  
 5 +3  
 6 Oregon R-C

Chromosome 1

100 B  
 101  $bb^1$   $w^e/ClB$  (with floating Y)  
 102 br  
 103  $br_{ec}/y$   
 104  $Bx^3$   
 106  $cm\ ct^6\ sn^3$   
 \* 108 ec  
 110  $Hw^{49c}/FM1, y^{31d}\ sc^8\ w^a\ lz^s\ B$   
 111  $In(1)dl-49, y\ Hw\ M^2\ g^4/y\ f$   
 113  $kz\ g^2\ B/y$   
 115  $Df(1)N^8/dl-49, y\ Hw\ m^2\ g^4$   
 116  $sc\ ec\ cv\ ct^6\ v\ g^2\ f/FM3, y^{31d}\ sc^8\ dm\ B\ 1$   
 117 sd  
 119  $sx\ vb^2\ sy/FM4, y^{31d}\ sc^8\ dm\ B$  (extra Y floating)  
 122 w  
 123  $w^a$   
 124  $w^{bl}$   
 125  $w^{bl}\ ec/FM4$   
 126  $w^{cf}$   
 127  $w^{cf}/FM4$   
 129  $w^{ch}\ wy$   
 130  $w^{ch}\ wy/FM4$   
 131  $w^{co}\ sn^2$   
 132  $w^{co}\ ec/FM4$   
 133  $w^e$   
 140 y  
 141  $y\ ac\ v/y\ v$   
 142  $y\ ac\ sn^3\ v$   
 143  $y\ ac\ sn^3\ B$   
 144  $y\ ac\ sn^3\ sx\ vb^2\ sy/y^2\ sc^{51}\ B\ v\ w^a\ sc^8$  (dl-49)  
 145  $y\ ac\ v$   
 150  $y\ ac\ Dp\ w^a, (w^a)_2/y^2\ sc\ w^{56l}\ ec$   
 151  $y\ f/y\ ac\ Dp\ (w^a)_2$   
 155 y sc  
 156  $y\ sc\ m\ f^5$   
 157  $y\ sc/y\ ac^+\ sc^+\cdot Y$   
 159  $y\ sn^3$   
 160 y w

161  $In(1)y^2\ In(1)w$   
 165  $y^2\ cv\ v\ f$   
 180  $X^{c2}\ f\ car/y\ f$   
 181  $y\ w/w^{vc}/w$  (ring)  
 183  $w^{vc}/In(1)dl-49, y\ w\ lz^s/sc^8\cdot Y$   
 185  $X\cdot Y^L, sc\ cv\ v\ f/y/Y^S$   
 186  $y\ w\ bb/X\cdot Y^S, y\ w\ Y^S/Y^L\cdot bb^+\ ac^+\ y^+\ sc^8$

Chromosome 2

200 a px sp  
 205 al dp b pr c px sp/Cy pr  
 206 al dp b pr cn vg c a px bw mr sp/ $S^2$  Cy  
 $lt^3\ pr^+\ Bl\ cn\ L^4\ sp^2$   
 208 b  
 212 bw  
 214 c  
 215 cg c/U  
 216 cl  
 218 cn bw  
 220  $In(2L)t\ esc\ c\ sp/SM5, al^2\ Cy\ lt^v\ sp^2$   
 225  $l(2)gl\ cn\ bw/Cy\ al^2\ lt^3\ L^4\ sp^2$   
 226  $L^4$   
 228 pr cn ix/SM5  
 229 pr en  
 230  $Sco/Ins(2L + R)Cy, dp^{txI}\ Cy\ pr\ Bl\ cn^2\ L^4$   
 232  $vg^{no}$   
 233  $vg^{no}$

Chromosome 3

301 cp in ri  $p^D$   
 302 cu  
 303  $cv-c\ sbd^2$   
 308 Gl Sb/LVM  
 310 gro/D  
 311 h  
 312  $Ly/D^3$   
 313 mwh  
 314 mwh e  
 315  $p^D$   
 316 ru h st  $p^D\ ss\ e^s$   
 317 ru h th st cu sr  $e^s\ ca$   
 319 se  
 320 se h  
 321 se dn Sb/LVM  
 322 se Ly dn/LVM  
 323 ss  
 324  $ss^a$

325 ss<sup>a-B</sup>  
 327 tet<sup>Bd</sup> bilat  
 328 th st cp  
 329 th st Pc Scx p<sup>p</sup> dd/TM1, Mé ri  
 340 In(3LR)TM; Mé/In(3LR)Ubx e<sup>s</sup>  
 350 Pc/T(2,3)Mé

Chromosome 4

402 bt ey<sup>R</sup> sv<sup>n</sup>  
 403 bt<sup>D</sup>/ci<sup>D</sup>  
 404 ci  
 405 ci<sup>w</sup>  
 408 ci<sub>2</sub>ey<sup>R</sup>  
 412 ey<sup>2</sup>  
 413 ey, ophthalmoptera  
 414 bubble-eye, eyeless  
 420 M-4/ey<sup>D</sup>  
 421 sv<sup>n</sup>

Multichromosomal

500 w; vg  
 501 x<sup>c2</sup>, t; en/y f  
 502 y; Cy/Pm  
 503 y f/Y; fes Dp(1)y<sup>+</sup> Tft/Cy x sc<sup>19</sup>/Y;  
       fes Dp(1)y<sup>+</sup> Tft/Cy

510 y; mwh  
 511 y; D/tra red  
 512 ec; sv<sup>n</sup>  
 513 y w f/y<sup>31d</sup> sc<sup>8</sup> g<sup>3n</sup> v; Df(4), Dp y<sup>+</sup>  
       ac<sup>+</sup>/ey<sup>D</sup>  
 514 b; p<sup>p</sup>  
 515 Cy/Pm; D/Sb  
 516 vg; se  
 517 se h; ci ey<sup>R</sup>  
 518 sn<sup>3</sup>; cn bw; ri  
 519 y; Gla/cn bw; ri  
 520 y f; bw; e; ci ey<sup>R</sup>

Triploid

552 y<sup>2</sup> sc w<sup>a</sup> ec/FM4  
 553 oc sn<sup>3</sup>/FM6, y<sup>31d</sup> sc<sup>8</sup> dm B  
 554 cm ct<sup>6</sup> sn<sup>4</sup>/FM6  
 555 g<sup>H</sup> wy<sup>2</sup>/FM6

Translocations

603 T(1;2)Bld/ClB  
 606 T(1;2)sc<sup>19</sup> y<sup>+</sup>/y f:=; fes sc<sup>19i</sup>; b pr/  
       Cy dp<sup>th</sup> pr  
 607 Xa/Sb Ubx  
 608 T(2;3)Met/dp

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Department of Biological Sciences

(Only unusual stocks listed)

1	D InsCXF/dsx; g sd Zw <sub>B</sub> . = & w m f Zw <sub>B</sub>	4	D InsCXF/h tra p; g sd Zw <sub>B</sub> . = & w m
2	D InsCXF/dsx; y Zw <sub>A</sub> . = & Zw <sub>A</sub>		f Zw <sub>B</sub>
3	D InsCXF/dsx; w m f Zw <sub>B</sub> /sc <sup>8</sup> .Y.B <sup>S</sup>	5	D InsCXF/h tra p; y Zw <sub>A</sub> . = & Zw <sub>A</sub>
		6	D InsCXF/h tra p; w m f Zw <sub>B</sub> /sc <sup>8</sup> .Y.B <sup>S</sup>
		7	D InsCXF/h tra p; y w sn <sup>3</sup> /sc <sup>8</sup> .Y.B <sup>S</sup>

EAST LANSING, MICHIGAN: MICHIGAN STATE UNIVERSITY  
Department of Zoology

Wild Stocks

Oregon-R  
 Crimea  
 Samarkand  
 Swedish-B

Chromosome 1

w  
 w<sup>a</sup>  
 w<sup>e</sup>  
 w  
 w m f  
 w m  
 v  
 g

f  
 m  
 pn  
 rb<sup>s1</sup>  
 y  
 Basc  
 m<sup>D</sup>/FM3  
 B

<u>Chromosome 2</u>	ca	y; bw; e; ci ey <sup>R</sup> (1;2;3;4)
	st	
bw	se	<u>Attached-XY</u>
dp	e se	
cn bw	ru h th st cu sr e <sup>S</sup> ca	Y <sup>S</sup> w y·Y <sup>L</sup> y <sup>+</sup> /0/y w/0
Cy/Pm		
vg	<u>Multichromosomal</u>	<u>Attached-X</u>
pk cn		
SD-5	bw; st (2;3)	y m f/ y w f:=
SD-72	v; bw (1;2)	
b pr	bw; e (2;3)	<u>Closed-X</u>
b	w; bw; st (1;2;3)	
<u>Chromosome 3</u>	Cy/Pm; H/In(3R)Mo, Sb, sr (2;3)	X <sup>c1</sup> , y v/y w f:=
	dp b Pm/Cy sp <sup>2</sup> ; Sb/D Ins CXF	
	(2;3)	
e		

CLEVELAND, OHIO: WESTERN RESERVE UNIVERSITY  
Department of Biology

<u>Chromosome 1</u>	b8 ltd	<u>Chromosomes 1 and 2</u>
	b9 net	
a1 B	b10 vg	e1 B; ho
a2 m		e2 B; vg
a3 sc cv v f	<u>Chromosome 3</u>	e3 v; bw
a <sup>4</sup> w		
a5 y w m f/y f:=	c1 e	<u>Chromosomes 2 and 3</u>
<u>Chromosome 2</u>	c2 gl <sup>3</sup>	
	c3 h	f1 b; se
b1 al dp b pr	c4 ri sbt e <sup>2</sup>	f2 bw; st
b2 b	c5 se	f3 dp; se
b3 bw	c6 st	f4 ltd; h
b4 cn	<u>Chromosome 4</u>	<u>Chromosomes 1, 2, 3 and 4</u>
b5 cn bw		
b6 dp	d1 ci ey <sup>R</sup>	g1 y; bw; e; ci ey <sup>R</sup>
b7 ho	d2 ey <sup>2</sup>	

PHILADELPHIA, PENNSYLVANIA: HAHNEMANN MEDICAL COLLEGE  
Graduate School: Department of Genetics and Anatomy

<u>Chromosome 1</u>	b23 w <sup>bl</sup>	b55 w <sup>i</sup>
	b24 w <sup>Bwx</sup>	b56 w <sup>i</sup> f <sup>3</sup> bb <sup>N</sup>
b10 w	b30 w <sup>cf</sup>	b60 w <sup>r</sup>
b11 w <sup>a</sup>	b32 w <sup>ch</sup> wy	b70 w <sup>sat</sup>
b12 w <sup>a2</sup>	b33 w <sup>co</sup>	b71 w <sup>sp</sup> (sp-w)
b13 w <sup>a3</sup>	b35 w <sup>col</sup>	b81 w <sup>t</sup> fw
b14 w <sup>a4</sup>	b36 w <sup>cp</sup>	b85 w <sup>w</sup> f <sup>5</sup>
b15 w <sup>a</sup> su-f/y f:=	b40 w <sup>e</sup>	b86 w <sup>w</sup> rb
b20 w <sup>bf</sup> f <sup>5</sup>	b41 w <sup>ec3</sup>	b90 z
b21 w <sup>bf2</sup>	b50 w <sup>h</sup>	b91 z w <sup>11E4</sup>
		b92 z w <sup>zm</sup>

1 Lexington, Kentucky, wild type

LAKE FOREST, ILLINOIS: LAKE FOREST COLLEGE  
Department of Biology

Wild Stocks

1 Urbana-1119.10

Chromosome 1

2 B  
 3 f  
 4 f odsy car  
 5 f odsy fu  
 6 odsy fu  
 7 sc cv v f  
 8 w  
 9 y  
 10 y w f

Chromosome 2

11 bw  
 12 dp  
 13 L<sup>4</sup>  
 14 M<sub>a</sub><sup>33</sup>/bal.  
 15 vg

Chromosome 3

16 e<sup>11</sup>  
 17 se  
 18 Ly Sb/bal.

Chromosome 419 ey<sup>2</sup>Multichromosomal

20 v e<sup>11</sup>  
 21 w vg e<sup>11</sup>

Attached-X

22 y f/f B car\*  
 23 y f/f odsy fu  
 24 y f/ odsy fu  
 \* see duplications

Duplications

25 f B car (long duplication:  
 f B<sup>+</sup> odsy<sup>+</sup> B odsy car)

Inversions

26 SM1, Cy/102; Ubx<sup>130</sup>/Sb  
 27 Cy/Pm; D/Sb  
 28 Muller-5

Translocations

29 T(Y;2)A

Unanalyzed

Four chromosome 1 lethals  
 (homozygous). All are over  
 Muller-5

HARWELL, DIDCOT, BERKS., ENGLAND: MEDICAL RESEARCH COUNCIL  
Radiobiological Research Unit

Wild Stock

1 Oregon-K

Inbred Lines

2 light (F<sub>150</sub>)  
 3 straw (F<sub>150</sub>)

Chromosome 14 sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup>Chromosome 2

5 a px  
 6 a px pd bw  
 7 b  
 8 bw  
 9 cn  
 10 Cy/Bl L<sup>2</sup>  
 11 el  
 12 el b  
 13 el b pr lt ltd cn/Cy

14 el b pr lt ltd cn a px pd bw/  
Cy

15 lt  
 16 ltd  
 17 ltd cn  
 18 lt stw<sup>3</sup>  
 19 pd  
 20 pd bw  
 21 pr  
 22 pym/Cy  
 23 stw

CLERMONT-FERRAND, FRANCE: FACULTÉ DES SCIENCES DE CLERMONT-FERRAND  
Laboratoire de Zoologie et de Biologie Cellulaire

Wild Stocks

Oregon-R-C

Chromosome 1

w  
 B  
 y ct f

Chromosome 2

vg

Chromosome 3

e

BIRMINGHAM, ENGLAND: UNIVERSITY OF BIRMINGHAM  
Department of Genetics

Wild Stocks

1 Edinburgh  
 2 Wellington  
 3 Florida

13 b cn vg  
 14 dp cn bw  
 15 Cy/al dp b pr c px sp

Chromosome 3

16 se  
 17 e  
 18 st  
 19 st p<sup>p</sup>  
 20 se cp e

Inbred for 70-700 Generations

4 Oregon  
 5 Samarkand  
 6 6 C/L

Chromosome 1

7 B  
 8 w  
 9 w m B  
 10 y v f

Chromosome 2

11 cn  
 12 vg

Inversions

26 ClB/+  
 27 Muller-5  
 28 M<sup>e</sup> Sb e/He  
 29 Cy L/Pm; H/Sb  
 30 y sc<sup>S1</sup> B dl-49 w<sup>a</sup> sc<sup>8</sup>  
 31 s g<sup>3</sup>/FM6 y<sup>31d</sup> sc<sup>8</sup> dm B  
 32 Df(3)ry<sup>k</sup>/Ubx<sup>130</sup>  
 33 M 34 Dfd ry<sup>1</sup>/M<sup>e</sup> Ins ri sbd<sup>1</sup>  
 34 Mn ma-l/y<sup>31d</sup> sc<sup>8</sup> dm B; TM3  
       Sb Ser y<sup>+</sup>/cu<sup>2</sup>kar  
 35 Ins(2L+2R)Cy cn<sup>2</sup>/In(2LR)Pm al<sup>4</sup>  
       ds<sup>33k</sup> lt bw<sup>vi</sup>; In(3LR)DcxF  
       D/Sb  
 36 y; Ins(2L+2R)Cy cn<sup>2</sup>/In(2LR)Pm  
       al<sup>4</sup> ds<sup>33k</sup> lt bw<sup>vi</sup>; In(3L)D,  
       D/Sb

Attached-X

37 y x w

SYDNEY, AUSTRALIA: UNIVERSITY OF SYDNEY  
Department of Animal Husbandry

Wild Stocks

5 strains from N. S. W.  
 and Victoria

y w  
 sc cv v f

Chromosome 2

b  
 b j  
 j  
 net  
 sca  
 vg  
 Cy/Pm  
 Cy L<sup>4</sup>/Pm  
 Bl L<sup>2</sup>/Cy dp<sup>2</sup>

al b c sp

Chromosome 3

e<sup>11</sup>  
 Ly Sb/LVM  
 se ss k e<sup>s</sup> ro

Multichromosomal

sc<sup>S1</sup> B Ins w<sup>a</sup> sc<sup>8</sup>; Ins SM1, al<sup>2</sup>  
 Cy Sp<sup>2</sup>/dp b Pm ds<sup>33k</sup>; C Sb/  
 Ubx<sup>130</sup> e<sup>s</sup>; pol

LYON, FRANCE: UNIVERSITÉ DE LYON  
Zoologie Expérimentale

Wild Stocks

Oregon R

Lyon

Algérie

JERUSALEM, ISRAEL: THE HEBREW UNIVERSITY  
Laboratory of Genetics

W. Wild Stocks

Berlin

Canton-S

Qiryat-Anavim

A. Chromosome 1

B

B &amp; y:=

br

br w<sup>e</sup> ec rb t<sup>4</sup>/FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> B

cx ct v g f &amp; y f:=

cv f

f

f B

f od car

f.Y<sup>S</sup>/Y<sup>L</sup> (Finland)g<sup>2</sup>Hw<sup>49c</sup>/sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup>In(1)rst<sup>3</sup> m v & y f:=In(X<sup>c2</sup>)w<sup>vc</sup> f & y f:=l J1<sup>+</sup>.Y/l J1 sc<sup>J1(+)</sup> & y f:=l(X)7 w sn/FM6, y<sup>34</sup> sc<sup>8</sup> dm B

lz/ClB

lx<sup>A</sup> & y f:=

m

N<sup>264-105</sup> (dm)/y Hw in49 m<sup>2</sup> g<sup>4</sup>pn<sup>2</sup>pn<sup>FG</sup>pn<sup>59j</sup>

rb cx v

sc t<sup>2</sup> v f & y f:=sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup> (Basc)sc<sup>L8</sup> g<sup>s</sup> v lz<sup>g</sup> sc<sup>8</sup> & y f:=

sn

spl t v f &amp; y f:=

t v g

v f Bx<sup>3</sup> ma-1 (Glassman)

v g f

v ma-1<sup>bz</sup>

v ras

w

w<sup>a</sup>w<sup>a</sup> vw<sup>e</sup>w<sup>m4</sup>w<sup>m-4000</sup> (Pavia)X<sup>c2</sup> y f & y w f:=Y<sup>L</sup>/f.Y<sup>S</sup> & sc v f:=Y<sup>S</sup> X.Y<sup>L</sup>, In(1)EN, Y<sup>S</sup> B y.Y<sup>L</sup> & y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup>

bb:=/0

Y<sup>S</sup> X.Y<sup>L</sup>, In(1)EN, Y<sup>S</sup> v cv y.Y<sup>L</sup> y<sup>+</sup> & y<sup>2</sup> su-w<sup>a</sup>w<sup>a</sup> bb:=/0Y<sup>S</sup> X.Y<sup>L</sup>, Ins(1)EN, dl-49, Y<sup>S</sup> car f v y.Y<sup>L</sup> &y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> bb:=/0Y ma-1<sup>+</sup> #2/y<sup>2</sup> v ma-1Y.w<sup>+</sup>/y w<sup>a</sup>Y.pn<sup>-</sup> w<sup>+</sup>/y w<sup>a</sup> & y pn:=

y

y<sup>56k</sup>

y ac sc pn sn

y ct<sup>n5</sup> In49 By f:= & Y.B<sup>S</sup>y pn<sup>54c</sup> spl

y pn:= &amp; FM6

y<sup>2</sup> sc w<sup>a</sup> ecy<sup>2</sup> sc w<sup>a</sup> ec & y<sup>+60d19</sup> sc w<sup>aRM</sup>:= (M. Green)

y sc

y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup>y<sup>S1</sup> sc<sup>8</sup>y<sup>S1</sup> sc<sup>8</sup> pn<sup>1</sup>/FM6y sc<sup>4</sup> InS B w<sup>a</sup> sc<sup>8</sup>/sc<sup>8</sup>.Y & XX ("S-5")y<sup>S1</sup> sc<sup>8</sup> sn<sup>3</sup> wy su-Hw Hw In49 m<sup>2</sup> g<sup>4</sup> & y w f bb:=y t<sup>2</sup> v fy<sup>2</sup> v f car

y w

y w sn

y w sn &amp; y:=

y w<sup>a</sup> spl & y f:=B. Chromosome 2

b cn

b lt bw

b pr vg

Bl L/Cy

bri

bs<sup>2</sup>

bw

bw<sup>D</sup> (Hinton)bw<sup>V4</sup>/SM1, al<sup>2</sup> Cy sp<sup>2</sup>bw<sup>V5</sup>/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>

cl

cn bw

Cy/Pm

Cy L/Pm  
 dp<sup>T</sup>  
 dp<sup>T</sup> Sp cn bw sp/S<sup>2</sup> (ls<sup>+</sup>) Cy, InL cn bw sp  
 dp<sup>T</sup> Sp cn InNSR mr/S<sup>2</sup> ls Cy pr Bl cn<sup>2</sup> L<sup>4</sup>  
 bw sp<sup>2</sup>  
 dp<sup>tx</sup> Sp cn/S<sup>2</sup> Cy, InCyL cn  
 fes ms b cn sp/dp<sup>txI</sup> Cy,05 pr cn<sup>2</sup>  
 fes ms cn bw/dp<sup>txI</sup> Cy,05 pr cn<sup>2</sup>  
 fes ms pr cn/dp<sup>txI</sup> Cy,05 pr cn<sup>2</sup>  
 fes ms cn sp/net dp<sup>txI</sup> Cy b pr Bl lt<sup>3</sup> cn<sup>2</sup>  
 L<sup>4</sup> sp<sup>2</sup>  
 lt bw  
 melanotic tumor q<sup>156</sup>  
 ms cn bw/dp<sup>txI</sup> Cy pr Bl lt<sup>3</sup> cn<sup>2</sup> L<sup>4</sup> sp<sup>2</sup>  
 net bw mr crs/al<sup>2</sup> dp<sup>txI</sup> Cy InMis1 pr Bl  
 lt<sup>3</sup> cn<sup>2</sup> L<sup>4</sup> sp<sup>2</sup>  
 pr  
 pr vg  
 S fes Sp ms ts cn mr crs/al<sup>2</sup> InMis1 dp<sup>txI</sup>  
 Cy pr Bl cn<sup>2</sup> L<sup>4</sup> sp<sup>2</sup>  
 stw bs

#### C. Chromosome 3

Antp<sup>R/D</sup>  
 ca<sup>nd</sup>/Tm3 Sb ser  
 ca<sup>572jIIa(3)</sup>/Mé, Ins ri sb<sup>1</sup>  
 ca bv  
 ca K-pn  
 D/Sb  
 Gl Sb/InLVM  
 e  
 gl<sup>2</sup> e<sup>4</sup>  
 gl<sup>G263</sup>  
 h  
 h<sup>2</sup>  
 Ly/Mé  
 p  
 p<sup>p</sup>  
 pyd  
 ri p<sup>p</sup>  
 ru<sup>h</sup> th st cu sr e<sup>s</sup> ca (rucuca)  
 ry<sup>2</sup>  
 se  
 se e  
 se ss K e<sup>s</sup> ro  
 sed  
 st  
 th st pp

#### D. Chromosome 4

bt<sup>D</sup>/ci<sup>D</sup>

ci ey  
 ey<sup>2</sup>  
 ey<sup>2</sup> ophthalmoptera  
 ey<sup>D</sup>/1(4)13  
 ey<sup>D</sup>/Df(4)M

#### M. Multichromosomal

##### 1;2

ab-1 Bld w<sup>a</sup>/w; Cy  
 ab-2 v; bw  
 ab-3 y; Cy L/Pm

##### 1;3

ac-1 y ac sc pn; h  
 ac-2 y pn:=; ca<sup>2</sup>

##### 2;3

bc-1 bw; st  
 bc-2 cn bw; ri e  
 bc-3 Cy/Pm; Sb/D  
 bc-5 Cy InL/cn bw; ri e  
 bc-6 dp; e  
 bc-7 fes ms cn sp/Cy O; h ri e<sup>s</sup>/Mé ri  
 bc-8 pr; st  
 bc-9 T(2;3)(606)Cy; Sb

##### 2;3;4

bcd-1 bw; e; ci ey<sup>R</sup>

#### T. Translocations

##### 2;3

SR-10 Lobe-like  
 SR-12 Variegated (whole arm translocation)  
 SR-14 bw  
 SR-40 (viable as homozygote)

##### Y;2;3

SR-1 aristopedia-like  
 SR-2 exceptionals  
 SR-8 exceptionals  
 SR-11 deformed tarsi

TURKU, FINLAND: UNIVERSITY OF TURKUDepartment of GeneticsWild Stocks

- 1 Canton-S  
2 Oregon-K  
3 Oregon-R-S  
4 Samarkand (Inbred)

Chromosome 1

- 101 B  
102 bb  
103 bi ct g<sup>2</sup>  
104 br w<sup>e</sup> ec rb t<sup>4</sup>/FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> B  
105 Bx  
106 car  
107 cm<sub>6</sub>ct<sup>6</sup> sn<sup>3</sup> & y f:=  
108 ct<sup>6</sup>  
109 cv  
110 cv dx sn  
111 cv lz/ClB  
112 cv sn  
113 ec  
114 ec ct v f  
115 ec cv  
116 f  
117 f<sub>2</sub>car  
118 g<sub>2</sub>  
119 g<sub>2</sub> f B & y  
120 g<sub>2</sub> f sd  
121 In(1)dl-49, y fa<sup>n</sup>  
122 In(1)sc<sup>8</sup>, sc<sup>8</sup>  
123 Ins(1)sc<sup>8</sup>1L, S, sc<sup>8R</sup>, w<sup>a</sup> B (Muller-5)  
124 In(1)w<sup>m4</sup>  
125 m  
126 m g<sub>2</sub>  
127 m wy g<sub>2</sub>  
128 ras<sub>2</sub>  
129 rb  
130 rb cx  
131 rsc and y f:=  
132 s  
133 sc  
134 sc cv  
135 sc cv v f  
136 sc ec cv ct<sup>6</sup> v g<sup>2</sup> f/y<sup>2</sup> sc<sup>S1</sup> B, In-49,  
v w<sup>a</sup> sc<sup>8</sup>  
137 sc w cv  
138 sd  
139 sn<sup>3</sup>

- 140 spl  
141 t<sup>4</sup>  
142 v  
143 w<sup>ch</sup>  
144 w<sup>ch</sup> wy  
145 wy  
146 y  
147 y ac v  
148 y cv  
149 y In49 B<sup>M1</sup> & y f:=  
150 y In49 f car & y f:=  
151 y sn<sup>3</sup> bb  
152 y w  
153 y w cv  
154 z

Closed- X

- 160 X<sup>c2</sup>, cv v f/ClB

Chromosome Y

- 191 f<sup>o</sup>Y<sup>S</sup>/Y<sup>L</sup>  
192 In(1)w<sup>m4</sup> & extra Y  
193 sc<sup>8</sup>Y/y ac sc oc ptg & y f:=  
194 v f B X<sup>X</sup>/y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> bb  
195 X<sup>o</sup>Y<sup>L</sup>/Y<sup>S</sup> & X<sup>o</sup>Y<sup>L</sup>/X<sup>o</sup>Y<sup>L</sup> (Neuhaus)

Chromosome 2

- 200 al dp b pr c px sp/Cy al<sup>2</sup> lt<sup>3</sup> L<sup>4</sup> sp<sup>2</sup>  
201 al<sup>2</sup> Cy, InL lt<sup>3</sup>/b pr Bl lt<sup>3</sup> cn<sup>2</sup> InCyR  
L<sup>4</sup> sp<sup>2</sup>  
202 ap<sup>4</sup>/Rvd, In2LR  
203 Bl L<sup>2</sup>/Cy  
204 bw<sub>2</sub>  
205 cn<sub>2</sub> InCyR cg sp<sup>2</sup>/InsNS px sp  
206 dp<sup>tx</sup> Sp cn<sup>2</sup>/s<sup>2</sup> Cy cn<sup>2</sup> (homoz. InCyR)  
207 ds<sup>38k</sup>/Cy(2L), dp<sup>2</sup> b pr  
208 In(2L)Cy, al<sup>2</sup> ast<sup>3</sup> b pr (Cy not present)  
209 NS, b mr/Cy  
210 pr cn ix/Sm5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>  
211 rl  
212 stw<sub>2</sub>  
213 stw<sub>2</sub> vg  
214 vg

Chromosome 3

- 301 Bd<sup>G</sup>/In(3R)C, 1(3)a

302 ca bv  
 303 D<sup>3</sup> Sb/InLP Dfd InRP ca  
 304 e  
 305 Gl Sb/LVM  
 306 h th st cp in ri p<sup>p</sup> sr e<sup>s</sup> ca/In(3LR)Ubx<sup>130</sup>,  
     ri Ubx<sup>130</sup> e<sup>s</sup> ca (hinca)  
 307 In(3LR)Ubx<sup>130</sup>, ri Ubx<sup>130</sup> e<sup>s</sup>/TM1, Mé ri p<sup>p</sup>  
     sbd<sup>1</sup>  
 308 In(3R)Antp<sup>B</sup>, ss<sup>a</sup>/TM1, Mé ri sbd<sup>1</sup>  
 309 In(3R)Dl<sup>B</sup>, st Dl<sup>B</sup>/In(3R)P<sup>W</sup>, st L(3)W ca  
 310 In(3R)P<sup>FLA</sup> (homozygous)  
 311 Ly Sb/LVM  
 312 Mé, InL Sb/ru h D InsCXF  
 313 ru h st Scx p<sup>p</sup> ss e<sup>s</sup>/TM1, Mé ri sbd<sup>1</sup>  
 314 (ru) h th st cp in ri p<sup>p</sup> ss<sup>a</sup> bx<sup>3</sup> sr e<sup>s</sup>/  
     TM1, Mé ri sbd<sup>1</sup>  
 315 ru h th st cu sr e<sup>s</sup> ca (rucuca)  
 316 ru h th st cu sr e<sup>s</sup> Pr ca/T(2;3)Mé  
 317 Sb Ubx/T(2;3)Xa  
 318 se app  
 319 se rt<sup>2</sup> th/Mé, InL  
 320 se ss k e<sup>s</sup> ro  
 321 ss bxd k e<sup>s</sup>/T(2;3)Xa  
 322 st sr H<sup>2</sup> ca/In(3R)P<sup>W</sup>, st l(3)W ca  
 323 th st cp in ri p<sup>p</sup>  
 324 th st cp in ri p<sup>p</sup> bx sr e<sup>s</sup>/TM1, Mé ri p<sup>p</sup>  
     sbd<sup>1</sup> (thrie)  
 325 th st cp in ri p<sup>p</sup> ss bxd sr e<sup>s</sup>/TM1, Mé ri  
     p<sup>p</sup> sbd<sup>1</sup>  
 326 tra/Mé, T23  
 327 W Sb/InsCXF

Chromosome 4

401 ci

402 ci<sup>W</sup>  
 403 spa

Multichromosomal

501 bw; e  
 502 bw; st  
 503 Pm, dp b/Cy, sp<sup>2</sup>; Sb/D, CxF (ru h ca?)  
 504 In(3) T(2;3)Antp<sup>Yu</sup>/Cy; Sb

Deficiencies

601 Df(2)MS-4/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 602 Df(2)MS-8/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 603 Df(2)MS-10/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 604 Df(2)r1<sup>10a</sup> lt cn/Pm ds<sup>33k</sup>

Translocations

701 Bl T(2;3)A; ru h D TA ss e<sup>s</sup>/Payne  
 --- T(2;3)Antp<sup>Yu</sup> (see 504)  
 702 T(2;3)B; ru h D TB ss e<sup>s</sup>/Payne  
 703 T(2;3)bw<sup>V4</sup>/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 704 al T(2;3)101 sp<sup>2</sup>/Cy L<sup>4</sup> sp  
 705 ru h T(2;3)101 e<sup>4</sup> ro ca/Payne, Dfd ca  
 --- T(2;3)Mé (see 316)  
 706 T(2;3)rn (several stocks with different  
     marker combinations)  
 --- T(2;3)Xa (see 317, 321)

Triploid801 y<sup>2</sup> sc w<sup>a</sup> ec/FM4, y<sup>31d</sup> sc<sup>8</sup> dm B; cn; hTÜBINGEN, GERMANY: MAX PLANCK INSTITUT FÜR BIOLOGIE (Abt. Reichardt)Chromosome 1

1z<sup>65d20</sup>  
 1z<sup>3</sup> ♂ & y f:= ♀

Chromosome 2

so

Chromosome 3

se

Chromosome 4ey<sup>2</sup>MITAKA, TOKYO, JAPAN: INTERNATIONAL CHRISTIAN UNIVERSITYWild Stock

Tokyo

Chromosome 1

w

y

y w m f

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Department of Zoology

Wild Stocks

a1 Canton-S  
a2 Oregon-R  
a3 P Ceylon  
a4 Nai-C

Chromosome 1

b1 B  
b2 B Bx<sup>r</sup> car/y f:=  
b3 B<sup>M1</sup> f<sup>B27</sup>/ClB  
b4 Bx  
b5 Bx<sup>J</sup>  
b6 ct  
b7 dm/y f:=  
b9 sn<sup>3</sup>  
b10 sn<sup>4</sup>  
b12 sn<sup>36a</sup>/y f:=  
b13 sta/y f:=  
b15 sx vb<sup>2</sup> sy/FM4  
b16 v<sup>2</sup> fw  
b17 v f Bx<sup>r49K</sup> car/y f:=  
b18 w  
b20 w<sup>a</sup>  
b21 w<sup>bo</sup>  
b22 w<sup>co</sup>  
b23 w<sup>co</sup> sn<sup>2</sup>  
b24 w<sup>e</sup>  
b25 w<sup>e</sup> sn/ClB  
b26 w<sup>h</sup>  
b27 w<sup>i</sup>  
b28 y  
b29 y ac sn<sup>3</sup> sx vb<sup>2</sup> sy/y sc<sup>S1</sup> Indl-49 B v  
w<sup>a</sup> sc<sup>8</sup>  
b30 y oc/y f:=  
b31 y w sn<sup>3</sup> f<sup>36a</sup>  
b32 y pn w cm ct<sup>6</sup> sn<sup>3</sup> oc ras<sup>2</sup> v dy g<sup>2</sup> f od  
sw/Ins(1)sc<sup>S1</sup>, y v B

Chromosome 2

c1 al dp b bw l(2)ax/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>  
c2 Bl L<sup>2</sup>/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>  
c3 bw  
c4 cg c/U  
c5 pr en  
c6 rdo<sup>2</sup> pr  
c7 sm px/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>

c8 vg

Chromosome 3

d1 D/dsx red  
d2 D/Mé  
d3 D/tra red  
d4 e<sup>s</sup>  
d5 red e  
d6 se h  
d7 th st Pc Scx p<sup>p</sup> ss/TM1, Mé ri

Chromosome 4

e1 ci<sup>w</sup>  
e2 ey<sup>2</sup>

Multichromosomal

f1 tu<sup>1</sup> with modifiers  
f2 tu<sup>2</sup> with modifiers  
f3 y<sup>2</sup> sc w<sup>a</sup> ec:=/FM4; cn/cn/cn (3N)  
f4 y; D/tra red

Attached X-Y and Altered Y Chromosomes

g1 FR1, Y<sup>S</sup> y cv v f/y f:=  
g2 In(1)EN, Y<sup>S</sup> B f v.Y<sup>L</sup>.y<sup>+</sup>/RM(15-DRP), y  
su-w<sup>a</sup> w<sup>a</sup> bb.Y<sup>L</sup>/y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> bb.Y: bb<sup>+</sup>  
g3 sc<sup>S1</sup>.Y<sup>L</sup>#2/y f.Y<sup>S</sup>/y w  
g4 sc<sup>V1</sup>.Y<sup>S</sup>/y v f bb, Y<sup>L</sup>/y f:=  
g5 y w.Y<sup>L</sup>.Y<sup>S</sup>/O/y w  
g6 y Hw.Y<sup>S</sup>.y<sup>+</sup>/Y<sup>CL</sup>/y w<sup>a</sup>:=  
g7 y w f.Y<sup>L</sup>.Y<sup>S</sup>/O/y w  
g8 Y<sup>CS</sup>: bw<sup>+</sup> bb<sup>+</sup>/g<sup>2</sup> B.Y<sup>L</sup>/y v bb; bw  
g9 Y<sup>L</sup>: bb<sup>+</sup>?/y ct<sup>6</sup> f.Y<sup>S</sup>/y v bb; bw  
g10 Y<sup>CL</sup>-15/y f.Y<sup>S</sup>/y w  
g11 Y<sup>S</sup>.w y.Y<sup>L</sup>.y<sup>55f10</sup>/O/y w  
g12 Y<sup>S</sup>.X.Y<sup>L</sup>.y<sup>+</sup>, y<sup>55e24-1</sup>/y v bb/O  
g13 Y<sup>S</sup>.Y<sup>S</sup>/sc.sc.Y<sup>L</sup>/y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> bb

Deficiencies and Duplications

h1 Df(1)g l f B/InAM  
h2 Df(1)N<sup>8</sup>/FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> B  
h3 Df(1)N<sup>268-105</sup>/FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> B  
h4 Df(1)N<sup>264-39</sup>, w<sup>ch</sup>/FM4, y<sup>31d</sup> sc<sup>8</sup> dm B  
h5 Df(1)M-4/ey<sup>D</sup>  
h6 y ac Dp w<sup>a</sup> (w<sup>a</sup>)2/y<sup>2</sup> sc<sup>56e</sup> w ec

h7 y f:=/y ac Dp(w<sup>a</sup>)2TranslocationsInversions

i1 Bd<sup>G</sup>/In(3R)C, 1(3)a  
 i2 tra/In(3LR)Ubx<sup>130</sup> (FMA3) w<sup>a</sup> f<sup>36a</sup>  
 i3 y Indl-49 sn<sup>X2</sup> B<sup>M1</sup>/y f:=  
 i4 (In sn)sc<sup>S1</sup> Indl-49 sn<sup>X2</sup> sc<sup>8</sup>/y f:=  
 i5 y Indl-49 sn<sup>X2</sup> bb/y f:=  
 i6 y<sup>S1</sup> sc<sup>8</sup> B InS sc<sup>S1</sup>/w sn<sup>5s</sup> bb

j1 T(1;2)N<sup>264-10</sup>/FM6, y<sup>31d</sup> sc<sup>8</sup> dm B  
 j2 T(1;2)sc<sup>19</sup>/y f:=; fes sc<sup>19i</sup> b pr/Cy, dp<sup>Th</sup> pr  
 j3 T(1;3)N<sup>264-6</sup>, y/y w dm (=N<sup>6</sup>)  
 j4 T(1;3)sn/y f:=  
 j5 T(1;4)B<sup>8a</sup> (16A1) & y f:=  
 j6 T(1;4)N<sup>8a</sup> (3C5-6&7)/FM6, y<sup>31d</sup> sc<sup>8</sup> dm B  
 j7 T(1;4)w<sup>13</sup>(9B&20)sc v<sup>m</sup> g?ClB  
 j8 T(1;4)w<sup>m5</sup> (3CZ) & y f:=  
 j9 T(1;4)(4C3) & y f:=  
 j10 T(1;4)w<sup>m5</sup>: T(1;3)sc<sup>J4R</sup> (ClB)

BERLIN-BUCH: DEUTSCHE AKADEMIE DER WISSENSCHAFTEN  
Institut für Experimentelle Krebsforschung

Wild Stocks

1 Berlin wild  
 2 England

15 sc  
 16 sc rb cv  
 17 m  
 18 B  
 19 cv

31 L<sup>2</sup>/Cy  
 32 vg  
 33 bw cn  
 34 al dp

Chromosome 1

3 w  
 4 w sn<sup>3</sup>  
 5 w<sup>bf</sup>  
 6 w<sup>e</sup>  
 7 w<sup>co</sup> sn<sup>2</sup>  
 8 w<sup>m4</sup>  
 9 y<sup>303</sup>  
 10 y  
 11 y w  
 12 y pn  
 13 y w bb  
 14 y fa wy<sup>2</sup> g<sup>2</sup>

20 car  
 21 fa<sup>n</sup>  
 22 ct  
 23 y w f/B  
 24 w<sup>e</sup> bb<sup>1</sup>/ClB  
 25 sc<sup>S1</sup> InS w<sup>a</sup> sc<sup>8</sup>  
 26 sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup>  
 27 sc ec ct v g f

Chromosome 2

28. j  
 29 bw<sup>pp</sup>  
 30 b cn vg

Chromosome 3

35 e<sup>11</sup>  
 36 st  
 37 p<sup>p</sup>  
 38 Dfd<sup>r-L</sup>  
 39 ri

Chromosome 4

40 ey<sup>2</sup>

Multichromosomal

41 cn ss  
 42 tu<sup>g</sup>

STRASBOURG (BAS-RHIN), FRANCE: THE UNIVERSITY  
Zoology Laboratory

Wild Stocks

Oregon-R-C

Chromosome 2

cn  
 b

Chromosome 4

ey

Chromosome 1

B  
 v  
 w  
 y

Chromosome 3

DcxF/Dfd  
 e  
 se

Multichromosomal

vg; cn  
 w; e  
 y; e  
 y; se

MÜNCHEN, GERMANY: ZOOLOGISCHES INSTITUT DER UNIVERSITÄT MÜNCHENWild Stocks

Berlin

Chromosome 1

br  
 cs<sup>53</sup>/<sub>6</sub> y w bb  
 ct<sup>6</sup>  
 cv  
 if<sup>3</sup> sc z w<sup>17G2</sup> ec ct<sup>6</sup>  
 sc z<sup>1</sup>  
 sc z<sup>m</sup>  
 sn<sup>65a</sup>/y f:=  
 w  
 w<sup>co</sup> sn<sup>2</sup>  
 w spl cv f  
 y pn  
 y sc z w<sup>a</sup> spl ec/y f:=  
 y sc w<sup>Bwx</sup>  
 y sc z w<sup>r,def.</sup> spl f/y f:=  
 y v f  
 y w spl  
 y w spl cv f  
 y z<sup>a</sup>  
 y<sup>2</sup> sc w<sup>a</sup> w<sup>ch</sup> fa/y f:=  
 y<sup>2</sup> w<sup>bf</sup> spl sn<sup>3</sup>  
 z  
 z w<sup>11E4</sup>

Chromosome 2

al b c sp  
 b dp  
 lgl cn bw/Cy  
 vg bw

Chromosome 3

e  
 ru<sup>g</sup>  
 ru<sup>g</sup> jv se by  
 ry  
 se h  
 th cu sr e<sup>s</sup> ro ca  
 ve h th

Multichromosomal Stocks

y v; bw (1;2)  
 ntg; px pd; su-pd (1;2;3)

w; se h (1;3)  
 w<sup>co</sup> sn<sup>2</sup>; ru<sup>g</sup> (1;3)  
 w<sup>co</sup> sn<sup>2</sup>; se h (1;3)  
 z; ru<sup>g</sup> (1;3)  
 w; ey (1;4)  
 b; se (2;3)  
 bw; e (2;3)  
 bw; st (2;3)  
 Cy/Pm; CxD/Sb (2;3)  
 SM1, al Cy sp<sup>2</sup>/In(2LR)102 ds<sup>w</sup> sp<sup>2</sup>; In(3LP,  
 3RC)Sb e<sup>s</sup>/Ubx<sup>130</sup> e<sup>s</sup> (2;3)  
 b; ey (2;4)

X Chromosomes with a Y Arm Attached

X.Y<sup>S</sup> (A-3), y w.Y<sup>S</sup>/y v f/Y<sup>Lc</sup>  
 X.Y<sup>L</sup> (A-2), y w.Y<sup>L</sup>/y/Y  
 X.Y<sup>L</sup> (U-8e), y w.Y<sup>L</sup>/y/Y

Attached-XY

X.Y<sup>S</sup>.Y<sup>L</sup> (115-9 Parker), y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> Y<sup>S</sup>.Y<sup>L</sup> y<sup>+</sup>/  
 y v bb/0  
 X.Y<sup>L</sup>.Y<sup>S</sup> (108-9 Parker), y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> Y<sup>L</sup>.Y<sup>S</sup>/  
 y v bb/0  
 X.Y<sup>L</sup>.Y<sup>S</sup> (2-10T13 Parker), y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> Y<sup>L</sup>.Y<sup>S</sup>/  
 S.Y<sup>S</sup>/y v f B.Y/y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup>/0

Closed-X

In(1)X<sup>c2</sup>, w<sup>vc</sup> f/y w lz

Deficiencies

Df(1)N<sup>264-105</sup>/FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> B  
 Df(1)rst<sup>2</sup>/FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> B<sub>4</sub>  
 Df(1)w<sup>258-11</sup>, y/dl-49, y Hw m<sup>2</sup> g<sup>4</sup>  
 Df(1)w<sup>258-42</sup>, y/FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> B  
 Df(1)w<sup>258-48</sup>, y sc<sup>5</sup> spl; Dp(1;3)w<sup>Vco</sup>/y f:=

Duplications

Dp(1;3)w<sup>m264-58a</sup>/Y<sup>S</sup>.w y.Y<sup>L</sup> y<sup>+</sup>/Y/y w; +<sup>Su-V</sup>

Inversions

In(1)C1B  
 In(1)N<sup>264-84</sup>, y/FM6, y<sup>31d</sup> sc<sup>8</sup> dm B  
 In(1)rst<sup>3</sup>  
 In(1)rst<sup>3</sup>, w m v f/M-5

In(1)rst<sup>3</sup>, w<sup>48-3</sup> v f/y f:=; bw  
 In(1)rst<sup>3</sup>, y car bb  
 In(1)rst<sup>3</sup>, y w<sup>60i29</sup> car bb  
 In(1)w<sup>m4</sup>  
 In(1)w<sup>m4</sup>, f  
 In(1)w<sup>m4</sup>; Cy/En-var-7  
 In(1)w<sup>m4</sup>; Y's extra

Translocations

T(1;3)N<sup>264-6</sup>, y/y w dm  
 T(1;4)w<sup>258-18(3C4)</sup>, y/dl-49, y Hw m<sup>2</sup> g<sup>4</sup>  
 T(1;4)w<sup>258-18(3C4)</sup>, y/y w dm  
 T(1;4)w<sup>258-21</sup>/y w spl  
 T(1;4)w<sup>m5</sup>/w; ey<sup>R</sup>  
 T(1;4)w<sup>m5</sup>/w; ci ey<sup>R</sup>

SANTIAGO, CHILE: UNIVERSIDAD DE CHILEInstituto de Biología "Juan Noé", Departamento de GenéticaWild Stocks

1 Chillán  
 2 Oregon-R-C  
 3 Rapel  
 4 Santiago  
 5 Swedish (free of inv.)  
 6 Valdivia

Chromosome 1

7 B  
 8 Bx  
 9 my

10 v  
 11 w  
 12 w m f  
 13 y

Chromosome 2

14 b  
 15 b vg  
 16 bw  
 17 dp  
 18 L<sup>2</sup>  
 19 L<sup>2</sup>/Cy  
 20 S/Cy; En-S  
 21 vg

Chromosome 3

22 D/Gl  
 23 e<sup>11</sup>  
 24 Bl Sb/LVM  
 25 se  
 26 st

Chromosome 4

27 ey<sup>2</sup>

Multichromosomal

28 dp; e<sup>11</sup>  
 29 w; vg

BRIGHTON, SUSSEX, ENGLAND: UNIVERSITY OF SUSSEXSchool of BiologyWild Stocks

Ore K  
 Ore +  
 Ore R  
 Ore S  
 N. B.

Chromosome 1

ma-l x y f:=  
 M-5  
 N<sup>8</sup>/dl-49 Min<sup>2</sup> y Hw g<sup>4</sup>  
 chieti vermilion  
 wild vermilion  
 v  
 w sn m  
 y f:= CaX pn x ma-l CaX pn  
 y pn XX x FM6 ♂

Chromosome 2

ab  
 b gp  
 dp ey<sup>2</sup>  
 tu A<sub>2</sub>  
 tu B<sub>3</sub>  
 tu<sup>k</sup> Ey OrK  
 tu 55g

Chromosome 3

ri  
 ri (cal)  
 rucua  
 tt wo

Chromosome 4

Ant x Ey k  
 ci ey<sup>R</sup>  
 Ci D/Spa<sup>cat</sup>

Multichromosomal

SM5/Bli<sup>2</sup>; st/st  
 tu bw; +sutu  
 tu bw su-er; st, sutu er  
 tu bw; TM3/sb  
 tu<sup>K</sup> TM3/sb  
 vg/m - vg

MILAN, ITALY: UNIVERSITA' DI MILANO  
Istituto di Genetica

Wild Stocks

1 Cagliari  
 2 Canton-S  
 3 Chieti-v  
 4 Crkwenica  
 5 Gaiano  
 6 Jaslo O. C.  
 7 Moltrasio  
 8 Oregon-R  
 9 Pavia  
 10 S. Maria  
 11 Sevelen  
 12 Suna  
 13 Urbana  
 14 Valdagno  
 14 Varese

45 So<sup>C</sup>  
 46 spt  
 47 sp<sup>2</sup> bs<sup>2</sup>

Chromosome 3

48 cp  
 49 gl<sup>3</sup>  
 50 mwh  
 51 mwh- se  
 52 mwh ri ss k e<sup>S</sup> ro  
 53 obt  
 54 ru b st p<sup>p</sup> as e<sup>S</sup>  
 55 ru  
 56 ve

Multichromosomal

57 px<sup>43j</sup> oo; ru jv se st ca  
 58 y; al bw sp

Not Localized

59 tg (formerly abab<sup>49</sup>)

InversionsChromosome 1

60 ClB/+  
 61 ClB y/y g<sup>4</sup>  
 62 1(1)7/dL-49 y Hw m<sup>2</sup> g<sup>4</sup>  
 63 Muller-5  
 64 Muller-5/lozenge

Chromosome 2

65 Cy sp/Pm  
 66 Cy E-S/S  
 67 Cy cn<sup>2</sup> bw sp / Gla InLR  
 68 Gla/spd gt-4

Chromosome 3

69 H/Sb sr In(3R)Mé  
 70 ltr/Sb sr In(3R)Mé  
 71 Mé ca/ru cu ca  
 72 ve h th C3G Sb Ubx/st C3G ca

Chromosome 1

16 B  
 17 N<sup>B</sup>-S  
 18 ptg<sup>2</sup>  
 19 sc ec ct v g f  
 20 v  
 21 sd  
 22 w<sup>a</sup>  
 23 w<sup>bl</sup>  
 24 w<sup>e</sup>  
 25 y w  
 26 ol  
 27 od  
 28 b cn  
 29 a px sp  
 30 ab  
 31 b cn vg  
 32 blt  
 33 blt<sup>S</sup>  
 34 bsp  
 35 bw ba  
 36 c wt px  
 37 cn  
 38 cn c wt px  
 39 dp cl b  
 40 ft  
 41 ll<sup>2</sup>  
 42 net  
 43 so  
 44 so<sup>2</sup> b cn

Multichromosomal

73 Cy L<sup>4</sup> sp/Pm; H/Sb sr In(3R)Mé  
 74 y sc<sup>S1</sup> In49 sc<sup>8</sup>; bw; st p<sup>p</sup>

Deficiencies

75 Df(1)N y/dl-49 y Hw m<sup>2</sup> g<sup>4</sup>  
 76 Df(2)Px<sup>2</sup> Df(2)Px, bw sp/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 77 Df(2)bw<sup>5</sup> Df(2)bw<sup>5</sup> sp<sup>2</sup>/Xa  
 78 Minute (2) Bridges  
 79 M(2)33a/al<sup>2</sup> InMis Cy cn<sup>2</sup> sp<sup>2</sup>

Special Stocks

80 "sz e" Y<sup>Lc</sup>/X.Y<sup>S</sup> & y v f:=; e  
 81 "szw" Y<sup>Lc</sup>/X.W.Y<sup>S</sup>  
 82 y<sup>2</sup> Sn<sup>w</sup> w<sup>a</sup> bb:= & sfB.Y

Stocks Selected for Tumor Manifestation

83 tu A1  
 84 tu B1  
 85 tu B3  
 86 tu C1  
 87 tu C2  
 88 tu C3  
 89 tu C4  
 90 tu C5  
 91 tu D  
 92 tu So<sup>c</sup>  
 93 tu Aspra  
 94 Freckled/Cy  
 95 q 156 melanotic  
 96 e 144 melanotic  
 97 lm  
 98 lnd

SÃO PAULO, BRAZIL: UNIVERSIDADE DE SÃO PAULO

Faculdade de Filosofia, Ciências e Letras, Dept. de Biologia Geral

Wild Stock

Canton-S

Chromosome 1

br  
 ec  
 f  
 g<sup>2</sup>  
 sc  
 sc cv v f  
 sc pn<sup>3</sup> g<sup>2</sup> (rv) Bx<sup>2</sup>  
 v  
 v f su<sup>w</sup>-f  
 w  
 y  
 y<sup>2</sup> wy<sup>2</sup> g<sup>2</sup> (g<sup>2</sup> partly reverted)  
 y<sup>2</sup> sc w<sup>a</sup> ec/FM4, y<sup>31d</sup> sc<sup>8</sup> dm B  
 Muller-5  
 Ins(1)FM-6, y<sup>31d</sup> sc<sup>8</sup> dm w B/y f:=/y<sup>+</sup> Y

Chromosome 2

b el rd<sup>s</sup> pr cn<sup>2</sup>  
 chl en/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>  
 b gp

bw

dp

fr/Cy, dp<sup>2</sup>

px

vg

al dp b pr Bl C px sp/SM1, al<sup>2</sup> Cy sp<sup>2</sup>  
 al dp b pr blt bw/SM5, al<sup>2</sup> Cy lt sp<sup>2</sup>  
 In(2L)T, esc c sp/SM5, al<sup>2</sup> Cy lt<sup>v</sup> sp<sup>2</sup>  
 SM-1, al<sup>2</sup> Cy sp<sup>2</sup>/Pu<sup>2</sup> (eye color)

Chromosome 3

e

st

tx

pc/TM1, Mé ri sbd<sup>1</sup>  
 Pr Dr/TM3, y<sup>+</sup> ac<sup>+</sup> ri p<sup>p</sup> sep bx<sup>34e</sup> e<sup>s</sup>  
 Sb<sup>Sp1</sup>/In(3LR) cx  
 l(3)ac e<sup>s</sup> M(3)w/LVM  
 In(3LR)Ubx<sup>130</sup>, Ubx<sup>130</sup> e<sup>s</sup>/st C3G ca

Multichromosomal

bw; st

dp; e

vg; e

y<sup>2</sup> AM; SM1; Ubx<sup>130c</sup>; pol  
 FM6 Be Vno pol

SALISBURY, RHODESIA: UNIVERSITY COLLEGE OF RHODESIA  
Department of Biological Science

<u>Wild Stocks</u>	v	<u>Chromosome 3</u>
	B	
Oregon-R	w m B	se
Stock collected at Inhaca Island, Mozambique, Sept. 1965, and maintained in mass culture.	y	st
	<u>Chromosome 2</u>	e
	vg	<u>Rearrangements</u>
<u>Chromosome 1</u>	bw	ClB/+
w <sub>e</sub>		
w		

BERLIN-DAHLEM, GERMANY: INSTITUT FÜR GENETIK DER FREIEN UNIVERSITÄT BERLIN

<u>Wild Stocks</u>	24 wy	<u>Multichromosomal</u>
	25 y cv v f	
1 Berlin wild K	26 y w	46 Bld w <sup>a</sup> /w; Cy
2 Berlin wild B	27 yy:=/+	47 bw; st
3 England	<u>Chromosome 2</u>	48 cn; ss
		49 vg; e <sup>11</sup>
<u>Chromosome 1</u>		50 sc <sup>S1</sup> B InS w <sup>a</sup> sc <sup>8</sup> ; Ins SM1
	28 al dp	al <sup>2</sup> Cy sp <sup>2</sup> /dp b Pm ds <sup>33k</sup> ;
4 B	29 b cn vg	C Sp/Ubx <sup>130</sup> e <sup>s</sup> ; pol
5 ClB/+	30 bw	51 y <sup>S1</sup> sc <sup>8</sup> InS y <sup>3P</sup> ; al <sup>2</sup> Cy lt <sup>3</sup>
6 car	31 L <sup>2</sup> /Cy	sp <sup>2</sup> /dp b Pm <sup>1</sup> ; ru h D <sup>3</sup>
7 cv	32 tu <sup>8</sup>	InCXF ca/Sb In(3R)
8 f	33 vg	52 y f:=; bw; e; pol
9 In(1)dl-49, ty-1 bb <sup>1</sup> /y v f car:=	<u>Chromosome 3</u>	<u>Altered Y</u>
10 l(1)7/dl-49, y Hw w lz <sup>s</sup>	34 bx <sup>34e</sup>	53 Multi ♂: sc <sup>8</sup> Y/y In49 B; bw <sup>D</sup>
11 m	35 Dfd <sup>r-L</sup>	& y f:=; bw <sup>D</sup> ♀
12 sc ec ct	36 e <sup>11</sup>	54 Multi ♀: X·Y InEN y; st
13 sc ec ct v f g	37 jv se	55 Sterilizer +: Y <sup>Lc</sup> /X·Y <sup>S</sup> ♂
14 sc <sup>S1</sup> inS B w <sup>a</sup> sc <sup>8</sup>	38 Ly/D <sup>3</sup>	& X·Y <sup>S</sup> ♀
15 v	39 ri	<u>Selected for DDT-Resistance</u>
16 w	40 ru h st Dfd p <sup>p</sup> ss e <sup>s</sup>	
17 w <sup>a</sup>	41 st	56 Berlin wild, DDT-resistant 1
18 w <sup>bf</sup>	42 Tu	
19 w <sup>e</sup>	<u>Chromosome 4</u>	<u>Unanalyzed</u>
20 w <sup>m4</sup>		
21 w sn <sup>3</sup>	43 bt <sup>D</sup> /ci <sup>D</sup>	57 CO <sub>2</sub> -sensitive
22 w <sup>co</sup> sn <sup>2</sup>	44 ci ey <sup>R</sup>	
23 w <sup>ch</sup> wy	45 ey <sup>2</sup>	

OBNINSK, USSR: INSTITUTE OF MEDICAL RADIOLOGY  
Department of General Radiobiology and Genetics

Wild Stocks

Berlin wild  
 Algeria inbred  
 Canton-S

y ac sc pn sn  
 y ec ct v f  
y f  
 y sc<sup>S1</sup> In49 v sc<sup>8</sup>/y f  
 y w  
 y<sup>2</sup> v f car su-f/y f

gl  
 h ri<sup>1</sup> ca  
 Hn<sup>r2</sup>  
 ri  
 st  
 st ss e<sup>11</sup>

Chromosome 1

B  
 CLB  
 ct v f  
 cv  
 f BB/y  
 m  
 ma-l  
 od car  
 rb  
 v f  
 w  
 w B  
 w/CLB  
 w<sup>ch</sup>  
 w<sup>m4</sup>  
 y  
 Y

Chromosome 2

b  
 b cn vg  
 b j pr cn  
 bw  
 c  
 cn  
 Cy/L<sup>2</sup>  
 sp  
 vg

Chromosome 3

ca  
 ca<sup>nd</sup>  
 cu

Chromosome 4

bt<sup>D</sup>/ci<sup>D</sup>  
 ci<sup>R</sup>ey<sup>R</sup>  
 ci<sup>D</sup>/ey<sup>D</sup>  
 sv<sup>n</sup>

Multichromosomal

cn bw; e<sup>11</sup>  
 cn; e<sup>11</sup>  
 Cy/L; D/Sb  
 e<sup>11</sup>; sv<sup>n</sup>  
 sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup>/sc<sup>8</sup>.Y  
 vg; e  
 X<sup>c2</sup> y v/sc<sup>8</sup>.Y

VARANASI, INDIA: BANARAS HINDU UNIVERSITY  
Department of Zoology

Wild Stocks

a Canton-S  
 b Oregon-R  
 c Kerala

Chromosome 1

CLB  
 X<sup>c2</sup>  
 i  
 w<sup>a</sup>  
 w<sup>bl</sup>  
 w<sup>e</sup>  
 w<sup>co</sup>  
 w<sup>h</sup>

82 - y sc<sup>S1</sup> B In49 ct<sup>ns</sup> sc<sup>8</sup> se cu

Chromosome 2

vg  
 g<sup>49</sup> - dp<sup>tx1</sup> Sp ab<sup>2</sup>/S<sup>2</sup> ls Cy  
 InCy L  
 B - fes ms (b) cn sp/dp<sup>tx1</sup>  
 Cy<sup>1</sup> cn<sup>2</sup>  
 135 - S fes Sp ms ta cn mr  
 crs/dp<sup>tx1</sup> Cy<sup>1</sup> cn<sup>2</sup>  
 g<sup>67</sup> - ls dp<sup>T</sup> Sp ms ta cn crs/  
 S<sup>2</sup> Cy Bl cn<sup>2</sup> L<sup>4</sup> sp<sup>2</sup>  
 g<sup>45</sup> - dp<sup>T</sup> Sp cn bw sp/S<sup>2</sup> (1st)  
 Cy, InL cn bw sp Cy/D

Chromosome 3

se h  
 ss<sup>a</sup>  
 Ly/D<sup>3</sup>

Chromosome 4

ey<sup>2</sup>  
 ci<sup>w</sup>

Multichromosomal

fs 13 - y<sup>+</sup> ac<sup>+</sup> sc<sup>8</sup>Y/y b; bw<sup>D</sup>; st<sup>+</sup> ♂  
 & y f<sup>±</sup>; bw<sup>D</sup> ♀  
 j102 - Y<sup>S</sup> X InEN In49 y Y<sup>L</sup>; st  
 (no free Y)

TÜBINGEN, GERMANY: MAX PLANCK INSTITUT FÜR BIOLOGIE (Abt. Beermann)Wild Stock

1 Berlin (Marburg)

Chromosome 1

2 B

3 Df(1) In(1)bb<sup>-</sup>, y sl<sup>2</sup> bb<sup>-</sup>/FM4, y<sup>31d</sup> sc<sup>8</sup>  
dm B\*/sc<sup>8</sup>.Y (\*FM4 chromosome carries  
recessive lethal factor)4 sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup> (Muller-5)5 sc<sup>8</sup> bb w<sup>61a</sup>6 sn<sup>3</sup>7 v/Y<sup>bb</sup>

8 w

9 w/y

10 w<sup>a2</sup>11 w<sup>bl</sup>12 w<sup>m4</sup>

13 y v f

14 y w lz/X<sup>c2</sup>, w<sup>vc</sup>/sc<sup>8</sup>.Y15 y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> f<sup>1</sup> su-fChromosome 2

16 al b cn sp

17 vg bw

Chromosome 3

18 D/G1

19 se

\* tra/D ....(20)

Multichromosomal20 y v f/w<sup>a</sup>; tra/D InsCXFTriploid21 y<sup>2</sup> sc w<sup>a</sup> ec/FM4, y<sup>31d</sup> sc<sup>8</sup> dm B/sc<sup>8</sup>.Y;  
cn/cn/cnAttached-X

\* y/w ....(9)

22 y f/Y<sup>S</sup> & g<sup>2</sup> B.Y<sup>L</sup>/Y<sup>S</sup>23 y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> bb/0 & v f B, X.Y24 y<sup>2</sup> su-w<sup>a</sup> w<sup>a</sup> bb/sc<sup>8</sup>.Y/X<sup>c2</sup>, y v

\* y v f ....(20)

\* y<sup>2</sup> sc w<sup>a</sup> ec ....(21)Closed-X\* X<sup>c2</sup>, y v ....(24)\* X<sup>c2</sup>, w<sup>vc</sup> ....(14)VEPERY, MADRAS, INDIA: MADRAS VETERINARY COLLEGE  
Department of Animal GeneticsWild Stocks

Oregon-K

Madras

Chromosome 11 sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup> (M-5)

2 w

Chromosome 23 Cy/Bl L<sup>2</sup>

4 dp b cn bw

5 vg

Chromosome 3

6 e

Attached-X7 sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup> & y v f:=Multichromosomal

8 bw; st

9 y sc<sup>S1</sup> In49 sc; bw; st

10 dp; e

NEW DELHI, INDIA: INDIAN AGRICULTURAL RESEARCH INSTITUTEWild Stocks

1 Oregon-K

Chromosome 1

1 B<sup>S1</sup> InS w<sup>a</sup> sc<sup>8</sup> (Muller-5)  
 2 y<sup>S1</sup> InS sc<sup>8</sup>  
 3 y<sup>S1</sup> InS sc<sup>8</sup>  
 4 y sc<sup>S1</sup> B in<sup>49</sup> ct n<sup>s</sup> sc<sup>8</sup> (Binsety)

Chromosome 2

1 vg  
 2 cn bw  
 3 b cn bw  
 4 dp b cn bw  
 5 dp b cn  
 6 Cy cn bw  
 7 bw  
 8 Cy/B1 L<sup>2</sup>  
 9 fes ms b cn Sp/Cyo  
 10 fes ms cn Sp/Cyo  
 11 fes Sp ms ta cn mr(Crs)/Cyo  
 12 dp<sup>tx</sup> Sp ab<sup>2</sup>/S<sup>2</sup> ls Cy In Cy L  
 13 dp b L<sup>4</sup> Pm<sup>1</sup>/dp tx<sup>1</sup> Cy, o<sup>5</sup> pr cn<sup>2</sup>  
 14 ls dp<sup>+</sup> Sp ms ta cn Crs/S<sup>2</sup> Cy lt<sup>3</sup> pr<sup>+</sup> B1  
 15 dp<sup>tx</sup> Sp cn bw sp/S<sup>2</sup> (ls<sup>+</sup>) Cy In L cn bw sp

Chromosome 3

1 e  
 2 st

Chromosome 4

1 ey

Multichromosomal

1 bw; st  
 2 y sc<sup>S1</sup> InS sc<sup>8</sup>; Cy/B1 L<sup>2</sup>  
 3 y sc<sup>S1</sup> InS sc<sup>8</sup>; bw; st  
 4 y sc<sup>S1</sup> InS sc<sup>8</sup>; cn bw  
 5 y sc<sup>S1</sup> InS sc<sup>8</sup>; vg  
 6 y sc<sup>S1</sup>  
 7 sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup>; Cy<sup>h</sup> b bw/dp<sup>txl</sup> Cy<sup>Pl</sup> B1  
 8 y sc<sup>S1</sup> In<sup>49</sup> v sc<sup>8</sup>; dp<sup>h</sup> b bw/dp<sup>txl</sup> Cy<sup>Pl</sup> B1  
 9 sc<sup>8</sup> Y/y B & y f:= Bw<sup>d</sup> cn<sup>2</sup> L<sup>4</sup> sp<sup>2</sup>

Attached-X

1 sc<sup>S1</sup> B InS w<sup>a</sup> sc<sup>8</sup> & y v f:=  
 2 sc<sup>S1</sup> f In<sup>49</sup> v w<sup>a</sup> sc<sup>8</sup> & y f:=  
 3 y sc<sup>S1</sup> f In<sup>49</sup> v w<sup>a</sup> sc<sup>8</sup> & y f:=

Altered Y with Mutants in X

1 1 j<sup>1</sup> Y/l j<sup>1</sup> sc<sup>j1</sup> (+) In<sup>49</sup> v Ptg<sup>oc</sup> B<sup>M1</sup>/  
 y sc<sup>S1</sup> car odsy f<sup>g2</sup> dy v ras<sup>2</sup> sn<sup>3</sup> ct<sup>6</sup>  
 cm rb ec w Pn<sup>1</sup> sc<sup>8</sup> (Maxy)

THESSALONIKI, GREECE: UNIVERSITY OF THESSALONIKIDepartment of BiologyWild Stocks

Oregon-K

Berlin wild

Chromosome 1

y  
 pn<sup>2</sup>  
 w  
 we  
 wbf  
 fa<sup>n</sup>  
 sn  
 v  
 m

f

Muller-5

XXY

Chromosome II

Cy  
 dp  
 b  
 pr  
 cn  
 vg  
 bat  
 bw

bs

Cy L<sup>4</sup>/PmChromosome III

ve  
 jv  
 se  
 st  
 cu  
 ry  
 ss  
 e  
 Bd

SHEFFIELD, ENGLAND: THE UNIVERSITY  
Department of Genetics

Wild Stocks: mass mated

	cn	ey <sup>2</sup> <sub>4</sub>
	bw	ey <sup>K</sup>
Florida	b cn vg	ey <sup>R</sup>
Edinburgh	bw cn	ci <sup>D</sup> ey <sup>D</sup>
Pacific	vg	ci <sup>D</sup> /ey <sup>D</sup>
Oregon-K	b	
Wellington	dp b cn bw	
	SM5, al <sup>2</sup> Cy lt <sup>v</sup> sp <sup>2</sup> /Bl L <sup>2</sup>	<u>Multichromosomal</u>

Chromosome 1

CLB

w<sup>a</sup>

w

y w

y v f

w<sup>a</sup> v

w m B

ma-l<sub>bz</sub>

ma-l

y w

cx<sup>tg</sup> t/FM1, y<sup>31d</sup> sc<sup>8</sup> w<sup>a</sup> lz<sup>s</sup> BChromosome 3e<sup>11</sup>

st

eyg<sup>2</sup>ry<sup>2</sup>

er

se cp e

Ly/Mé

th st cp

ve h eyg cp

TM3, ri p<sup>p</sup> bx<sup>34e</sup> Ser/SbCy L<sup>4</sup>/Pm; H/ SbTumour Strains: tu(2-83.9)

tu-B3

tu bw

al b pr tu px sp

tu bw; st su-tu

v; tu bw; st su-tu

y; tu bw; st su-tu

b tu bw; st su-tu

Translocations (1;4)T(1;4)<sub>w</sub><sup>258-21</sup>, y w<sup>a</sup>/FM4, y<sup>31d</sup><sub>sc<sup>8</sup> dm B</sub>T(1;4)<sub>w</sub><sup>258-21</sup>, y w/Ins(1)dl-49,  
sc<sup>8</sup> y<sup>31d</sup> w<sup>a</sup> lz BChromosome 2

dp

Chromosome 4

ey

BOGOTÁ D. E., COLOMBIA: UNIVERSIDAD DE LOS ANDES  
Instituto de Genética

Wild Stocks

San Andrés, Colombia

San Jorge, Colombia

Santa Marta, Colombia

Monteria, Colombia

Caracolisito, Colombia

Soledad, Colombia

Bogotá, Colombia

Duitama, Colombia

Sasaima, Colombia

Stephenville

Barranquilla, Colombia

Rio Meta, Colombia

Florian, Colombia

Medellin, Colombia

COPENHAGEN, DENMARK: UNIVERSITY OF COPENHAGEN  
Institute of Genetics

Amylase Stocksa1 c Amy<sup>1</sup> wt (Phil.)a2 Amy<sup>2</sup> (58-105)a3 Amy<sup>2</sup> Amy<sup>3</sup> (58-105)  
a4 Amy<sup>2</sup> Amy<sup>6</sup> (58-105, ad<sup>60</sup>)  
a5 Amy<sup>4</sup> Amy<sup>6</sup> (ad<sup>60</sup>)a6 c Amy<sup>4</sup> Amy<sup>3</sup> wt (ad<sup>60</sup>, 58-105)

For other stocks see DIS 41:33

SEOUL, KOREA: SEOUL NATIONAL UNIVERSITY  
Department of Zoology

Wild Stocks

Canton-S

Seoul-S

Chromosome 1

bo

br

cm

ec

fa

sc cv v f

t<sup>2</sup> v f

v

w

w<sup>a</sup>w<sup>e</sup> bb<sup>l</sup>/ClB

y

Chromosome 2

ab

al dp b pr c px sp/Cy, pr

b lt wxt bw

b vg

c

c wt px

Cy/Pm

L

L<sup>4</sup>

pd

pr

Chromosome 3

aa h

cv-c sbd<sup>2</sup>

gl

Gl Sb/LVM

h

jv

p

ro

ru

ru h th st cu sr e<sup>s</sup> ca

se h

ss

st

th st cp

Chromosome 4

ci gvl bt

ey

ci gvl ey<sup>R</sup>/sv<sup>n</sup>

spa

Multichromosomal

v;bw (1;2)

w; vg (1;2)

bw;st (2;3)

Cy/Pm;D/Sb (2;3)

Cy/Pm;Sb/Ubx (2;3)

lys rc;ss (2;3)

vg;se (2;3)

Inversions

Muller-5

VIENNA, AUSTRIA: INSTITUT F. ALLG. BIOLOGIE

Wild Stocks

1 Oregon-S

2 Lipari

3 Wien

4 Kairo

Chromosome 1

5 BB

6 ClB/v ptg oc sn

7 fa<sup>n</sup>

8 Muller-5

9 pn

10 w

11 w sn B

12 y

13 y/f

14 y w

Multichromosomal

28 bw; e

29 bw; st

Chromosome 2

15 b cn vg

16 bw

17 dp b

18 L<sup>2</sup>19 L<sup>4</sup>

20 L Cy/Pm

21 L/Cy

22 vg

23 vg<sup>p2</sup>Chromosome 3

24 e cu

25 jv se

26 Ly/D<sup>3</sup>

27 st

BLINDERN, NORWAY: UNIVERSITY OF OSLO  
Institute of General Genetics

The stock list remains unchanged from DIS 40:27 with correction as in DIS 41:54.

## NEW MUTANTS

Report of M. K. Nelson

c62i: curved 62i E. Ehrlich, 62i. 2-75.5. Found in new Mad. 1 gr 5#3 in a Muller-5 fly in 1962. From stock irradiated in Wisconsin by S. Abramhamson. Thin textured wings which become crinkly with age, and which often fail to fully expand after pupation. Curved slightly downward throughout entire length, with strong tendency to invert. Narrow with age due to crumpling at edges. Originally set at about 30° and widely spread from body, but tend to rise to almost 75° when active. Allele of curved. RK2.

Additional information to Report of C. Tokunaga on Multiple sex comb (Msc), DIS 41:57.

In Table 1 b, 100 units of length = 0.65mm.

Report of C. W. Clancy

This is a correction as to the identity and localization of a gene originally named pteridine modifier (ptm) and reported as a new mutant in DIS 39:65. The "mutant" gene behaves as if it were a wild type isocallele of the bw locus. The original report indicates the way in which it can be differentiated from the bw<sup>+</sup> allele of the + Oregon-R strain. A summary of additional observations is given under Research Notes in this issue.

Report of M. Whittinghill and Mother M. Clancy

upt: upturned bristle S. Bryan 63f Of spontaneous origin, chromosome 2 in isogenic stock of dp pr stw px; recessive; dorsocentral bristles and/or anterior scutellar setae are curled upward. Penetrance at 40% at 21 ± 1° C. and up to 60% at 25 ± 1° C.; variability of expression. Determined as being half way between dumpy and purple, or perhaps nearer purple.

Report of Sara H. Frye

Frye (1966, DIS 41:175) reported that several singly-occurring, male-viable, forward-mutant phenotypes were recovered from pilot X-ray experiments (sc<sup>8</sup>·Y/y ac In49 BM1 ♂♂ 4 kr X Y/y f:=<sup>VV</sup>♀♀) designed to test for recovery of singly-occurring, male-viable, reverse-mutant phenotypes from yellow-and/or-achaete regions. Results of the progeny test (i.e., forward-mutant ♂ X 2 or 3 Y/y f:=<sup>VV</sup>♀s) of each forward-mutant are given below where the mutant phenotype is represented by the traditional genotypic symbol and placed in parentheses. (All cultures were discarded and no tests for allelism were conducted since facilities were not available.)

- |  |  |
|--|--|
| 1. y ac (w) In49 BM1 ♂ - sterile<br>....                     | 5. y ac In49 (r) BM1 ♂ - fertile and trans-<br>.... missible                 |
| 2. y ac (w) In49 BM1 ♂ - fertile and trans-<br>.... missible | 6. y ac In49 (f <sup>ex</sup> ) BM1 ♂ - sterile                              |
| 3. y ac (m) In49 BM1 ♂ - sterile<br>....                     | 7. y ac In49 (f <sup>ex</sup> ) BM1 ♂ - sterile                              |
| 4. y ac In49 (1z) BM1 ♂ - sterile<br>....                    | 8. y ac In49 (f <sup>ex</sup> ) BM1 ♂ - fertile and trans-<br>.... missible. |

Report of W. D. Kaplan and Nancy Hayes

fx: frostex 1:2.6. Recovered as male following the feeding of  $H^3TdR$  to first instar male larva. Strong echinus-like eye, darkening with age, with glistening frosted appearance. Originally set up in stock with attached-X females. Homologous females subsequently found to be sterile. Sterility factor not associated with a chromosomal rearrangement, but may be separable from fx gene.

Report of George E. Brosseau, Jr.

In(1)601 (2b; 6A1-2), B<sup>r601</sup>. X-ray induced. B<sup>r601</sup> is a recessive allele of Bar. Allelism was determined in the heterozygote with B<sup>M1</sup>, which is also recessive. The eyes of B<sup>r601</sup> homozygotes and hemizygotes are 1/2 to 2/3 normal size. The lower half of the eye is reduced a little more than the upper half; the phenotype of B<sup>r601</sup> resembles that of Lobe. B<sup>r601</sup> is inseparable from In(1)601 which has the proximal break at 16A1-2 and the distal break in the bulb of 2B. In(1)601, B<sup>r601</sup> has excellent viability and fertility as a homozygote; B<sup>r601</sup> shows little variability in phenotype and is easily classified. RK1

Report of H. A. Bender

spa<sup>en-lz</sup>: sparkling-enhancer of lozenge Bender, 65b23. 4-(iso or pseudoallele of spa). Spontaneous in lz<sup>D</sup>/dl-49, m<sup>2</sup> g<sup>4</sup>; Cy/Pm (1;2) stock which was found to be homozygous for spa<sup>en-lz</sup>. Homozygotes for the enhancer which are at the same time lz<sup>k</sup>/+, where lz<sup>k</sup> stands for lz<sup>3</sup>, lz<sup>36</sup> or lz<sup>D</sup>, have somewhat smaller, strongly roughened eyes. Slight eye roughening occurs in lz<sup>k</sup>/+ heterozygotes when the enhancer is heterozygous. spa<sup>P</sup>/spa<sup>en-lz</sup> and spa<sup>p65</sup>/spa<sup>en-lz</sup> have very rough eyes but normal tarsal claws and spermathecae. In the absence of lozenge, spa<sup>en-lz</sup> homozygotes are wild type. The eye morphology of lz<sup>D</sup>/+ in the absence of the enhancer appears near wild type.

spa<sup>p65</sup>: sparkling-poliert Bender, 65j11. Spontaneous in lz<sup>y4</sup>/FM6 stock. Rough, partially glazed eyes moderately reduced in size. More extreme allele than spa<sup>P</sup> (new symbol for the original sparkling-poliert mutant, formerly pol - Bickenbacher, DIS 27:59, and spa<sup>pol</sup> - Sturtevant, DIS 35:47) but less extreme than spa<sup>p61</sup>. Tarsal claws unpigmented and possibly reduced, reminiscent of certain lozenge mutants. However, pulvilli and accessory female reproductive structures appear normal. spa<sup>p65</sup> is phenotypically allelic to spa<sup>P</sup> and spa<sup>p61</sup> with heterozygotes having affected tarsal claws as well as rough eyes. spa/spa<sup>p65</sup> heterozygotes have slightly roughened eyes at 25°C. but markedly roughened eyes at 18°C., intermediate in degree of expression between that of the respective homozygotes, with females being somewhat more extreme than males. Viability and fertility of spa<sup>p65</sup> are good. RK1

Report of S. Mittler

eyg<sup>64e</sup>: eye-gone<sup>64e</sup> Mittler, 64e15. 3-35.5. Found in offspring of x-rayed male heterozygous for "rucuca" and homozygous "rucuca". Eye is smaller and less variable than eyg. Can be detected in some heterozygous flies by a slight reduction of eye size. RK1.

pn<sup>63d</sup>: prune 63d. Mittler, 63d5.1-0.8. From offspring of x-rayed Oregon-R male mated to attached-X y cv v f. Similar in color to pn<sup>2</sup>. RK1.

Report of A. H. Sturtevant

spa<sup>p61</sup>: sparkling poliart Sturtevant, 1961. Spontaneous in closely inbred (bristle selected) sc, st stock. Small, rough, glazed eyes. More extreme allele than spa<sup>p</sup> or spa<sup>p65</sup> but possibility of modifiers not excluded. Non-pigmented tarsal claws. Heterozygotes with spa<sup>p</sup> and spa<sup>p65</sup> show the mutant phenotype. RK1

Report of P. T. Ives

If: Irregular facets. Lucy Casey, 65 l 16. 2-107.6 U. A spontaneous rough eye mutant in the ho \$, If was observed by Mrs. Casey and studied by P. T. Ives. If/+ has about half the + eye area, is oblong and generally pointed ventrally. The facets are strongly irregularly distributed, are frequently missing across the middle of the eye, and are sometimes fused or absent in the ventral portion. If/If is generally easily distinguished from If/+ and is a narrow slit of an eye with a smooth glossy surface. In heterozygous If the eye colors bw and or are easily distinguished from + but or<sup>45a</sup> is much less readily classed. If has been localized by tests with b vg bw, or<sup>45a</sup> sp<sup>2</sup> and a px or. Its locus rests on 20 crossovers between sp<sup>2</sup> and If in 3367 flies and on 51 crossovers between or or<sup>45a</sup> and If in 5702 flies. The locus order was clearly bw or sp If in this study. Viability and fecundity of If/+ and If/If are good to excellent. No marked pleiotropy has been noticed. If is easier to score than Pin. The order of If and Pin has not been determined, nor has If been studied cytologically. RK1.

sca<sup>65131</sup>: scabrous. P. T. Ives. U. Arose in the generation 450 stock bottle of inbred Oregon-R. Allelism indicated in mating to sca l (2) C. Looks like sca but its bristle effects have not been studied quantitatively.

T(2;3), Bl vg; se. P. T. Ives. U. Induced by X-rays, this translocation was synthesized for the purpose of delivering specific pairs of non-translocated chromosomes (+; +/vg; se) to lethal tests with Cy SM5: TML. It breeds well when balanced with Cy bw<sup>45a</sup> or <sup>45a</sup> sp<sup>2</sup>; se, which pair of markers serves to deliver T(2;3)+; + pairs to similar lethal tests. It does not breed well when balanced with a Cy and either TML or TM3. It has not been studied cytologically.

Report of R. C. Gethmann

Dp(1;f)65; Duplication(1;f)65 Thompson 1965. From X-irradiated wild type males. Covers the deficiency of XX, Df(1)60g (Mohler DIS 34) and contains wild type alleles of the following loci: y, ac, sc, (but not pn), and su<sup>+</sup>-f (but not car).

Dp(1;f)65X<sup>c2</sup>; Duplication(1;f)65X<sup>c2</sup> Thompson 1965. From X-irradiated X<sup>c2</sup> males. Covers the deficiency of XX, Df(1)60g and contains the wild type alleles of the following loci: y, ac, sc (but not pn), and su<sup>+</sup>-f (but not car). The duplication is somatically unstable, mosaics frequently occur.

Y, su<sup>+</sup>-f (=Dp(1;Y)su<sup>+</sup>-f; Duplication (1;Y)su<sup>+</sup>-f Wong 1965. From X-irradiated T(XY<sup>L</sup>.YS:4)BS(16A1), XD, BSYL.YS males. Covers the deficiency of XX, Df(1)60g, and contains su<sup>+</sup>-f locus, but no loci from X distal or the tips of any of the autosomes. The tested loci were y, ac, sc, pn, al, sp, ru, ca, and ey.

Report of H. J. Becker

sn<sup>65a</sup>: singed<sup>65a</sup> 1 - 21.0. X-ray induced in Berlin wild male. Males fertile, homozygous females entirely sterile. Most bristles on head, thorax and scutellum bent, wavy and occasionally forked. Hairs wild type. Viability good.

## LINKAGE DATA

Report of P. T. Ives

Location of new mutant, If<sup>65</sup> 1 16, and relocation of the or locus.

A. From b vg bw +/+++ If.

O= 248+335; 1= 44+33; 2= 120+164; 3= 8+18; 1,2= 18+10; 1,3= 1+0; N= 1009; R1= 10.5; R2= 30.9; R3= 2.7.

B. From a px or +/+++ If.

O= 1011+1118; 1= 16+12; 2= 67+92; 3= 9+11; N= 2335; R1= 1.2; R2= 6.8; R3= 0.9.

C. From or<sup>45a</sup> sp<sup>2</sup> +/++ If.

O= 1555+1781; 1= 5+6; 2= 9+11; N= 3367; R1= 0.3; R2= 0.6.

In these data the order is clearly bw or sp If, and the distances are or - sp, 0.3 (11 in 3367); sp - If, 0.6 (20 in 3367); and or - If, 0.9 (51 in 5702). Given sp at 107.0 this indicates 106.7 for or and 107.6 for If.

Report of C. Tokunaga

Recombination frequency between Tufted and Bristle. Tufted (Tft) is known to be located somewhere on the second chromosome between pr and b. The following experiment shows that the locus of Tft is to the left of Bl. The recombination frequency is 1.2 per cent. P: y/y, sc<sup>19i</sup> Tft/ Bl ♀♀ X y ♂♂

F<sub>1</sub>: (N=11,953)

non crossovers	crossovers		
	sc <sup>19i</sup> -- Tft	Tft -- Bl	Double crossovers
Tft : 3978	y,Tft: 1979	Tft,Bl: 69	y,Tft,Bl: 8
y,Bl: 3911	Bl: 1936	y: 55	+: 17

## LINKAGE DATA

Report of E. Bahn

Crossing over between structural genes for amylases (Amy). Recombination analysis (see research note, this issue) has demonstrated spatial separation with a linkage intensity of the order of 1/10,000 (0.01 map units) for a pair of structural genes each determining a single band in the electrophoretic pattern. The terminology employed is based on agar gel electrophoresis and conforms with that of Kikkawa (1964, Jap. J. Genet. 39:401) and Doane (1966, DIS 41: 93).

Strains representing three different banding patterns were analysed, viz. Amy<sup>4.6</sup> established by Dr. Doane from an adipose<sup>60</sup> line, Amy<sup>2.3</sup> isolated from a Bennett population (58-105) by the author, and Amy<sup>1</sup> from The Institute for Cancer Research, Philadelphia (DIS 39: 20 g99). From heterozygotes Amy<sup>1</sup>/Amy<sup>2.3</sup> two recombinant chromosomes were recovered showing only one band Amy<sup>2</sup> in the electrophoretic pattern and one chromosome with the amylase pattern Amy<sup>1.3</sup>. From heterozygotes Amy<sup>2.3</sup>/Amy<sup>4.6</sup> two recombinants Amy<sup>2.6</sup> and one recombinant Amy<sup>4.3</sup> were recovered. Concordant recombination was observed in two outside marker genes (c and wt). Six recombinant chromosomes were found among ca. 77,500 scored.

These data suggest that different amylase electrophoretic bands are determined by separate structural genes. For the strains in question it is evident that the loci of the Amy<sup>3</sup> and Amy<sup>6</sup> alleles are placed to the right of the loci of the Amy<sup>1</sup>, Amy<sup>2</sup>, and Amy<sup>4</sup> alleles. Accordingly it is suggested to alter the symbols for the amylase genes: Stock Amy<sup>4.6</sup> should hereafter be designated Amy<sup>4</sup> Amy<sup>6</sup>, and Amy<sup>2.3</sup> designated Amy<sup>2</sup> Amy<sup>3</sup>, and Amy<sup>4.3</sup> designated Amy<sup>4</sup> Amy<sup>3</sup> etc. corresponding to the linkage data. The importance of distinction between stocks of independent origin possessing identical electrophoretic patterns is evident.

Report of George E. Brosseau, Jr.

The salivary chromosome breakpoints of In(1)S are: distal break in region 6F, proximal break in 10F just distal to the dark band S 11A1-2.

Report of Steven R. Thompson

Localization of the major gene in a crossveinless-like strain of Drosophila melanogaster. The major effect in cvl-5 (a crossveinless-like selected strain) was localized to chromosome III by Mohler (1965, Gen. 51: 641). In order to determine if this effect was primarily due to a major gene or a number of modifiers, a more precise localization was undertaken. Crossveinless-like females (Orinda X, Orinda II, cvl-5-hi III) were mated to ru h th st cu sr e<sup>s</sup> ca ("rucuca") males. F<sub>1</sub> females were mated to "rucuca" males, and single crossovers were selected (as males) and test crossed to Orinda X, Orinda II, cvl-5-hi III females to determine the presence or absence of crossveinlessness. From this study it was learned that the cvl-5-hi major gene was located between st and cu. Next, recombinant chromosomes with a crossover located between st and cu were selected to refine the localization. Of the st cu<sup>+</sup> 8 were cvl<sup>+</sup> and 18 cvl; of the st<sup>+</sup> cu 20 were cvl<sup>+</sup> and 9 cvl; thus, 69% (38/55) of these crossovers were located between st and the cvl-5-hi major gene and the rest were located between the cvl-5-hi major gene and cu. Sixty-nine % of the map distance between st and cu (6.0 map units) is approximately 4.1 map units. Thus cvl-5-hi lies 4.1 map units to the right of st (44.0) at 48.1 on chromosome III.

AUSTIN, TEXAS: UNIVERSITY OF TEXAS  
Department of Zoology

DROSOPHILASubgenus Dorsilopha

busckii (12 strains)

Subgenus Drosophilavirilis group

americana (16)

a. texana (7)

borealis (6)

ezoana (3)

laticola (1)

littoralis (4)

montana (18)

novamexicana (8)

virilis (23)

subtilis group

subtilis (1)

robusta group

lacertosa (5)

robusta (6)

sordidula (1)

melanica group

euronotus (3)

melanica (5)

micromelanica (3)

nigromelanica (3)

paramelanica (5)

pengi (1)

annulimana group

araicas (2)

gibberosa (1)

undet. spp. (5)

canaline group

canaline (7)

mesophragmatica group

gaucha (2)

pavani (2)

repleta groupfasciola subgroup

fascioloides (2)

fulvalineata (1)

moju (1)

mojuoides (2)

hydei subgroup

bifurca (4)

hydei (35)

eohydei (5)

neohydei (2)

nigrohydei (2)

melanopalpa subgroup

canapalpa (2)

fulvimacula (12)

fulvimaculoides (2)

f. falvorepleta (2)

limensis (1)

melanopalpa (19)

repleta (1)

mercatorum subgroup

mercatorum (17)

m. pararepleta (3)

paranensis (14)

mulleri subgroup

aldrichi (4)

anceps (1)

arizonensis (6)

buzzatii (8)

hamatofila (1)

longicornis (1)

martensis (1)

meridiana rioensis (6)

meridionalis (1)

mojavensis (2)

mulleri (16)

nigricruria (10)

pegasa (1)

peninsularis (15)

stalker (1)

tira (1)

nannoptera group

nannoptera (1)

polychaeta group

polychaeta (2)

immigrans group

curviceps (4)

hypocausta (2)

immigrans (20)

nasuta (spinofemora) (9)

nasutoides (hypopygialis) (12)

pararubida (4)

rubida (4)

setifemur (6)

funnebris group

funnebris (9)

macrospina (11)

m. limpiensis (3)

subfunnebris (1)

quinaria group

brachynephros (1)

falleni (3)

guttifera (4)

innubila (3)

munda (2)

nigromaculata (4)

occidentalis (5)

palustris (1)

phalerata (1)

quinaria (1)  
subpalustris (2)  
transversa (1)  
unispina (1)

testacea group

putrida (3)  
testacea (2)

guarani group

griseolineata (5)  
guaraja (1)  
guaramunu (3)  
guarani (2)  
subbadia (2)

calloptera group

ornatipennis

cardini group

acutilabella (28)  
belladunni (1)  
cardini (38)  
cardinoides (27)  
dunni (21)  
neocardini (3)  
neomorpha (6)  
parthenogenetica (9)  
polymorpha (10)  
procardinoides (1)

rubrifrons group

parachrogaster (2)

macroptera group

submacroptera (1)

pallidipennis group

hyalipennis ? (1)  
pallidipennis (10)  
p. centralis (2)

tripunctata group

crocina (6)  
mediodiffusa (13)

mediopunctata (1)  
mediopictoides (3)  
medionotata? (2)  
medionotata (1)  
mediostriata (10)  
metzii (1)  
paramediotriata (1)  
tripunctata (11)  
unipunctata (10)

bizonata group

bizonata (1)

histrion group

histrion (1)  
undet. sp. (1)

Unplaced spp.

castanea (4)  
daruma (1)  
para? (2)  
peruviana? (5)  
serenensis (1)  
sternopleuralis (1)  
sticta (1)  
tumiditarsus (1)  
tumiditarsus? (1)  
virgata (1)

Subgenus Hirtodrosophila

duncani (4)  
pictiventris (3)

Subgenus Lordiphosa

undet. sp. (1)

Subgenus Scaptodrosophila  
(=Pholadoris)

bryani group

bryani (1)

coracina group

cancellata (1)  
coracina (3)  
lativittata (1)  
novopaca (1)

latifasciaeformis group

latifasciaeformis (6)

victoria group

lebanonensis (1)  
l. casteeli (1)  
pattersoni (1)  
victoria (1)

Subgenus Sophophora  
obscura group

affinis (12)  
algonquin (1)  
ambigua (1)  
athabasca (4)  
azteca (1)  
bifasciata (2)  
narragansett (1)  
pseudoobscura (4)  
subobscura (2)  
tolteca (4)

melanogaster group

ananassae (41)  
auraria (3)  
bipectinata (2)  
birchii (5)  
kikkawai (14)  
lutea (2)  
mayri (3)  
melanogaster (10)  
nikanaru (1)  
rufa (2)  
seguyi (1)  
serrata (12)  
simulans (13)  
suzukii (1)  
szentivani (1)  
takahashii (1)  
yakuba (1)

willistoni group

capricorni (6)  
equinoxialis (6)  
fumipennis (10)  
nebulosa (25)  
paulistorum (4)  
sucinea (17)  
tropicalis (4)  
willistoni (13)

saltans group

austrosaltans (1)  
emarginata (17)  
lusaltans (1)  
milleri (2)  
neocordata (2)  
neoeleptica (2)  
nigrosaltans (1)  
parasaltans (1)  
prosaltans (25)  
pseudosaltans (1)  
saltans (1)  
septentriosaltans (1)  
sturtevantii (42)  
subsaltans (1)

Hawaiian species

adiastola (1)  
crucigera (9)  
disjuncta (1)  
engyochracea (1)

eurypeza (1)  
fasciculisetae (1)  
grimshawi (4)  
mimica (8)  
picticornis (1)  
pilimana (1)  
takadai (2)

## CHYMOMYZA

amoena (2)  
procnemis (2)

# DETTOPSOMYIA

nigrovittata (1)

## LTODROSOPHTLA

aerea (1)

## MYC ODR OS OPH TLA

dimidiata (1)

stalker (1)

## SCAPTOMYZA

adusta (2)  
anomala (5)  
australis (2)  
hamata (1)  
pallida (8)  
palmae (7)  
quadridentata (2)  
varifrons (3)  
xanthopleura (4)  
cryptoloba (2)

## ZAPRIONUS

ghesquierei (1)  
multistriata? (1)  
tuberculatus (1)  
vittiger (6)

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA  
Department of Zoology

700 simulans wild 801 virilis scarlet 950 Zaprionus vittiger  
800 virilis Pasadena wild 900 montium Abidiaw

EAST LANSING, MICHIGAN: MICHIGAN STATE UNIVERSITY  
Department of Zoology

robusta: East Lansing 1964      immigrans: East Lansing 1964      pseudoobscura: Rochester  
melanica: East Lansing 1964      virilis: California      funebris: Yale

BOSTON, MASS.: NORTHEASTERN UNIVERSITY

[illegible]

NEW HAVEN, CONN.: YALE UNIVERSITY  
Department of Biology

See DIS 41:67, delete acanthoptera, mirim, quinarina.

BOGOTÁ D. E., COLOMBIA: UNIVERSIDAD DE LOS ANDES  
Instituto de Genética

saltans: Costa Rica  
 prosaltans: Sao Joao Island, State of Sao Paulo; Rio Grande do Sul; U. S. A.  
 neocordata: Boas Esperancas, Minas Gerais  
 nigrosaltans: Estado del Paraná  
 septentriosaltans  
 parasaltans  
 austrosaltans  
 lusaltans: Haití  
 emarginata: Costa Rica  
 sturtevantii: Cuba; U. S. A.  
 milleri  
 funebris: Bilbao, Spain  
 cardini: Barranquilla, Colombia  
 immigrans: Gigante, Colombia; Sasaima, Colombia; Barcelona, Spain  
 repleta: Barcelona, Spain; Puente Nacional, Colombia; Neiva, Colombia; Bogotá, Colombia; San Agustín, Colombia  
 hydei: Barcelona, Spain; Copiapó, Chile

willistoni: Sasaima, Colombia; Huston, U. S. A.; Manizales, Colombia; San Agustín, Colombia, Gigante, Colombia  
 nebulosa: Huston, U. S. A., Gigante, Colombia; San Agustín, Colombia  
 gaucha: Valparaiso, Chile  
 pavani: Valparaiso, Chile  
 viracochi: Santiago, Chile  
 brancici: Neiva, Colombia  
 mesophragmática: Manizales, Colombia  
 pseudoobscura: Bogotá, Colombia  
 subobscura: Barcelona, Spain  
 busckii: Barcelona, Spain  
 ambigua: Lagrasse, France  
 phalerata: Cantonigrol, France  
 ananassae: Amazonas, Colombia; Turbe, Colombia; San Andrés, Colombia, Caracolisito, Colombia; Barranquilla, Colombia; Florian, Colombia; Sincelejo, Colombia; Medellin, Colombia

SÃO JOSÉ DO RIO PRÊTO, SÃO PAULO, BRAZIL: FACULDADE DE FILOSOFIA, CIÊNCIAS E LETRAS  
Departamento de Biologia Geral

willistoni  
 paulistorum  
 guarani  
 campestris  
 mediotriata  
 nigricruria  
 pallidipennis  
 latifasciaeformis  
 polymorpha  
 simulans  
 nebulosa  
 ananassae  
 ararama  
 immigrans  
 guaranumu  
 mediopunctata

fumipennis: Mirassol (Est. de São Paulo)  
 montium: São José do Rio Preto (Est. de São Paulo)  
 prosaltans: Brazil (El Dorado, Belém, Guarapari, Ilha São João); Costa Rica (Palmar, Piedras Blancas, San Isidro, Turrialba); Panamá (Balboa, Chanquinola); Trinidad (Sangre Grande); Colombia (Bucaramanga)  
 saltans: El Salvador (San Salvador); Guatemala (Guatemala); Mexico (Huichihuayan - San Luiz Potosi, Chilpancingo)  
 austrosaltans: Brazil (Pirassununga)  
 septentriosaltans: Colombia (sevilla)  
 parasaltans: Brazil (Uapes)  
 neocordata: Brazil (Boa Esperanca)  
 emarginata: Panamá (Cerro Campano); Perú (Tingo Maria); Costa Rica (Turrialba)  
 milleri: Porto Rico (El Yunque)  
 sturtevantii: Brazil (I. das Cobras, I. Queimada Grande, I. Queimada Pequena, I. São João, Amazonas, Maranhão, Pará, Paraná, Espírito Santo, Rio de Janeiro, Mato Grosso); Cuba, Porto Rico, Guadalupe, Santa Lucia, Trinidad, Mexico, Honduras, El Salvador, Costa Rica, Panamá, Colombia, Venezuela, Perú, Guiana Britanica.

SANTIAGO, CHILE: UNIVERSIDAD DE CHILE  
Instituto de Biología "Juan Noé", Departamento de Genética

busckii: Chile (La Serena)  
 camaronensis: Chile (Azapa)  
 funebris: Chile (La Serena, Valdivia, Tierra del Fuego and Punta Arenas)  
 gasici: Chile (Arica), Bolivia (Cochabamba), Colombia (Bogotá)  
 gaucha: Brazil (M. Capoes, C. de Jordan and Taimbas), Argentina (Córdoba)  
 hydei: Chile (Camarones and El Tabo), Bolivia (Cochabamba)  
 immigrans: Chile (El Tabo and Valdivia)  
 mercatorum: Chile (Arica)  
 mesophragmatica: Bolivia (La Paz), Perú (Machu-Picchu, Cuzco)  
 pavani: Chile (Copiapó, Vallenar, La Serena, El Tabo, Viña del Mar, Olmué, Bellavista, Arrayán, Los Alpes, Colbún, Los Queñes, Chillán), Argentina (Mendoza)  
 simulans: Perú (Lima), Chile (Arica)  
 viracochi: Perú (Machu-Picchu), Colombia (Bogotá)  
 virilis: Chile (Santiago)

SÃO PAULO, BRAZIL: UNIVERSIDADE DE SÃO PAULO  
Departamento de Biologia Geral

ananassae: Brazil	prosaltans: Brazil, Colombia, Costa Rica
austrosaltans: Brazil	Panamá
emarginata: Costa Rica, Panamá, Perú	repleta: Brazil
equinoxialis: Brazil	saltans: El Salvador, Guatemala, Mexico
immigrans: Brazil	simulans: Brazil, South Africa
montium: Brazil	sturtevantii: Brazil, Colombia, Costa Rica
nebulosa: Brazil	El Salvador, Guiana, Honduras, Mexico,
neocardini: Brazil	Panamá, Perú, Puerto Rico, Trinidad,
neocordata: Brazil	Venezuela

NOTE: For some species we have strains from several places.

willistoni

Wild Stocks

Ituitaba, Natal, Angra dos Resi, São Paulo,  
 Pirassununga (Brazil), Lima (Perú)

Chromosome 1

<sup>e</sup>  
 w y sn ru  
 se  
 w

Chromosome 2

Em/abb bw  
 S Hk abb bw (In)/cn  
 S Hk abb bw (In)/leth  
 S Hk abb bw (In)/br

Chromosome 3

Delta pink (In)/leth  
 e

ST. LOUIS, MISSOURI: WASHINGTON UNIVERSITY  
Department of Biology

americana Missouri	immigrans (various)	pengi Japan
buzzatii (various)	lacertosa Japan	pseudosordidula Japan
carsoni Vermont	macrospina Georgia	repleta (various)
colorata Michigan	melanica (various)	robusta (various)
duncani South Carolina	melanura (various)	sordidula Japan
euronotus (various)	mercatorum (various)	stalker (various)
funnebris Minn.	micromelanica (various)	subfunnebris California
gibberosa Mexico	nigromelanica (various)	thoracis Texas
hydei (various)	paramelanica (various)	virilis Mexico

CHICAGO, ILLINOIS: UNIVERSITY OF ILLINOIS AT CHICAGO CIRCLE  
Department of Biological Sciences

*persimilis*: Wild strains from localities: White Wolf (Yosemite, 8,000 ft.), Timberline (10,000 ft.), Mather (4,000 ft.) and Humboldt (Redwoods forest).

Mutant strains: Chromosome 3 Multichromosomal

or Bl Sc pr/or pr Delta; or; Curly

*pseudoobscura*: Wild strains from Mather (AR - 10 strains, ST - 10 strains, TL - 10 strains PP - 14 strains, CH - 7 strains)

DAYTON, OHIO: UNIVERSITY OF DAYTON  
Department of Biology

<i>affinis</i> - Dayton, Ohio, 1966	<i>repleta</i> - Dayton, Ohio, 1966	<i>tripunctata</i> - Dayton, Ohio, 1966
<i>melanogaster</i> - Wild Type Dayton, Ohio, 1966	<i>robusta</i> - Dayton, Ohio, 1966	

LINCOLN, NEBRASKA: UNIVERSITY OF NEBRASKA  
Zoology and Physiology Department

*affinis*: Arkansas, Florida, Nebraska, Ontario  
*algonquin*: Minnesota, Ontario, Vermont  
*athabasca*: Alaska, British Columbia, Colorado, Idaho, Massachusetts, Minnesota, New Mexico  
 North Carolina, Ohio, Oregon, Ontario, Pennsylvania, Vermont, Washington  
*azteca*: Arizona, California, Mexico (Guerrero, D. F.)

POUGHKEEPSIE, NEW YORK: MARIST COLLEGE  
Department of Biology

D. pseudoobscura

Payson, Ariz. (3 strains)  
Pine Creek, Ariz. (3)  
Baker Butte, Ariz. (3)  
Flagstaff, Ariz. (1)  
Lake Mary, Ariz. (3)  
Grand Canyon, N. rim, Ariz. (3)  
Prescott, Ariz. (6)  
Sierra Ancha Mtns., Ariz. (1)  
Portal, Ariz. (3)  
Crystal Lake, Calif. (3)  
Sequoia Nat. Pk., Calif. (3)  
Yosemite Nat. Pk., Calif. (3)  
Nederland, Colo. (1)  
Montrose, Colo. (1)  
Black Canyon, Colo. (1)  
Custer, S. Dakota (3)  
Logan, Utah (2)

D. persimilis

Crystal Lake, Calif. (1)  
Sequoia Nat. Pk., Calif. (2)  
Yosemite Nat. Pk., Calif. (3)

D. busckii

Princeton, N. J.

D. melanogaster

Princeton (1)  
Poughkeepsie (1)

D. hydei

Poughkeepsie, N. Y. (1)

D. duncani

Poughkeepsie (1)

D. putrida

Poughkeepsie (1)

D. funebris

Poughkeepsie (2)

D. robusta

Princeton (2)  
Poughkeepsie (1)

D. immigrans

Princeton (1)  
Poughkeepsie (1)

D. affinis

Princeton (5)  
Poughkeepsie (3)

D. narragansett

Jackson St. Forrest, N. J. (1)

LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY  
Department of Zoology

D. affinis: Lexington, Kentucky  
D. busckii: Lexington, Kentucky  
D. hydei: Lexington, Kentucky  
D. putrida: Lexington, Kentucky  
D. robusta: Lexington, Kentucky  
D. tripunctata: Lexington Kentucky

SYDNEY, NEW SOUTH WALES, AUSTRALIA: UNIVERSITY OF SYDNEY  
Department of Animal Husbandry

ananassaeWild Stocks

- 1 Cristobal, Panamá  
 2 Texas

Mutants

- 3 st f  
 4 st w  
 5 se  
 6 se cv  
 7 or

persimilisWild Stocks

- 1 Porcupine Flat  
 2 Quesnell

## 3 Sequoia

Mutant

- 4 Delta or Cy  
pseudoobscura

Wild strains homozygous for  
Chromosome 3 Inversions

- Arrowhead (8 strains) Piñon,  
 Calif.  
 Chiricahua (8 strains) Piñon,  
 Calif.

Mutant

gl

simulans

2 wild strains from N. S. W. and  
 Victoria

Mutants

- 1 y  
 2 y v  
 3 v  
 4 st  
 5 p  
 6 st p  
 7 net pm (b py sd)

Other Species

montium  
 nebulosa

VARANASI, INDIA: BANARAS HINDU UNIVERSITY  
Department of Zoology

Wild Stocks

bipunctinata (Calcutta)  
 ananassae (Howrah, Kerala,  
 Mughalsarai, Bhagalpur)

malerkotliana  
 nasuta

immigrans

Mutants - ananassaeChromosome 1

y  
 w<sup>a</sup>  
 vs

Chromosome 2

cu b se  
 cu se  
 b se

cu b

b  
 cu  
 se  
 ic  
 cu bw  
 blo  
 ss<sup>a</sup>

Chromosome 3

px pc  
 stw pc  
 stw px  
 stw  
 px  
 pc

Unlocated Mutants

sp  
 ci  
 arch

ZURICH, SWITZERLAND: UNIVERSITY OF ZURICH  
Zoological Museum

1. Several cytologically defined homokaryotypic stocks of *D. subobscura* are maintained. In the following list, the chromosomes not mentioned are of the standard (St) order, as known for stock "Kusnacht".

- |   |   |
|---|---|
| 1 | Kusnacht  |
| 2 | Zurich: U <sub>1+2</sub>  |
| 3 | Zurich: U <sub>1+2</sub>  |
| 4 | Zurich: I <sub>1</sub>  |
| 5 | Tunis: I <sub>1</sub> , E <sub>1+2</sub> , U <sub>1+2+8</sub> , O <sub>3+4+8</sub>                        |
| 6 | Tunis: A <sub>1+2</sub> , I <sub>1</sub> , E <sub>1+2+9+4</sub> , U <sub>1+2+8</sub> , O <sub>3+4+8</sub> |

2. Mutant stocks of *D. subobscura*

- |     |              |     |               |
|-----|--------------|-----|---------------|
| 7:  | y            | 11: | pl pp pt      |
| 8:  | sj pl pp otp | 12: | m ct bnt v sc |
| 9:  | oc           | 13: | ho            |
| 10: | pm ct sn     | 14: | Bx            |

TÜBINGEN, GERMANY: MAX PLANCK INSTITUT FÜR BIOLOGIE (Abt. Beermann)

athabasca	fulvimacula	repleta
azteca	miranda	simulans
bifurca	neohydei	victoria
eohydei	negrohydei	virilis
funebria		

hydei (see report in DIS 40:37)

1	wild	17	w lt	<u>Chromosome 6</u>	
		18	w <sup>m1</sup>		
	<u>Chromosome 1</u>	19	w <sup>m2</sup>	25	Ci Ok/Ci
		20	w <sup>m3</sup>		
2	ch <sup>to1</sup> y <sup>t1</sup> m <sup>t1</sup>			<u>Multichromosomal</u>	
3	delta				
4	f <sup>2</sup>			26	bb; p vg (1;2;3)
5	g y m			27	scarlet, scabrous, javelin (2;3;5)
6	mt <sup>2</sup>	21	e <sup>Du</sup>		
7	Df(1), N/w lt			<u>Attached-X</u>	
8	to <sup>2</sup>			28	w lt/Y & +/Y
9	to <sup>3</sup>			29	v f/Y & w lt/Y
10	vt <sup>1</sup>	22	cn	30	y m ch/Y & w lt/Y
11	v <sup>t2</sup>	23	heart cn ro		
12	v <sup>t3</sup> T(X;2) homozygous lethal			<u>Y Chromosome</u>	
13	v f				
14	v sc sn y m ch bb	24	red eye, brown thorax		
15	w			31	H 194.23
16	w <sup>a</sup>			32	tube-proximal
				33	tube-distal

SAPPORO, JAPAN: HOKKAIDO UNIVERSITYDepartment of Zoology

histrioides (4 strains)	brachynephros (1)	lacertosa (4)
busckii (1)	anugularis (2)	moriwakii (1)
ananassae (1)	unispina (1)	neokadai (2)
kikkawai (1)	nigromaculata (1)	sordidula (3)
auraria race A (1)	immigrans (1)	pseudosordidula (4)
race B (1)	funnebris (1)	Scaptomyza pallida (2)
race C (2)	virilis wild (2' mutant (1)	S. concimilis (1)

CALCUTTA, INDIA: UNIVERSITY OF CALCUTTADepartment of ZoologyananassaeWild Stocks

1 a6 Calcutta  
 2 a7 Behala  
 3 a8 Kolaghat

Chromosome 2

4 bw vs ss<sup>a</sup>

Chromosome 3

5 pc

Multichromosomal

6 In(3)M pr pc px fu  
 7 stw px pc fu

Special Selected Strains with Crossing Over in Males

8 px pc (6a)  
 9 px pc (6a) high  
 10 px pc (6a-13)  
 11 px pc (3)  
 12 px pc (6a-4)

Other Species

bipectinata  
 simulans

LYON, FRANCE: UNIVERSITE DE LYONZoologie Experimentale

funnebris

busckii

MILAN, ITALY: UNIVERSITA' DI MILANOIstituto di GeneticasimulansWild Stocks

1 Aspra  
 2 Morro Bay  
 3 Pavia

Stocks Selected for Tumor Manifestation

4 tu B1  
 5 tu Aspra

JERUSALEM, ISRAEL: THE HEBREW UNIVERSITY  
Laboratory of Genetics

hydei	funnebris
immigrans	subobscura
simulans	subobscura Kusnacht

TURKU, FINLAND: UNIVERSITY OF TURKU  
Department of Genetics

<u>simulans</u>	<u>Chromosome 3</u>
wild	jv se st pe
	st pe

SALISBURY, RHODESIA: UNIVERSITY COLLEGE OF RHODESIA  
Department of Biological Science

seguyi: Salisbury  
 seguyi complex: 2 undescribed species from Rhodesia  
 simulans: Salisbury

BARQUISIMETO, VENEZUELA: CENTRO EXPERIMENTAL DE ESTUDIOS SUPERIORES

mesophragmatica of Columbia & Venezuela

Collections in Venezuela are just being started - soon many species will be available.

BERLIN-DAHLEM, GERMANY: INSTITUT FÜR GENETIK DER FREIEN UNIVERSITÄT BERLIN

58 funnebris	61 simulans: v
59 busckii: wild	62 virilis: wild
60 hydei: wild	63 pseudoobscura A 333
	64 pseudoobscura C 325

BRIGHTON, SUSSEX, ENGLAND: UNIVERISTY OF SUSSEX  
School of Biology

simulans

Wild Stocks

1 AG  
 Jer X

subobscura

Wild Stocks

B (inbred)  
 K (inbred)

## NEW MUTANTS

pseudoobscuraReport of D. W. Crumpacker

New genes on the third chromosome of *Drosophila pseudoobscura*. During a study of genetic loads and gene arrangements in a naturally occurring Colorado population of *D. pseudoobscura*, several visible third chromosome recessive mutants were isolated. These were observed in chromosomes made homozygous by use of the balanced lethal stock, B1 Sc pr (ST)/pr : (SC). Two of the mutants, both on AR (Arrowhead gene arrangement) chromosomes, appear to be useful markers for genetic studies, and are described below.

1. bv: brevis. Bristles shortened, especially the dorsocentrals and scutellars. Expression good in most individuals. Viability good. The name *brevis* is proposed because of similarity in bristly phenotype to that described for the *brevis* gene in *D. melanogaster* (Bridges, C. B., and Brehme, K. S., 1944). The monogenic nature of the mutant was determined from the following data:

		<u>+</u>	<u>bv</u>	<u>Total</u>	<u>P(X<sup>2</sup>)</u>
Testcross	Observed	83	76	159	0.50-0.75
	Expected	79.5	79.5	159	
F <sub>2</sub>	Observed	97	31	128	0.90-0.95
	Expected	96	32	128	

2. stw: straw. All hairs and bristles bright yellow. Wings often appear fragile and crumpled. Expression excellent. Viability fair. Apparently homologous to *stw* at 55.1, adjacent to the centromere, on the right arm of chromosome 2 of *D. melanogaster*. Inheritance of *stw* in *D. pseudoobscura* is complicated in two ways: (1) *stw* flies often exhibit a scute phenotype similar to *Sc*, a dominant gene located at 28.3 on chromosome 3 of *D. pseudoobscura*, which lowers the number of dorsocentral and especially scutellar bristles (Donald, H. P., J. Genet. 33:103-122, 1936; Sturtevant, A. H., and Tan, C. C., J. Genet. 34:415-432, 1937.); and (2) *stw* was first isolated on a semilethal chromosome. It is probable that neither the scute condition nor the semilethal viability is a property of the *stw* locus. This conclusion is based on the following evidence.

F<sub>1</sub> flies were obtained by outcrossing *stw* females to males from the same population which were homozygous for an AR chromosome which carried wild type genes and displayed normal viability. These F<sub>1</sub> individuals were then used as parents to produce testcross and F<sub>2</sub> progenies. Two types of progenies were obtained. When the parents were scute, there was a preponderance of scute to non-scute flies, and a striking deficiency of *stw* flies. When the parents were non-scute, the proportion of scute to non-scute flies was roughly 1:1 in the backcross and 1:3 in the F<sub>2</sub>, and many more *stw* flies occurred. These results suggest that two kinds of scute genes are involved. In the case of crosses involving scute parents, the dominant gene *Sc*, located originally on the B1 Sc pr (ST) chromosome of the balanced lethal stock has been transferred to the wild chromosome carrying *stw*. This requires a recombination of ST and AR chromosomes, a phenomenon known to occur at a low rate (Dobzhansky, Th., and Epling, C., Proc. Natl. Acad. Sci. 34:137-141, 1948.). In crosses involving non-scute parents, a recessive scute gene, perhaps newly arisen, is segregating. In the data given below, only the *stw* segregation is considered.

The viability of *stw* mutants is greatly reduced in crosses between scute parents. This suggests a deleterious interaction between the section of laboratory chromosome carrying *Sc* and the section of wild chromosome carrying *stw*. Viability is greatly improved in crosses between non-scute parents, even though scute progeny occur in both *stw* and + classes. A similar deleterious interaction evidently does not exist between *stw* and the recessive scute gene. (See table below)

Stocks of both *bv* and *stw* will be maintained at the Rockefeller University, where they can be obtained by writing to Mr. Boris Spassky. Before *stw* can be used in genetic studies, it will be necessary to obtain a purified stock which is free of the suspected dominant and recessive scute genes.

		<u>Scute Parents</u>			
		<u>+</u>	<u>stw</u>	<u>Total</u>	<u>P(X<sup>2</sup>)</u>
Testcross	Observed	83	18	101	<0.005
	Expected	50.5	50.5	101	
F <sub>2</sub>	Observed	122	4	126	<0.005
	Expected	94.5	31.5	126	

		<u>Non-scute Parents</u>			
		<u>+</u>	<u>stw</u>	<u>Total</u>	<u>P(X<sup>2</sup>)</u>
Testcross	Observed	22	13	35	0.10-0.25
	Expected	17.5	17.5	35	
F <sub>2</sub>	Observed	100	28	128	0.25-0.50
	Expected	96	32	128	

This report was partially supported by Public Health Fellowship 1-F3-CM-28, 365-01, from the National Institute of General Medical Sciences. The author is on leave from Colorado State University, Fort Collins, Colorado.

Note: the stw mutant is unofficially but affectionately known in the laboratory as "Ehrman yellow."

VIENNA, AUSTRIA: INSTITUT F. ALLG. BIOLOGIE

<u>D. ambigua</u>	Ponza, Italy	<u>Other Species</u>
<u>Wild Stock:</u> one strain	Thessaloniki, Greece	D. buzzatii: Ponza
<u>Chromosome 1</u>	Drobak, Norway	D. funebris: Lipari
or	<u>Chromosome 0</u>	D. hydei: Wien
y	Ba	D. mercatorum: Las Palmas
v	cu ch	Zaprionus Ghesquierei
	Va	<u>Megaselia scalaris</u>
<u>Autosomes</u>	Va Inv 8/Ba	(Phoridae)
pn	<u>Wild Strains Homozygous for Gene</u>	There are several stocks
Bd	<u>Arrangements in the Following</u>	in the collection with
bs	<u>Autosomes: (subobscura)</u>	recessive and dominant
dl	Küsnacht, homozygous in St for all	markers for chromosome I,
	chromosomes	II and III associated with
<u>D. subobscura</u>		the dominant male determining
<u>Wild Stocks</u>	A <sub>1</sub> /A <sub>1</sub>	factor on chromosome I, II
	A <sub>2</sub> /A <sub>2</sub>	and III respectively.
	A <sub>1+2</sub> /A <sub>1+2</sub>	
Wien, Austria	J <sub>1</sub> /J <sub>1</sub>	
	O <sub>3+4+7</sub> /O <sub>3+4+7</sub>	

Nöthel, H. Freien Universität Berlin, Berlin-Dahlem, Germany. An estimation of the number of active oogonia per ovariole in *D. melanogaster* females.

In spite of the fact that there is a lot of dividing cells in each germarium, the amount of active oogonia is quite unknown. It seems to be rather difficult to distinguish between oogonia and follicular cells by means of histological

methods. Hence, some indirect attachments have been made by using external influences on oogonia.

Since the development from oogonia to mature eggs lasts up to 10 days, the effects induced by such influences are seen in eggs laid 10 days after exposition and later. After X-irradiation the number of eggs laid at that time is decreased according to dose. This decrease is probably caused mainly by induced chromosomal aberrations in the oogonia and/or by affecting its DNA synthesis (Nöthel, unpublished). The radiation-induced reduction of egg laying, relative to unirradiated controls, therefore, is equal to the frequency of oogonia destructed. Without oogonial divisions an ovariole will become empty when the more mature stages have completed development. It diminishes and may be absorbed. The extent to which the ovariole number is decreased by a given reduction in the frequency of oogonia depends on the number of active oogonia per ovariole and is a matter of probability. Given in frequency numbers relative to the unirradiated controls (control values set 1) the equation assumed is: reduction in ovariole number = number of oogonia destructed potentiated with the number of oogonia/ovariole. Since in unirradiated controls as well as after X-irradiation with different doses the ovariole number is simply found by preparation, the destruction of oogonia by means of egg laying, the equation can be solved to find the number of oogonia/ovariole.

Eggs were counted 24 hours prior to preparation which was performed between the 10th and 16th day after onset of the experiment. Experimental conditions are to be published in detail elsewhere. The results are summarized in the table below.

unirradiated controls	number of w <sup>e</sup> -♀♀ tested	mean number of		number of oogonia per ovariole (x) calculated from
		eggs/♀/day	ovarioles/♀	
	99	41.7	38.3	
X-ray dose in kR		frequency of induced mean re- duction, relative to controls		x = log b : log a
		(a) eggs/♀/day	(b) ovarioles/♀	
4	97	0.089	0.099	0.96
5	106	0.153	0.164	0.96
6	96	0.590	0.433	1.59
8	90	0.887	0.817	1.69

As a result based on the assumptions made above it has to be stated that there are 1 to 2 active oogonia per ovariole. The differences between the 4 values calculated have proved statistically significant since the egg to ovariole number relations in the F-test have shown a high degree of inhomogeneity between dose classes. Therefore these differences are to be explained by methodological reasons. Moreover, they seem to be systematically in the way that with increasing doses and decreasing egg numbers the oogonia/ovariole number tends to increase from 1 to 2. This may be caused by some oversimplifications in the assumptions made above. Firstly, the experiments point to a decrease in the speed of egg deposition when the support of immature stages is lowered. Secondly, the duration of egg development may be enhanced by irradiation. Finally, at 6 or 8 kR damage in follicular cells may contribute to the reduction of egg development. Since egg number is measured per female per day as well in the controls as in the irradiated groups, all 3 factors mentioned can increase the postirradiation reduction in egg number relative to controls and will, therefore, increase the number of oogonia calculated. Hence, the number of 1-2 oogonia/ovariole resulting from these experiments may be reduced to only 1 by interpretation.

Hoenigsberg, H. F. and L. A. Granobles.  
 Universidad de les Andes, Bogotá D.E.,  
 Colombia. Comparative frequencies of  
 detrimental recessives in European and  
 neo-tropical populations of *D. mel.*

In continuing studies of natural popu-  
 lations of Diptera, certain general  
 findings concerning the presence of sig-  
 nificantly different quantities of re-  
 cessive genes which in double dose cause  
 death (lethal and semilethal genes are  
 grouped into this class) in central

European (Hungary) and in neo-tropical (Colombia) demes may be of interest to workers in the  
 marvels of natural selection and mutation. Table 1 presents the number of homozygous second  
 chromosomes classed according to the viability exhibited. For instance: we consider lethals  
 those chromosomes which produce from 0 to 10% of the normal viability. Normal viability is  
 the average viability of the wild-type in the heterozygous crosses (picked and crossed at  
 random using the table of random numbers). Semilethals are those chromosomes which in double  
 dose produce from 10 to 50% of the same normal viability; finally, quasinormals are those  
 chromosomes which in the homozygous condition produce from 50% up to normal viability. This  
 last group contains subvital, normal and supervital genes or gene complexes. To mark the  
 second chromosome the Cy L/Pm balanced lethal stock was used. From crosses between Cy L/Pm  
 and males collected from nature, F<sub>1</sub> progeny containing Cy L/+ heterozygous for the male  
 second are recovered and recrossed to Cy L/Pm females to get Cy L/+ and Pm/+ heterozygous  
 for the same second chromosomes; when these sibs are allowed to breed they produce four geno-  
 types: +/+, Cy L/+, Pm/+ and Cy L/Pm in equal proportions unless the lethal appears and  
 eliminates partially or completely the +/+ class. Random heterozygotes are made by crossing  
 F<sub>2</sub> individuals heterozygous Cy L/+ and Pm/+ from different cultures. All experiments were  
 performed at 24°C. Table 2 shows the percent of drastics in different populations while in  
 table 1 only one of the three replicates represent each population just to indicate the  
 approximate distribution of the three most important viability classes; in table 2 all  
 chromosomes from the three replicates are used to compute mean percentage of drastics.

Table 1. The homozygous second chromosomes per viability class.  
 One of the three replicates represent each deme of *D. melanogaster*.

Populations	Lethals	Semilethals					Quasinormals					Totals
	0	10	20	30	40	50	60	70	80	90	100+	
Hungary I	8	0	0	1	1	1	3	4	16	17	14	15 = 80
Hungary II	11	0	1	0	1	0	0	8	11	39	31	27 = 129
Hungary III	8	0	1	0	0	0	1	3	25	39	31	26 = 134
San Agustín I	25	3	1	3	3	3	3	9	14	21	9	15 = 109
San Agustín II	21	0	4	4	2	7	3	4	10	11	11	11 = 88
Sasaima II	20	1	0	1	2	2	1	6	11	11	9	8 = 72
Florian I	7	0	3	0	2	3	2	3	6	6	10	6 = 48

Table 2. Percent of drastics (lethals and similethals)  
 present in European and in neo-tropical populations of  
*Drosophila melanogaster*. N. stands for the total number  
 of chromosomes tested.

Populations	N	% drastics
Hungary I	307	9.12 ± 0.519
Hungary II	280	14.64 ± 2.112
Hungary III	374	7.22 ± 0.424
San Agustín I	274	39.05 ± 2.770
San Agustín II	257	37.74 ± 3.023
Sasaima II	208	41.35 ± 3.414
Sasaima III	240	25.42 ± 2.812
Florian I	134	29.10 ± 3.924

Clancy, C. W. University of Oregon, Eugene, Oregon. Identification of ptm (pteridine modifier) as a wild type isoallele of the bw locus.

Under special genetic conditions, the gene, ptm, reported as a new mutant in DIS 39:65, gives a positive test for allelism with the mutant gene bw<sup>1</sup>. As noted elsewhere in this issue (see under New Mutants), the original

localization of ptm to the third chromosome is incorrect. The probable reason for this error will be suggested after the special conditions required to demonstrate the gene's existence, and its allelism to bw have been described.

In order to simplify presentation, the original symbol, ptm, will be used throughout this note. In addition, the symbol, +, when used opposite ptm or bw in a genotype will represent the allele, bw<sup>+</sup>, as it occurs in the + Oregon-R wild type, the only standard strain against which comparisons have thus far been made.

The action of ptm on eye color is not detectable by ordinary methods of observation unless the fly is a compound of either of the two white alleles, w<sup>sat</sup>, or w<sup>cf</sup>, plus one of the genetic blocks to ommochrome (brown pigment) formation, v, cn, or st. For example, the genotypes, ptm/ptm, ptm/bw, ptm/+, and +/+ (+ Ore-R) are inseparable on the basis of eye color when observed one to five days after eclosion under a binocular microscope at ordinary magnifications. The same is true for corresponding arrays in which the brown pigment has been eliminated by the substitution of any one of the mutant genes, v, cn, or st. Corresponding compounds with w<sup>sat</sup> or w<sup>cf</sup> are likewise indistinguishable when the blocks to ommochrome formation are omitted. On the other hand, each member of the following two series of compounds is readily separable from any other within its group: w<sup>sat</sup> v;ptm/ptm (white, tinged with brown, and inseparable in color from the compound, v<sup>1</sup>;bw<sup>1</sup>); w<sup>sat</sup> v;ptm/+ (yellow, and clearly lighter than the medium orange of w<sup>sat</sup> v;+/+). The corresponding compounds with w<sup>cf</sup> are even more useful as indicators of the presence and dosage of ptm because the visible pigment differences are more striking, eg., w<sup>cf</sup> v;ptm/ptm (pale yellow), w<sup>cf</sup> v;ptm/+ (medium bright orange), and w<sup>cf</sup> v;+/+ (dark reddish orange).

The relation of ptm to the bw locus can be shown in several ways but a comparison of the results of the following two crosses emphasizes the genetic conditions required to obtain a positive test for allelism with bw. If w<sup>sat</sup> v;ptm ♀♀ are mated to v;bw ♂♂, the daughters (w<sup>sat</sup> v;ptm/bw) are phenotypically inseparable from their parents, ie., their eye color is tinged white. The sons (v;ptm/bw) are vermilion in eye color. In contrast, when y v;bw ♀♀ are mated to v;ptm ♂♂, all offspring are vermilion in eye color.

These gross phenotypic results suggest that ptm is a wild type isoallele of bw slightly hypomorphic to the bw<sup>+</sup> allele of Oregon-R with respect to the visible eye pigments (drospterines).

The assignment of ptm to a locus not significantly different from that of bw is also based on results of linkage studies utilizing the special genetic background required to follow it. The first of two recent experiments was a two-point in which the recombination fraction from a test cross of w<sup>cf</sup> v;ptm sp/+ ♀♀ was 51/2089 = 2.44, a number not significantly different from 2.5, the published value for the bw--sp interval. The second experiment was a test cross of w<sup>cf</sup> v;a px ptm sp/+. If the arc locus is ignored because of difficulties in classification, the results are: 0 = 2855 + 3844; 1 = 129 + 159; 2 = 83 + 75; N = 7145; R-1 = 4.03, and R-2 = 2.21. Although the values for the intervals, px--ptm, and ptm--sp, agree with what one might expect to obtain in a similar experiment involving bw, this experiment shows a significant shortage of the triple recessive non-crossovers.

An additional linkage experiment was performed in which a wild type recombinant chromosome for the bw locus was sought among the offspring of the cross: w<sup>cf</sup> v;px bw sp/ptm X w<sup>cf</sup> v;px bw sp. Such a recombinant, if it occurred, should be easily detected, since its carrier would have a bright orange eye color as opposed to the colorless eyes of its sibs. None was found among 12,860 offspring counted. Thus, if ptm is a pseudoallele of bw, the probability is less than 0.01 that recombination between them will exceed 0.05 map units.

It is now possible to understand how the original error of localizing ptm to the third chromosome occurred, for it is likely that the Cy chromosome of the tester stock carried either bw or ptm. This would account for the fact that from the test cross of w<sup>sat</sup>;Cy/-;D/st ♀♀ the critical class occurred with the Cy marker alone; not with Cy and D, or with D. (Supported by PHS grant GM-09802)

Momma, E. and A. Kaneko. Hokkaido University, Sapporo, Japan. Drosophilidae from three localities in Hokkaido and in Aomori Prefecture, based on collections in 1965.

Collections were made during the period from the 3rd of August to the 2nd of September, 1965, in Ohnuma, southern part of Hokkaido, Ikomabetsu (above 1,000 m from sea level), Taisetsu mountain region, central part of Hokkaido

and Yunosawa, Aomori Prefecture, northern part of Honshu. A total of 5,249 specimens was obtained which were mostly collected with fermented banana traps, at one hour intervals from sunrise to sunset for three successive days. They were represented by 33 species as given in the accompanying table. Abundant species in each collection sample were as follows: *D. auraria* with 55.43% in Ohnuma, *D. bifasciata* with 67.71% in Ikomabetsu and *D. auraria* with 72.99% in Yunosawa.

Table 1. Numerical data of drosophilid flies collected from three localities of Hokkaido and Aomori Prefecture.

Species	Yunosawa		Ohnuma		Ikomabetsu		Total
	♀/♂	(♀/♂)	♀/♂	(♀/♂)	♀/♂	(♀/♂)	
<i>Amiota</i>							
<i>variegata</i>	-		-		0/1		1
<i>Microdrosophila</i>							
<i>cristata</i>	-		1/0	(1/0)	-		1 ( 1)
<i>Scaptomyza</i>							
<i>pallida</i>	1/0	(1/0)	9/16	(9/16)	4/5	(2/4)	35 ( 32)
sp. like <i>graminum</i>	-		-		0/2	(0/2)	2 ( 2)
<i>concimilis</i>	-		-		6/6	(5/5)	12 ( 10)
<i>Polygonia</i>	-		0/3	(0/3)	5/8	(5/8)	16 ( 16)
<i>Okadai</i>	-		-		9/6	(9/6)	15 ( 15)
<i>Drosophila</i>							
<i>histrioides</i>	0/2		-		-		2
<i>coracina</i>	-		79/97		16/17		209
sp. of <i>fenestrarum</i> gp.	-		1/0	(1/0)	2/2	(1/1)	5 ( 3)
<i>bifasciata</i>	1/1		11/8		390/520		931
<i>helvetica</i>	-		-		4/0	(3/0)	4 ( 3)
<i>suzukii</i>	16/43	(0/2)	-		-		59 ( 2)
<i>lutea</i>	3/3		-		-		6
<i>nipponica</i>	1/0	(1/0)	-		1/0	(1/0)	2 ( 2)
sp. like <i>mommai</i>	-		-		7/6	(7/6)	13 ( 13)
<i>auraria</i> race A	875/596	(45/55)	68/119	(1/0)	-		1658 (101)
<i>auraria</i> race B	88/109	(0/6)	275/426	(0/2)	0/10		908 ( 8)
<i>auraria</i> race C	-		3/7		-		10
<i>brachynephros</i>	13/12	(1/0)	18/17		-		60 ( 1)
<i>unispina</i>	0/1		-		3/2		6
<i>nigromaculata</i>	9/5	(2/2)	43/37	(3/2)	54/17	(8/8)	165 ( 25)
<i>testacea</i>	1/0		68/30		203/18	(1/0)	320 ( 1)
<i>histrio</i>	5/5		6/5		2/3		26
<i>immigrans</i>	4/7	(3/1)	9/18		-		38 ( 4)
<i>pengi</i>	-		2/0		-		2
<i>virilis</i>	1/0		1/0		-		2
<i>ezoana</i>	-		1/0		2/3		6
<i>lacertosa</i>	194/264	(1/0)	36/45		-		539 ( 1)
<i>moriwakii</i>	0/3		4/3		-		10
<i>okadai</i>	3/2		2/0		5/5		17
<i>neokadai</i>	6/1		-		-		7
<i>sordidula</i>	5/4		79/60		-		148
<i>pseudosordidula</i>	-		6/7		-		13
sp. of <i>robusta</i> gp.	1/0		-		-		1
	1227/1058	(54/66)	722/898	(15/23)	714/630	(43/39)	
	2285	(120)	1620	(38)	1344	(82)	5249 (240)

The numerals in parentheses denote the number of specimens collected with the use of net sweeping.

Hochman, B. The University of Tennessee, Knoxville, Tennessee. EMS and ICR-100 induced chromosome 4 lethals in *D. mel.*

Two chemical mutagens, ethyl methanesulfonate (EMS) and quinacrine mustard (ICR-100), when injected separately into Oregon-R males in our laboratory, have each induced fourth chromosome

recessive lethal mutations at a frequency approximating 4%. This mutational response of the microchromosome is about four times that produced by X-ray doses of 3 or 4 Kr (Hochman, Gloor and Green, 1964).

The experiments utilizing ICR-100 were performed by Thomas B. McCune (1965, unpub. thesis). When an ICR-100 solution of 0.5% in saline was used, 10 lethal-containing fourth chromosomes were found out of 266 examined (3.75%). The same treatment induced sex-linked lethals at a 12% rate (112 per 924 X chromosomes tested).

Allelism tests of the ten chromosome 4 lethals show that they are situated at only four loci. There were four occurrences at two of the loci and a single mutation at each of the other two sites. The possibility of "clusters" was avoided by discarding the treated males after three days of mating with tester stock females. Moreover, eight injected males each "contributed" one lethal chromosome, while a ninth male was responsible for two of the ten lethals. Since we now know of the existence of more than 25 microchromosomal loci, these findings suggest that the mutational sites of chromosome 4 may vary widely in their sensitivity to ICR-100.

Experiments involving EMS were conducted on a much larger scale than those described above. Thirteen temporally distinct series of Oregon-R males were injected with a 0.05 M solution of EMS in saline. Of 1,640 fourth chromosomes analyzed, 65 (4%) were lethal in the homozygous state. As table I reveals, rather dissimilar mutation frequencies characterize some of the series.

Table I. Chromosome 4 lethals induced by 0.05 M EMS on saline.

Series	1	2	3	4	5	6	7	8	9	10	11	12	13	Total
No. chromosomes tested	248	121	57	82	40	25	120	50	305	83	98	187	224	1,640
Lethal chromosomes	5	3	3	2	0	1	6	0	14	7	5	10	9	65
% lethal	2.0	2.5	5.3	2.4	0	4.0	5.0	0	4.6	8.4	5.1	5.3	4.0	4.0

Although the data cannot be subjected to rigorous statistical treatment, we are confident that much of the variation seen above is due to chance. Nevertheless, mention can be made of other factors that might be responsible for some of the variability noted. Since EMS breaks down rapidly in aqueous surroundings, there may have been differences in the efficacy of the solutions injected at the various times. Secondly, no attempt was made to measure precisely the amount of fluid received by each treated male; injected flies were merely given a dose sufficient to cause a slight distention of the abdomen. Dr. J. L. Epler (pers. comm.) has observed this swelling effect following injections of about 0.5  $\mu$ l. Despite these methodological limitations, it is felt that the 4% mutation rate obtained, being based on 1,640 chromosomes tested, rests on solid ground.

The EMS lethals are currently being tested for allelism inter se and with previously obtained lethal and visible mutants. Results to date indicate that this chemical mutagen has induced genetic changes at some twenty chromosome 4 loci.

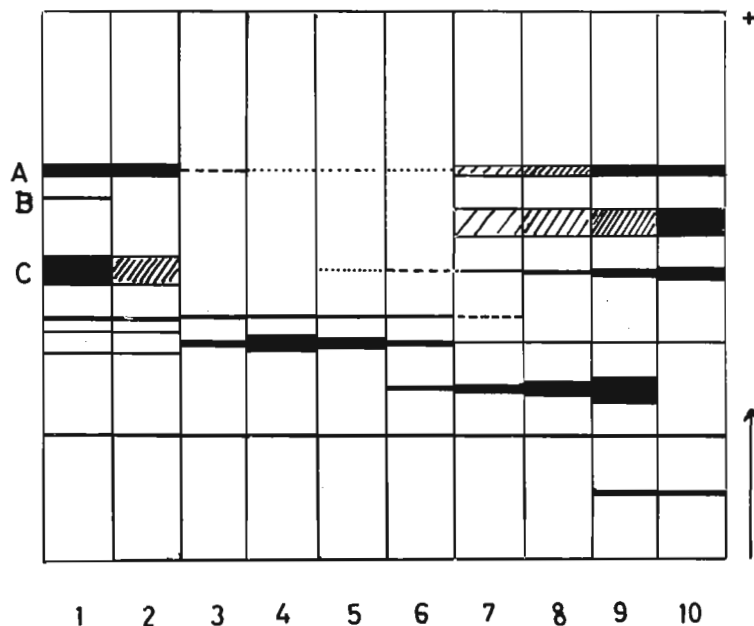
References: Hochman, B., H. Gloor and M. M. Green 1964, *Genetica* 35:109-126. McCune, T. B. 1965, M. S. Thesis, The University of Tennessee, Knoxville. (Research supported in part by NSF Research Grant GB 5144.)

Nilsson, L. R. University of Uppsala, Sweden. On the ontogeny of alkaline phosphatases in *D. m.*

phatase-patterns were developed with alpha-naphtylphosphate and Fast Garnet GBC salt in boric acid buffer at pH 7.8. Two larval stages ( $48 \pm 3$  hrs. and  $72 \pm 3$  hrs.), five pupal stages (0 - 3,  $12 \pm 3$ ,  $36 \pm 3$ ,  $60 \pm 3$  and  $84 \pm 3$ ) and imagoes of two ages (0 - 3 and  $24 \pm 3$  hrs.) were studied. Patterns specific to the stage of development were found in all stages studied. Among the strains the two genetic variations described by others were present. The inter-strain variation of alkaline phosphatase-bands was greater than in the other stages of development, AE showed four bands while AT had nine bands. All strains, except A-KAS and KAS which had identical patterns, varied with respect to time and disappearance of the strong band in the second larval stage. Differences of activities of certain pupal stages were also noticed between the strains.

Adult flies 0 - 3 hrs. old showed the unaltered pattern of the final pupal stage. In flies  $24 \pm 3$  hrs old a new pattern appears. These new alkaline phosphatases are of abdominal origin but not connected with the gonads. In the strain Be there is a difference in patterns of males and females. Females have a faint band B, absent in males. Females also have a higher activity of band C. In males this band is not so highly stained. The other strains showed the same change of the adult pattern, but had no "sex-difference" in their alkaline phosphatases.

Figure: The patterns of alkaline phosphatases of the strain Be. 1. Adult female 24 hrs. 2. Adult male 24 hrs. 3. Adults of both sexes 0 - 3 hrs. 4. Pupae 84 hrs. 5. Pupae 60 hrs. 6. Pupae 36 hrs. 7. Pupae 12 hrs. 8. Pupae 3 hrs. 9. Larvae 72 hrs. 10. Larvae 48 hrs.



Giesel, Betty Jean. University of Oregon, Eugene, Oregon. Ring loss in  $X^{c2} In(1)w^{vc}$ ,  $w^{vc} f$  at two temperatures.

Females from  $X^{c2} In(1)w^{vc}$ ,  $w^{vc} f/y w^{m4}/Y$  stocks which have been selected for several years for high production of gynandromorphs were crossed individually to  $y w^{m4}$  sib males. Most individuals

carried extra Y chromosomes. Flies were raised at room temperature and  $25^{\circ}C$ . Several generations (6 at  $25^{\circ}C$  and 5 at room temperature) of single female matings were made.

Gynandromorphs were scored for male tissue in two areas of the body, left front metatarsal segment (sex comb) and the more posterior tergites on the right side. The total numbers of gynandromorphs and apparently perfect females were also noted.

In the flies raised at  $25^{\circ}C$ , an average of 50.7% of the tissue of a gynandromorph is apparently male, and 47.1% in flies raised at room temperature. This suggests that  $X^{c2} In(1)w^{vc}$  is lost almost exclusively at the first cleavage division.

The number of gynandromorphs in both cases almost exactly equals the number of females. At  $25^{\circ}C$ , 49.9%, and at room temperature, 49.1% of the presumptive females in fact became gynandromorphs due to ring chromosome loss. Thus,  $X^{c2} In(1)w^{vc}$  loss may be due to some regular process.

Brosseau, George E., Jr. University of Iowa, Iowa City, Iowa. The effect of low temperature on the mitotic chromosomes of *Drosophila*.

One of the problems associated with the use of larval brain squash preparations for cytogenetical investigations is the usually low frequency of suitable mitotic figures. The observations reported in this note were made in the

course of experiments carried out to determine if the frequency of mitotic metaphases could be increased by subjecting 3rd instar larvae to low temperature treatments. Mated Oregon-R females were allowed to oviposit on standard cornmeal-agar medium for 3 days at 25°C. When 3rd instar larvae began to crawl free of the medium, the cultures were transferred to 6°C. Brain squash preparations made after 9, 15 and 24 hours of cold treatment. The larval brain was dissected in 1% Na citrate and transferred immediately to 2% orcein in lactic-acetic (50-50) acid solution. Fresh preparations were sealed and examined with phase contrast.

Low temperature was apparently successful in blocking mitosis. The frequency of metaphases after cold treatment was approximately twice (450 vs 247 metaphases per slide, 4 slides counted) that found in the 25°C controls. In addition there were no anaphase or prophase figures in the cold treated tissue. These results were considered to be only partially successful, however, because the cold treatment also causes a considerable degree of contraction of the metaphase chromosome (Fig. 1). Thus the chromosomes would generally be unsuitable for morphological analysis although they would be useful for counts. The longer periods of cold treatment did not appear to increase the frequency of mitotic metaphases when compared to the shorter treatments. After 24 hours at 6°C, most of the larvae died. Some C-mitotic like figures were found after 24 hours in the cold suggesting that polyploid cells could be produced by this treatment.

If the cultures are returned to 25°C after 9 or 15 hours at 6°C, the larvae will continue development. Therefore an attempt was made to find out if a high frequency of more suitable division figures could be found subsequent to the cold treatment. Preparations were made from larvae subjected to cold temperature for 9 hours and then transferred to 25°C for 3 hours. Casual observation of three preparations indicated that a higher frequency of favorable mitotic figures might be obtained in this fashion although more work would be necessary to verify this effect. It is possible that a more favorable sequence of treatment times and temperatures can be found.

A curious effect of the cold temperature on the centromere region of the X chromosome was found (figure 1). While the X is as highly contracted as the other chromosomes, a portion of the proximal heterochromatin is rather highly extended. Neither the autosomes nor the Y chromosome show this effect. It is not seen in 100% of the mitotic metaphases although a high proportion of them show it. This cold induced negative heteropycnosis of a portion of the X chromosome is reminiscent of the effect of cold treatment on portions of the chromosomes of *Trillium* (Darlington and La Cour, 1940). Darlington termed this effect "nucleic acid starvation" although these observations would now be interpreted in terms of effects on the degree of coiling or compaction of the chromosome. Apparently a portion of the X heterochromatin responds to cold treatment by becoming more extended (or failing to contract) while the remainder of the chromosomes is coiling tightly. (Work supported by grant RG-06508 from the USPHS). Reference: Darlington, C. D. and L. La Cour, 1940, Nucleic acid starvation of chromosomes in *Trillium*. *J. Genet.* 40:185-213.

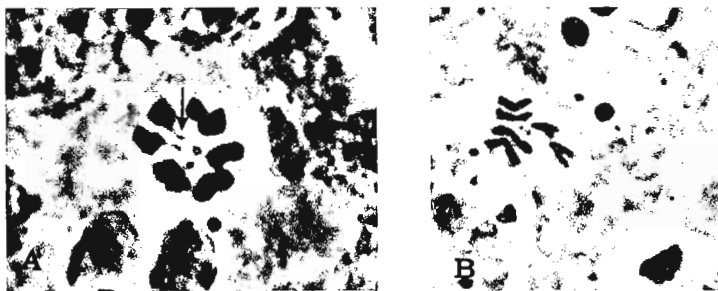


Figure 1. Photomicrograph of a mitotic metaphase from a cold treated (24 hours at 6°C) 3rd instar larval brain showing the highly compacted nature of the chromosomes and the extended portion near the centromeres of the X chromosomes (A). A normal metaphase is shown for comparison (B). A and B are not of comparable magnification.

Datta, Rajat Kumar. University of Calcutta India. Influence of  $y^+$  and  $sc$  alleles in suppression of yellow color in the sexcomb of *Drosophila melanogaster*.

The sexlinked mutant 'yellow' ( $y$ ) causes the production of yellow body, hairs and bristles in *D. melanogaster*, and results in a brownish-yellow hue in the sexcomb teeth which are black in normal strains. The  $y^+$  allele in  $y^+/y$  females or  $y^+Y/y$

males, and many  $sc$  alleles in  $y/y$  ( $sc/sc$ ) females or  $y/sc \cdot Y$  males suppress the yellow color of body and bristles. However, these alleles behave differently in the sexcombs. The behavior is variable and the variability in the color of the sexcomb teeth, seen in various combinations, can be classified in to three classes, (a) black, (b) intermediate color of brownish yellow and (c) yellow. The  $y/Y^S \cdot y^+ bb^+-7$  males as expected, had normal body color and black bristles. But pigmentation of the teeth in their sexcombs show three shades of color, most of which being of an intermediate (brownish yellow) hue (Table 1). The  $sc^{V1} \cdot Y^S/y$  and  $sc^{S1} \cdot Y^L \#2/y$  males on the other hand, show still different expression.  $sc^{V1}$  does not suppress the yellow ( $y$ ) and, therefore, the body color of  $y/sc^{V1} \cdot Y^S$  males are yellow. However, among the sexcomb teeth examined, 22% are dark brown, 78% yellow and none are black (Table 1). In addition, some of the yellow teeth also show occasional black pigments at the basal part, while the upper portion remains yellow. Finally, in the  $y/sc^{S1}$  males the yellow body color is completely suppressed but the sexcomb shows all the three classes of pigment distribution in considerable proportions.

Genotype	% of colored teeth			No. of teeth examined *
	Black	Intermediate	Yellow	
1. $y/Y^S \cdot y^+ bb^+-7$	11	83	6	276
2. $y/sc^{V1} \cdot Y^S$	0	22	78	274
3. $y/sc^{S1} \cdot Y^L \#2$	31	23	46	276

\* Total number of teeth in 30 males.

Van Delden, W. University of Groningen, Netherlands. Adaptation in *D. melanogaster* populations started from an inbred line.

Cage populations were started with flies derived from an inbred line kept by single-pair brother-sister mating for hundreds of generations. The original inbred line had a very low fitness

using such measures as productivity, egg hatchability, longevity, competitive ability, etc. Five cages were held at a constant temperature of 25°C and three cages were held at the same mean temperature but undergoing a regular oscillation of 10°C during each 24-hour period. Populations 1-5 were kept at 25°C and populations 6-8 under the fluctuating temperature. A control population (C) which originated from the same inbred line, but was started two years earlier, was also kept at 25°C. Productivity of the populations was measured by withdrawing at regular intervals foodcups with eggs from the cages and counting the numbers of emerging adults.

Only mutation, followed by recombination, can bring about improvement of fitness in the populations started with homozygous material. There were great differences in increase of productivity between the populations (Table 1).

Table 1. Regression of productivity on time (months) during the first 14 months.

Cages:	1. + 15.2	4. + 34.5	7. + 51.7
	2. - 5.3	5. + 36.4	8. + 45.3
	3. + 21.4	6. - 11.4	C. - 1.6

After an initial increase, all populations except the control suffered a temporary decline in productivity. This began at the 6th-8th month and continued for 2-3 months. Populations 2 and 6 nearly became extinct. However, after 14 months half of the populations equaled the control population in productivity. The heritability of sternopleural bristles in these populations equaled or surpassed the control value (0.30). There was good correlation between productivity and other fitness measures as population size, egg hatchability, larval survival to adulthood, adult longevity, and productivity in different new environments.

Danieli, G. A. and E. Rodinò. University of Padua, Italy. Biochemical estimate of DNA content in *D. hydei* salivary glands.

Practically all the investigations on nucleic acids content in salivary glands are carried out by means of cytochemical and histophotometric methods. Actually the biochemical approach to the problem

is quite infrequent in literature. The average content of DNA and RNA per cell was determined by Patterson and Dackerman (1952) in *D. melanogaster* salivary glands by chemical methods. The determinations were made on 18 pairs of glands by a microtechnique (Linderström-Lang and Holter apparatus) using a modification of the Schneider method.

We have decided to investigate the same problem using the improved Burton's DPA method. In recent years this method was applied successfully on various materials for evaluations of extreme sensitivity coupled with low reagent blank. In order to obtain a further increase in sensitivity, we followed the Giles and Myers (1965) suggestions. Preliminary experiments pointed out that the best results are obtained with a 2.5% DPA concentration. *Drosophila hydei* salivary glands have been studied at various larval stages from the late second to the late third instar.

From synchronized cultures, 100 larvae per point were dissected in Ephrussi-Ringer solution at 4°C temperature in order to reduce metabolic activity. The glands were collected and immediately frozen on dry ice, and then stored at -30°C until used.

Extractions and determinations were carried out at the same time on all the groups of glands.

After thawing, the glands were homogenized in 10% TCA and the material was subsequently treated according to the standard Schneider procedure with centrifugation at 4°C, 4,000 r.p.m. for 30 minutes in 5% TCA followed by treatment with ethanol and ether.

The nucleic acids were extracted from the dried residue with 1.6 ml. of 5% TCA at 90°C for 15 minutes, the material extracted was then centrifuged at 4,000 r.p.m. for 45 minutes.

1 ml. of supernatant was used for DNA determination. The colorimetric reaction was that of Burton's modified DPA method.

Some of our first results are reported in Table 1.

A comparison between the absolute values of DNA content per cell found by several authors in

*D. melanogaster* and in *D. hydei* is given in Table 2.

Table 1

age of larvae (hrs. after ovoposition)	average DNA content per gland expressed in $\mu\text{g.} \times 10^{-2}$
96	2.85
144	8.12
192	12.02

Table 2

Patterson and Dackerman 1952 (biochemical evaluation)	<i>D. melanogaster</i>	2.8
Kurnick and Herskowitz 1952 (histophotometric evaluation)	<i>D. melanogaster</i>	0.712
Plaut 1963 (quantitative autoradiography)	<i>D. melanogaster</i>	about 10
Berendes 1965 (histophotometric evaluation)	<i>D. hydei</i>	7.05 (distal nuclei)
Our results (biochemical evaluation)	<i>D. hydei</i>	9.2

The values are expressed in  $\mu\text{g.} \times 10^{-4}$

References: Berendes, H. D. 1965. *Chromosoma*, 17:35; Burton, K. 1956. *Biochem. J.*, 62:315; Giles, K. W. and Myers, A. 1965. *Nature*, 206:93; Kurnick, N. B. and Herskowitz, I. H. 1952. *J. Cell. Comp. Physiol.* 39:281; Patterson, E. K. and Dackerman, M. E. 1952. *Arch. Biochem. and Biophys.*, 36:97; Plaut, W. 1963. *J. Mol. Biol.* 7:632.

Lefevre, G. San Fernando Valley State College, Northridge, California. Lethal and visible mutation frequencies.

In experiments where the frequency of sex-linked recessive lethal mutations was determined following irradiation with 4000r of male germ cells at different stages of maturity, all F females were inspected for the presence of visible changes at the y, w, v, and f loci, as well as for Notch and white-mottled mutations. Further, the occurrence of hyperploid males was noted. Altogether, 187,381 F<sub>1</sub> males and 194,775 F<sub>1</sub> females were examined, and a total of 461 visible mutants and 224 hyperploid males were found. The fertility of both phenotypically normal and mutant F<sub>1</sub> females was compared, as was the frequency of mutants in the fertile and sterile classes of F<sub>1</sub> females.

In general, the frequency of both visible mutants and recessive lethals is high following the irradiation of mature sperm in the male, somewhat lower when mature sperm are irradiated in the female, much lower when immature sperm are irradiated, but high again following irradiation of early spermatids. At the same time, sterility is highest when mutation frequencies are highest, except in the case of mature sperm irradiated in the female. There, sterility is increased although mutation frequencies are lower.

The frequencies of different categories of mutation do not all follow the same pattern. Chromosomal mutants (hyperploid males and white-mottled) are most sensitive, sex-linked lethals (a mixture of chromosomally normal and abnormal mutants) next, and male-viable y, w, v, and f mutants least sensitive to the effect of irradiating different germ cell stages. Further, visible mutants are 8 times more frequent among sterile than among fertile F<sub>1</sub> females.

These results indicate that differential chromosome breakage or restitution of breaks, not differential gene mutation rates, is primarily responsible for the differences in lethal mutation frequency detected at successive intervals following irradiation; the frequency of cytologically normal mutants is relatively constant. Further, chromosome mutants contribute to both inviability and sterility of F<sub>1</sub> females. Thus, variations in recessive lethal mutation frequencies do not necessarily reflect true differences in the mutability of different germ cell stages exposed to given doses of X-rays.

Yoon, Jong Sik and R. H. Richardson. The University of Texas, Austin, Texas. A study of the relationship between ovariole number and egg production in *Drosophila pseudoobscura*.

The variation of ovariole number and egg production was examined among four laboratory strains. Average ovariole number per ovary was constant (20.14 with range of 19.66 to 20.36) among different strains, but there was great variation among ovaries. There was

also a significant difference of average ovariole numbers among flies. Estimates of components of variance of ovariole number indicated about half as large a component among flies as within flies. During six consecutive days (8 through 13 days after eclosion), the mean egg production per female per day differed among strains, with a range of  $24.22 \pm 2.71$  through  $35.60 \pm 4.18$ . There was no correlation between ovariole number and egg production among strains (correlation coefficient = 0.02), while there were positive correlations within strains (average within-strain correlation of 0.44 with a range of 0.10). Apparently the strains each have a characteristic rate of oogenesis per ovariole. This rate is relatively constant within the strain and variations of ovariole numbers of flies within a strain is reflected in a similar variation in their egg production. An examination of stages of oogenesis within the ovaries of each fly confirms the synchrony among ovarioles during resting and egg-laying stages observed by Wolfsberg (1959), and further, indicates synchrony during yolk deposition between these two stages. At the egg-laying stage there are both mature eggs being laid and pre-yolk eggs, but no stages in between. The characteristic rate of oogenesis for the various strains may reflect either a difference in the lag-time between expulsion of mature eggs and initiation of yolk deposition, or a difference in the rate of yolk deposition among strains. (Supported by PHS Grants GM 11609 and 2-T1-337.)

Pipkin, Sarah B. Howard University, Washington, D.C. Polymorphism of octonol dehydrogenase in neotropical *Drosophila* species.

Isozymes of octonol dehydrogenase have been studied in 30 neotropical *Drosophila* species using the method of Ursprung and Leone, 1965. When mass homogenates of 200 flies per species

were subjected to electrophoresis, presented with a substrate of hexanol, heptynol, or octonol, in a formazan staining mixture, 18 species showed a single isozyme. The monomorphic species included *D. neomorpha* and *D. carmargoi*, each represented by 5 strains of different geographic origin, and *D. briegeri* and an annulimana group species, each represented by two strains. The remaining 14 monomorphic species were so characterised from the study of only one geographic strain per species. In the 12 polymorphic species, using mass homogenates, three isozymes, representing slow, fast, and hybrid enzymes of ODH usually were shown. In one strain of two different species, four isozymes were identified using mass homogenates. ODH polymorphism was found in 5 among 5 strains tested of *D. metzii*; 3 of 3 strains of *D. pellewae*; 6 of 9 strains of *D. mediotriata*; 4 of 5 strains of *D. medionotata*; 1 of 3 strains of *D. capricorni*; and a single strain of *D. calloptera*, *D. sticta*, *D. crocina* and an undescribed member of the flavopilosa species group. The widespread polymorphism of ODH has been found in five different neotropical species groups. A detailed study of *D. metzii* and *D. pellewae* using single flies instead of mass homogenates has revealed as many as 7 different alleles of the ODH gene were responsible for isozyme patterns in certain strains.

Tedeschi, M. Valéria and Luiz Edmundo de Magalhães. Universidad de São Paulo, São Paulo, Brazil. Sex-limited effect of detrimental autosomic chromosome in *D. melanogaster*.

In an analysis of the viability of homozygotes for the second chromosomes of *D. melanogaster* from São Paulo City, Brazil, two chromosomes were found with sex-limited detrimental effect. One of these kills all the pre-adult males but the females show normal viability. The

other one has an opposite effect: it is semi-lethal for the females. In three cultures we counted 79♂ to 56♀;  $x^2 = 3.9$ ;  $P < 0.05$ . (Work supported by FAPESP; Contract 66/066.)

Hunter, Alice S. Centro Experimental de Estudios Superiores, Barquisimeto, Venezuela. Preference in mating time of *Drosophila mesophragmatica*.

Courtship behaviour and discrimination among five species of the mesophragmatica group of *Drosophila* was studied by Koref-Santibañez (1963) who reported that *D. mesophragmatica* is relatively inactive in courting. She also noted that copulation

of this species never took place while they were under observation.

In our laboratory preliminary studies of isolated pairs of virgin *D. mesophragmatica* have been made and it has been observed that they are sexually inactive during the first few days after hatching. A study of virgin pairs of two weeks of age has given the following data:

<u>Copulations of Bogotá line 22 <i>D. mesophragmatica</i></u>		
	% copulation	number of pairs tested
6 AM	33	64
10 AM	18	130
3 PM	9	80
7 PM	6	20

These data lead us to believe that there is a preferred time of day for copulation in this species. Additional experiments are being carried out in order to test this hypothesis.

Burla, H. University of Oregon, Eugene, Oregon. Induction of mutations in homokaryotypic *D. subobscura*.

In an attempt to obtain marker genes for the U chromosome in *D. subobscura*, flies of separate sexes from 2 homokaryotypic stocks, one containing  $U_{St}$ , the other  $U_{1+2}$ , were submitted to EMS treatment. For each

series anew, the substance was dissolved in 5% sucrose and offered to flies after they had been starved during the first 24 hours of their imaginal life. Upon feeding on EMS, they were kept on normal food for 48 hours to complete maturation, and then mated individually to homokaryotypic partners, which were either treated themselves, or left untreated. A sample of 35 males, 20 of which were kept for 24 hours on EMS, the others for 48 hours, left no offspring. A dosage of .03 molar, offered to several later series for 30 hours, did not reduce fertility much below normal. Judging from a lack of daughters and grandsons, EMS was effective in inducing sex linked dominant and recessive lethals, and was consequently trusted to induce visible mutations as well. In previous experiments, DES had failed in this respect, and EMS was used thereafter, following a suggestion by Dr. E. B. Lewis.

By repeated pair matings between full sibs, treated chromosomes were expected to become homozygous in the 3rd filial generation at reasonable frequencies. A total of 620 F3 sibships were scanned for mutants. Actually a variety of mutant phenotypes showed up, 46 of which could be maintained in stocks and transferred to Zurich, Switzerland. They will be included in the stock list as soon as the autosomal mutants are assigned to their respective linkage group, and as the possible identity, or allelomorphism, of the new mutants with those obtained by former workers are tested. To mention some of the mutants gained, there are phenotypes suggesting identity with bubble, Beadex, cut, Net, rotund, tuck, thin, shaven, vermilion, white, antennapedia, aristapedia, dachs and many-jointed. Probably some mutants were present in the stocks before treatment, especially abnormalities like scute, broken-bristle, thin short vein, and spot.

Okada, T. Tokyo Metropolitan University, Japan. Character continuities in the caenogenetic organs of *Drosophilidae*.

Although the caenogenetic organs do not indicate the ancestral adult forms, they frequently undergo ontogenetic development parallel to the sequences of the adult phylogeny. This kind of onto-

genetic development, which was named 'general caenogenesis' by the present author, can be distinguished from 'special caenogenesis' which is a caenogenetic development of an organ adaptive to the specific environment, not reflecting at all the adult phylogeny.

The anterior spiracles of the larvae and puparia of *Drosophilidae* are likely to follow a general caenogenetic trend in the shape, size and the number and arrangement of the branches. The posterior spiracles tend, on the contrary, to show a special caenogenetic change, their size and shape being adaptive to the specific habitats in which the larvae live and breathe. At the stage of puparium formation, the anterior spiracles are connected to the pupal mesothoracic spiracles by means of a temporarily produced tracheal system (Bodenstein, 1950), while the posterior spiracles lose respiratory function after that time.

The character continuities from the larvae to the adults seem to be higher in degree in the organs of general caenogenesis such as anterior spiracles than in that of special caenogenetic organs such as posterior spiracles.

The caenogenetic sequences and the character continuities of the larval mouth hooks are intermediate in degrees. This organ is general-caenogenetic in the earlier larval stages and specific-caenogenetic in the later larval stages, and it has indirect continuities from the larval to the adult stages, the homology between this organ and the adult mouth parts being recognizable.

Although the egg filaments of *Drosophilidae* are known to have systematically important morphological features reflecting the adult phylogeny to a great extent, they are developmentally not continuous to the larvae and adults. They continue, however, to the adults of the parent generation. For such an organ the term 'pseudocaenogenesis' is proposed here. Finally, the true palaeogenetic organs, such as the Malpighian tubules, which do not undergo metamorphic changes, are directly and completely continuous from the larvae to the adults.

Wasserman, Marvin. Queens College, New York. Collections of *Drosophila* from Central Mexico.

During the summers of 1963 and 1964, *Drosophila* populations were sampled from eleven arid or semi-arid localities in Central Mexico. We were interested in collecting repleta group species, and

therefore cactus areas were heavily baited with bananas, oranges, pineapples, and other tropical fruits; little or no sweeping was attempted. Table 1 gives the altitude and major geographical region for each locality. Collections were limited to the higher altitudes (1213 to 2446 meters). In Table 2 the relative frequencies of the various species at each locality is indicated. The region is obviously rich in *Drosophila* species: forty-two species plus unidentified members of the caridini and willistoni group were found. The identification of twenty repleta group species (in Table 2, hydei through nigrohydei) was later confirmed by a cytological analysis of their offspring. (Supported by research grant GB 709 from NSF.)

Table 1. Collection localities

	Locality	Altitude	Geographical Region
A	Cuernavaca, Morelos	1225 m	Sierra Volcanica Transversal
B	Acatlan, Puebla	1213 m	Balsas Depression
C	Tehuacan, Puebla	1676 m	Oaxacan Highlands
D	Pachuca, Hidalgo	2446 m	Sierra Volcanica Transversal
E	Zimapan, Hidalgo	1813 m	Central Plateau
F	Matehuala, San Luis Potosi	1615 m	Central Plateau
G	Tequila, Jalisco	1218 m	Central Plateau
H	Guadalajara, Jalisco	1552 m	Central Plateau
I	Patzcuaro, Michuacan	2114 m	Sierra Volcanica Transversal
J	San Miguel de Allende, Guanajuato	1870 m	Central Plateau
K	San Juan del Rio, Guereataro	1978 m	Central Plateau

Table 2. *Drosophila* collections from Central Mexico

	Loc. A	Loc. B	Loc. C	Loc. D	Loc. E	Loc. F	Loc. G	Loc. H	Loc. I	Loc. J	Loc. K
melanogaster & simulans	7983	3984	334	8	44	57	287	601	266	203	551
cardini group	47	64	2	-	1	1	1	15	18	-	5
pseudoobscura	21	47	134	135	28	46	-	58	275	48	50
nebulosa	46	58	27	-	-	1	3	3	1	1	-
willistoni group	120	-	22	-	-	-	14	4	4	4	1
hydei	24*	12*	75*	2	11*	14	1	15*	10	50*	24
nigricurria	5	74*	7*	-	1	-	-	8*	5*	-	3*
hexastigma	7	40*	-	-	92*	14	-	-	-	1*	1
aldrichi	24*	12*	-	-	-	-	-	-	-	-	-
linearepleta	3	8	1	-	-	-	-	-	-	-	-
anceps	33*	40*	-	-	-	-	-	-	-	-	-
eremophila	-	120*	28	-	-	-	-	-	-	-	-
racemova	-	20	18*	32*	35	-	2	-	23	-	4
m. rioensis	-	2*	-	-	-	-	-	14*	-	1	-
pegasa	-	15*	4*	-	-	14*	-	-	-	-	-
paranaensis	-	3	5*	-	-	-	2*	-	2	-	-
leonis	-	1	-	24	4*	-	1	96*	52*	10*	20*
bifurca	-	2	163*	-	53	8	-	-	15	4	25
tira	-	-	99*	-	7*	-	1*	9*	52*	-	-
ritae	-	-	-	-	6*	6	1*	-	32*	1	-

Continued on next page.

Table 2 continued:

	Loc. A	Loc. B	Loc. C	Loc. D	Loc. E	Loc. F	Loc. G	Loc. H	Loc. I	Loc. J	Loc. K
fulvilineata	-	-	1	-	-	-	-	-	2*	-	-
pachuca	-	-	-	100*	-	12*	-	-	-	-	-
propachuca	-	-	5*	10*	-	-	-	-	-	-	-
longicornis	-	-	20*	-	15	-	80	86*	-	1189*	1639*
hamatofila	-	-	-	-	5	638*	18	-	-	50*	76*
nigrohydei	-	-	10*	34*	1	-	-	-	32	-	-
carbonaria	-	-	1	-	-	2	-	-	-	-	-
alafumosa	-	-	1	-	-	-	-	-	5	-	-
capricorni	-	-	-	-	-	-	2	2	-	-	-
latifasciaformis	4	-	8	-	-	7	-	-	-	1	-
uninubes	1	1	1	-	-	-	-	-	1	-	-
gibberosa	1	1	-	-	-	-	-	2	-	-	-
trapeza	2	-	-	-	-	-	-	1	1	-	-
flexa	8	2	-	-	1	-	-	-	-	-	-
Gitona americana	9	1	1	-	9	12	-	2	-	4	-

\* identification confirmed cytologically

Other species collected: Loc. A: crocina (2), floricola (1), bromeliae (3); Loc. B: floricola (1), biopaca (1); Loc. C: submacroptera (8), setapex (1), parabocanaensis-like (2); Loc. G: ananassae (3); Loc. H: alagitans (1); Loc. I: macroptera (1).

Shamay, E. Hebrew University, Jerusalem, Israel. Induced spermatogonial crossing over in the centromere regions of chromosomes II and III of *D. melanogaster*.

One day old males of *D. melanogaster* heterozygous for a second chromosome marked with J, cn and bw and for a third chromosome marked with ri and e were irradiated with an X-ray dose of 2500 r. Subsequently, they were mated daily to a

fresh supply of females homozygous for the four recessive markers, utilizing two females per male. Starting from day VIII after irradiation the offspring were scored for induced recombinants.

The standard map distances between the markers employed are 16.5 for J-cn, 47.0 for cn-bw and 23.6 for ri-e. The regions J-cn and ri-e span the centromeres of chromosomes II and III, respectively.

The results of two experiments presented in the Table, confirm those of previous workers who found an excess of induced, as compared with spontaneous, crossing over in the centromere regions. Moreover, the present results indicate that the centromere region of chromosome II is more susceptible to induced crossing over than that of chromosome III. On each day in both experiments the rate of recombinants in the region J-cn exceeded that in the region ri-e with the exception of day XV in experiment 2. This difference may be ascribed to the larger amount of heterochromatin present in the centromere region of chromosome II.

Day after Irradiation	Total no. of Offspring	Experiment 1			Total no. of Offspring	Experiment 2		
		Percentage of recombinants				Percentage of recombinants		
		J-cn	cn-bw	ri-e		J-cn	cn-bw	ri-e
VIII	1009	2.18	0.99	0.69	93	1.1	1.1	-
IX	1451	2.69	0.28	0.69	1777	2.26	0.51	1.19
X	1389	2.16	-	0.43	1740	2.36	0.46	0.92
XI	1967	0.61	-	0.36	1942	0.41	0.77	0.10
XII	1312	0.84	-	0.15	2140	0.05	0.56	-
XIII	978	0.61	-	0.30	2364	0.30	0.17	0.25
XIV	1088	3.30	-	1.19	3290	0.49	-	-
XV	1330	1.50	0.38	0.38	3063	0.20	-	0.42

Arking, Robert and Ralph Hillman. Temple University, Philadelphia, Pennsylvania. Analysis of the mode of action of the eyeless-Dominant allele.

Of the eight known loci on the fourth chromosome which produce an eyeless phenotype, only the  $ey^D$  locus is neither allelic nor hypomorphic when combined with any of the others (Hinton, Amer. Nat. 76:219, 1942). Its major phenotypic

effect is the gross reduction in the size of the eye (Patterson and Muller, Genetics 15:495, 1930) with secondary malformations of the antennae and head capsule.

Reciprocal pair matings have been performed utilizing the balanced lethal,  $sv^{de}/ey^D$ , and an inbred Oregon-R strain which has been maintained in this laboratory for thirty-five generations by brother - sister matings. The results of these crosses (Table 1) show the existence of three major classes of  $F_1$  progeny: I. Those flies which possess a normal eye; II. Those flies which exhibit a severe decrease in the number of facets/eye; III. Those flies that never eclose and upon dissection can be seen to be either completely headless or else possess a very malformed and assymetrical head capsule.

Table 1. The number of facets/eye in the different classes of  $F_1$  progeny

Class	$sv^{de}/ey^D \times +/+$			$+/+ \times sv^{de}/ey^D$		
	Range	Median	Mean $\pm$ S.E.	Range	Median	Mean $\pm$ S.E.
I	692 - 806	764	755 $\pm$ 12.4	722 - 884	801.5	806.7 $\pm$ 14.3
	668 - 779	721	718 $\pm$ 8.9	736 - 791	749.5	756.8 $\pm$ 5.3
II	0 - 364	0.0	35.6 $\pm$ 20.5	0 - 232	0.0	22.8 $\pm$ 6.1
	0 - 301	0.0	46.5 $\pm$ 9.5	0 - 81	0.0	23.9 $\pm$ 30.3
III	Lethal - Fails to Eclose			Lethal - Fails to Eclose		

In class II there is a highly non-parametric distribution of facets/eye. The Wilcoxon-White test shows that there is no significant difference in the frequency distribution of facets/eye between (a) the reciprocal crosses, or (b) males and females of the same mating. Although the scored flies had either two normal eyes or two abnormal eyes, those flies that were assymetrically abnormal exhibited no preference as to whether the most severely damaged eye would be on the right or on the left side of the head capsule. Similar genetic and developmental results are found when  $sv^{de}/ey^D$  flies are crossed to Canton-SA wild types and when  $+/ey^D$  flies are crossed to flies from either Oregon-R or Canton-SA inbred stocks.

Most of the work thus far has involved the third, or lethal, class. Histological examination of the abnormally developing pupae has revealed the following syndrome: 1. normal differentiation and development of the imaginal legs, wings, hypoderm, etc. 2. the retention of the larval salivary glands, some in an apparently healthy condition, for periods of time ranging up to 156 hours post-pupation; 3. a partial to complete development of the imaginal salivary glands which occurred even in those organisms retaining the larval salivary glands; 4. a failure of the gonads to develop (cessation of development usually occurred at approximately the 0 - 4 hour pupal stage and by the 27 hour pupal stage at the latest.) 5. an abnormal eversion of the head capsule and its associated structures.

Similar histological evidence for the lethality of  $ey^D$  was obtained from the  $F_1 \times F_1$  cross of  $ey^D/+ \times ey^D/+$ . Developmental data from this cross can be seen in Table 2. Although the histological picture in non-eclosed pupae is similar to that described above, the phenocritical phase actually begins in the late third larval instar. The non-pupating larvae do not undergo further larval molts, but remain in the third larval instar for periods of time ranging up to 120 hours. At this time they either die or undergo an abortive pupation. The effective lethal phase in the  $ey^D$  mutation therefore occurs in the beginning and early stages of metamorphosis.

Table 2. Developmental data of  $ey^D/+ \times ey^D/+$  cross.

Larvae Hatched	Non-pupating Larvae	Pupae	Non-eclosed Pupae	Total Adults	$+/+$	$ey^D/+$	Lethal
89	15	74	8	66	25	41	23

continued

The above syndrome suggests that the ecdyson/juvenile hormone balance in the  $ey^D$  flies has been upset. Whether the primary lesion affects the ecdysial glands, the corpora allata, the corpora cardiaca or other neuro-secretory elements is not known at this time. Neuro-secretory staining and implantation experiments are presently being performed in order to further study the mode of action of the  $ey^D$  locus. (The research reported above was supported by a NSF Predoctoral Cooperative Fellowship and in part by grant GM 10480 from the USPHS.)

Gethmann, Richard C. Oregon State University, Corvallis, Oregon. A reduced viability effect of a ring duplication,  $Dp(1;f)65X^{c2}$ .

In crossover tests of a reversed acrocentric, a reduced viability of one of the duplications used was observed. The reversed acrocentric used in the experiments was deficient for a large block of interstitial heterochromatin, and is

lethal in the absence of the heterochromatin. The missing heterochromatin was supplied by different duplications.

Since many of the exchanges within a reversed acrocentric result in lethal bridges, a reduction in the number of recovered female progeny would be expected. However, in parallel experiments, quite different sex ratios were recovered. An examination of the genotypes of the progeny from these crosses show that there is a marked reduction in the number of recovered males when the male zygotes receive  $Dp(1;f)65X^{c2}$  (of crosses 1, 2, 3, and 4, also see Report of R. C. Gethmann, this DIS, for description of  $Dp65X^{c2}$ ).

If the reversed acrocentric is heterozygous for  $In(1)dl49$ , one would expect a reduction in exchanges, and hence, a higher sex ratio. This was the case, however, again the apparent lethal effect of the ring duplication was found (of crosses 5, 6, 7, and 8). Since lethal exchange classes are absent in a reversed metacentric, one would expect a 1:1 sex ratio from crossover tests with this type of a compound X chromosome. However, as can be seen from cross 9, there was a reduction in the number of recovered males. Again, these male zygotes received  $Dp65X^{c2}$ .

Finally, the duplication can be induced to segregate randomly if a Y chromosome is present as a pairing partner for the reversed acrocentric. An examination of the regular and exceptional progeny from this cross (cross 10), shows that in the regular progeny, there is a reduction of males (cross 10a), which is comparable to that found in crosses 1 or 2. However, in the exceptional progeny (cross 10b), the duplication is included in the female zygotes, rather than the males, and here, the female class is the one which is greatly reduced.

In conclusion,  $Dp(1;f)65X^{c2}$  is lethal in a fraction of the zygotes, it appears that approximately 30% of the zygotes receiving the duplication do not survive to adulthood. (This work was supported by NSF grant GB-1864 to J. D. Mohler.)

Cross number	genotype of female progeny	number	genotype of male progeny	number	female to male ratio
1	RA/Dp60	471	$\overline{XY}/Dp65X^{c2}$	638	0.74
2	RA/Y, $su^+-f$	549	X/Dp65X <sup>c2</sup>	765	0.72
3	RA/Y, $su^+-f$	2862	X/Dp60	5921	0.49
4	RA/Y, $su^+-f$	2870	X/Y, $su^+-f$	5274	0.54
5	RA,dl49/Dp60	831	$\overline{XY}/Dp65X^{c2}$	790	1.05
6	RA,dl49/Y, $su^+-f$	1704	X/Dp65X <sup>c2</sup>	1733	1.00
7	RA,dl49/Y, $su^+-f$	977	X/Dp60	1246	0.78
8	RA,dl49/Y, $su^+-f$	1680	X/Y, $su^+-f$	2058	0.82
9	RM/Dp60	439	$\overline{XY}/Dp65X^{c2}$	333	1.32
10a	RA/Dp60	341	$\overline{XY}/Dp65X^{c2}$	469	0.73
10b	RA/Dp60/Dp65X <sup>c2</sup>	317	$\overline{XY}$	719	0.44

DeMarinis, F. Cleveland State University, Cleveland, Ohio. A comparison of the effects of 5-bromouracil and uracil on the facet number of the Bar eye.

It has been shown that a large number of amides can increase the number of facets in the Bar eye *Drosophila* (DIS 41:149). In the present study it has been found that 5-bromouracil has an opposite effect. In brief, each compound tested

was added to Pearl's standard formula in proportionate amounts and Bar stock eggs were deposited and permitted to complete development on it. The effect on the eye-size of males and females was determined by counting the number of facets. All tests were carried out at 25°C.

Table 1 compares the effect of uracil and 5-bromouracil on the size of the Bar eye of the males and females. Uracil increases the number of facets while its homologue, 5-bromouracil, markedly reduces the number of facets. These two compounds apparently have an antagonistic effect on the rate of facet formation.

Table 2 shows the effect of various concentrations of 5-bromouracil on facet number. It is apparent from these data that the number of facets decreases with an increased concentration of the compound. It shows a maximum effect at approximately 1% concentration level. Table 3 shows the effect of various concentrations of uracil on eye-size ♂♂ only. In this case the number of facets increases with an increase in concentration of uracil. The results listed in this table were taken from an earlier test where a lower average Bar stock was used. More detailed studies have indicated that, in general, uracil as well as other amides modifies Bar to a typical wild type eye, while 5-bromouracil modifies Bar to a typical double Bar type.

Table 4 compares the effect of other homologues of uracil. Only thymine seems to have a moderate effect in increasing the size of the eye; other homologues are either highly toxic or have no apparent effect on the eye.

It should be pointed out that while the main study of 5-bromouracil has been on its effect on the eye, many other effects were also noted. Among these the wings were noted to be markedly affected. They were smaller, stretched out and cupped under towards the ventral part of the thorax. The flies are unable to fly but creep very much like the vestigial type.

Table 5 shows the effect of various concentrations of 5-bromouracil on the proportion of abnormal-winged flies.

In general, higher concentrations of 5-bromouracil tend to produce greater proportions of abnormal-winged flies, but at the same time tend to reduce the total number of adults that emerge. Each experimental batch of media tested was initially seeded with approximately 400 eggs.

Table 1

A comparison of 0.75% uracil and 0.75% 5-bromouracil on the development of the Bar eye in ♂♂ and ♀♀ at 25°C.

	Bar ♂♂ Facet number	Bar ♀♀ Facet number
Uracil (0.75%)	265 ± 19.2	211 ± 20.0
5-bromouracil (0.75%)	47.7 ± 3.6	29.1 ± 2.7
Control	106.4 ± 4.4	75.9 ± 3.7

Table 2

The effect of 5-bromouracil on the size of the Bar eye ♂♂ and ♀♀ at 25°C.

% 5-bromouracil	Bar ♂♂ Facet number	Bar ♀♀ Facet number
0.10	68.5 ± 5.3	51.7 ± 4.5
0.25	60.6 ± 3.5	36.6 ± 3.2
0.50	-----	30.6 ± 2.9
0.75	47.7 ± 3.6	29.1 ± 2.7
1.00	-----	26.6 ± 1.6
1.25	-----	31.4 ± 3.0
1.50	-----	35.4 ± 2.4
1.75	-----	42.2 ± 3.7
2.00	-----	47.4 ± 3.3
2.25	-----	52.7 ± 4.4
Control	106.4 ± 4.4	75.9 ± 3.7

Table 3

The effect of uracil on the size of the Bar eye ♂♂ at 25°C.

% Uracil	Bar ♂♂ Facet Number
0.75	244 ± 18.1
1.50	310 ± 15.0
1.75	450 ± 13.8
2.00	575 ± 17.9
Control	88 ± 3.9

Table 4

The effects of other uracil homologues on the Bar eye ♂♂ at 25°C.

	Bar ♂♂ Facet Number
5-methyl uracil (Thymine)(0.25%)	155 ± 10.1
5-nitrouracil (2.25%)	no effect
5-aminouracil (0.10%)	highly toxic
6-methyl uracil (0.50%)	no effect
6-aminouracil (1.00%)	no effect

Table 5

The effects of 5-bromouracil on wings of fB stock at 25°C.

% 5-bromouracil	Total Number of Adults Emerged	Number With Abnormal Wings	% Abnormal Wings
0.10	235	16	6.8
0.25	141	87	61.7
0.50	16	16	100
0.75	63	62	100
1.00	19	19	↓
1.25	35	35	
1.50	16	16	
1.75	28	28	
2.00	13	13	
2.25	15	15	
Control	350	0	0

Rasmuson, B. University of Uppsala, Sweden. Modulation of the puff in the tip of the X-chromosome in D. m.

In a duplication of white-locus, containing the mutant ch in the distal duplicate and sp-w in the proximal one (Rasmuson: Hereditas 53), a series of salivary chromosome analyses was performed in order to

determine the length of the duplication. The original ch- and sp-w stocks were analysed together with the duplication, which had occurred independently in two females, heterozygous for these marker genes. It was found that the duplication had arisen as a cross-over after unequal pairing in such a way that a segment from 3 A to 4 C was duplicated. This means that the bands 3 A 1234, including the zeste band 3 A3, which is known to pair regularly - but very seldom - with the 3 C1 band within or very close to the white-locus, also have an affinity to pair with some structure between 4 C and 4 D. The band 4 D does not seem to be duplicated. Further, the size and the shape of the puff near the tip of the X-chromosome, including the sections 2 A and 2 B, is modulated from the normal appearance found in the two original strains. In males, the puff in the duplicated chromosome is elongated in proximal direction to an ellipsoidal shape and appears to include the 3 A12 bands within its border. In a recombinant where the duplication had been eliminated, the normal shape of the puff reappeared. Thus, the duplication seems to contain some factor which induces the puffing in a region outside of the duplication what under normal conditions does not show any puffing activity. Studies are under way to investigate whether this factor is a special gene or a heterochromatic region and whether it is possible to obtain the same phenomenon with other chromosome rearrangements.

Bowman, J. T. Utah State University, Logan, Utah. Another "super-reversion" at the white locus of *D. melanogaster*.

Among the revertant progeny of homozygous attached-X females of the constitution  $w^1/w^1$  was a single female which on progeny testing proved to be of the genotype  $w^+/"w"$ . Attempts to map this

new white allele ( $w^{62k}$ ) have been largely unsuccessful since it shows the same type of recombinational abnormality as the ivory mutant from which it was derived (Lewis 1959; Bowman 1965). Only one exception was recovered from 67,000 offspring of heterozygous  $y w^{62k}/y^2 sc w^{bf} w^{ch} spl$  females; a single young  $y^2 sc w^+ spl$  male. Since no equivalent stock is maintained in the laboratory, it is extremely unlikely that this exception was a contaminant.

This event resembles, at least superficially, Lewis' recovery of a  $y^2 fa$  male from females of the constitution  $w^1/y^2 w^1 w^{ch} fa$  (Rasmuson 1962). Of particular interest is the fact that in both cases the chromosomes that did not contribute the outside markers recovered with the exceptions carried related, recombinationally aberrant mutants. A significant difference is that  $w^{62k}$  has not been observed to revert in homozygotes while  $w^1$  reverts frequently (Lewis 1959, Bowman 1965). Rasmuson (1962) has offered a recombinational explanation for the Lewis "reversion". An additional hypothesis based on a single event would be unwarranted. References: Bowman, J. T., 1965. Spontaneous reversion of the white-ivory mutant of *D. melanogaster*. *Genetics* 52:1069-1079. Lewis, E. B., 1959. Germinal and somatic reversion of the ivory mutant in *D. melanogaster*. *Genetics* 44:522. Rasmuson, B., 1962. Evidence for a compound nature of the mutant alleles  $w^a$  and  $w^1$  in *D. melanogaster*. *Hereditas* 48:612-618. (Supported by NSF Grant No. GB-4539.)

Paterson, H. E. and L. Tsakas. University College of Rhodesia, Salisbury. The identification of *Drosophila seguyi* Smart.

Paterson (DIS 34) drew attention to the fact that *Drosophila seguyi* is a member of a complex of sibling species. It was not then possible to decide which species represented the true *D. seguyi*: so, pro-

visionally, the form figured by Burla (1954a) (Rev. Suisse Zool. 61, Fasc. suppl., p. 158) was designated "Species A" and the form figured by Burla (1954b, Rev. Brasil Biol. 14(1) p.45 figs. 3 and 6) as "Species B".

This contribution is to report that the type of *D. seguyi* Smart has now been examined by one of us and found to be identical with males of species B as defined above. The examination included the genital arch and the sexcombs, and on both characters agreement with males of species B was complete. The form of the metatarsal sexcomb differs between species A and *D. seguyi* in that in the former species all the teeth are in line whereas in the latter species the last two teeth are displaced from the line on which the others lie. This character was brought to our attention by Professor Burla.

It is to be hoped that laboratories holding stocks of "*D. seguyi*" will now examine them and decide on their identity.

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

In August 1961, one ether-resistant female fly appeared from Mino-H stock in our laboratory. After 13 generations of selection with ether, this stock was ready for experiments. The phenotype of

this ether-resistant strain showed yellow finally, so we named it  $y^{ER}$  (yellow from ether resistant). Adult flies of 24 hr. after emergence were treated with a definite quantity of ether for definite minutes, then the dosage-mortality curves after 24 hr. of etherization were constructed, and the median lethal time (in minutes) was estimated.

The  $LT_{50}$  of  $y^{ER}$  was 4.5 min. and 4.3 min. in females and males respectively; on the other hand, ether-sensitive stocks of Quick Sand and  $bw;st;sv^n$  showed the  $LT_{50}$  of 2.6 min. in females and 2.0 min. in males. Genetical analysis revealed that the ether-resistance is completely dominant and the major gene(s) is located on the right end of the 3rd chromosome. Furthermore, minor genes were found on the X-chromosome (otherwise Y-chromosome) and on the 4th chromosome. Thus the ether-resistance in *Drosophila* was recognized as polygenic.

Barigozzi, C. and Sari Gorla M. University of Milan, Italy. Mitotic recombination in *Drosophila melanogaster*.

Freckled (Frd; 2; 102) in male sex, when heterozygous in cis or in trans with several chromosomal abnormalities of the 2nd chromosome (Cy, Gl<sub>a</sub>, M(2)33a), gives rise erratically to unexpected segregation

tion which sometimes comprise recombinants. As example, the following cases are here presented: 1) 8 pairs  $\sigma$   $\frac{\text{Frd M}(2)33a}{+}$  x  $\phi$   $\frac{+}{+}$  produced in 7 cases normal offspring (Frd M(2)33a and + + in equal proportions) and in 1 case 106 + + and 60 Frd (one parental and one recombinant class lack, and the other recombinant is unexpectedly frequent). If one assumes that mitotic recombination has occurred very early in the embryo, the results quoted above can be easily understood: mitotic recombination followed by sorting out of the two chromatids + + and Frd + (according to Stern mechanism) can have produced a gonadic tissue consisting only of two classes of cells (higher advantage of + + gametes over the Frd + ones can explain the significant difference between 106 and 60).

2)  $\sigma$   $\frac{\text{Cy Frd}}{+}$  x  $\phi$   $\frac{+}{+}$  (1 pair) produced 3 Cy Frd, 1 Cy +, 55 + Frd and 54 + +. These results can be interpreted as the gonadic tissue being derived from two cells. Most of them may have derived from a cell characterized by one parental chromosome and by one of the two recombinants. A minority, however, must come from one cell containing the other two chromosomes (Cy Frd and Cy +); there Cy is present in double dose, so the genotype is nearly lethal: its rarity is thus explained.

These types of segregation never occurred when Frd is heterozygous with Cy L, which is well known for its ability to eliminate recombinants.

A somewhat different effect gives heterozygosity with Pm. Crossing-over in male meiosis seems here to occur homogeneously in all cultures at low rate, in the chromosome section located between the right break of Pm and Frd.

During the feminine meiosis the phenomenon occurs, but more weakly. In the absence of structural abnormalities, Frd behaves normally, and no recombinations occur in males. In the presence of inversions in the 1st chromosome (M-5) or in the 3rd (Sb M<sub>e</sub>/H) no mitotic recombination has been found.

We may conclude that Frd has the peculiar property of inducing mitotic recombination in the early development. The presence of Frd male sex and some structural abnormalities, which can be responsible for disturbance in chromosome contact, seem to be critical in causing mitotic recombination.

The reason why Frd is capable of such activity in those conditions remains unexplained.

Di Pasquale, A. and L. Baratelli Zambruni. University of Milan, Italy. Histological study of bsp phenotype.

"brown spots" (bsp) is characterized by superficial pigmented areas restricted to pleurae (DIS 37:40). These brown areas are variable in size and can be reduced to dot-like spots (dottings);

without exception, they appear only in the female after copulation. Following artificial stimulation (introduction into the vagina of a thin glass needle) "brown masses" appear, consisting of small pigmented masses, which are located deeper than the spots. They are scattered throughout the whole body.

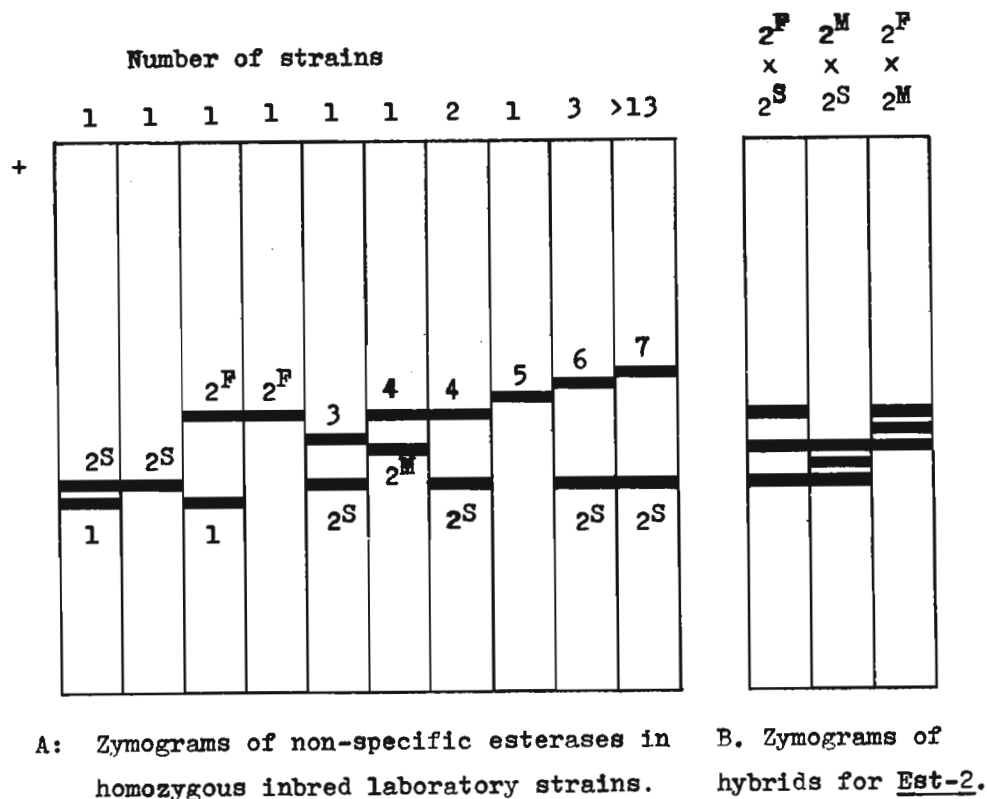
Brown spots begin to appear at the 2nd hour after copulation, and their manifestation is complete at the 6th hour. Brown masses begin again at the 2nd hour after artificial stimulation, but require 24 hours to complete their formation. They are more precocious when located in the abdomen.

Histological examination of spots revealed that the structure of the cuticula is normal, and only the cupulae are thicker and pigmented. The difference between spots and dottings consists only in a different number of thick cupulae, which is very low in dottings. Spotted flies show sometimes a brown layer added to the vaginal wall.

The histological examination of the brown masses has shown that pigmented small masses are present not only in the cuticula, but also in the epidermis, in the connective tissue and, sometimes, in fat and muscle. Pigmented masses are found also (in variable number and size) in different abdominal organs (intestine, uterus, tracheae). The pigmented masses in the epidermis and the other tissues mask the histological structure. Small pigmented masses are found attached to cuticula, to the muscle elements and to the intestinal wall of the thorax.

By means of thin layer agar gel electrophoresis (Ogita, DIS 37), flies of several days old were individually examined as to the electrophoretic variants of non-specific esterases. As a standard technique, the following were

various conditions of genes controlling esterase activities in each strain. About thirty inbred laboratory strains could be classified into ten different zymograms, each of which showed one or two clear zones of esterase activity (Figure A). Analysis of progenies ( $F_1$ ,  $F_2$  and back-crosses) from various inter-strain crosses suggested that there might be several (probably seven) different loci which control the main zones of esterase zymograms discussed here and that all of them are located on the 2nd chromosome. They were tentatively labeled Est-1 to Est-7. One locus, Est-2, was quite unique, involving four alleles ( $2^S$ ,  $2^M$ ,  $2^F$  and  $2^O$ ), among which  $2^O$  was a "silent" gene producing no detectable



zone in homozygotes and no detectable effect in heterozygotes. The other three alleles revealed marked hybrid enzymes in three heterozygous combinations (Figure B).

In a previous issue (Carlson, J. H., DIS 34:74-75) it was reported that a new stock containing an interruption of the second longitudinal vein ( $L_2$ ) had been produced. By selection it was possible to increase expression

of this mutant to a maximum average L<sub>2</sub> absence of 47%, at which time selection was no longer effective for increased absence. Early in 1964, while doing some additional work on possible isoalleles in various wild stocks, the selected L<sub>2</sub> mutant stock was reciprocally out-crossed to a Samarkand wild type stock. The F<sub>1</sub>'s were all wild type. However in the F<sub>2</sub> a few flies were produced without L<sub>2</sub> veins. These flies were used as parents to obtain a strain of L<sub>2</sub> deficient flies. For the past two years this stock has been maintained at room temperature by mass matings (about twice a year selection is practiced). At the present time this stock is characterized by all flies having interrupted L<sub>2</sub> veins. At the last count, 44 of 48 males (92%) and 46 of 54 females (86%) showed complete absence of the L<sub>2</sub> for both wings. Further studies of interaction with other venation mutants are planned.

Ménsua, J. L. University of Barcelona, Spain. Some factors affecting pupation height of *Drosophila*.

Sokal et al. (1) have studied some factors affecting pupation site of *Drosophila*, observing only central or peripheral sites, with independence of the distance to the medium. We considered it

important to see if the pupation height (p.h.) on the wall of vials was affected by different environmental factors and by disruptive selection for p.h.

Prat de Llobregat (near Barcelona) and Caracolisito (2) strains were used, both kept at 17°C. in population boxes. Eggs from these boxes were collected and 70 eggs were put in the middle of the food in vials of 80 mm. H., 24 mm. Ø and containing 8 mm. H. of medium, and 90 eggs in bottles of 100 mm. H., 45 mm. Ø and containing 10 mm. H. of medium. Inside the vials millimetric paper was put around from the top to the medium, in order to facilitate the measurements. The results, expressed in millimeters, were as follows:

a) Selection experiment: A Prat strain was used, with three vials each generation, selecting 8 males and 8 females from each vial for higher and lower p.h. at 17°C. The parent generation p.h. measured  $\bar{x} = 14.63 \pm 0.93$ , and the F<sub>2</sub>, in which were counted six vials in both directions,  $\bar{x} = 24.38 \pm 0.75$  and  $\bar{x} = 14.56 \pm 0.60$  respectively ( $P < 0.001$ ).

b) Temperature effect: Four temperatures, with five vials each one, were proved in Caracolisito strain. The results gave at 13°C.  $\bar{x} = 11.77 \pm 0.43$ , at 17°C.  $\bar{x} = 13.29 \pm 0.43$ , at 25°C.  $\bar{x} = 15.47 \pm 0.54$  and at 29°C.  $\bar{x} = 4.26 \pm 0.27$ . The differences among the various temperatures are significant at the level of at least  $P < 0.01$ .

c) Recipient size effect: By using three bottles instead of vials, the p.h. in Caracolisito strain gave  $\bar{x} = 25.03 \pm 1.19$  at 17°C. (instead of  $\bar{x} = 13.29 \pm 0.43$  in vials;  $P < 0.001$ ).

d) Crowding effect: By putting 35 eggs per vial and 45 eggs per bottle, the results at 17°C. were as follows: three vials of Prat strain gave  $\bar{x} = 9.07 \pm 0.76$  (instead of  $\bar{x} = 15.75 \pm 0.81$  with 70 eggs;  $P < 0.001$ ); three bottles of Caracolisito strain gave  $\bar{x} = 17.21 \pm 1.34$  (instead of  $\bar{x} = 25.03 \pm 1.19$  with 90 eggs;  $P \leq 0.001$ ).

e) Aeration deficiency: By using cork instead of cotton stoppers, the results with Prat strain at 17°C. in three vials were  $\bar{x} = 28.17 \pm 1.93$  (instead of  $\bar{x} = 14.63 \pm 0.93$  with cotton stoppers;  $P < 0.001$ ).

As we can see, the preliminary findings reported here suggest that the disruptive selection was only effective in one direction, moving the pupae away from the medium, but not toward it. The high temperature (29°C.) clearly diminishes the p.h., the highest value being at 25°C. The p.h. is higher when the number of individuals per recipient increases. When the recipient size (or the diameter) is increased, the p.h. also increases. When there is a good aeration inside vials the p.h. diminishes.

For these reasons we can consider the p.h. as an ethological and adaptative character with genetic variability (due to polygenes) because it responds to selection pressure and to different environmental factors, being exposed to natural selection in natural populations.

This work is presently continued in order to verify the results reported here, to make a selection experiment in detail, and to see if pupae size, sex and moisture have some influence in pupation height.

(1) Sokal, R. R., et al., 1960. Some factors affecting pupation site of *Drosophila*. *Annals Entomol. Soc. of America*, Vol. 53, 2:174-182. (2) I am grateful to Dr. Hoenigsberg for providing the Caracolisito strain. (Supported by a Predoctoral Fellowship (P.I.O.). I am in debt for advice and suggestions to Dr. A. Prevosti.)

Ives, P. T. Amherst College, Amherst, Massachusetts. Relocation of the or locus closer to pd.

Crossing over tests in March and April 1966 using b vg bw, or<sup>45a</sup> sp<sup>2</sup>, and a px or, in that sequence, with a new mutant, If<sup>65</sup> 1 16, have established an order of bw or sp If, which reverses the previ-

ously reported order for the or and sp loci. Based on 11 crossovers between or<sup>45a</sup> and sp<sup>2</sup> in 3367 flies a locus of 106.7 is suggested for or, placing it close to pd, another brownish eye color mutant. Earlier crosses (Ives and Evans, DIS 25:107) of pd and bw to each other and to or and or<sup>45a</sup> gave + offspring in all cases, indicating that these now fairly closely bunched loci probably function independently of each other in spite of the phenotypic similarity of many of their mutants.

Ohba, S. and F. Sasaki. Tokyo Metropolitan University, Japan. Polymorphisms of electrophoretic variants of esterase in a natural population of *D. virilis*.

In August 1965, 131 *D. virilis* flies were sampled from a large natural population in Niigata City. Zymograms of non-specific esterases were examined by means of thin layer agar gel electrophoresis.

Clear polymorphisms were found at all Est

loci. For Est-2, which shows four alleles ( $2^S$ ,  $2^M$ ,  $2^F$  and  $2^O$ ), flies were classified into seven electrophoretically different phenotypes as shown in the following table. Gene frequencies of these four alleles could be estimated in the following way:  $2^O = (\sqrt{S+O} + \sqrt{M+O} + \sqrt{F+O} - 1) / 2$ ,  $2^S = \sqrt{S+O} - 2^O$ ,  $2^M = \sqrt{M+O} - 2^O$ ,  $2^F = \sqrt{F+O} - 2^O$ . Expected numbers of each phenotype calculated from estimated gene frequencies coincided with observed ones.

Phenotype	Genotype	Observed		Gene frequency	Expected	
		No	%		%	No
S	S/S + S/O	80	61.1		61.5	80.6
M	M/M + M/O	1	0.8	$2^S = 0.734$	1.2	1.6
F	F/F + F/O	4	3.0	$2^M = 0.071$	3.5	4.6
O	O/O	1	0.8	$2^F = 0.143$	0.3	0.4
SM	S/M	13	9.9	$2^O = 0.052$	10.4	13.7
SF	S/F	28	21.4		21.0	27.5
MF	M/F	4	3.0		2.0	2.7

Lefevre, G. and L. Moore. San Fernando Valley State College, Northridge, California. Sperm transfer and storage.

The processes of sperm transfer and storage in *Drosophila* have been investigated by dissection of 1, 2 and 3-day-old males (both wild-type and  $v f^{3n} car$ ) and their consorts (3-day-old virgins) at

accurately timed intervals after the initiation of mating. In 264 dissections completed to date, the first evidence of sperm transfer was seen at 6.0 minutes after the initiation of mating by 3-day-old males, and at 8.0 minutes by 1-day-old males. The first evidence of sperm storage in the ventral receptacle or spermathecae was found at 8.5 minutes for 3-day-old males and at approximately 1 minute after completion of mating for 1-day-old males. A decided preference for initial storage in the receptacle, rather than in the spermathecae, was exhibited by sperm from males of all ages. Matings allowed to go to completion lasted an average of 18.2 minutes for 3-day-old males and 15.9 minutes for 1-day-old males. Sperm from 1-day-old males seem to be transferred and stored somewhat more slowly than do those from 3-day-old males. Maximum storage appears to occur within 15-20 minutes or less, regardless of the source of sperm.

To explore the effects of diminished sperm transfer on the storage process, females were dissected following second and third consecutive matings of 3-day-old males. Storage was found to be accomplished in these cases as promptly as in the case of large transfers (i.e., first matings).

The males were dissected following completion of their mating programs and inspected for condition of testes, seminal vesicles, ejaculatory duct, and accessory glands. Males dissected immediately after interruption of matings in which sperm transfer had begun were observed to have many motile sperm in the duct, but this rarely, if ever, occurs when matings go to completion. Dissection of 3-day-old males following second and third matings showed a progressive depletion of fluid in their accessory glands. Accessory gland fluid was usually found to be present in the glands of males dissected following second matings; whereas, following third matings the glands were nearly always collapsed and empty of secretion. Sperm were still present in the vesicles of 3-day-old males after 3 matings, but young males sometimes had virtually empty vesicles after only one mating.

Imaizumi, T. Kyoto University, Japan.  
A new embryonic lethal strain originated from a tip mutation of the X-chromosome.

A new embryonic lethal strain, named 1(1)3063, was found from a male of the Hiroshima wild strain by X-ray radiation to 3000r in 1963.

The genetic analysis is as follows:

Crosses	♀		F <sub>1</sub>	♂		Ratios in male
y/1-3063 x y	+	y		+	y	
	1089	1070		5	931	5/936 = 0.0053
sc/1-3063 x sc	+	sc		+	sc	
	550	530		2	558	2/560 = 0.0036
pn <sup>2</sup> /1-3063 x pn <sup>2</sup>	+	pn <sup>2</sup>		+	pn <sup>2</sup>	
	552	591		0	569	0/569 = 0
w/1-3063 x w	+	w		+	w	
	907	930		7	971	7/978 = 0.0072
car/1-3063 x car	+	car		+	car	
	550	336		136	214	136/350 = 0.389

Thus, the strain possesses a genic mutation or perhaps a small deficiency near pn, and it is of interest that the mutation is adjacent to or included in the tip lantern of the X chromosome. Hemizygotes are killed in the post-middle or late stages of embryonic development. Further studies are in progress at present.

Wagoner, Dale E. Metabolism and Radiation Research Laboratory, USDA, Fargo, North Dakota. The linkage group - karyotype relationship in the house fly (*Musca domestica* L.).

The house fly, *Musca domestica* (L.) has five linkage groups found by genetic methods (Hiroyoshi, 1960) and six pairs of chromosomes (Stevens, 1908). The normal female has an X-X sex chromosome complement and the normal male an X-Y complement.

Several exceptional strains have been recovered, however, where the Y chromosome has undergone a translocation with an autosome, and the males in these stocks may be X-X or X-0 and carry the Y-autosome translocation chromosome. The linkage groups have been arbitrarily numbered in two different ways by two independent groups without regard to karyotype relationship. The karyotype has been worked out (Boyes, 1962), but has not been related to the linkage groups, so that some linkage groups have as many as three different numbers with respect to chromosome designation. The present work has employed translocations and their cytological analysis as a method of determining which linkage groups should be assigned to the various chromosome pairs in the house fly karyotype. Sex-linked visible and lethal mutation tests were also performed in order to test for the presence of markers on the X-chromosome. Neither lethal nor visible mutations have been found as a result of these tests. This indicates that the five linkage groups belong to the five pairs of autosomes and that markers for the sex chromosomes are as yet unavailable. The X- and Y-chromosomes appear to be completely heterochromatic in metaphase of mitosis. A scarcity of visible or lethal genes on the sex chromosomes would be expected on the basis of results obtained from other insect species.

Kuhn, D. T. and G. D. Hanks. University of Utah, Salt Lake City, Utah. Suppression of RD (Recovery Disrupter) effect due to a 4th chromosome carrying Minute.

Previous work has shown that the autosomes help control the rate of recovery of the X and Y chromosomes in the RD system (Hanks 1964). Now it appears that a particular 4th chromosome marked with Minute suppresses

the usual RD effect. In 7 out of 7 experiments the percentage of females produced by males carrying all RD background but heterozygous for a 4th chromosome marked with Minute produced the following percentages of females: 52.2, 52.5, 50.7, 50.9, 53.2, 51.5 and 51.8. Controls gave 68.0 and 68.2% females. Table 1 gives results of 2 experiments testing individual males along with simultaneous sibling controls. The 4th chromosome marked with Minute does have a differential viability effect on the two sexes (about 6% greater effect on the female) but does not account for the reduced percentages of females. Minute males with RD background were crossed to RD females and the Minute and non-Minute flies were classified separately as to sex. Out of a total of 1,307 flies in the non-Minute class of progeny only 53.6% were females, indicating that the differential viability of the sexes carrying Minute does not account for the suppression of the RD effect. Another Minute, M(2)S7, shows no similar suppression effect.

Table 1. Representative data showing results of testing males carrying a M(IV) chromosome. (Each % female value represents at least 200 progeny)

Experimentals		Controls	
Y <sup>RD</sup> ♀(5) x Minute ♂(1)	Y <sup>RD</sup> ♀(5) x Minute ♂(1)	Y <sup>RD</sup> ♀(5) x RD ♂(1)	Y <sup>RD</sup> ♀(5) x RD ♂(1)
% ♀	% ♀	% ♀	% ♀
60.2	49.4	70.4	69.8
42.5	56.2	65.6	65.9
52.8	50.2	67.4	67.9
52.9	54.7	68.7	68.7
55.3	54.6	69.3	72.6
50.0	53.9	68.0	68.0
54.0	$\bar{X} = 53.2$	69.6	64.3
$\bar{X} = 52.5$	$n = 6$	67.4	$\bar{X} = 68.2$
$n = 7$	$S^2 = 6.19$	66.0	$n = 7$
$S^2 = 25.12$		$\bar{X} = 68.0$	$S^2 = 7.13$
		$n = 9$	
		$S^2 = 2.62$	

Reference: Hanks, G. D. 1964, Genetics 50:123-130. Details of this study may be found in: Kuhn, D. T. "Fourth Chromosome Studies of a Case of Meiotic Drive in *Drosophila melanogaster*." M.S. Thesis, University of Utah Library, Salt Lake City, Utah. (Supported by NSF Grant No. GB-456)

Jones, L. P. University of Sydney, Australia. Instability of an ebony polymorphism of *D. melanogaster*.

Jones and Barker (1966, Genetics 53:313-326) describe the breakdown of an apparently stable polymorphism between ebony (*e*<sup>11</sup>) and its wild-type allele.

The frequency of ebony homozygotes remained at 1 to 3% in each of two cages between weeks 12 and 46 after initiation. The frequency then declined so that by week 150, no ebony homozygotes were found among 2000 flies from either cage, and only 3 heterozygotes in each of 144 and 136 adults' progeny tested from the two cages. Since then, the frequency of ebony has continued to decline. At week 189, egg samples were taken from the cages and the emergences were progeny-tested. No heterozygotes were found among 197 and 187 flies respectively. This was repeated at week 210 with larger samples (669 and 659). Again, no heterozygotes were found. The ebony gene has thus declined to a negligible frequency. This indicates that the initial equilibrium was probably due to linkage disequilibrium and not overdominance.

Frye, Sara H. P.O. Box 267, Irvine, Kentucky. Correction in the report of Sara M. Frye (DIS 41:205).

I have been informed (as of late February) that the double marker mutant stock ( $Y/y, ac$  In49  $B^{M1}$  ♂ &  $Y/y$  f: ♀) reported by Frye (1966, DIS 41:61, 175 and 205) is not available from the Pasadena stock center.

I would suggest that anyone interested in this stock for the purpose of cytological analysis write to Dr. E. Grell of Oak Ridge, Tenn. This stock is important for the following reasons:

First, the double marker mutant did not originate in a scute-8 or scute-8-like inverted X-chromosome, but it did originate in an irradiated (2 kr) In49  $B^{M1}$  chromosome which is like a normal X-chromosome from the distal tip of the X-chromosome up to the left break (between 1, 7.5 and 1, 13.7) of In49 which is the more distally located of the two inversions. This X-chromosomal area (1, 0.0 - 7.5 or 1, 0.0 - 13.7) includes the normal alleles of the more useful mutant markers of lethal-Jacobs-1 ( $l J1^+$ ), yellow ( $y^+$ ), achaete ( $ac^+$ ), scute ( $sc^+$ ), and lethal to right of scute ( $li^+$ ). The origin of In49 of Muller and Stone is not given in the 1944 edition of Bridges and Brehme (see p. 97), however, the origin of  $B^{M1}$  (In $B^{M1}$ ) is "from X-rayed wild type" (ibid. p. 20).

Second, the origin of the scute-19i chromosome is "from X-rayed In49, bb" (ibid. p. 167). Scute-19i chromosomes contain an insertion of the normal alleles of the more useful mutant markers yellow ( $y^+$ ), achaete ( $ac^+$ ), scute ( $sc^+$ ), and lethal to the right of scute ( $li^+$ )\* from the distal tip of an irradiated In49 bb chromosome, and these normal alleles are re-located between the normal alleles of the recessive markers, dumpy (2, 11.0) and clot (2, 16.5) in the second chromosome.

In both chromosomal environments, i.e., In49  $B^{M1}$  (or In49 bb) and scute-19i the yellow region is free of chromocentral heterochromatin and therefore not like a scute-8 chromosome where chromocentral heterochromatin is adjacent to the yellow-achaete region. Unfortunately, I am not trained in the cytological analysis of the yellow-achaete-scute region and therefore I am not qualified to give an opinion as of the presence of intercalary heterochromatin in the neighborhood of the y-ac-sc region in either chromosomal system.

\*see Serebrovsky, Ivanova, and Ferry, 1929, Journal of Genetics 21:287-314.

Whittinghill, M. University of North Carolina, Chapel Hill, North Carolina. Somatic spot confirmation of the chromosome arm loci of in ri and p.

The relation of the in and ri loci to each other and to the kinetochore has been in doubt, and they are variously placed on different maps and lists. Other workers have established that the gene order is st in ri p, before we ob-

tained further confirmation in ordinary testcrosses. Now a single specimen confirms the location of the spindle attachment as being between st and in.

A mosaic female from a testcross with st in ri pP females and a male showing G1 st Ubx produced the expected offspring in both sexes; some were scarlet and the remainder showed Glued orange eyes and were Ultrabithorax. No sib of the mosaic showed intumed or radius incompletus. Most of the mosaic resembled her st sibs, except that less than half of the right eye showed st alone. The remainder of that eye and the entire left eye were orange, st pP. Recessive intumed showed on the entire right abdomen and on the right mesothorax. The right wing had ri clearly expressed in vein II. The contiguous presence of in and ri on the same side of the thorax and of pP in part or all of the eye would be explained by somatic crossing over between the spindle attachment and the in locus. No deletion as long as this would be likely to survive let alone multiply to occupy so much of the classifiable regions of the body. No double fertilization seems possible here, because the mother was homozygous recessive st in ri pP and the father contributed + alleles for all six marked loci except scarlet. It would be too much to suppose that somatic crossing over and segregation took place in both arms of the third chromosome in this fly, so all three somatically segregating loci must be in the right arm.

Moriwaki, D. and Y. N. Tobar. Tokyo Metropolitan University, Japan. Male crossing over in *Drosophila ananassae*.

It has been known that male crossing over in *D. ananassae* could occur habitually without any agency but by means of some heritable enhancing power of crossing over (Moriwaki, 1937, '38, '40; Kikkawa,

1937). In Kikkawa's case, an enhancer or enhancers were believed to induce male crossing over with a considerably high ratio in the 3-chromosome, while Moriwaki's case was related to the 2-chromosome in which a dominant gene, En-2, was able not only to increase somewhat the female crossing over, but also to induce male crossing over. Unfortunately all these strains were lost during the war, leaving further investigation interrupted. On the other hand, another case with an abnormally high rate of spontaneous male crossing over was reported in a local strain from India of *D. ananassae* (Mukherjee, 1957; Ray Chaudhuri and Mukherjee, unpubl.) Mukherjee (1961) investigated effect of selection on crossing over in the males, using two markers in the 3-chromosome.

Recently, while studying crossover frequencies between two markers, st (scarlet) and se (sepia) in the 3-chromosome, firstly we could observe a recombination range of zero to several per cent in different individual males, with an average value of 2.64%. The recombination value in females was once estimated as 47.06% (1505/3198) by Moriwaki and Ono (unpubl.). Next, crossing the homozygous double recessives with different wild flies, the F<sub>1</sub> females and males obtained were individually backcrossed with the double recessives respectively. The crossover frequency between st and se amounted to 7.75% (1152/14872) in total offspring from the mating, st se ♀ x st se/+ + ♂, whereas the reciprocal mating, st se/+ + ♀ x st se ♂, gave a recombination value of 47.99% (5983/12466). In the former, however, when the data were separately analysed for the different wild strains, it appeared that the degree of male crossing over might depend on which wild chromosome would pair heterozygously, varying from 0.29% to 36.26%.

As for these results, what kind of causes would act, e.g. a dominant gene, polygenes, or some other factors, is still uncertain. However, a possibility can not be excluded that there may exist a trend to induce a little male crossing over fairly often, this being a characteristic of this species. In reality, similar phenomena were also encountered regarding the 2- and 4-chromosomes, just lately.

Poulson, D. F. Yale University, New Haven, Connecticut. Developmental effects of mutants at the Notch locus in *D. mel*.

A number of point mutants localized by Welshons in the Notch region of the X have been examined for developmental disturbances. Those mutants giving Notch phenotype in the heterozygote,

N<sup>55e11</sup>, N<sup>264-40</sup>, N<sup>264-103</sup>, N<sup>Co</sup>, and N<sup>60g11</sup>, show essentially identical embryonic disturbances in the hemizygous males, indistinguishable from those shown by deficiency for all or part of the Notch region. Thus these five sites distributed throughout the length of the Notch region are essential to normal neurogenesis and separation of hypoderm. On the other hand, those recessive lethals lacking the Notch effect in the heterozygote, and designated by Welshons (1965) as 1(1)N<sup>1</sup>, 1(1)N<sup>2</sup>, and 1(1)N<sup>3</sup>, show related but much more limited effects in male embryos. 1(1)N<sup>B</sup> is not egg lethal but appears to have its effect in early larval life. A detailed study of these and other Notch mutants is in progress.

Poulson, D. F., Thomas Hyde and Kugao Oishi. Yale University, New Haven, Connecticut. Interactions between SR spirochetes from different species of *Drosophila*.

SR spirochetes from *D. equinoxialis* interact both in vivo and in vitro with those from *D. willistoni* and *D. nebulosa*. When spirochetes are introduced from *equinoxialis* into SR strains of *nebulosa*, or vice versa, the spirochetes of *equinoxialis* origin eliminate those of *nebulosa* origin.

Indications are that *equinoxialis* spirochetes also eliminate those from *willistoni*. The nature of the substances involved is under investigation as is the question of their possible relationship to male lethality in SR strains.

Muhammed, Amir and James Trosko. Oak Ridge National Laboratory, Oak Ridge, Tennessee. A search for photoreactivating enzymes in *D. mel.* extracts.

Photoreactivating (PR) light given as a post-treatment increases embryo survival and adult fertility, and decreases recessive lethal mutation rate after low doses of ultraviolet (UV) radiation to polar cap cells of

*Drosophila* (Meyers, 1951, Genetics 36:565; Browning and Allenburg, 1962, Genetics 47:361). Also, UV-induced phenocopies in pupae can be photoreactivated (Perlitsch and Kelner, 1953 Science 118:165). The question of whether these phenomena were dependent on a photoreactivating enzyme similar to that in yeast and *Escherichia coli* (Rupert, 1960, J. Gen. Physiol. 43:573) was investigated. Eggs, larvae, pupae, and adult *Drosophila* were grown aseptically and homogenized in cold 0.05 M phosphate buffer (pH 7.3). The homogenates were sonicated for 20 sec. to rupture cells and were then filtered through miracloth to remove chitinous debris.

The photoreactivating enzyme activity of different extracts was tested by their ability to photoreactivate UV-irradiated transforming DNA as described earlier (Muhammed, in press, J. Biol. Chem.). A large excess of calf thymus DNA was added to the reaction mixtures in order to protect the transforming DNA against DNases. The results of a typical experiment are summarized in Table 1.

Table 1. Photoreactivating enzyme activity of various extracts from *Drosophila*.

No.	Extract	No of Streptomycin Transformants		Ratio of PR/dark Transformants
		Dark Control	PR	
1	Growth medium	1081	1065	0.99
2	Eggs	1201	1220	1.02
3	Larvae, 3-day	1112	1184	1.06
4	Larvae, 7-day	930	899	0.97
5	Pupae, 1-day	1093	1129	1.03
6	Pupae, 2-day	1109	1110	1.00
7	Adult	1373	1323	0.96

No photoreactivating enzyme activity was detected under the assay conditions used in these experiments. It was realized that the lack of PR activity in the extracts might be due to the presence of some substances which could strongly inhibit the enzyme activity. When purified yeast PR enzyme was added to the extract, however, there was no loss in activity, indicating the absence of any inhibitory material in *Drosophila* extracts. This suggests that the reported photoreactivation phenomena in *Drosophila* are mediated through an indirect effect (Jagger and Stafford, 1965, Biophys. J. 5:75). Another possibility is that the photoreactivating enzyme is present in too small amounts, or that the enzyme in *Drosophila* is of a different type than the one in micro-organisms, and is not detectable by the assay used in this work. The extracts were diluted to a final protein concentration of approximately 0.5 mg/ml in 0.04 M phosphate buffer (pH 7.2). The reaction mixture contained 0.2 ml of UV-irradiated *H. influenzae* transforming DNA (10  $\gamma$ /ml, 1% survival 2480 ergs/mm<sup>2</sup>), 0.2 ml calf thymus DNA (1.80 mg/ml), and 0.4 ml of the diluted extract. 0.4 of the mixture was kept as a dark control, and the remaining photoreactivated for 20 minutes. Conditions for photoreactivation and assay of transforming DNA have been previously described (Muhammed, in press, J. Biol. Chem.). (Research sponsored by the U.S. Atomic Energy Commission under contract with Union Carbide Corporation and American Cancer Society Fellowship to J. E. Trosko, PF-253)

Tsacas, L. Centre National de la Recherche Scientifique, Gif-sur-Yvette, France. Drosophilids of the Ivory Coast.

During research work on the fauna of the Palmtree Borasus ethiopum done by M. R. Vuatoux at the Laboratory of Lampto (between Tiasalé and Toumodi) a large number of Drosophilids was

captured. The bait used was: a) fruits, b) terminal buds and c) sap of *B. ethiopum*. The collecting was made March 5 to November 22, 1962. The following species were identified on material kept in alcohol. 1. *Chymomyza avikam* (a); 2. *Zaprionus tuberculatus* (a,c); 3. *Z. vittiger* (a,b,c); 4. *Z. sp. cf. armatus* (a,c); 5. *Drosophila latifasciaeformis* (b); 6. *D. saba* (b); 7. *D. melanogaster* (a); 8. *D. seguyi*, species A (a); 9. *D. ananassae* (a); 10. *D. yukuba* (a); 11. *D. sp. melanogaster gr.* (a).

Mittler, Sidney and J. R. Gerdy. Northern Illinois University, DeKalb, Illinois. The effect of various maternal chromosomes on the spontaneous occurrence of XO males.

J. Arnesen, working in our laboratory in the spring of 1965, found that more spontaneous XO males occurred in the offspring of a cross of  $X^{c2} y^B$  males to  $y sc^{S1} In^{49} sc^8, bw; stp^P$ , than in the offspring resulting from the mating to

a  $y w f$  female (Mittler, Arnesen and U in press in *Int. J. Rad. Biol.*). To determine what effect the maternal parent had upon the spontaneous occurrence of XO males, gynandromorphs and XXY females, the  $X^{c2} y^B/sc^8 y^+$  males age 2 - 6 hours old were mated at the ratio of one male to three females every other day or 12 days to females of  $y sc^{S1} In^{49} sc^8, bw, stp^P$ ;  $y w f$ ;  $y sc^4$ ;  $y fa^n dl-49$ ; and  $y^{31d} sc^8 wa$ . The data are presented in Table 1.

Table 1.  $X^{c2} y^B/y^+ sc^8$  X various females with respect to spontaneous XO males in the offspring.

Female parent	No. of gametes	% XC	% Gynandromorph	%XXY
$y w f$	40,875	.503	.038	.084
$y fa^n dl-49$	19,248	.620	.047	.314
$y sc^4$	37,905	.659	.034	.110
$y^{31d} sc^8 wa$	54,236	.694	.124	.110
$y sc^{S1} In^{49} sc^8, bw, stp^P$	44,425	.776	.074	.121

There was no significant difference in offspring of the various crosses described in Table 1 with respect to the number of gynandromorphs, production of XXY females, productivity per male, and sex ratio. However, there was a significant difference between the offspring of the  $y w f$  mating (in which the lowest percentage of XO's resulted) and those of females of other stocks. These data are presented in Table 2.

Table 2. Comparison by means of a 2 x 2 contingency table of XO males produced by various females mated to  $X^{c2} y^B/y^+ sc^8$

	Chi-square
$y w f$ vs $y fa^n dl-49$	5.577
$y w f$ vs $y sc^4$	9.643
$y w f$ vs $y^{31d} sc^8 wa$	14.673
$y w f$ vs $y sc^{S1} In^{49} sc^8, bw, stp^P$	29.290
$y fa^n dl-49$ vs $y sc^{S1} In^{49} sc^8, bw, stp^P$	6.424
$y sc^4$ vs $y sc^{S1} In^{49} sc^8, bw, stp^P$	6.42

The appearance of XO male indicates a loss of the ring X or Y chromosome or the  $y^+$  portion usually before fertilization and if this loss occurred with the same frequency in the spermatogenesis of all the  $X^{c2} y^B$  males, then the differences in the number of XO's obtained may be explained by the variation in the viability of the different XO's produced. The  $y w f$  XO male is thus less viable than the XO male,  $y sc^{S1} In^{49} sc^8$ . More gynandromorphs resulted in crosses involving  $y^{31d} sc^8 wa$  and  $y sc^{S1} In^{49} sc^8, bw, stp^P$  than with  $y w f$ .

Bahn, E. University of Copenhagen, Denmark. Crossing over within "alleles" determining multiple amylases in *D. mel.*

Zymograms of amylase activity of individual whole flies were obtained by means of agar gel electrophoresis. The patterns observed were identical to those described by Kikkawa (1964, Jap.

*J. Genet.* 39:401). Thus five different major isoamylase bands could be recognized. Following Kikkawa these isoamylases are designated Amy<sup>1</sup>, Amy<sup>2</sup>, Amy<sup>3</sup>, Amy<sup>4</sup>, Amy<sup>6</sup> according to the electrophoretic migration velocity towards the anode, Amy<sup>1</sup> being the fastest migrating component and Amy<sup>6</sup> the slowest.

Some highly inbred strains reveal the coexistense in individual flies of two different amylases, e.g. nos 4 and 6. In crosses these two bands segregate together and are therefore inferred to be determined by a single allele, viz. Amy<sup>4.6</sup>. The possibility also exists that two closely linked structural genes are responsible for this phenotype. In the latter case it should be possible to demonstrate recombination of Amy alleles. With this in mind the following experiments were carried out, and indeed, crossing over was found to occur in a strain Amy<sup>4.6</sup> obtained from Dr. Kikkawa (originally established from an adipose<sup>60</sup> line by Dr. Doane). Crossing over was also observed in a strain Amy<sup>2.3</sup> isolated together with Amy<sup>1</sup> and Amy<sup>1.3</sup> from a Bennett population cage (58-105) established in 1958 by Dr. Frydenberg. A third strain c Amy<sup>1</sup> wt px was obtained from Miss Rose Schwarz, The Institute for Cancer Research, Philadelphia (DIS 39:20 g99).

Females heterozygous for the Amy genes in question and for two closely linked outside marker genes curved (c map position 75.5) and welt (wt 82.0) were mated to c Amy<sup>1</sup> wt males. The expected 6.5% recombinant male offspring were isolated and stored for 8 - 10 days on a starch medium. They were then mated in single cultures to Cy L/Pm females in order to recover chromosomes in which crossing over between the Amy loci had occurred. Finally the amylase zymogram of each individual outside-marker-recombinant male was determined. Among 5039 flies 6 were found to be recombinants also with regard to the Amy loci. The results are given in table 1.

Table 1. Crossover data

Maternal genotype	No. of ♂♂ zymogramed (6.5%)	Estim. total ♂♂ offspring (100%)	Crossover <sup>+</sup> in reg. I	Crossover <sup>+</sup> in reg. II	Crossover <sup>+</sup> between Amy loci
c 1 wt + 46 +	1,034	15,908	(c+) 368 (+wt) 94	(c+) 457 (+wt) 115	0 0
c 1 wt + 23 +	947	14,569	(c+) 159 (+wt) 81	(c+) 557 (+wt) 150	(1.3) 1 0
c 1 + + 23 wt	1,128	17,354	(++) 409 (cwt) 11	(++) 686 (cwt) 22	(2) 2 0
+ 23 wt c 46 +	1,618	24,892	(++) 583 (cwt) 54	(++) 918 (cwt) 63	(2.6) 2 (4.3) 1
c 23 + + 46 wt	312	4,800	(++) 95 (cwt) 8	(++) 197 (cwt) 12	0 0
TOTAL	5,039	77,523	1,862	3,177	6

+ The type of recombinants are given in parentheses.

The recombination analysis has demonstrated the amylase "alleles" to be spatially separable with a linkage intensity of the order of 1/10,000 (0.01 map units). Furthermore, the crossover produced two new banding patterns, Amy<sup>2</sup> and Amy<sup>4.3</sup>. For the strains in question it is evident that the loci Amy<sup>3</sup> and Amy<sup>6</sup> are placed to the right of the loci Amy<sup>1</sup>, Amy<sup>2</sup>, and Amy<sup>4</sup> on the chromosome. These data suggest that the different amylase isozymes of *D. mel.* correspond to different structural genes, and it seems reasonable to suppose that occasionally a duplication of the Amy region has taken place. Accordingly, it is suggested to alter the symbols for the amylase genes: stock Amy<sup>4.6</sup> should hereafter be designated

Amy<sup>4</sup> Amy<sup>6</sup>, and Amy<sup>2.3</sup> designated Amy<sup>2</sup> Amy<sup>3</sup>, and Amy<sup>4.3</sup> designated Amy<sup>4</sup> Amy<sup>3</sup> etc., corresponding to the linkage data. The importance of distinction between stocks of independent origin possessing identical electrophoretic patterns is evident.

Mather, Wharton B. University of Queensland, Australia. Inter-yearly fluctuation of *D. rubida* inversion polymorphism.

It has been shown previously that certain inversions vary significantly in frequency in different ecological niches and between sexes (Mather, 1963b), at different times of the year (Mather,

1964) and in different geographical regions (Mather, 1966b). It is the purpose of this report to record inversion frequency at the same time of the year over a three year period at two different stations in Papua - New Guinea.

Material was collected from fermenting banana baits at Bulolo in August 1963 - 1965 and at Bisianumu (Port Moresby) in May 1963 - 1965.

The material was analysed by mating males from the wild against a standard strain and scoring seven larvae from each mating against a photographic map (Mather, 1961). Salivary chromosomes were prepared by the method given in Strickberger (1962).

The inversions recorded are described in a number of publications (Mather, 1961, 1963a and c, 1966a).

The results are set out in Tables I and II. In 1963 and 1964 at Bisianumu inversions IIRB and I and IIRD, H and G were confounded. These tables clearly show that there is no detectable trend in the inversion frequencies over this three year period.

Acknowledgements are due to Sheridan Butler for technical assistance and Mr. D. Angus who collected the Bulolo flies.

References: Mather, W. B. 1961. Chromosomal Polymorphism in *Drosophila rubida* Mather. Genetics, 46:799. Mather, W. B. 1963a Notes on the Inversions of *Drosophila rubida*. DIS, 37:104. Mather, W. B. 1963b. Ecological and Sexual Variation in *Drosophila rubida* inversion polymorphism. Heredity, 18:109. Mather, W. B. 1963c. Further Inversions in *Drosophila rubida*. DIS, 38:55. Mather, W. B. 1964. Temporal variation in *Drosophila rubida* inversion polymorphism. Heredity, 19:331. Mather, W. B. 1966a. New Inversions in *Drosophila rubida*. DIS, 41:125. Mather, W. B. 1966b. *D. rubida* inversion polymorphism. DIS, 41:126. Strickberger, M. W. 1962. Experiments in Genetics with *Drosophila*. John Wiley.

Table 1. Port Moresby

Chromosome	1963 %	1964 %	1965 %
II +	44.1	35.2	49.4
LA	0.9	3.1	1.1
RA	5.4	4.9	5.1
B	] -12.7	] -23.5	10.1
I			2.8
C	28.9	29.6	19.1
D	] -26.5	] -34.0	14.6
H			0
G			15.7
III +	54.9	56.2	51.1
A	3.4	0.6	5.1
B	3.4	1.8	3.9
D	4.4	4.3	3.9
E	32.4	34.0	38.8
F	10.3	7.0	6.2
H	0	0.6	1.1
I	0	0	0
Flies scored	102	181	89

Table 2. Bulolo

Chromosome	1963 %	1964 %	1965 %
II +	0	0.3	0
LA	14.4	16.8	7.4
RA	35.6	41.6	41.2
B	] -44.9	25.8	14.19
I		17.7	27.03
C	0.4	0.3	0.7
D	] -99.6	76.5	73.0
H		22.3	25.7
G		0.6	0.7
III +	27.1	28.3	32.4
A	0	0.3	1.4
B	0	0.3	1.4
D	53.4	52.0	50.0
E	49.6	51.7	50.0
F	0	0.3	0
H	12.3	13.6	10.8
I	21.6	16.5	14.9
Flies scored	121	172	74

Hughes, Ann M. and Philip E. Hildreth.  
University of California, Berkeley, California. The production of a new mutant in *D. melanogaster* by low doses of tritium irradiation.

On two separate occasions, during the course of an investigation in which *D. melanogaster* larvae were being raised on tritiated medium, apparently identical mutants affecting adult coloration were recovered. Since these mutants originated among only a few thousand flies and only

in the treated series, it appeared that tritium might be repeatedly causing mutations at a specific locus. Therefore, further experiments were conducted in two series (1961-62, 1965-66) in order to test whether this one type of mutant could be routinely produced.

Homozygous yellow ( $y/y$ ) females that had mated with  $y/B^{SYy+}$  males oviposited on control or tritium-supplemented medium where the eggs hatched, the larvae and pupae developed. For the treated series, the standard cornmeal-molasses-agar-yeast medium contained either 0.1 or 1.0  $\mu\text{C } ^3\text{H}$  per gram. On the basis of the table of Tolbert (1960) and assumptions of rapid equilibration between the larval body water and that of the medium, the two concentrations of tritium used would give a maximum total body irradiation of 0.5 and 5.0 r per fly during the time from hatching through eclosion. The actual irradiation is probably less.

The  $F_1$  males ( $y/B^{SYy+}$ ) were then individually mated to virgin yellow nontreated females and the offspring raised on control medium. The expected offspring would again be yellow females and nonyellow Bar-eyed ( $y/B^{SYy+}$ ) males. Among the second-generation offspring exceptional males were recovered; they had bar-shaped eyes and wildtype-colored wings as in the expected class, but their bristles were yellow and bodies yellowish (but not as yellow as  $y/Y$  males) instead of wildtype. The mutant has been transmitted with the Y chromosome ( $B^{SYy^{61d}}$ ) through many generations. The results are shown in Tables I and II.

In order to determine whether other types of irradiation would produce similar mutation, experiments involving the above mating procedure, but using X rays as the irradiation source, were carried out. In the first experiment, virgin males were given 2800 r by use of a 250-kV X-ray machine with a 0.5-mm Cu and a 1.0-mm Al filter. The second experiment repeated the first except that a smaller dose of X-ray was used. (A 150-kV machine with an inherent filtration of 1.5 mm Al was used to give the males 1000 r.) In the third experiment, prepupae were collected from culture bottles, irradiated with the 150-kV machine (735 r), and then allowed to pupate and eclose. In these experiments the expected offspring were the same as in the tritium experiments. Among 25,120 males from the control and 22,476 from the treated series, no mutants were recovered. Therefore it appears likely that it is not irradiation per se but some specific property of tritium that caused the mutations.

According to Dr. Irwin I. Oster, who kindly did salivary gland analyses, the  $B^{SYy^{61d}}$  chromosome was not different from the original  $B^{SYy+}$  chromosome. Thus no gross chromosomal aberration was associated with the mutation. It is likely that the change was within the  $y^+$  locus, although an undetected alteration in some other region of the  $B^{SYy+}$  chromosome cannot be ruled out.

To determine interactions with other alleles, males carrying  $B^{SYy^{61d}}$  were crossed with the following stocks:  $y, y^{2S}, y^{62a} sc cv$  and  $y f; br ec/y^{3d}, y^2 cv v f$  and  $M(1)n/FM6 y^{31d}$ .

When heterozygous,  $y^{61d}$  acted as a dominant gene over all alleles in regard to wing color, over  $y^2$  and  $y^{3d}$  in regard to body color.

The alleles which normally produce dark bristles are dominant over  $y^{61d}$ . In hemizygous condition, both  $y^{3d}$  and  $y^{61d}$  produce yellow bristles, yet  $y^{3d}/B^{SYy^{61d}}$  males have dark bristles.

Combining the control and the X-ray totals, the mutation rate is 0.016/1000. Since both mutants were found in the same culture, the minimum number of mutant events could be 0.008/1000. In the tritium-treated group, the total mutation rate is 0.148/1000, and the minimum mutant event rate 0.058/1000. In addition, in the tritium treatment, in one culture a gonosomal mosaic for this mutant was recovered, and in another culture one mutant in the third generation. At present there is no explanation for the lack of recovery of mutants in each experiment in the 1965-66 series, as was observed in 1961-62. (The work described in this paper was sponsored, in part, by AEC Contract No. W-7405-eng-48.)

Tolbert, B.M.: Self-Destruction in Radioactive Compounds. *Nucleonics*, Aug. 1960:74-75.  
Continued on next page.

Table I. The occurrence of B<sup>Sy</sup>61d mutant in *Drosophila* grown on medium containing tritium (1961-62).

Experiment	Date	Treatment	Number cultures	Total males	Total mutants	Mutant events <sup>a</sup>
I	6/61	Control	199	6487	0	0
		0.5 r	189	6044	5	2
II	8/61	Control	385	15574	0	0
		0.5 r	388	15954	1	1
III	1/62	Control	240	7260	2	1
		0.5 r	263	8474	6	3
		5.0 r	246	7419	1	1
IV	4/62	Control	199	7817	0	0
		0.5 r	206	7673	0	0
		5.0 r	394	13903	10	2
Totals		Control	1023	37138	2	1
		0.5 r	1046	38145	12	6
		5.0 r	640	21322	11	3

a. In some cultures, more than one mutant was recovered. Thus, "mutant event" refers to the number of cultures in which one or more mutants were found.

Table II. The occurrence of B<sup>Sy</sup>61d mutant in *Drosophila* grown on medium containing tritium (1965-66)

Experiment	Date	Treatment	Number cultures	Total males	Total mutants	Mutant events <sup>a</sup>
V	10/65	Control	28	7941	0	0
		20 r <sup>b</sup>	69	18085	0	0 <sup>c</sup>
VI	11/65 12/65	Control	200	15745	0	0
		0.5 r	495	41178	0	0
VII	2/66	Control	117	9853	0	0
		0.5 r	338	26270	0	0
VIII	3/66	Control	100	2923	0	0
		1 r	319	10463	1 <sup>d</sup>	1

a. In some cultures, more than one mutant was recovered. Thus "mutant event" refers to the number of cultures in which one or more mutants were found.

b. Tritium solution was inadvertently contaminated with <sup>14</sup>C.

c. One mutant found in third generation.

d. Phenotypically like original mutant, but germ cells B<sup>Sy</sup>61d<sup>+</sup>.

Hijikuro, S. Osaka University, Osaka, Japan. Studies on the content of beta-alanine and the body color of *Drosophila*.

Seki (1962, DIS 36:115) first reported that beta-alanine was detected in the pupal sheaths of wild type strain of *D. virilis*, but not in those of dark color mutant strain, ebony (eb). Further-

more, Fukushi (1966, Japan. J. Genetics: In press) revealed the fact that the pupal sheaths of a pale body color mutant, yellow (y), of *D. melanogaster* contained more beta-alanine than those of the wild type.

In the present study, the content of beta-alanine in adult flies (whole body) was analyzed after acid hydrolysis with an amino acid analyzer. In a wild type strain (Oregon-R) and y of *D. melanogaster*, molar ratios of beta-alanine to leucine were 0.05 and 0.08, respectively. In *D. virilis*, a wild type strain (Pasadena) contained the same amount of beta-alanine as y. The ratio was 0.06. The content of beta-alanine in ebony mutants of both species was negligible.

These results suggest some relationship between the body color and the content of beta-alanine in adult flies as well as in pupal sheaths.

Bösigger, E., A. Pentzos and A. Kanellis.  
C. N. R. S., Gif-sur-Yvette, France.  
Frequency of visible mutants in natural  
populations of *D. subobscura*.

The frequency of visible mutations in  
the offspring of ♀♀ of *D. subobscura*,  
captured in natural populations of  
Litochoron village at the foot of the  
mountain Olympus and on the isle of Samo-  
thrace in Greece has been established as

a basic information for further studies on the genetical variability of Greek populations of this species.

In this first report we are giving the distribution of the number of visible mutant characters in the  $F_2$  offspring of ♀♀ and ♂♂. In some cases we utilized for practical reasons also  $F_3$  and  $F_4$  cultures.

An aberrant phenotype has been considered as a mutant character when it occurred at least three times in one single  $F_2$  culture. The table gives for each population on the first line the observed frequency of ♀♀ revealing in their offspring from 0 to 12 mutants; and on the second line the corresponding theoretical frequency, calculated on the basis of a Poisson distribution. The following populations are listed: A: Offspring of 47 single ♀ caught at Litochoron (Olympus). B: Offspring of 48 ♀ caught on the isle of Samothrace. C: Offspring of 32 groups of 5 to 10 ♀♀ in each vial, from Samothrace. D: Offspring of 55 ♂♂ captured on Samothrace and crossed with ♀♀ from the laboratory standard strain "Küssnacht". E: Analogous figures for the offspring of 218 ♀♀ of *D. melanogaster*, captured in a natural population of Banyuls, France, (Bösigger, 1962).

Table: Frequency of mutants in Greek populations of *D. subobscura*.

Popula- tion	n ♀	n mut.	m̄ per ♀	Frequency of ♀ revealing from 0 to 12 mutants												X <sup>2</sup>	P
				0	1	2	3	4	5	6	7	8	9	10-12			
A Lito- choron	47	217	4.6	0 0.5	2 2.2	8 5	15 7.7	3 8.8	4 8.1	2 6.2	4 4	5 2.3	0 1.2	4 0.8	15.2	< 0.01	
B Samo- thrace	48	213	4.4	0 0.6	3 2.6	3 5.7	12 8.4	10 9.2	8 8.1	5 5.9	3 3.7	2 2	1 1	1 0.6	3.7	< 0.50	
C Samo- thrace	32	89	2.7	1 2.1	5 5.8	10 7.8	8 7	5 4.7	1 2.6	1 1.1	0 0.4	0 0.1	1 0.1	0 0	1.6	< 0.50	
D Samo- thrace	55	132	2.4	0 5	17 12	23 14.4	5 11.5	5 6.9	2 3	1 1.3	0 0.5	1 0.1	0 0	1 0	19.2	< 0.001	
E Bany- uls	218	971	4.5	5 2.5	7 11	20 24.9	30 37.1	55 41	41 37	27 28	21 17.9	9 10	2 4.9	1 2.2	14.2	< 0.05	

The results presented in the table are leading to the following preliminary conclusions:

1. The heterogeneity observed in the offspring of single ♀ of *D. subobscura*, captured in the populations of Litochoron (A) and Samothrace (B) is as high as the heterogeneity observed in the offspring of single ♀ of *D. melanogaster*, caught in the population of Banyuls (E). The mean number of 4.4 to 4.6 mutants in the offspring of one ♀ is rather high and indicates an important degree of heterogeneity of the populations of *D. subobscura*. It is interesting to notice that the two species and the three populations are giving the same mean number of mutants.

2. The offspring of the 32 groups of ♀ (C) shows a lower number of mutants. The following two factors might be responsible for the lower mutation rate. Since several females had been placed together in each vial, the probability of the constitution of homozygote combinations for recessive mutations is smaller than in the offspring of single females with an obligatory brother-sister mating for the  $F_2$ . Modifiers may also partly be responsible for concealed genes in the population C. For these two reasons it seems plausible that the mutation rate is lower in population C, in spite of the presence of several ♀♀ in each vial, sharing together more mutants than the single ♀ per vial in populations A and B.

3. In the offspring of single males (D), crossed with females of another strain, we observe also a smaller number of mutants. The following three factors may be partially

responsible for the lower gene frequency in population D. The selection pressure against genes on the X-chromosome is much higher in  $\sigma\sigma$  than in  $\varphi\varphi$ , since these genes are in a hemizygous state and always expressed in  $\sigma\sigma$ , if there are no modifiers concealing them. In a natural population of *D. melanogaster* from Banyuls (Bösiger, 1962) the frequency of mutants is twice as high in  $\varphi\varphi$  than in  $\sigma\sigma$ . The greater fragility of  $\sigma\sigma$  could add to the lower mutant gene frequency. The effect of modifier genes, introduced by the crossing of the Samothrace  $\sigma\sigma$  with the foreign Küssnacht  $\varphi\varphi$ , may have prevented in some cases the phenotypic expression of present mutants.

4. The  $X^2$  test shows, that the observed distribution of frequencies of mutant genes fits well with a Poisson distribution for the populations B and C, at the 1% level only for population A and not at all for population D.

5. A preliminary morphological comparison of the mutant phenotypes found by Gordon, Spurway and Street (1939) in England, by Buzzati-Traverso (1941) in Italy and by Prevosti (1952) in Spain with those found in Greece shows that the great majority of the phenotypes are common in the four countries. The list and description of the mutants found in Greece will be given later.

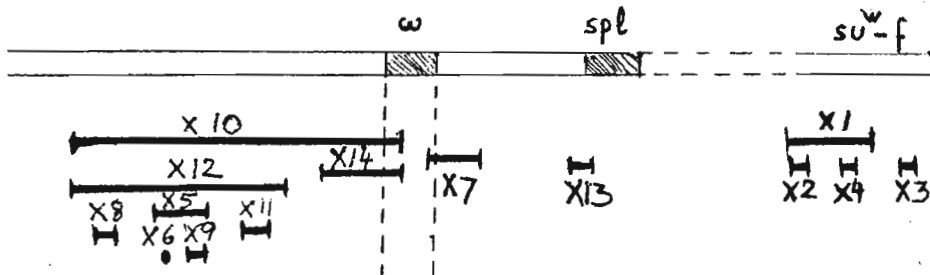
Bösiger, E., 1962, Bull. Biol. France et Belgique, 96:3-122. Buzzati-Traverso, A., 1941, Scient. Genetica, II:1-34. Gordon, C., Spurway, H. and Street, P., 1939, J. Genetics, 38:37-90. Prevosti, A., 1952, Genetica Iberica, 4:95-128.

Lifschytz, Eliezer. Hebrew University, Jerusalem, Israel. Induced X-chromosome lethals covered by  $Y \cdot w^+$ .

7100 y ac sc chromosomes were irradiated with a 2000 r dose of X-rays. 363 chromosomes carried lethal mutations. 14 of these lethals (3.9%) were covered by a  $Y \cdot w^+$  chromosome produced by Brosseau

et al, (1961).

The lethals covered by the  $Y \cdot w^+$  chromosome were crossed among themselves in all possible combinations in order to determine their allelism. Lethals that were suspected to be "point mutations" after the allelism test were checked further for crossing-over disturbance in the y-pn-w region. One group of lethals showed free recombination with the markers and proved to be proximal to f. They are also covered by  $Y \cdot ma-1^+$  and by  $Y \cdot B^S$ . These lethals were thus located in the most proximal region of the X-chromosome, and are probably covered by  $su^w-f$ . All the lethals except one showed disturbances in the crossing over frequency in their immediate vicinity. The following "complementation map" is consistent with the data collected so far:



Since we selected for lethals covered by the  $Y \cdot w^+$  compound, deficiencies extending beyond the region of the X-chromosome included in this compound were automatically excluded. This may explain the fact that only 3.9% of the lethals were found in a section comprising 10% of the cytological length of the X chromosome.

Most of these lethals do not disturb crossing-over between distant markers, such as y-sn, and would have been classified as "point mutations" by routine genetic procedures. These results suggest that many X-ray induced lethals that pass for "point mutations" are actually small aberrations.

Note that lethals X10, X14 and X7 are allelic to w, but X7 complements the lethal effect of both X10 and X14. This is in line with Lefevre's (1965) findings, and suggests that a deficiency for the w-locus is non-lethal.

We started to accumulate chemically induced lethals covered by  $Y \cdot w^+$  in order to compare the "complementation map" of lethals induced by the various mutagenic agents.

Forward, K. J. and B. P. Kaufmann. University of Michigan, Ann Arbor, Michigan. The bipartite nature of the salivary-gland X chromosome.

Sturtevant, Bibliog. Genet. 2, 1925). A cytological examination of third-instar larval salivary-gland chromosomes from the cross, *D. melanogaster*, Sw-b ♀ x *D. simulans* ♂ revealed that most individuals were female, although occasional males, having a single unpaired X chromosome, were detected. The diameter of this X usually equals or surpasses that of the paired autosomal homologues within the complex (as is shown on the accompanying photomicrograph), and may be assumed - as noted by Dobzhansky in *Chromosoma* 8, 1957, following a study of hybrids from the cross *D. insularis* x *D. tropicalis* - to present a "visible counterpart of the genetic phenomenon of dosage compensation." Spectrophotometric studies by Aronson, Rudkin, and Schultz (*J. Histochem. & Cytochem.* 2, 1954) indicated that in *D. melanogaster* the amount of DNA in the salivary-gland X chromosome of the male is about half as much as in the paired homologous X's of the female. A similar quantitative relationship is suggested by the faint stainability with acetic-orcein of the X in the *melanogaster-simulans* hybrid as compared with the more intense colorability of the arms of the autosomes (see photograph).

When *Drosophila melanogaster* females are mated with *D. simulans* males, the adult hybrid progeny are ordinarily female, although exceptional (non-disjunctional) males are obtainable (Sturtevant, *Genetics* 5, 1920; Morgan, Bridges and



We have observed that unpaired X chromosomes in third-instar salivary gland cells of *malanogaster-simulans* hybrid males vary greatly in length, breadth, and puffing pattern. The significance of these variations with respect to developmental processes will be discussed in another paper. What we would like to point out here is that the X, when well extended as shown in the accompanying photomicrograph, often reveals with striking clarity a bipartite structure (see intertwining strands at arrows). The X of the male fly has sometimes been designated as "unipartite," but it is apparent that a subsidiary order of organization can be recognized at the level of resolution afforded by the light microscope. By analogy, chromosomes composed of paired homologues should at times reveal a quadripartite structural pattern. That this is indeed the case was reported, for example, by Frolova for *D. robusta* (*Biolog. Zhurnal* 6, 1937), by Melland for a chironomid (*Proc. Roy. Soc. Edinb.*, B, 1942),

and has been observed occasionally by us in squash preparations of salivary-gland chromosomes of *D. melanogaster* (although more commonly the limits of the subsidiary units are masked by the close apposition of the strands that constitute the giant chromosomes). These microscopically discernible subunits assumedly reflect the basic pattern of organization of the chromosomes in the mitotically-active cells from which the salivary-gland progenitors are derived, and they undergo *pari passu* a series of endomitotic replications to produce the giant polytene chromosomes. (Supported by N.I.H. Grant GM-10499).

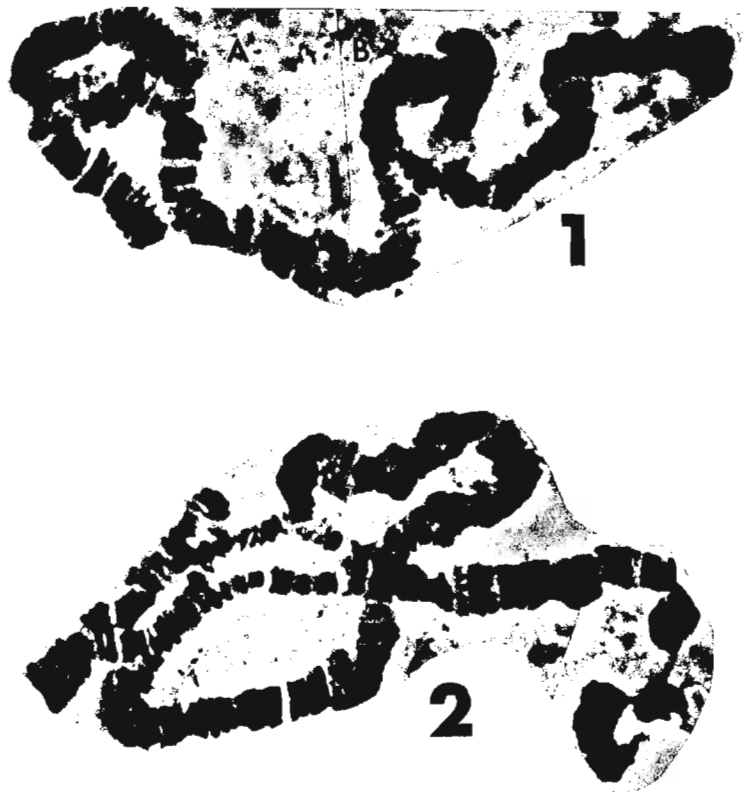
Miller, D. D., N. J. Westphal and R. A. Voelker. University of Nebraska, Lincoln, Nebraska. A preliminary note on gene sequence variation reinvestigation in the C chromosome of *Drosophila athabasca*.

Novitski (1946, Genetics 31:508) reported that the salivary gland chromosomes of *D. athabasca* revealed the C chromosome to be relatively variable and that certain of its sequences were restricted to the west, others to the east. Therefore, it seemed desirable to

reinvestigate gene sequence variation in this chromosome for a possible relationship between structural heterozygote configurations and the partially isolated western-northern subdivisions of the species, distinguished by Y chromosome type, duration of copulation, and pigmentation (Miller and Westphal, 1965, Genetics 52:459). Numerous C chromosome configurations have been observed, all evidently based on one or more inversions. Their analysis is incomplete, but early results seem significant.

Figure 1 (Raton Pass, New Mexico) shows two configurations that appear to be widespread in the western strains at our disposal but have not been found in the eastern ones. The small subterminal double configuration (1A) has been encountered in strains from British Columbia (Okanagan), Idaho (Boise), Minnesota (Hallock), New Mexico (Raton Pass), Oregon (Eel Creek, Eugene), and Washington (Sequim Bay). This resembles the M/O heterozygote configuration of Novitski (1946, Fig. 5A); coexistence of the M and O sequences was reported by him from British Columbia, Oregon, Washington, and Wyoming. The single submedian inversion configuration (1B) has been found in our strains from British Columbia (Okanagan), Minnesota (Duluth, Hallock), New Mexico (Raton Pass), Oregon (Eugene), and Washington (San Juan Islands). All these western strains are characterized by a Type I or Type V (Sequim Bay) Y chromosome, long copulation time, and dark pigmentation; they also demonstrated partial or complete failure to mate with laboratory strains of *D. athabasca* with Types II and III Y chromosomes. Other configurations found in these western *athabasca* strains were all relatively short and/or single inversion dependent.

None of the western configurations is clearly present in our eastern strains. Although these strains do have relatively small, simple inversion configurations, most also



have large ones that are quite complex. Figure 2 illustrates a double (overlapping) inversion configuration found in our strain from Jim Thorpe (Carbon County), Pennsylvania; one similar to this (if not identical) has been found in strains from Massachusetts (Amherst) and Ontario (Owens Sound). Large, complex configurations, covering most of the euchromatic arm and involving three, four, or five inversions have been found in strains from Massachusetts (Amherst), Minnesota (Lake Shamineau), North Carolina (Highlands), Ontario (Owens Sound), Pennsylvania (Jim Thorpe), and Vermont (Poultney). Of our eastern strains, only those from Oberlin, Ohio, and Philadelphia, Pennsylvania, have failed so far to reveal such large configurations. This may be significant inasmuch as these two eastern strains behaved differently from other eastern ones in sexual isolation tests, mating relatively easily with western strains but less so with other eastern ones (Miller and Westphal, 1965).

Recent contributions of wild flies and stocks by Dr. Max Levitan (Univ. Virginia, Fairfax) and Dr. D. L. Williamson (Woman's Med. Col. Pennsylvania, Phila.) are much appreciated. This work is supported by National Science Foundation Grant GB 5011.

Jost, P. University of Oregon, Eugene, Oregon. Observation of an unexpectedly high frequency of Bar revertants.

In an experiment involving females carrying a compound X chromosome, heterozygous for  $In(1)S$  and Bar, a  $sc^8 \cdot Y$ , and a compound fourth chromosome AF, (with no free 4) 11 out of 74 pair matings produced a single  $B^+$   $F_1$  female (total  $F_1$  females = 2403). The parental females were of the constitution  $RA, y^2 su-w^a w^a --- M-5/sc^8 \cdot Y; AF, ci ey^R/ci ey^R$ , where  $M-5 = Muller-5 (Ins(1)s sc^{SIL}, S, sc^{8R})$ , and were mated to males that also carried a compound-4.

The original stock, carrying free 4's and with no free Y, has not produced Bar revertants in the year and a half it has been maintained in this laboratory. George Brosseau, who supplied us with this particular stock, confirms that stocks of this chromosome, carried both with and without a  $sc^8 \cdot Y$  for several years in his laboratory, have not produced any  $B^+$  individuals detected in routine stock inspections.

The introduction of  $sc^8 \cdot Y$  and the compound 4, and removal of the free 4's, in our laboratory gave a stock that accumulated a high frequency of  $B^+$  females, necessitating periodic selection for B to maintain the original phenotype  $sc$ . This stock also shows a wide variability in the Bar phenotype, consistent with the presence of  $B/B$ .

Individual stocks were established from 7 of the original 11 independently arisen  $B^+$  revertants and an additional stock of  $B^+$  was established by culling  $B^+$  females from the B stock containing the compound 4. Salivary gland chromosome preparations of several larvae from each of these 8 stocks have been examined by George Brosseau in anticipation that some of the recombinational events involved would have resulted in a structurally homozygous compound X, i.e., a compound-X that either contained  $In(1)S$  in both X elements or lacked it in both elements. All of the preparations examined showed an inversion loop, indicating retention of the structural heterozygosity of the chromosome. If these revertants are the result of a 3-strand double crossover, then in all cases, both crossovers occurred distal to  $In(1)S$ , although it would be reasonable to expect that in some cases one of the crossovers would have occurred proximal to this inversion, yielding a compound homozygous for sequence in this central region of the X elements.

The presence of the compound 4 per se, or as an unpaired element (or the introduction of some unknown factor in stock synthesis) appears to produce an unexpected inter-chromosomal effect in addition to the increased recombination in the reversed acrocentric compound X expected from the presence of the  $sc^8 \cdot Y$ . Such an effect was not observed for homozygosis for w in experiments involving Muller's  $y f = (RA, In(1)d1-49, y w f -- In(1)sc^8? f sc^8.)$  that gave a total of 5803  $F_1$  females, although a single  $y w f$  female has been recovered from the stock bottles. (This derivative chromosome shows no inversion loop in salivary gland preparations.) Since homozygosis for w would involve a crossover between the centromere of the  $sc^8$  element and the w locus of the distal X, i.e., effectively a crossover between y and w, the rarity of this event is undoubtedly a reflection of the short map distance involved between markers, and may not indicate the absence of an inter-chromosomal effect associated with the presence of the compound-4.

Beatty, R. A. and N. S. Sidhu. Edinburgh University, Scotland. Spermatozoan nucleus length in minutes of *D. melanogaster*.

Six minute stocks originating from and continuously back-crossed to the Pacific mass-mated strain were obtained from Dr. F. W. Robertson. Spermatozoan nucleus length was

measured in aceto-orcein preparations under a projection microscope. The sampling was: 10 spermatozoa per seminal vesicle, 2 seminal vesicles (1 preparation from each) per male, 5 M/+ and 5 +/+ males per stock (1 culture bottle per stock; the individuality of a culture bottle is known to be unimportant). Detailed procedures are given by Sidhu (1963 Ph.D. Thesis, Edinburgh University, U.K.). Various analyses showed no significant levels of differences among the 12 means (in microns) of the following Table, where the S.E. per mean is  $\pm 0.23$  (48 d.f.). Overall means are 8.41 (M/+) and 8.25 (+/+), the non-significant

Genotype	Stock $\rightarrow$	M(2)17	M(2)18	M(2)20	M(3)11	M(3)12	M(3)14
M/+		8.08	8.42	8.56	8.47	8.64	8.30
+/+		8.10	8.01	8.71	8.34	8.38	7.98

difference being  $0.16 \pm 0.13$  (48 d.f.). The subsampling mean squares were: spermatozoa within seminal vesicles, 0.84; seminal vesicles within males, 3.66 (significant,  $P < .001$ ); males within groups of the Table, 5.31 (48 d.f.) (not significant relative to preceding mean square). Necessarily, the one significant level concerns variation between seminal vesicles and between preparations. Comparable analyses of the variance within seminal vesicles indicated homogeneity throughout the whole sampling structure; pooled variances were 0.9 for M/+ and 0.8 for +/+.

The possibility of a "haploid genetics", envisaging phenotypic effects on a spermatozoon according to its own haploid genetic content, is supported in mammals by the results of Braden (1960, J. Cell. comp. Physiol., 56 (Suppl. 1), 17) and Bhattacharya (1962, Zeit. Wissenschaft. Zool. 166:207). Other work in mammals, centering on genetic factors known to affect the diploid cell phenotype, has not revealed definite "haploid effects" in spermatozoa. The present work extends the enquiry to *Drosophila*, minutes being chosen because their well-known effect of reducing diploid cell size might be reflected in reduced spermatozoan nucleus size, and the variance of spermatozoa within seminal vesicles might be greater in M/+ males (segregating two kinds of spermatozoa) than in +/+ (segregating one kind). The results show, however, with some precision, that no effect on spermatozoon nucleus length is attributable to the minute factors, either severally or collectively. It would be logical to study a more direct measure of spermatozoon size, such as total spermatozoon length, but technical difficulties exist.

Kumar, Sushil, R. P. Sharma and M. S. Swaminathan. Indian Agricultural Research Institute, New Delhi, India. Chromosomal rearrangements resulting from action of a monofunctional alkylating derivative of acridine in salivary gland chromosomes of *Drosophila melanogaster*.

Mutations induced by the monofunctional nitrogen mustard derivative of acridine, 2-methoxy-6-chloro-9-(3-(ethyl-2-chloro-ethyl) aminopropylamino) acridine dichloride (ICR-170) in *Drosophila* and *Neurospora* have been found not to involve chromosomal deletions (Carlson and Oster, Genetics 47:561, 1962, and Brockman and Goben, Science 147:750, 1965). In tests

on *Vicia faba* chromosomes, ICR-170 has been shown to be a potent chromosome breaking agent inducing chromatid and isochromatid deletions, and subchromatid, chromatid and chromosome exchanges (Kumar, Aggarwal and Swaminathan, Mutation Research in press). The present experiments show that inversions result in the salivary gland chromosomes of *Drosophila* after treatment with ICR-170.

Eggs of Oregon-K strain were laid on the ICR-170 medium (200  $\mu$ g of ICR-170/ml mixed with basic medium, (agar, 3%; yeast, 10%; glucose, 10%; propionic acid, 0.4%; water, 100ml), 1:1). Third instar larvae from this medium were harvested for studying salivary gland chromosomes. Four spontaneous and 52 ICR-170 induced inversions were recovered in 500 cells scored for control and treated larvae. Most of the inversions were complex. Treated chromosomes were puffed. The concentration of ICR-170 used in these experiments yielded about 6% sex-linked lethals.

Cole, Thomas A. Wabash College, Crawfordsville, Indiana. Alcohol dehydrogenases in the pupae of *D. melanogaster* (Oregon R-C).

Alcohol dehydrogenases of carefully aged pupae of *Drosophila melanogaster* (Oregon R-C) were studied by polyacrylamide gel electrophoresis in small tubes (disc electrophoresis). This method reveals

that alcohol dehydrogenase is a highly polymorphic enzyme. A total of fourteen different types have been detected. Nine different bands have been identified with eight bands being the largest number in any one type and two being the smallest number. This system in Oregon R-C, when studied with disc electrophoresis, appears to be more complex than the alcohol dehydrogenase system in other strains as reported in the literature. (Supported by Research Grant GM-11860, United States Public Health Service)

MacIntyre, Ross J. and M. R. Dean. Cornell University, Ithaca, New York. In vitro dissociation and reconstitution of acid phosphatase-1 from *D. melanogaster*.

Zymograms of heterozygotes containing alleles specifying acid phosphatase-1 (Acph-1) molecules with different electrophoretic mobilities show three bands or zones of activity (MacIntyre, Genetics 53: 461). This probably means that the enzyme,

in its active form, is at least a dimer. However, in order to verify the multimeric nature of this enzyme and to obtain some information about the bonding between the polypeptide subunits, we attempted to reversibly dissociate the extracted enzyme. These attempts, which utilized a variety of known dissociating agents, were unsuccessful until the enzyme was partially purified by ammonium sulfate fractionation. The procedure is outlined in Figure 1.

Figure 1

1. Ten grams (wet weight) of flies homogenized in 50 ml 0.025 M Citrate-phosphate buffer, pH 7.0, with 0.5 g Norit A.
- ↓
2. Homogenate centrifuged 45 minutes (27,000 x g).
- ↓
3. Supernatant dialyzed 16 hours (4°C) against 0.025 M Citrate-phosphate buffer, pH 5.2, containing  $4 \times 10^{-4}$  M EDTA.
- ↓
4. Dialysate centrifuged 30 minutes (27,000 x g).
- ↓
5. Dry  $(\text{NH}_4)_2 \text{SO}_4$  added to supernatant to bring to 60% of saturation (calculated for 0°C). Stirred at 4°C for 30 minutes.
- ↓
6. Centrifuged 20 minutes (27,000 x g).
- ↓
7. Precipitate suspended in 20 ml of 0.025 M Citrate-phosphate buffer, 45% saturated with  $(\text{NH}_4)_2 \text{SO}_4$ . Stirred at 4°C for 1 hour.
- ↓
8. Centrifuged 20 minutes (27,000 x g).
- ↓
9. Supernatant dialyzed successively against distilled water for 4 hours and against 0.05 M Na Cl for 10 hours.

This results in about a 40 - 50 fold increase in the specific activity of acid phosphatase. Enzyme activity was measured during purification and in these experiments by the spectro-

photometric determination of *p*-nitrophenol released by the enzymatic hydrolysis of *p*-nitrophenyl phosphate at pH 5.0 (Bergmeyer, *Methods of Enzymatic Analysis*, p. 783). The ability of Acph-1 enzymes to hydrolyze this substrate was demonstrated by developing zymograms in stain solutions containing *p*-nitrophenyl phosphate,  $\text{Mg Cl}_2$ ,  $\text{Pb (NO}_3)_2$  and  $(\text{NH}_4)_2 \text{S}$  (Allen and Hyncick, *J. Histochem. Cytochem.* 11:169).

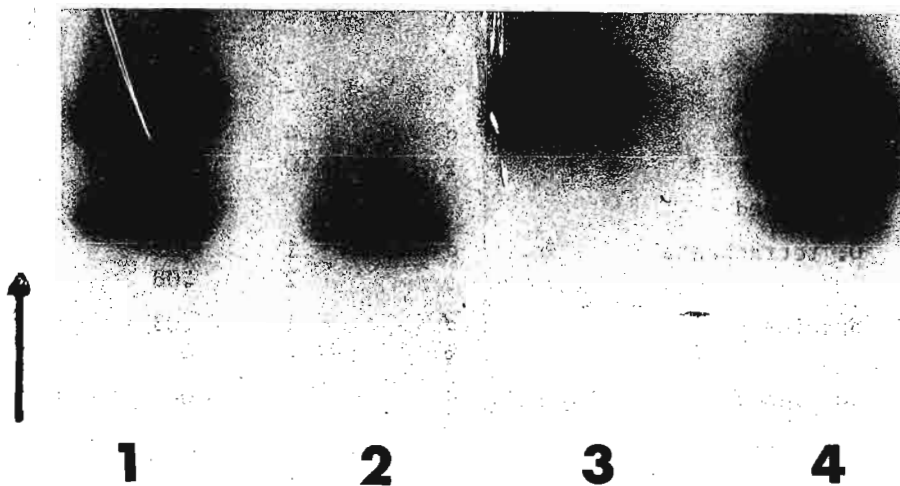
It was found that acid phosphatase activity of partially purified preparations from stocks monomorphic for the "slow" electrophoretic variant (Acph-1<sup>AA</sup>) decreases sharply between pH 2.9 and 2.5 when dilute HCl is added. No detectable activity was found in preparations below pH 2.5. On the other hand, the acid phosphatases in preparations from stocks monomorphic for the "fast" variant (Acph-1<sup>BB</sup>) are more susceptible to hydrogen ion concentration. Activity begins to decrease at pH 3.3 and is gone at pH 2.9.

When partially purified and acid-inactivated extracts are dialyzed against or diluted in various buffers between pH 6.0 and 7.5, some acid phosphatase activity can be regained. Tris-maleate at pH 6.5 seems to be the best buffer for the recovery of activity from acid-inactivated preparations. At best, however, only about 30% of the activity of the untreated control has been obtained.

If solutions of acid-inactivated extracts from stocks monomorphic for Acph-1<sup>AA</sup>, Acph-1<sup>BB</sup> and a mixture of the two are dialyzed (72 hours, 4°C) against Tris-maleate buffer at pH 6.5, concentrated and electrophoresed, the patterns shown in Figure 2 are obtained. This zymogram clearly shows that the electrophoretic mobilities of the reconstituted Acph-1<sup>AA</sup> and Acph-1<sup>BB</sup> enzymes do not differ from those of the untreated molecules. In addition and most significant is the appearance of the "hybrid" enzyme or Acph-1<sup>AB</sup> in the treated extract of a mixture of flies from stocks monomorphic for the two electrophoretic variants, Acph-1<sup>AA</sup> and Acph-1<sup>BB</sup>. It seems certain, then, that acid-inactivation of Acph-1 involves dissociation into inactive but intact polypeptide subunits, and, with the increase in pH, reactivation is brought about by the reaggregation of these subunits into enzymatically active molecules. Furthermore, since high hydrogen ion concentrations alone will dissociate Acph-1, there are probably no disulfide bonds holding the subunits together (Schachman, *Cold Spring Harbor Symp. Quant. Biol.* 28:409).

Figure 2

Zymogram of reconstituted Acph-1 enzymes. Slot 1 is an untreated, partially purified extract of a mixture of flies from stocks monomorphic for Acph-1<sup>AA</sup> and Acph-1<sup>BB</sup>. Slots 2 - 4 are inactivated, reconstituted and concentrated extracts of flies from stocks monomorphic for Acph-1<sup>AA</sup> (slot 2), Acph-1<sup>BB</sup> (slot 3) and Acph-1<sup>AA</sup> and Acph-1<sup>BB</sup> together (slot 4). Treatment consisted of the fol-



lowing steps. Ten ml of the partially purified extracts were taken to pH 2.4 by adding 0.1 N HCl. After 30 minutes at 0°C, the extracts were assayed to confirm the absence of acid phosphatase activity. They were then dialyzed at 4°C first for 72 hours against 0.1 M Tris-maleate buffer at pH 6.5 and then for 4 hours against the same buffer containing 15% (by weight) polyethylene glycol (carbowax 4000). The final volume of each extract was about 2 ml. The arrow indicates the direction of migration.

Angus, D. University of Queensland, Brisbane, Australia. *Drosophila* collection from British Solomon Islands Protectorate and the Territory of Papua-New Guinea.

The following are the results of three surveys of the genus *Drosophila* attracted to banana baits placed on the forest floor at 1. Mount Austen, 7 miles S-E of Honiara, Guadalcanal; 2. In rubber plantation, Bisianumu, 25 miles

east of Port Moresby, T.P.N.G.; 3. In Teak plantation, near Brown River, 20 miles north of Port Moresby, T.P.N.G. Flies were collected 2 days after placing the baits. The results are based on approximately one tenth of the total catch at each site during February 1966.

Species		Mt. Austen		Brown River		Bisianumu	
		No.	%	No.	%	No.	%
<i>D. ananassae</i>	♂	833	31.4	499	31.4	164	22.0
<i>D. serrata</i>	♂	-	0	22	1.38	5	.67
<i>D. szentivanni</i>	♂	-	0	13	.82	4	.54
<i>D. melanogaster</i> group	♀	1007	38	759	47.7	330	44.4
<i>D. rubida</i>	♂	156	5.9	20	1.3	25	3.36
	♀	122	4.6	18	1.1	13	1.75
<i>D. setifemur</i>	♂	252	9.55	102	6.45	62	8.34
	♀	263	9.95	115	7.28	67	9.01
<i>D. pararubida</i>	♂	-	0	1	.06	12	1.62
	♀	-	0	2	.126	15	2.02
Sub-genus <i>Pholadoris</i>							
Species A	♂	-	0	3	.189	1	.013
	♀	-	0	9	.568	1	.013
Species B	♂	-	0	1	.063	-	0
	♀	-	0	6	.378	1	.013
Species C	♂	-	0	7	.441	5	.673
	♀	-	0	4	.252	2	.027
Species D	♂	4	.15	-	0	-	0
	♀	10	.38	-	0	-	0
Ungrouped							
<i>D. tetrachaeta</i>	♂	1	.03	-	0	1	.013
	♀	2	.08	-	0	-	0
Species E	♂	1	.03	2	.126	1	.013
	♀	-	0	-	0	-	0
Species F	♂	-	0	2	.126	15	2.02
	♀	-	0	-	0	5	.673
Species G	♂	-	0	-	0	7	.945
	♀	-	0	-	0	6	.807
Species H	♀	-	0	-	0	1	.013
Species I	♀	1	.03	-	0	-	0
Total		1587	100	745	100	2652	100

Rainfall for	-	+Mt. Austen	++Bisianumu
January 1965		18.75 ins.	6.88 ins.
February 1965		14.67 ins.	15.63 ins.
January 1966		1.08 ins.	9.30 ins.
Feb. 1-16 1966		0.12 ins.	9.27 ins.

Continued on next page.

Figures supplied by +Department of Forests B.S.I.P., and ++Department of Agriculture Stock and Fisheries Rubber Research Station Bisianumu T.P.N.G.

It was noted that Mount Austen had received less than 1/10 of its average rainfall during the 1966 wet season. It is suggested that the relatively poor species diversity found in that area was related to the dry conditions.

Further, only one specimen of *D. tetrachaeta* was recorded from 18,000 flies caught at Bisianumu and none was found at Brown River. Unpublished records show that this species is common in previous catches made in this season at these places. A prolonged dry period occurred during the seven months from May to November 1965. Only 18.11 inches of rain fell at Bisianumu - the wettest part of the Port Moresby area. It is suggested that *D. tetrachaeta* is dependant on factors particularly related to rainfall to maintain its populations.

The range of *D. tetrachaeta* has been extended to Guadalcanal.

Malich, C. W. and R. M. Binnard. NASA Ames Research Center, Moffett Field, California. Variations in the transmission of broken chromosomes of *D. melanogaster*.

The dominantly marked Y chromosome  $y^+ \cdot Y \cdot B^S$  of G. Brosseau has been used by a number of workers to study chromosome breakage and loss, by inspection of adult *Drosophila* for transmitted markers. Chromosome breakage may

result in bridge formation causing cell death at division, eliminating the damaged nucleus. Selective killing can thus affect the observed rates of transmission of the markers in such an experiment. We have used the  $y^+ \cdot Y \cdot B^S$  chromosome as incorporated by I. I. Oster in one of his multipurpose stocks to compare the breakages induced by irradiation with a variety of heavy particles. Males with the treated Y are routinely crossed to females with rod X chromosomes homozygous for yellow. There has been little variation among the particles in the apparent losses of one or both markers using this stock. The results tabulated for alpha particles are typical.

Recently we increased the fertility of the exceptional flies by crossing the treated males to special females having both arms of the Y attached to the X chromosome:  $Y^S \cdot X \cdot Y^L$ , having no free Y. The rate of exceptional flies increased as well as the fertility. The results listed in the table show no significant change in the rate of loss of single markers for either X-rays or alpha particles. Loss of both markers has almost doubled with this new stock. The ratio of double marker losses to single marker losses shown in the last column shows with greater precision the similarity between types of radiation and the variation between the two stocks.

Table 1. Transmission of chromosomes broken by different radiations. Rates are the total observed ones, including induced and spontaneous losses.

Radiation	#F <sub>1</sub> ♂	No. of Losses			Rate of Losses X10 <sup>6</sup>			Ratio of Double to Single Losses
		y <sup>+</sup>	B <sup>S</sup>	Both	y <sup>+</sup>	B <sup>S</sup>	Both	
REGULAR STOCK								
Alpha	30,288	45	138	268	0.8	2.3	4.5	1.47
x-ray	27,102	49	145	301	0.8	2.3	4.7	1.55
SPECIAL STOCK (Y <sup>S</sup> •X•Y <sup>L</sup> , no free Y)								
Alpha	3,534	9	26	90	0.9	2.6	9.1	2.57
X-ray	8,263	20	45	184	0.9	2.0	8.0	2.83

Fertility with the special stock is 72% for loss of  $y^+$  (21 of 29 tests), 87% for loss of  $B^S$  (61 of 70 tests), and 94% for loss of both markers (245 of 260 tests). Loss of both markers is usually taken as an indication of loss of the whole Y, so the lower fertility of single marker losses with the  $Y^S \cdot X \cdot Y^L$  chromosome containing all fertility factors could indicate selective killing of cells with a broken fragment. However, cytological studies of 9 of the double marker losses disclose a more likely cause for the higher rate of transmission. Five of the 9 flies had ring Y chromosomes, far more than the usual fraction found with the standard stock. Only 1 of these ring Y chromosomes is fertile when crossed to standard stock. The other four ring Y's must contain deletions

which are lethal unless covered by the Y in the  $Y^S \cdot X \cdot Y^L$  chromosome. Since these are nearly half of the small sample of flies tested cytologically, we conclude that most if not all of the doubling in rate of the double losses is due to these normally lethal ring Y chromosomes being transmitted in the special cross. This illustrates one problem of germinal selection even at low induced rates.

Keller, E. C. Jr., H. E. Keller and E. Liner. University of Maryland, College Park, Maryland. Heterosis in xanthine dehydrogenase activity levels.

Males of ten highly inbred wild-type strains of *D. melanogaster* and their  $F_1$  interstrain hybrid progeny were tested for their respective levels of xanthine dehydrogenase (XDH) activity. The ten strains were collected in 1954 from the

same locality and have been previously classified as within the "normal" XDH activity range (Keller, 1964). The method of assay was the fluorometric technique similar to that described by Keller and Glassman (1964). Progeny from the two genetic types ( $F_1$  interstrain progeny and parents) were reared at two temperatures (18°C and 26°C) with the same parents being used for both temperature experiments. The total sample size of the balanced experiment was 604 flies.

Table. Average Xanthine Dehydrogenase Activity Levels and Variances for Ten Wild Type Inbred Strains and Their  $F_1$  Progeny.

Genetic Type	TEMPERATURE				Average
	18°C		26°C		
	Average	Variance	Average	Variance	
Inbred Parents	4.03	0.542	3.16	1.463	3.59
F <sub>1</sub> Progeny	4.22	0.802	3.86	1.608	4.04
Average	4.12	-----	3.51	-----	3.82

The Table shows the mean XDH activity levels and their respective variances (for the experimental groups only) over the two temperature conditions for the two genetic types. On the average, the  $F_1$  progeny showed significantly greater XDH activity than their parents (at both temperatures). Also, there was a greater activity differential between the  $F_1$ 's and their parents at 26°C than at 18°C. One-tailed unpaired "t" tests were used, within temperatures, to complete the statistical tests. Preliminary tests showed that the  $F_1$  progeny always had greater XDH activity levels than their inbred parents. The "t" value for the difference between the means of the parents and their offspring was 3.74 (285 d.f.) at 26°C and "t" = 1.75 (314 d.f.) in the 18°C experiment. The former "t" value is significant beyond the 0.1% probability level, the latter "t" value is significant beyond the 5% probability level. Examination of the average XDH activity levels at different temperatures revealed that flies reared at 18°C showed significantly greater enzyme activity than those

flies reared at 26°C. This difference could be due to the significant average size difference that exists between the different temperature-reared flies (similar to that XDH activity sex difference that exists in *D. melanogaster*, Keller, 1964). This temperature difference could also be due to certain developmental homeostatic mechanisms.

Within the temperatures there was a very strong familial association of the XDH activity levels (specific combining abilities) however, the data as reported here completely confound these family differences and only the overall averages are reported. A complete diallel analysis is currently being completed and will be reported later.

As seen in the Table the variances of the XDH activities among the 18°C reared flies (within genetic type) were significantly lower than those of the 26°C reared flies for both the inbred parent and the  $F_1$  progeny groups. The variance ratios (F values) for the differences between the variances of the temperature groups were  $F = 2.702$  (significant beyond the 1% probability level) for the inbred parents and  $F = 2.005$  (significant beyond the 1% probability level) for the two  $F_1$  groups. Further, the variances of the  $F_1$  progeny, at both temperatures, might have been greater than the variances of the parents. In this experiment, the differences between the variances of the XDH activity of the parents were not significantly different from the variances of the  $F_1$  progeny, at either temperature.

The significance of these findings include: first, that there are significant average "heterotic" effects in XDH activity among the  $F_1$  progeny of certain inbred strains of *D. melanogaster*. Secondly, there appears to be a developmental homeostatic phenomenon that is highly associated with developmental speed (or temperature). Thirdly, the variation among the XDH activity of  $F_1$  progeny of inbred strains might be greater than that variation present among the inbred parents. Of specific interest in this respect is the fact that the variances in the  $F_1$  progeny were not less than that of the parents. However, this could be a simple consequence of the greater average XDH activity of  $F_1$  progeny which might have been predicted under the theory of genetic homeostasis. (Supported by USAF/OSR Contract #AF 49(638)-1603).

Waldner-Stiefelmeier, R. and P. S. Chen.  
University of Zürich, Switzerland. Proteolytic digestive enzymes in *D. mel.*

In connection with our studies on the biochemical effects of lethal factors in *Drosophila*, analyses have been carried out on the proteolytic digestive enzymes in both the wild type and lethal mutants.

Using azocasein as substrate at an assay temperature of 38°C, maximum digestion was found to occur in the alkaline range at pH 8.3. When azoalbumin and haemoglobin were used instead of azocasein, no enzyme activity could be detected in the acid range from pH 1.0 to pH 5.8. Extracts of wild type midgut from 4-day-old larvae show the highest value, but homogenates of whole larvae indicate a distinct drop from the 3rd to 4th day, apparently due to the rapid increase of unspecific proteins in the haemolymph and tissues at this later period of larval life. With the beginning of pupation the activity rapidly declines to a hardly detectable low level, but rises again shortly before adult emergence. Thus the enzyme activity during pupal development follows a U-shaped curve. In female adult flies maximum digestion has been observed at about 24 hrs. after emergence, corresponding to the period of intensive ovarian growth. The maximum for male flies also occurs at about the same time, but the absolute values amount to only 30% of those for females. Furthermore, we found that after a starvation period of 6 and 8 hrs. the enzyme activity was reduced to 75-89% of the normal level in fully grown larvae, and to 45% in the 3-day-old females flies. However, it seems that starvation has no effect on the digestive ability of adult males at similar ages.

In order to analyse the individual proteolytic components, midgut extracts from 4-day-old wild-type larvae were incubated with various synthetic substrates. The presence of trypsin is suggested by the hydrolysis of N-benzoyl-L-arginine ethylester. Since L-tyrosine ethylester is not attacked, chymotrypsin is probably absent. Likewise the hydrolysis of both N-carbobenzoxycyl-L-phenylalanine and L-leucine- $\beta$ -naphthyl-amide demonstrates the occurrence of carboxypeptidase A and aminopeptidase respectively, whereas the lack of activity for N-benzoylglycyl-L-lysine indicates that carboxypeptidase B is not involved. By means of polyacryl-amide gel electrophoresis we found that the mobility of the tryptic component in *Drosophila* differs from that of bovine trypsin, suggesting that the structure of this insect enzyme is not the same as that of vertebrates. These results provide evidence that the

pattern of proteases in *Drosophila* is heterogeneous, and at least 3 components contribute to the total proteolytic activity observed.

Based on azocasein hydrolysis the proteinase activity in the lethal lme/lme larvae aged 3-4 days has been determined to be only 48-58% of that in the wild type of corresponding ages. This is in agreement with our previous findings from both in vitro experiments (Chen and Hadorn 1955) and histo-chemical analyses (Meyer-Taplick and Chen 1960). On the other hand, the enzyme activity in 4-day-old homozygous larvae of the lethal mutant ltr has been found to be as high as 95% of the normal value. This indicates that the deficiency of proteolytic digestive activity of the mutant lme is probably locus-specific.

Shima, T., A. Kaneko and E. Momma.  
Hokkaido University, Sapporo, Japan.  
On some aspects of the copulation, insemination reaction and sperm storage in two species of quinaria group.

The first mating between virgin females and males of *D. brachynephros* and of *D. unispina* were observed during the period from the 4th day to the 12th day after the emergence, at the temperature of 20°C. Mating occurred in most cases on the 8th day. Copulation times were recorded for

100 pairs in the two species. The average time was 9 minutes and 36 seconds (ranged 5' 10" - 11' 42") for *D. unispina*, and 5 minutes and 6 seconds (ranged 3' 43" - 6' 51") for *D. brachynephros*. The reproductive organ was dissected out in a saline solution under the binocular microscope. A total of 998 females (478 for *D. unispina* and 420 for *D. brachynephros*) was dissected at varying times, starting immediately after copulation and extending through for about 40 days. The results of insemination reaction and sperm storage are summarized in the table. The evidence presented suggests that both species belong to a group of species which develops a large insemination reaction in homogamic matings (Wheeler, 1947).

Table 1. Insemination reaction and survival of sperm within the storage organs of females in *D. unispina* and *D. brachynephros*.

Time of dissection	D. unispina					D. brachynephros				
	Sperm storage					Sperm storage				
	insemination reaction	uterus	seminal receptical	spermathecae	remarks	insemination reaction	uterus	seminal receptical	spermathecae	remarks
immediate	a	+++	-	-	A	a	+++	-	+	A
2-minutes	b	+++	+	+	B	b	+++	+	+	B
10-minutes	c	++	+	++		c	++	+	++	
1-hour	d	+	+++	+++	C	d	+	+++	+++	C
3-hours		+	+++	+++			+	+++	+++	
6-hours	e	-	+++	+++			+	+++	+++	
7-hours		-	+++	+++	D	e	-	+++	+++	
9-hours		-	+++	+++			-	+++	+++	D
5-days		-	++	++	F		-	+++	+++	E
10-days		-	++	++			-	++	++	F
20-days		-	++	++			-	+	+	G
30-days		-	+	+	G		-	-	+	I
40-days		-	+	+	H		-	-	-	J

+++ : large amount of sperm, ++ : less sperm, + : few sperm, - : no sperm. a : beginning to enlarge. b : reaction mass in uterus c : small mass opaque. d : maximum size. e : mass reduced. A : end of coitus. B : highly motile sperm. C : both organs full of sperm. D : uterus normal. E : sperm few reduced in both organs. F : sperm reduced in both organs. G : sperm more reduced in both organs. H : very few sperm in both organs. I : no sperm seminal receptable. J : all gone.

David, J. and M. F. Clavel, University of Lyon, France. Variations of the rate of egg chambers production in the ovarioles of *Drosophila melanogaster*.

According to the strain, the daily egg production of young females ranges from 40 to more than 100. The ovariole number, for both ovaries, is between 40 and 60 in the great majority of strains.

To estimate the rate of egg chamber production in each ovariole, it is possible to divide the daily egg laying by the total ovariole number. This calculation gives a highly variable result, ranging from less than 1 to a little more than 2 egg chambers per day. But this estimation is often biased by the frequent occurrence of non-functional ovarioles which do not produce eggs. Their occurrence is particularly striking in strains with poor fecundity and in old females.

For a correct estimation, only the functional ovarioles are taken into account. These ovarioles, in active vitellogenesis, may be recognized by dissection, and it is then possible to get a fairly stable value for the rate of egg chamber production in all strains of *Drosophila melanogaster*.

Another more reliable way of estimation is to use a strain in which the ovarioles are all functional. This is generally the case for highly heterozygous flies in their first 10 days of life. Using such heterozygotes, it is possible to get an accurate estimation of the rate and to study its eventual variations. Some results, obtained at a temperature of 25°, are presented here.

The normal rate: The normal rate was estimated in 8 independent experiments with highly heterozygous animals fed on an axenic killed yeast medium (1). The results are presented in table 1. All values lie between 1.81 and 2.29, giving a mean of  $1.96 \pm 0.06$ . This is in agreement with a previous result from a vestigial strain (2) and with the latest value calculated by King (3).

Table 1: Influence of yeast autolysate on the daily rate of egg chamber production per ovariole.

Experiment No.	mean daily egg production (from 3rd to 8th day of life)		mean ovariole number		daily rate of egg chamber production		
	I	II	I	II	I	II	difference II - I
1	95.1	105.0	41.5	44.0	2.29	2.39	+ 0.10
2	93.3	100.6	47.5	41.0	1.96	2.45	+ 0.49
3	83.6	94.7	46.0	43.5	1.81	2.17	+ 0.36
4	84.5	102.5	45.5	48.0	1.85	2.14	+ 0.29
5	87.0	92.3	44.2	42.5	1.97	2.14	+ 0.17
6	88.4	99.6	46.0	45.7	1.92	2.18	+ 0.26
7	86.5	100.1	47.3	42.0	1.83	2.38	+ 0.55
8	94.6	103.5	45.7	46.7	2.07	2.21	+ 0.14
means	89.1	99.8	45.5	44.2	1.96	2.26	+ 0.30

I. *Drosophila* fed with the standard, killed yeast medium; II. *Drosophila* fed with the same medium supplemented with yeast autolysate. For each experiment, the fecundity of 4 females was studied on each medium during 9 days.

Diminution of the rate of egg chamber production: The rate possibly decreases as females become older (2). But this effect, if significant, is not very important.

In experiments where flies were fed with a folic acid antagonist, the rate of egg chamber production was clearly slowed down (4). This effect, which seems to concern primarily the follicular cells, results finally in a complete cessation of egg chamber formation.

Increase in the rate of egg chamber production: When the surface of the normal

medium is covered with a solution of yeast autolysate, the fecundity of the flies is improved. In heterozygous females, the rate of egg chamber production appears to be increased by this addition of yeast autolysate, as is shown in table 1. The mean rate becomes  $2.25 \pm 0.05$  and the difference between normal and autolysate fed females ( $+ 0.30 \pm 0.06$ ) is highly significant.

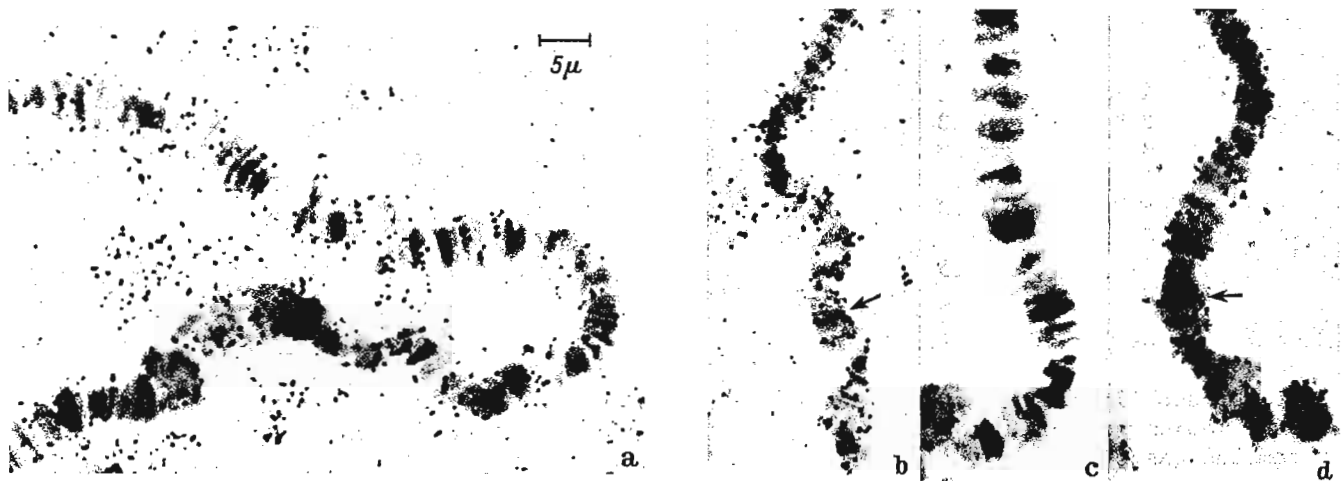
Conclusion: If only the functional ovarioles are considered, the rate of egg chamber production is fairly constant for all strains of *Drosophila*. But accurate measurements of this character demonstrates some variation, either below or above the normal rate. These variations could be an interesting test character in nutritional studies.

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Berendes, H. D. Max Planck Institut für Biologie, Tübingen, Germany. Amino acid incorporation into giant chromosomes of *D. hydei*.

Staining salivary gland chromosomes with Fast green at pH 2.14 revealed that normally occurring puffs as well as those produced by a temperature shock contain a high amount of proteins which are absent in the non-puffed condition of these

regions. Various tritiated amino acids were injected into larvae to trace their incorporation into the chromosomes and the possible relation of protein synthesis to the origin of puffs. The following experiments were performed:  $1 \mu\text{l}$  of tryptophan  $\text{H}^3$  (Spec. act.  $3.02 \text{ C/mM}$ ) was injected into 140 hour old larvae. The larvae were kept at  $25^\circ\text{C}$  for 10, 30, 60 minutes or 5 hours and then transferred to  $35^\circ\text{C}$  to induce temperature specific puffs. After the temperature treatment of 15 minutes the salivary glands were dissected, autoradiographs prepared and the number of grains occurring over the induced puffs was compared with that over a neighboring non-puffed part of the chromosome. No preferential labeling of the induced puffs was ever observed. In many cells the number of grains over the puffed region was even lower than over the neighboring part of the chromosome. In many cases, however, certain unpuffed bands showed a reproducible preferential labeling (see arrow in b and d). Naturally occurring puffs were studied similarly. Larvae of 145 hours old were injected with the



- part of chromosome 4 labeled with tryptophan  $\text{H}^3$  after 5 hours of incubation. The puffs showed no preferential uptake of the amino acid.
- histidine label over a part of chromosome 5 in a proximal cell of the salivary gland after 15 minutes of incubation.
- the same part of chromosome 5 in a distal cell of the same gland as in b.
- the same part of chromosome 5 as shown in b in a distal cell of the salivary gland after 5 hours of incubation.

labeled amino acid and the salivary glands were dissected after 8 or 12 hours of incubation. None of the puffs specific for puparium formation which arise during the period of incubation showed a preferential uptake of the labeled amino acid (see a). Similar results were obtained with proline, histidine, leucine and arginine. On account of these data it is clear that the regional increase in protein content during puffing is not caused by a synthesis in the puff itself, but is presumably due to an accumulation of pre-existent proteins from someplace in the cell. It is also conceivable that these proteins cannot be synthesized in the cell during the 15 minute temperature treatment since they appear as soon as swelling of the regions occurs. It might be suggested that the puff protein has a uniform composition in all puffs and has some bearing on the structural constitution of the puff and/or is involved in transport of the RNA produced.

A comparison of glands dissected from larvae 15 minutes after injection of either tryptophan or histidine with those dissected 5 hours after injection showed that in the first series of glands the cytoplasm and the chromosomes of the proximal cells was far stronger labeled than in the distal cells (see b and c). After an incubation period of 5 hours the difference in labeling intensity between the two types of cell was less obvious (see d). In most cells a positive correlation between the labeling density of the cytoplasm and of the chromosomes was observed. Furthermore, the possibility of a correlation between chromosomal protein synthesis and replication was studied. Larvae of 136 hours showed amino acid labeling in 100% of the salivary gland nuclei after 5 hours of incubation. After the same period of incubation with thymidine  $H^3$  only 37 - 46% of the nuclei of similar glands were labeled. It therefore might be suggested that there is no apparent correlation between chromosomal protein synthesis or accumulation and chromosomal replication.

Duffy, John P. and John Stiles Jr.\* St. John's University, Jamaica, New York. Esterase 6<sup>S</sup> isozyme in alaful-1 and gluful-1 mutants of *D. melanogaster*.

Starch gel electrophoresis (per method of Beckman and Johnson 1964, Wright 1963) was performed on 3rd instar larval homogenates of alaful-1 and gluful-1 along with the 3rd larval homogenates of either Est 6<sup>S</sup> or Est 6<sup>F</sup>, in order to compare the esterase

isozymes present in these amino acid mutants. It was found that the esterase of the alaful-1 and gluful-1 mutants electrophoretically migrated to the same positions as the Est 6<sup>S</sup> mutants. Using 3rd instar larval homogenates of the Est 6<sup>I</sup> mutant (*D. simulans*) (Wright and Mac Intyre 1963), we compared the esterases of alaful-1 and gluful-1 with Est 6<sup>I</sup> in the same manner as before. We found Est 6<sup>I</sup> esterase migrated further than that of gluful-1 and alaful-1 but less than the esterase of Est 6<sup>F</sup>. Thus, the alaful-1 and gluful-1 mutants appear to possess the Est 6<sup>S</sup> isozyme rather than the Est 6<sup>F</sup> or Est 6<sup>I</sup> isozyme.

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Rezzonico Raimondi, G. and A. Gottardi. University of Milan, Italy. Growth behavior of embryonic cells of *Drosophila melanogaster* cultured in vitro.

The behavior of embryonic cells of *Drosophila* in vitro has been analyzed during 8-day experiments, comparing the growth curves of three different wild stocks. The three stocks (Aspra, Varese and S. Maria) can be considered genetically diversified, although phenotypically indis-

tinguishable, as a consequence of their distant geographical origin and the strict inbreeding to which they have been submitted for several years.

The technique used for growing the cells obtained from dechorionated eggs is that by

Horikawa and Fox (1964). Cell countings have been made at the beginning of the experiment and at 24, 48, 96, 144 and 192 hours, using the Bürker haemocytometer. Four independent repetitions have been run for every stock. In all experiments an increase of cell number was found up to 96 hours. Afterwards the cell number decreases, always remaining higher than the initial one, undoubtedly because of accumulation of catabolites and exhaustion of the medium (which has never been replaced).

Within each stock the behavior is similar; statistical analysis has nevertheless proved a certain heterogeneity. Among stocks the differences are statistically significant at 96 hours: Aspra shows the highest increase, while Varese and S. Maria grow less.

Cell viability at the end of the experiments is still good, as proved by active incorporation of  $H^3$ -thymidine seen on autoradiographs.

The differences in growth among stocks is interpreted as an expression of genotypic differences.

Kaneko, A., E. Momma and T. Shima.  
Hokkaido University, Sapporo, Japan.  
Frequencies of abundance of robusta  
species group in Hokkaido.

Collections were extensively made in many  
localities of Hokkaido during the years  
from 1958 to 1965. From 24 localities,  
more than 50 specimens of robusta species  
group were obtained in collection samples  
as shown in the accompanying table.

Among the robusta group, *D. lacertosa* was very frequent in occurrence in all localities listed in the table, except two localities, Yamada-Onsen and Mt. Raus.

Table 1. Percentage frequency of six species of robusta group  
in 24 localities of Hokkaido.

Locality	No. of flies of robusta sp. group	Percentage frequency of each species					
		<i>lacertosa</i>	<i>moriwakii</i>	<i>okadai</i>	<i>neokadai</i>	pseudo- <i>sordidula</i>	<i>sordidula</i>
Sapporo	1326	73.36	2.49	0.75	-	-	19.38
Nopporo	10145	84.32	5.33	2.88	2.02	5.42	0.03
Iwamizawa	209	94.74	-	-	-	-	5.26
Matsumae	376	98.14	1.63	0.27	-	-	-
Ohnuma	242	33.47	2.89	0.83	-	5.37	57.44
Taisei	175	99.43	-	0.57	-	-	-
Gunai	382	67.80	12.83	17.80	-	-	1.57
Utasutsu	89	95.50	-	1.12	3.37	-	-
Toya	1443	83.09	15.94	0.14	0.35	0.42	0.07
Jozankei	301	43.52	48.84	6.64	0.33	0.66	-
Hiroo	232	99.14	-	0.86	-	-	-
Fuyushima	279	86.74	8.24	0.14	-	-	3.58
Yamada-Onsen	104	3.85	93.27	2.88	-	-	-
Utoro	257	61.87	0.39	34.63	-	-	3.11
Okkope	103	100.00	-	-	-	-	-
Toikanbetsu	2262	90.32	5.61	2.74	1.28	0.04	-
Naebutoro	874	72.88	1.14	25.97	-	-	-
Mt. Toyoni	71	87.32	12.68	-	-	-	-
Mt. Raus	75	28.00	61.33	10.67	-	-	-
Shakotan	90	72.22	17.78	7.78	-	-	2.22
Rebun Isl.	229	65.07	10.04	14.41	6.99	3.49	-
Rishiri Isl.	177	80.23	19.77	-	-	-	-
Yagishiri Isl.	194	96.91	-	1.03	2.06	-	-
Okushiri Isl.	273	98.90	0.73	0.37	-	-	-
Total flies	19908	16391	1402	835	263	580	437

Novitski, E., M. E. Myszewski and H. Goldin.  
University of Oregon, Eugene, Oregon. Di-  
centric chromosomes in *Drosophila*.

The genetics of centromere behavior in *D. melanogaster* has been previously studied by testing the action of centromeres against each other in dicentric chromosomes formed by crossing over. Classifi-

cation of centromeres into "strong" and "weak" categories suggests that a greater diversity of centromeric strengths may exist. The range of the diversity may extend to include centromeres which are less capable than even "weak" centromeres in effecting chromosome movement by virtue of a particular array of adjacent heterochromatin. The extreme case would be the physical presence of a centromere on a chromosome incapable of any movement, i.e., the chromosome would act as an acentric.

Females heterozygous for a long inversion provide the system where dicentric chromosomes are formed with regular frequency: Irradiation of these females would introduce random damage to the centromere regions of these crossover-produced dicentrics. In the instance where one of the centromeres is altered in the manner described previously, the second centromere could act to carry the new chromosome to the pole. This would provide a class of progeny generally lost and would also provide chromosomes with particularly interesting properties. Should dicentric chromosomes such as these be recovered, their analysis may provide useful information concerning centromere strength, activity of heterochromatin and chromosome structure.

A simple experiment was designed to generate dicentric chromosomes. Females heterozygous for two  $XY^S \cdot Y^L$  chromosomes, one in normal sequence, the other carrying an inversion of the entire X chromosome were used (Fig. 1) (In(1)EN,  $XY^S \cdot Y^L$ , car f v cv y  $Y^S \cdot Y^L$  (110-8 Parker)  $y^2$  su-wa wa  $Y^S \cdot Y^L$   $y^+$ ). The  $y^+$  and  $B^S$  markers, on the ends of their respective chromosomes,

A simple experiment was designed to generate dicentric chromosomes. Females heterozygous for two  $XY^S \cdot Y^L$  chromosomes, one in normal sequence, the other carrying an inversion of the entire X chromosome were used (Fig. 1) (In(1)EN,  $XY^S \cdot Y^L$ , car f v cv y  $Y^S \cdot Y^L$   $B^S/XY^S \cdot Y^L$  (110-8 Parker)  $y^2$  su-wa wa  $Y^S \cdot Y^L$   $y^+$ ). The  $y^+$  and  $B^S$  markers, on the ends of their respec-

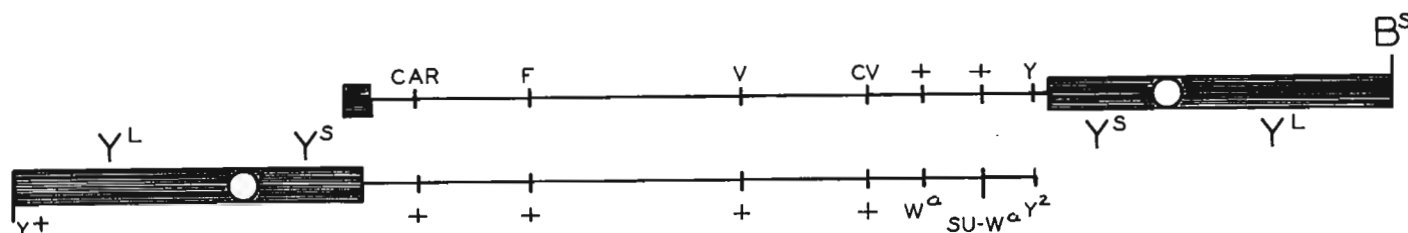


FIGURE 1.  $XY^S \cdot Y^L$  CHROMOSOMES UTILIZED IN CROSS DESIGNED TO PRODUCE DICENTRIC CHROMOSOMES

tive chromosomes, would be joined by a single crossover to form a  $y^+Y^L \cdot Y^S XY^S \cdot Y^L B^S$  chromosome. Progeny carrying both the  $y^+$  and  $B^S$  markers would be of interest, for while the  $y^+ B^S$  offspring may represent a fortuitous juxtaposition of the markers, they may also indicate a particular type of dicentric chromosome which is not lost during cell division.

Four-day-old females were X-rayed with 2700r and mated immediately to X/Y males carrying a yellow mutant. Among 37,370 progeny from irradiated females, 324 flies carrying  $y^+ B^S$  and intermediate markers indicative of single crossing over were observed. No flies of the  $y^+ B^S$  phenotype appeared among 16,626 progeny from non-irradiated females. Of the original 324 exceptional  $y^+ B^S$  flies, 186 (62 males, 124 females) proved to be fertile. These fertile exceptions are currently being tested to ascertain whether the  $y^+ B^S$  markers are actually carried by a dicentric chromosome or if they merely represent a tantalizing rearrangement of the markers.

Koref-Santibañez, S. and M. Lamborot.  
University of Chile, Santiago, Chile.  
The effect of temperature on mating  
activity in *D. gaucha* and *D. pavani*.

*D. gaucha* and *D. pavani*, two sibling  
species of the mesophragmatica group,  
are mostly allopatric in nature, overlap  
in one region of North Western Argentine  
and under laboratory conditions give  
abundant, but sterile hybrids. Their

courtship activity is very similar in pattern, although *D. gaucha* is more active under laboratory conditions, while copulation time is of around 60 min. in *D. pavani* and only about 30 min. in *D. gaucha* (1964, *Biológica* 36:17-26).

Several studies have been undertaken in order to analyze the biological and physiological differences between both species in nature. One of them bears relation to the effect of temperature on mating activity in them and in their reciprocal crosses. For this purpose the temperatures of 8°C, 12°C, 14°C, 16°C, 25°C, 29°C, 31°C were used. These could be indicative of the ranges at which the species may usually exist in nature.

Ten adult virgin females of either *D. pavani* or *D. gaucha* were placed together with an equal number of males of the same or the other species in separate vials at each of the temperatures mentioned, over a period of 6 hours. The spermathecae and ventral receptacles of the females were then examined for the presence of sperm. At least 100 pairs of each group were analyzed.

The results are summarized in Table 1. It may be observed that the optimal temperature for mating seems to be about the same for both species. Nevertheless, *D. pavani*, which

Table 1. Percentage of females inseminated

Crosses		Temperature													
♂	♀	8°C		12°C		14°C		16°C		25°C		29°C		31°C	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%
gaucha	gaucha	200	5	100	24	125	39	100	31	200	79	200	36	100	0
pavani	pavani	200	31	200	37	100	23	100	34	100	48	200	37	100	0
pavani	gaucha	121	4.1	100	18	116	26.7	100	45	200	63.5	200	35	100	0
gaucha	pavani	200	0	100	0	100	1	200	13	200	24	200	15	100	0

is in general less active than its sibling species, seems to tolerate lower temperatures more. This may bear relation to their geographic distribution, as *D. gaucha* lives in general in regions with warmer climate than does *D. pavani*. On the other hand, the great differences between the reciprocal crosses may indicate that the receptivity of females is one of the determining factors in mating success.

Palomino, H. and E. del Solar. University  
of Chile, Santiago, Chile. Density, fertility and gregarian tendency in *D. mel.*

The gregarian tendencies of some species, as *D. melanogaster*, may be revealed by the fact that females tend to lay their eggs in only some of the available breeding sites, or in areas which have previously

been occupied by larval forms (1966, *Am. Nat.* 100:27-133).

In order to analyze the effect of density and fertility on this type of gregariousness, oviposition was studied in *D. melanogaster* inseminated females placed in population cages, in numbers ranging progressively from 6 to 768. Each cage had 6 numbered vials containing food medium additioned with carbon, equidistant from each other and in fixed positions. In each vial the number of eggs was counted every 24 hours, and replaced by a new one over a period of 10 days.

From the results, it can be concluded that the site of maximum oviposition is independent of the spatial position of the vial within the cage; nevertheless 72% to 92% of the eggs are found in 50% of the total number of available vials, although this percentage decreases with increasing density. The maximum fertility was found at lower density (for example for 6 females per cage, the mean oviposition per day was of 4.72 eggs, and for 768 females it decreased to 0.55 eggs per day). If maximum oviposition is considered when density is lowest, it is possible to construct a regression line whose slope falls regularly as density increases. The observed facts seem to indicate that the number of individuals is important for the aggregative tendencies of oviposition in *D. melanogaster*.

Pasztor, Linda M. University of Oregon, Eugene, Oregon. A tandem metacentric which generates unstable rings.

fashion and ring loss, as evidenced by the presence of gynandromorphs, is relatively infrequent. It was, then, of considerable interest when gynandromorphs were found with a very high frequency in the progeny of a tandem metacentric compound X chromosome.

The tandem metacentric was recovered when females of the constitution  $Y^{SX} \cdot Y^L$ ,  $In(1)EN$ ,  $Y^S B f v w y \cdot Y^L y^+ / XY^L \cdot Y^S$ ,  $y Y^L \cdot Y^S y^+$  were irradiated with 3,000 r (Lucchesi, J. C., S. Mills and R. Rosenbleeth, DIS 1965). In the course of experiments designed to determine which, if any, fertility factors were present adjacent to the centromere of these compound X chromosomes, it was recognized that gynandromorphs occurred with a high frequency in one line of a TM stock. Only loss of the ring chromosomes regularly generated by the TM could account for

Chromosomes which are circular in shape present fascinating problems in chromosome behavior. With the exception of  $X^C$ ,  $w^{VC}$ , ring chromosomes in *D. melanogaster* have generally appeared to behave in a regular

the gynandromorphs.

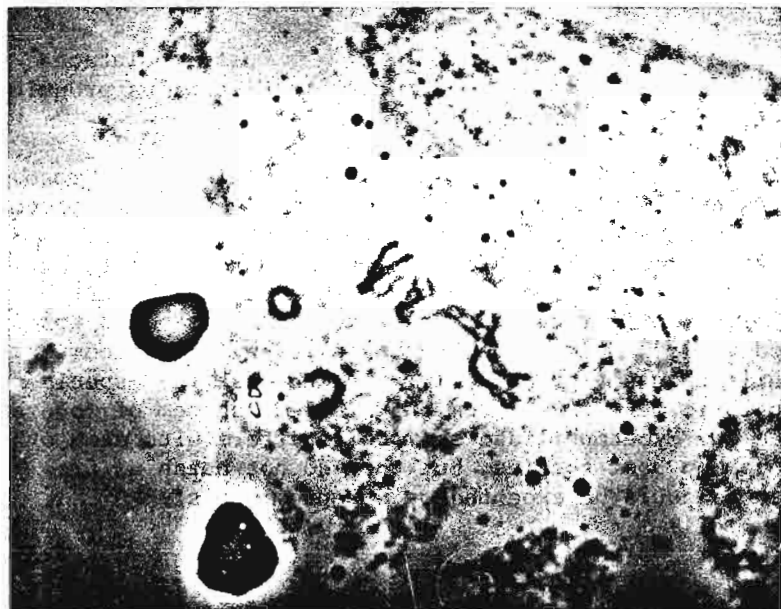
In that fertility factors adjacent to the centromere were found to be absent and homozygosity for vermilion has not been observed, it is proposed that the structure of the TM is  $Y^{SX} \cdot X$ ,  $In(1)EN$ ,  $Y^S B f w y \cdot y$ .

Subsequent studies of this stock revealed that gynandromorphs constitute from 26% to 46% of the female ring-bearing class (Gynandromorphs/((Ring/Rod) + Gynandromorphs)). Further evidence of ring loss is the extremely low c value (Novitski, E. and L. Sandler, Genetics, 1956) of .15. Ring-bearing males were not recovered.

In contrast to these data are those obtained from another stable line of the same origin. Gynandromorphs constitute from .05% to 1.9% of the female ring-bearing class (as computed above) and a c value of .65 is calculated. Ring-bearing males comprise about 45% of the total rings recovered.

It was later discovered that ring-bearing males could be recovered (Fig. 1) when  $Y^{BS}$  was introduced by way of the male parent; this chromosome apparently covers a deficiency found in the newly generated rings. This ring class is somewhat depressed: about 34% of the total rings found are in this group. Most recently, however, it has been revealed that they may also be recovered by the use of  $Y^{suw-f+}$ . It should be noted that, although the viability of the rings is good in males, when these males are tested their offspring show high frequencies of gynandromorphs with ring loss (Hinton, Genetics, 1955) about .42.

Because the phenomenon of a tandem metacentric which regularly produces unstable rings has not been reported previously, it is believed that experiments now in progress to characterize the behavior of these chromosomes may be of general genetic significance. It should be pointed out also that this stock is an extremely valuable and effective one for experiments in which numbers of gynandromorphs are desired.



Bächli, G. and H. Burla. University of Zürich, Switzerland. Breeding *Drosophila* from mushrooms.

During the summers of 1963 and 1964, 1246 mushrooms were collected in forests on the outskirts of Zürich, Switzerland, and brought to the lab, where the pre-adult insects they contained were allowed to develop. The mushrooms belonged to 121 different species of fungi. In total, 7118 insects hatched, 6960 of which were Diptera belonging to 12 different families. Of these, Mycetophilidae were most abundant with 3564 specimens. Drosophilidae came second with 2454, Phoridae third with 491 and Limnobiidae fourth with 168 specimens. The Drosophilidae, harvested from 34 species of mushrooms, were all members of the genus *Drosophila*, as follows:

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<i>Drosophila</i> species	No. of specimens	No. of host mushroom species	Flies predominantly in
phalerata	971	30	- - -
busckii	709	9	3 species of <i>Russula</i>
testacea	477	13	3 species of <i>Lactarius</i>
histrion	113	4	<i>Lactarius</i>
kuntzei	96	12	- - -
funnebris	58	3	<i>Russula alutacea</i>
pallida	29	4	<i>Clitopilus prunulus</i>
limbata	1	1	- - -

Mushrooms of *Russula linnaei* yielded on the average not less than 28.7 specimens of *Drosophila*, *Russula alutacea* 17.1, and *Oudemansiella platyphylla* 10. Whereas 1084 mushrooms contained no member of *Drosophila*, 84 yielded 1 specimen each, 51 were occupied by 2, 21 by 3, 5 by 4, and 1 by 5 different *Drosophila* species. These frequencies do not conform to expectations based on a Poisson distribution, the mushrooms in which 2, 3 or 4 different *Drosophila* species developed side by side being more frequent than expected. This seems to indicate that competition for food played a minor role. The food supplied by a mushroom may nourish many more specimens of *Drosophila* than emerge commonly. In one case, 298 specimens of *D. busckii* and 56 specimens of *D. funnebris* were taken from one single mushroom of moderate size. On the other hand, Drosophilidae and Mycetophilidae seem to interfere with each other. Both families develop in a variety of common host species, but they do not hatch as frequently from the same mushroom specimen as could be expected on a random basis.

Jungen, H. University of Zürich; Switzerland. Evidence of spontaneous inversion in *D. subobscura*.

From a mating of a Tunisian male of *D. subobscura* with a homokaryotypic female of stock c 6 (see stock list of the University of Zürich, Zoological Museum), one out of 16 larvae showed a hitherto unde-

scribed inversion,  $E_{(15)}$ . Its breaking points are at 54 C and 67 A/B, respectively (map by Kunze-Muehl and Sperlich, *Chromosoma* 9, 1958), the latter point being probably identical to the distal break of  $E_{(12)}$ . The inversion must have occurred spontaneously in the analyzed larva, as can be inferred from the following: (1) as mentioned above, 15 sibs of the larva did not carry  $E_{(15)}$ ; (2) the inversion was not found in the offspring of 200 males collected at the same locality, nor in 340 males from other collecting sites in Tunisia; (3)  $E_{(15)}$  was present in only a part of the salivary gland nuclei, the larva being a mosaic in this respect.

To understand why in *D. subobscura*, several different inversions have breaking points in common, one may assume a mechanical pre-disposition of certain chromosome regions for breaking and healing. The observation of  $E_{(15)}$  supports such a view. The incident shows, in addition, that somatic inversions do occur. They have a chance of becoming established in a population, provided that cells descending from the nucleus in which the inversion took place, will form part of the gonads.

The inversion altered the sequence  $E_{1+2+9+4}$ , abundant in Tunisia, to  $E_{1+2+9+4+15}$ . Inversion  $E_{(12)}$ , which shares its distal breaking point with  $E_{(15)}$ , is present in the sequence  $E_{1+2+9+12}$ , which is distributed over the whole Tunisian coast at low frequencies.

Jungen, H. University of Zürich, Switzerland. Abnormal sex ratio, linked with inverted gene sequence, in populations of *D. subobscura* from Tunisia.

Male samples of *D. subobscura* from Tunisia contained a new complex structural type (gene arrangement) of the A chromosome. It has not been fully analyzed as yet, but seems to differ from the standard sequence by about 5 inversion steps covering almost

the whole of the chromosome. Provisionally the sequence will be named "complex A". Males carrying complex A leave offspring of predominantly female sex, as seen from the data below which are based on samples from 2 localities in Tunis. The males collected were crossed with homokaryotypic females carrying either  $A_{St}$  or  $A_{1+2}$ . From the offspring of these matings, 8 larvae were determined as to sex. Similar abnormal sex ratios were found among hatched flies.

X-chromosome of male parent	$A_{1+2}$	$A_2$	complex A	other sequences
number of male parents	86	26	22	6
female/male larvae among offspring	355/333	115/93	159/17	27/21
$\chi^2$ based on 1:1 expectation	.7	2.3	114.5	.7

Daughters produce offspring with normal sex ratio, and so do granddaughters when crossed with males not carrying complex A. However, grandsons carrying complex A have again predominantly females among their offspring.

So far, samples have been studied from Tunis as well as from Gabès, 310 kms south of Tunis, and Tabarka, 120 kms west of Tunis. Complex A was found in all these samples, at an average frequency of 0.2.

The induction of abnormal sex ratio seems to be a concomitant of the complex A, either of its structural sequence, or of its gene content.

Parkash, O. University of Vienna, Austria. On the so-called conditioned (incomplete) and absolute lethals in *D. melanogaster*.

The different types of lethals and the terms used for them have been discussed by Hadorn (1949). In 'conditioned lethals' the penetrance of lethality is dependent on additional

genic or environmental factors, the penetrance for the absolute lethals being independent of any such changes. Dobzhansky (1946) reared a genotype of *Drosophila pseudoobscura* at different temperatures and found that its viability was almost normal at 16.5°C, whereas at 21°C it behaved as a semi-lethal and at 25.5°C as a full lethal. Though very many similar cases have been reported for other organisms (see Hadorn 1955) for *Drosophila melanogaster* the reports are rather scanty.

In the present pilot-experiment, a group of 8 sex-linked lethals was reared at 16°C, 23°C and 28°C respectively. Whereas at 16°C and 23°C all of these behaved as full lethals, at 28°C one out of the present group behaved as a normal, representing, so to say, a conditioned lethal. In another parallel experiment, the females heterozygous for the lethal factor ( $y\ sc^{S1}\ In49\ sc^{8/1}; Cy\ or/B1\ L^2$ ) from various cultures were separately mated to FM6 males (Grell and Lewis, 1956) and a set of 8 cultures each reared at the above mentioned temperatures, thus changing both the genic and the environmental factors. At 16°C two out of the 8 lethals behaved as normal. The normal males in these cases were sterile and showed anomalies of the wing-veins. At 23°C one of the lethals (the same one which behaved as normal at 28°C in the first experiment) behaved as semi-lethal and at 28°C as almost normal. Further, it was found that this particular lethal could be repeatedly converted into normal when reared at 28°C; however, the reversal from normal to lethal could not be obtained.

It will be interesting to determine the percentage of such incomplete lethals in lethals induced by radiation and other chemical means. Further, this phenomenon may play some part in population genetics.

Whitten, M. J. CSIRO, Canberra, Australia.  
Quantitative measurement of the effect of temperature on the penetrance of the eye mutant, witty, in *D. melanogaster*.

Penetrance of the witty character is dependent on background genotype and a number of environmental conditions (Whitten, 1966 and in press). In characters of this type penetrance has been measured in several ways. One can simply measure the propor-

tion of abnormal flies or abnormal eyes (Hansen and Gardner, 1962). Sang et al. (1963) have quantified the measurement of penetrance by reference to an underlying scale and two thresholds separating the three phenotypic classes. An alternative method requiring only one threshold was outlined in Whitten (loc cit). This method assumes that the abnormal phenotype is produced when some morphogenetic substance (m.s.) surpasses a threshold. In the witty case, it was argued from the presence of asymmetrical flies and the frequency of their occurrence in certain inbred strains that the amount of m.s. is independently determined for each eye. Since each fly has two values of m.s., each in part determined by developmental noise, a new penetrance parameter was introduced which is wholly determined by genotype and environment and has one value for each combination of genotype and environment. By measuring  $Z_1$ ,  $Z_2$  and  $Z_3$  where  $Z_1$  is the proportion of flies with both eyes normal ((++) in Table 1),  $Z_2$  the proportion of asymmetrical flies ((L+) and (+R)) and  $Z_3$  the proportion of flies with both eyes abnormal (LR), it is possible to calculate the mean (x) and SD (y) of the penetrance parameter for any population. Table 1 and figure 1 show the response to temperature using this method.

The mean, x, increases with temperature but the SD shows no consistent change. Similar results have been obtained with replica experiments. It can be shown that the mean change in m.s. for a population is equal to the change in x which is thus a direct measure of the response of the morphogenetic substance to temperature changes.

These results support the reasonableness of the penetrance parameter model described by Whitten (1966). The sigmoid response probably indicates a degree of developmental stability for the production of morphogenetic substances over the temperature range normally encountered by *Drosophila*.

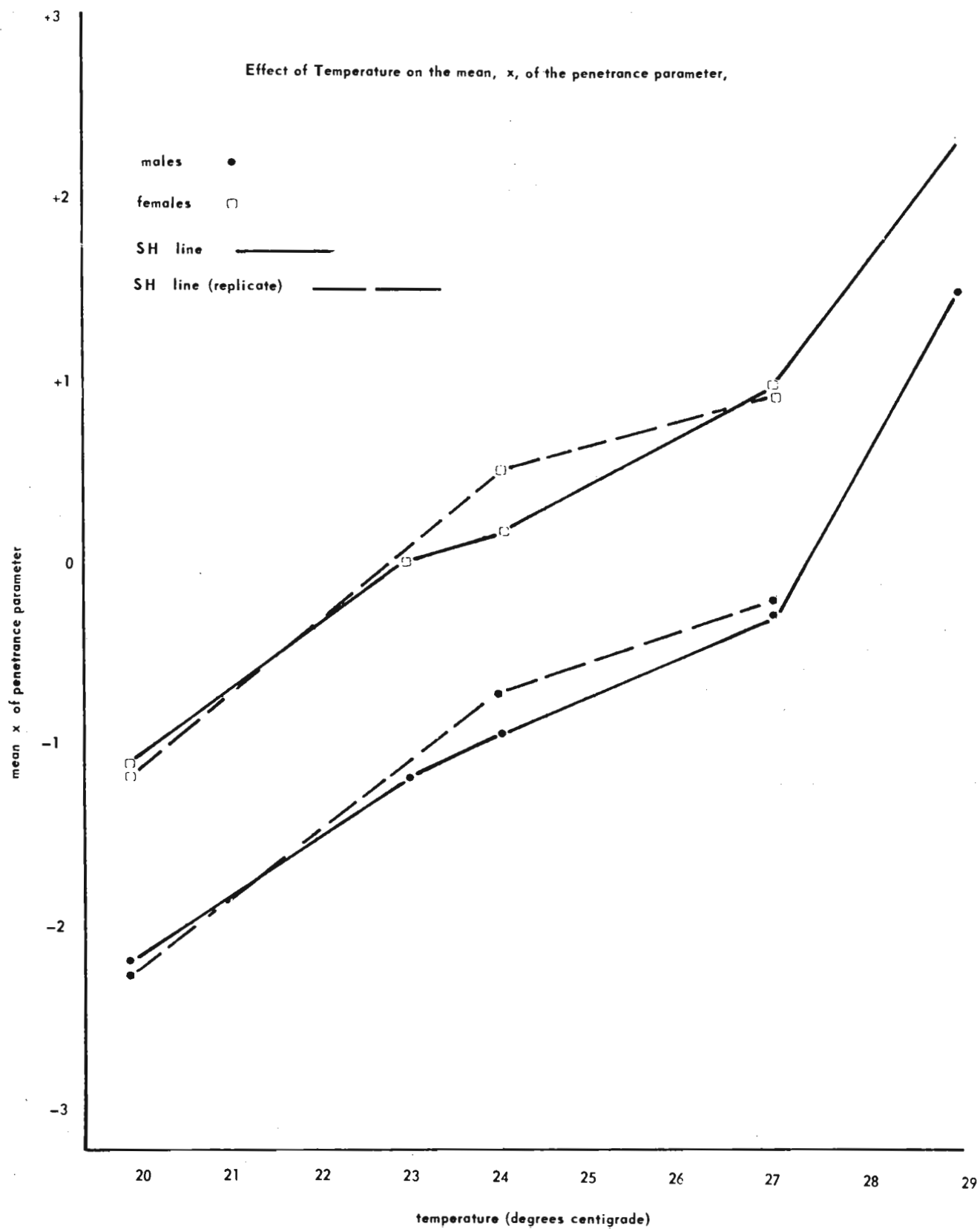
References: Hansen, A. and E. J. Gardner, 1962, *Genetics*, 47:587-598; Sang, J. H., 1963, *J. Hered.*, 154:143-151; Whitten, M. J., 1966, *Genetics*, 54:465-483.

Table 1: Effect of temperature on mean, X, and standard deviation, Y, of the penetrance parameter for the inbred line, SH, homozygous for witty.

Temperature	Sex	(++)*	(L+)	(+R)	(LR)	Sample Size	mean (X)		S.D. (Y)	
							males	females	males	females
20°C	m	296	8	11	0	315	-2.2	-1.11	0.40	0.61
	f	237	43	39	16	335				
23°C	m	178	26	29	11	244	-1.2	0.0	0.67	0.37
	f	91	84	73	92	340				
24°C	m	102	27	16	11	156	-0.96	+0.15	0.64	0.58
	f	40	36	33	57	166				
27°C	m	167	105	86	64	422	-0.33	+0.96	0.22	0.19
	f	14	77	59	329	479				
29°C	m	2	13	8	200	233	+1.5	+2.3	0.28	0.40
	f	0	4	3	211	218				

\* (++) indicates flies with both eyes normal, (L+) with right eye only normal, etc.

Continued on next page.



Angus, D. University of Queensland, St. Lucia, Brisbane, Australia. Cytological evolution in the quadrilineata sp. group.

The cytological evolution of the Australian and New Guinea representatives (*D. tetrachaeta*, *D. pseudotetrachaeta* and *D. nigrilineata*) of the quadrilineata species group of the subgenus *Chaetodrosophilella* is

being investigated. All three species have a metaphase plate consisting of 5 pairs of rods and a pair of dots. In each species there is a giant chromosome configuration of 5 arms and a dot. *D. nigrilineata* will not hybridize with the other two species.

*D. pseudotetrachaeta* females have produced inviable hybrid pupae when crossed with *D. tetrachaeta* males. Analysis of the banding pattern of hybrid larvae for intraspecific differences resulted in the detection of poor synapsis of homologous chromosomes and 9 paracentric inversions (1 on 1, 1 on 2, 2 on 3, 2 on 4, 3 on 5).

By comparison of banding patterns among the three species, 11 more paracentric inversions have been detected (6 in 1, 2 in 2, 1 in 4, 2 in 5). The inversion polymorphism of the group is being further investigated.

Uda, F. and T. Taira. Waseda University, Tokyo, Japan. Cyclic nucleotides and adenylosuccinic acid found in *D. m.*

The change of the pattern of nucleotide pool in *Drosophila* has been studied through the metamorphosis. In order to avoid the contamination of yeast nucleotides, the 90 hrs. old larvae of OR strain were starved

in the period of 2 and 4 hrs. on the wet cellulose powder washed clean prior to contact.

Nucleotides were isolated from hot ethanol extracts and were purified by means of the combination techniques of column chromatography on Dowex-1, paper chromatography and paper electrophoresis. Cyclic nucleotides were obtained in yield of 2.8  $\mu$  moles (Ip!), 1.0  $\mu$  mole (Gp!), 3.6  $\mu$  moles (Cp!) and 1.4  $\mu$  moles (Up!) from 10 g of the 2 hr. starved larvae. However, they were not detectable in the nucleotides from untreated larvae, 4 hr. starved larvae and prepupae. They were identified by the characteristics of their mobilities on paper-chromatography and paper electrophoresis. Their UV absorption spectra were nearly identical to those of the 5'-nucleoside monophosphate. The proportion, base to total phosphate, was 1:1.

Adenylosuccinic acid was isolated from untreated larvae, but it was not found out of both 2 hr. and 4 hr. starved larvae. UV absorption spectrum of the isolated compound was the same as that of authentic adenylosuccinic acid. This compound contained one phosphate group. After the hydrolysis with saturated  $\text{Ba(OH)}_2$  for 20 hrs. at 100°, a compound which showed the same ninhydrin color and chromatographic behavior as aspartic acid, was released.

#### TECHNICAL NOTES

Forbes, Clifford. University of Idaho, Moscow, Idaho. Plastic planchets as radiation exposure holders for *Drosophila*.

A nylon planchet used in isotope studies serves as a convenient chamber for radiation exposure. The planchets are one inch in diameter and one quarter inch deep. Flies are placed in the cup-shaped

planchet with a cover of lens tissue or cellophane held in place by the ring supplied with the planchet. (Available from Atomic Products Corporation, Center Moriches, L.I., New York.)

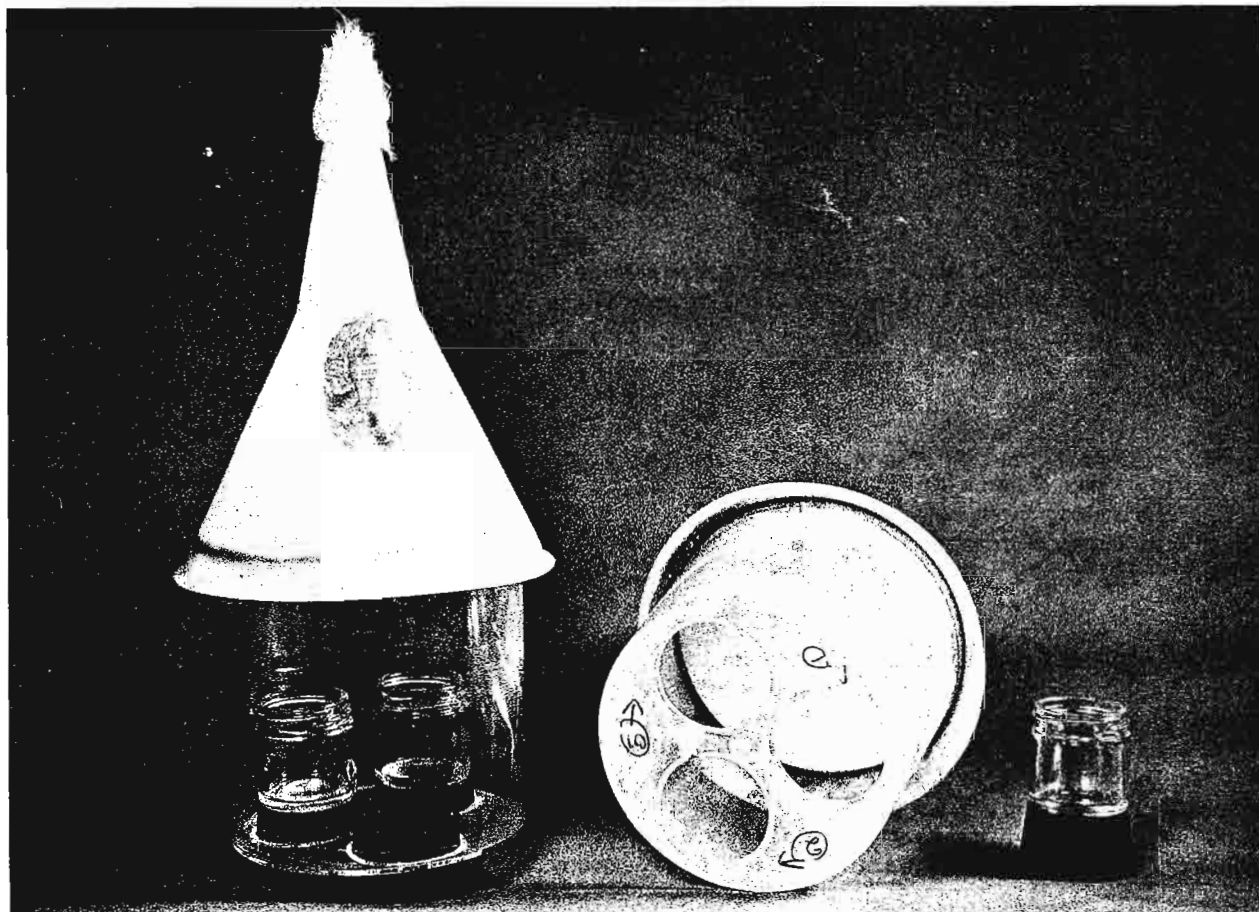
Blaylock, B. Gordon. Oak Ridge National Laboratory, Oak Ridge, Tennessee. A population cage for counting adult *Drosophila* populations.

A small population cage that will hold approximately one thousand flies has been successfully in competition experiments. The total adult population can be counted and transferred with minimum effort to another cage.

The cage is made from an acrylic plastic container that is 12.5 cm in diameter and 13 cm in height. Three holes, 4.5 cm in diameter, a size that will accomodate a no. 10 rubber stopper, are made in the bottom of the plastic container. The holes can be bored by melting the plastic with a hot metal lid of the desired diameter. The top of the cage is a short stem, polyethylene funnel 15.5 cm in diameter. Two openings approximately 3 by 5 cm are made on opposite sides of the funnel for ventilation. The openings are covered with a 40 mesh screen which is easily secured in place by pressing the overlapping edges into the polyethylene with a small, hot metal spatula. Pliobond adhesive (The Goodyear Tire & Rubber Company) is used to cement the funnel to the plastic container. After drying the rubber stoppers are inserted and the cage filled with water to test for tightness. Buxseal (The Johns-Manville Company) or the Pliobond adhesive can be used to fill leaks. If Duxseal is used, the cage is ready for immediate use since no drying time is required.

The food cup is a 40 ml glass jar that is glued onto the rubber stopper with the Pliobond adhesive. To count the adult population and change cages, the food cups containing the larvae and pupae are removed and placed in a clean cage. Plain rubber stoppers are inserted in place of the stoppers holding the food cups. Few flies will be lost in the transfer if the cage is placed under a bright light and the food cups tapped several times to drive away adults. The cage is inverted, and a small number of flies are shaken out of the cage through the funnel stem into an etherizer. Flies are then counted and transferred to another cage, and the process repeated until the entire population has been counted.

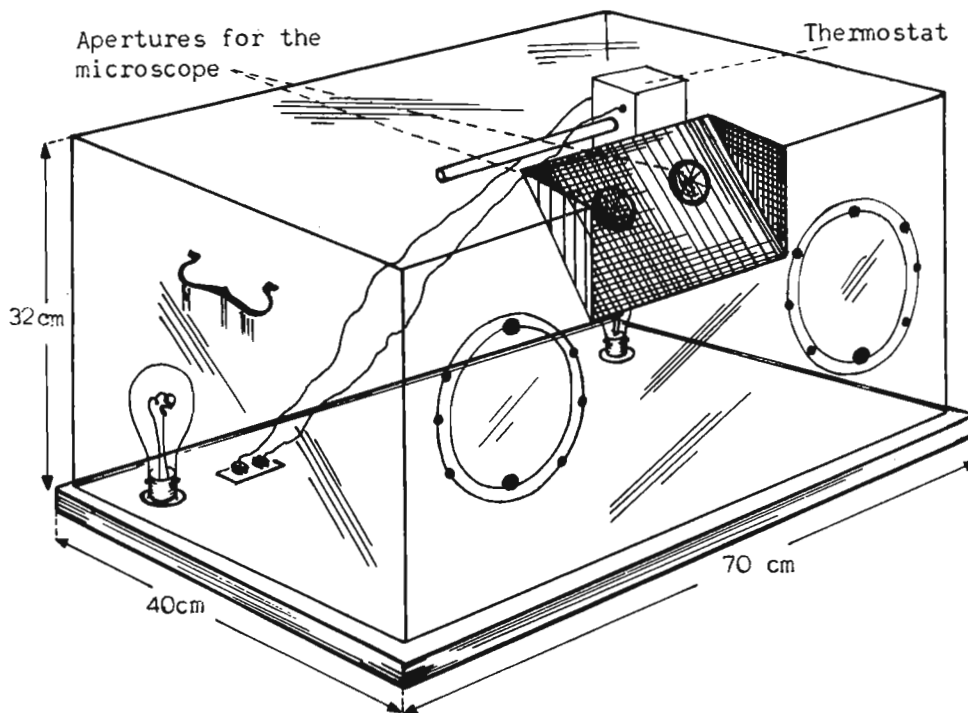
(Research sponsored by the US Atomic Energy Commission under contract with the Union Carbide Corporation.)



Petit, C. Université de Paris, France.  
An isotherm chamber for behavioral observations.

Sexual behavior is sensitive to temperature, so it is important to experiment within constant temperature conditions.

For this purpose, it is possible to use an isotherm chamber that looks much the same as those used by the bacteriologists: it is a plexiglass chamber with a cant at the top of the anterior face, in which temperature is kept constant by two carbon filament lamps controlled by a thermostat. The tubes or boxes containing animals to be observed are introduced by two circular windows on the anterior face. In the cant are set two apertures which enable a binocular microscope to be inserted for studying detailed behavior.



Such a chamber allows a constant temperature to be kept in a very small volume and avoids one remaining for a long time in the confinate isotherm rooms.

Hess, Oswald. Max Planck-Institut für Biologie, Tübingen, Germany. New "one way" *Drosophila* culture containers made of plastics.

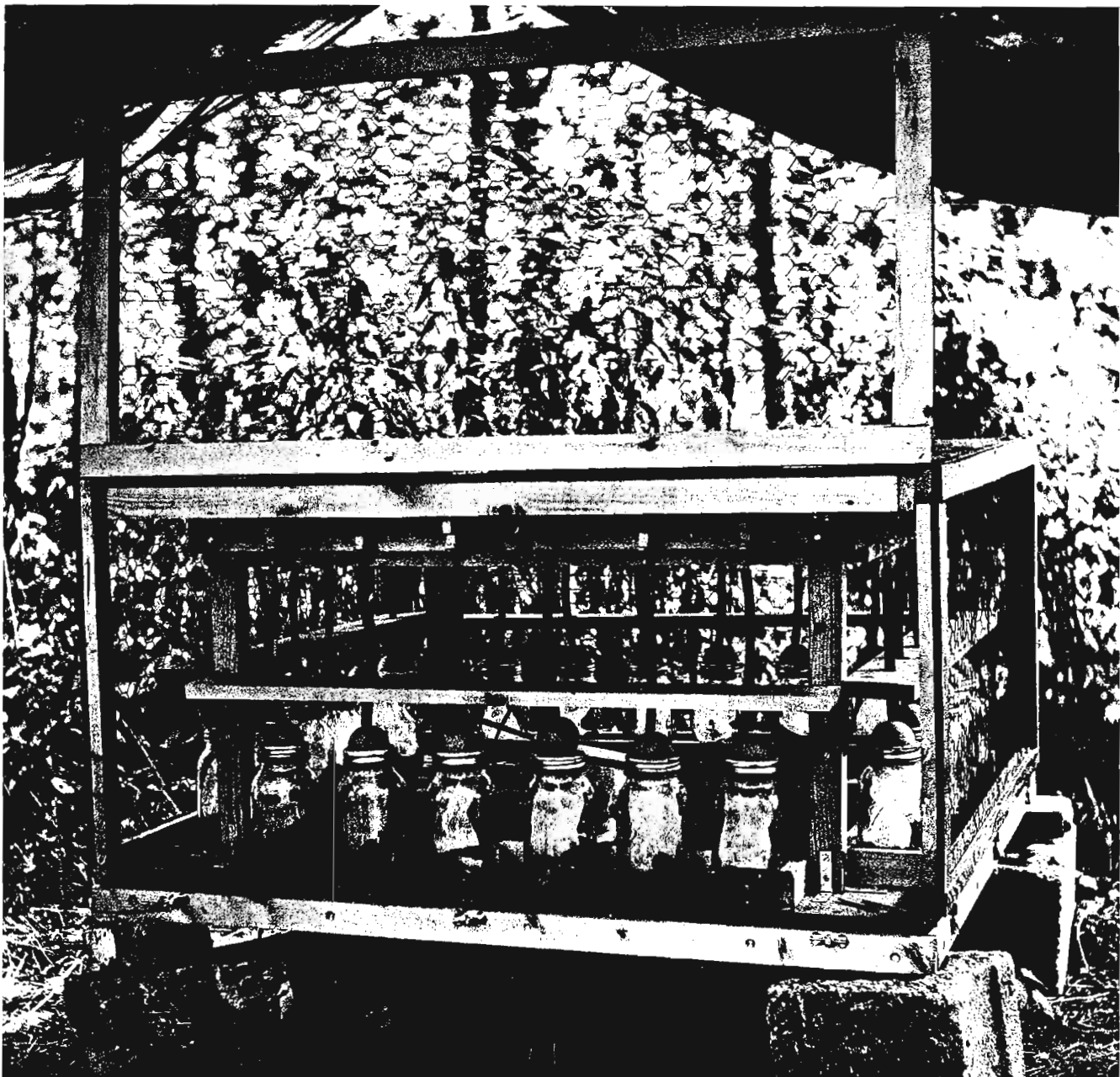
In order to avoid time-consuming and expensive washing of culture bottles new containers which can be thrown away after use have been developed. The containers are made of transparent and colorless polystyrol plastics. Bottles for mass

cultures are 50 mm in diameter and 100 mm high, vials for single matings etc. are 35 mm in diameter and 80 mm high. For both types of containers stoppers made of ceaprene foam are available in different colors. Such containers and stoppers have been used for the culture of *D. melanogaster* and *D. hydei* stocks with good success in our institute for six months. Prices are DM 175.- for 1,000 bottles (= US dollars \$44.00), DM 50.- for 1,000 vials (= US dollars \$12.50), DM 26.- for 1,000 stoppers for bottles (= US dollars \$6.50), and DM 25.- for 1,000 stoppers for vials (= US dollars \$6.25). The manufacturer is Fa. C. A. Greiner und Söhne, 744 Nürtingen/Neckar, Postfach 67, Germany.

Hooper, G. B. Marist College, Poughkeepsie, New York. An automatic trapping apparatus for *Drosophila*.

To facilitate the daily collection of drosophilids an automatic trapping apparatus was designed to collect flies continuously on an hourly basis. The unit (see accompanying photograph) con-

sists of 24 one-quart mason jar traps containing fermenting bananas that are opened on a sequential basis around the clock. Each trap is sealed by a 2 1/2 inch diameter hollow rubber ball that fits into the space normally occupied by the central disc of the cap. The ball in turn is attached to a solenoid (Herbach & Rademan, Phila., Pa. #TM9419) above the trap by a beaded chain which is covered with rubber tubing to minimize the bouncing of the ball when the solenoid is activated. Activation of the solenoid raises the ball approximately a half inch allowing flies to enter the trap. In the photograph the fourth trap from the left is open. Flies are collected in a basket 5 1/2 inches long and 2 1/4 inches in diameter made of



finely meshed nylon curtain material. The top of the basket fits snugly into the neck of the jar while the bottom rests on the banana mash to allow flies to feed while in the trap. To ventilate the closed trap, small holes  $\frac{1}{8}$  inch in diameter have been drilled around the periphery of the top of the cap and covered with organdy. The sequential opening of traps is controlled by a stepping relay (H & R #G4-101) housed in the protected cover of the apparatus.

Traps are sealed prior to removing them from the apparatus by replacing the metal with plastic caps. With a little practice this can be done with almost no loss of flies. Flies are readily removed from the traps later in the laboratory by anesthetizing them through a hole in the plastic caps and removing the baskets. During an active hour of the day, upwards of 250 flies have been collected in a single trap.

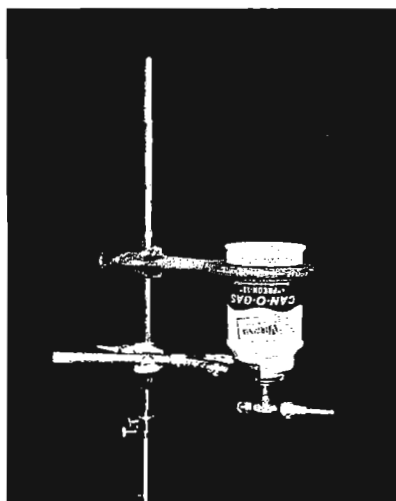
The 'trapper' operates on 110 volts and has three separate circuits: one powers the hourly timer (H & R #HR-027c) that activates the stepping relay; a second activates the solenoid of the stepping relay; and the third brings current to the 24 solenoids. The entire unit measures 47 inches square and is 24 inches high. The traps are protected from small mammals by a chicken-wire screen, while a 12 x 16 foot tarpaulin protects them from rain, falling leaves and branches.

Hoch, Floyd. Ohio Northern University,  
Ada, Ohio. Cover glass removal.

Dry ice has been used extensively for the removal of cover slips when preparing microscope slides by the squash technique. Several disadvantages ex-

perienced in using dry ice have been eliminated in a method developed in our Laboratory Technique course. These problems are availability, storage, and difficulty in removal of the cover slips.

Freon 12 is a non-inflammable, colorless, odorless, non-toxic gas that boils at  $-22^{\circ}\text{F}$  at atmospheric pressure. It is used in many household refrigeration systems and is available in 1 and 2 pound disposable cans from any refrigeration supply. They can be stored on the laboratory shelf indefinitely with no problems. Beginning students have had very little difficulty in removing cover slips using the following method.



The freon can is fitted with a reusable valve which punctures the sealed can and controls the release of the gas. With the can in the upright position, only gas escapes and very little cooling is accomplished, but if the can is inverted, the liquid freon is expelled. We used an ordinary ring stand with a clamp at the bottom and a ring at the top to hold the can in the inverted position. A  $1\frac{1}{2}$ " piece of  $\frac{1}{4}$ " tubing was flared and attached to the valve with a  $\frac{1}{4}$ " flare nut. The tubing acts as a nozzle in directing the liquid onto the slide.

To remove a cover slip, hold the slide at a slight angle in front of the nozzle with the cover slip down. Open the valve releasing liquid freon onto the slide for about 10 seconds. Flip off the cover slips immediately. To avoid frostbitten fingers do not allow the liquid freon to contact the skin.

Other refrigeration gasses boil at even lower temperatures than Freon 12, but it works well and is less expensive than the others. A one pound can costs about one dollar and can be used to remove about 35 to 40 cover slips.

Johnson, W. W. University of New Mexico,  
Albuquerque, New Mexico. A simple  
Drosophila activity maze.

A number of devices for measuring locomotor activity of *Drosophila* and other insects have been described in the literature (see Barton-Browne and Evans, 1960; Ewing, 1963). One general type

that has been used in studies of spontaneous locomotor activity is known as the activity maze. Two advantages of the maze are that any number of flies can be tested at the same time, and that continuous observation and recording of movements are not required. Although the mazes that have been employed in experimental work have differed in details of construction, their basic design has been the same. In essence, an activity maze consists of a series of cells or chambers that are connected linearly by passages through which flies can migrate from one cell to another. Flies are placed in a cell at one end of the series, and after a period of time the flies are scored according to the number of cells they traverse. Flies reaching cells progressively more distant from the starting point are considered logically to have exhibited correspondingly higher levels of activity. Individuals that move more rapidly than others, or that sustain activity for longer periods, would be expected to locate more readily the passages between cells, and thus pass farther along the series in a given period of time.

An activity maze for *Drosophila* can be constructed easily and inexpensively from a polyethylene ice cube tray, a 12" x 6" piece of masonite, six large paper clamps, and ten corks. The only specific requirement is that the tray be of one-piece construction and have adjacent cells separated from one another by common walls.

Into the bottom of each cell in the tray a 1/4" hole is drilled and fitted with a size 00 cork stopper. These holes are used for introducing flies into the maze at the beginning of a run and for inserting ether-soaked cotton to anesthetize the flies at the end of the test period. When the maze is in operation the tray is inverted on a flat surface and in this position the corks can be removed and replaced rapidly with little chance that any of the test flies will escape.

Communication between the cells is made by punching a hole in each of the internal dividers with a hand paper punch. This gives pathways for movements not only between cells in a row, but also between rows of cells. Different patterns for the movement of flies can be assigned by merely sealing off certain of the passages with adhesive tape. One of the most useful arrangements is obtained by isolating the two rows of cells, thus giving two cell chains in which the activity of males and females can be tested concurrently.

Added convenience in handling the maze and scoring the flies can come from placing the tray on a masonite board that has had the latticed outline of the tray cells drawn on it. A very satisfactory means of fastening the tray securely in register with the outline is to clamp the shoulder of the tray to the board with paper clamps. When the flies in each cell are etherized at the end of a run they will fall onto the corresponding rectangular area marked off on the base board. The tray can then be lifted from the board and the number of flies in each cell can be determined quickly by tallying the flies in each area.

The fact that this maze lacks certain of the refinements built into other activity mazes does not limit to any great extent its application to studies of locomotor activity in *Drosophila*. For example, in an attempt to increase discrimination between activity levels, funnel-shaped apertures have been used in other mazes to encourage transits from cells of lower to higher order. Although the effectiveness of this modification never actually has been evaluated, it appears certain that it does reduce the frequency with which flies will reverse direction and re-enter cells previously passed through. However, there is no reason to expect that flies with higher activity will have a predisposition for retrogressive movements and flies with lower activity a predisposition for progressive ones. Therefore, even in the simple maze I have described in which funneled passages have not been used, there should be a positive correlation between the activity of a fly and the rank of the cell in which it is found at the end of a test period. The validity of this supposition is born out by the success with which high and low activity lines have been developed through the use of this maze. (See Ewing, 1963, for a discussion of reactivity, or the interaction among flies in a crowded condition, as a factor in the performance of flies in an activity maze.)

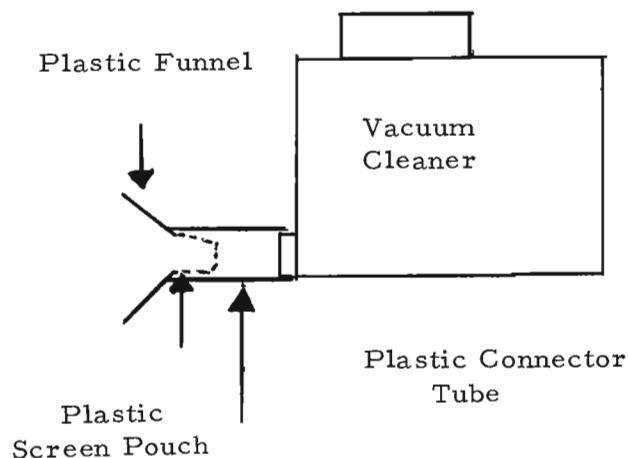
Random locomotion and reactivity are not the only behavioral traits that can be investigated with this maze. The translucent quality of the polyethylene tray makes the maze suitable for a variety of studies dealing with phototactic response. In such work, the light transmitted through the tray from a light source located at one end of the maze would

act as a directional stimulus for the movement of the flies through the cells of the maze.

References: Barton-Browne, L., and Evans, D. R., 1960, Locomotor activity of the blowfly as a function of feeding and starvation. *J. Insect Physiol.* 4:27-37; Ewing, A. W., 1963, Attempts to select for spontaneous activity in *Drosophila melanogaster*. *Anim. Behav.* 11:369-378.

MacMahon, J. A. and D. Taylor. University of Dayton, Dayton, Ohio. Another method for removing *Drosophila* from traps.

relative abundance estimates in studies of natural populations. It is, therefore, necessary to have a foolproof method of collecting all flies in a trap.



Differential flight characteristics of flies in traps may allow fast, agile species to escape, during the trap emptying process, easier than more sedentary species. Such activities may affect Traps similar to those described by Miller (DIS 37:141, 1963) have been emptied, without loss of flies, by utilizing a small, modified, battery powered vacuum cleaner (Fig. 1). The cleaner is equipped with a plastic funnel of a diameter which will completely cover the trap. The funnel is inserted into a plastic tube which fits the cleaner's hose connection. Affixed to the funnel is a plastic screen pouch which prevents the flies from being sucked into the cleaner. The procedure is to turn the cleaner on before the trap is disturbed. The funnel is placed over the trap, which is then tapped lightly. The flies are sucked into the collecting pouch. The funnel opening is stoppered and the funnel plus the attached pouch are removed. The pouch is inserted into an etherizer whose diameter is the same as the plastic tube. Flies may then be transferred to vials.

Shelton, E. E. Utah State University, Logan, Utah. A density method of collecting and cleaning eggs from *D. mel.*

yeasted spoons. Eggs and yeast were washed onto a piece of black cloth cut to fit inside a Buchner funnel. Most of the yeast washed through the cloth. The eggs and the remaining debris were washed from the cloth into a 12 ml. conical centrifuge tube. Sedimentation was facilitated by spinning briefly in an International Clinical centrifuge. The water was sucked off with an aspirator and the pellet resuspended in approximately 4 ml. of 16% sucrose. Approximately 2 ml. of 20% sucrose solution then was layered under the suspension. This was accomplished by placing the tip of a 9-inch Pasteur pipette at the bottom of the tube and allowing the 20% solution to flow slowly into the tube. The tube with its layered contents was spun in the centrifuge for 3 min. at 1610 X g. Most of the debris and all of the first instar larvae formed a pellet at the bottom of the tube. Debris of relatively low density remained at the top of the solution while the eggs accumulated at the interface between the 16 and 20% sucrose solutions and were easily removed with a pipet.



Stalker, H. D. Washington University, St. Louis, Missouri. Techniques for improving salivary chromosome preparations.

in lactic-acetic-orcein, and squashed on siliconed slides. The staining time is adjusted so as to give preparations that are a little too light to be studied with comfort. The preparations are then heated gently (placed on a black table top approximately 9" from a gooseneck lamp with a 100 watt bulb) for three to ten minutes. During the heating process the slides are inspected about once a minute. The heating causes the bands to darken and become thinner, thus increasing the contrast and improving the fine detail. This technique seems to improve all good squashes; it has little effect on poor ones. A second consideration in preparing satisfactory squashes is the time of day: many species produce satisfactory squashes in the morning, but not in the afternoon, or vice versa. Even closely related species may differ in this characteristic.

For many (possibly all) species, the fine banding detail can be greatly improved by controlled heating. Glands are dissected in 40 - 60% acetic acid (the concentration depending on the species), stained

Danieli, G. A. and E. Rodinò. University of Padua, Italy. A homogeneous medium for studies of labelled precursors incorporation in growing larvae.

A problem in metabolism studies approached by autoradiographic technique is the procedure used in labelled precursors administration. In small sized animals there is the possibility of microinjection, but the extreme accuracy

required by this procedure may lead to significant errors in final concentration of the isotope in the organism. Therefore many investigators, working on *Drosophila* salivary gland metabolism, choose incubation in vitro of isolated organs (Arnold, 1965; Plaut, 1966) or labelled precursors administration directly in the food (Taylor et al., 1955; McMaster et al. 1957, 1959, 1960; Woods et al., 1961). In the latter method, however, the normal food is scarcely suitable for experimental approach, because it is not homogeneous and hence leading to a nonrandom distribution (very important, in fact, in short term incubations) and a decrease of the culture synchronization.

In order to overcome these disadvantages we tried to obtain a medium as homogeneous as possible. We found that the most suitable and successful medium is a thick dead yeast suspension. In our experiments the labelled precursor was  $H^3$ -Thymidine and was administered to larvae from a fairly uniform culture at different stages as follows:

In a heat killed yeast suspension (50 gr. in 100 ml.) the isotope (Schwarz-Biores. sp.act. 19 mC/mM) was dissolved to a final concentration 8.3  $\mu$ C/ml. This solution was mixed vigorously by magnetic stirring for 2 minutes at room temperature. In order to maintain exactly the same feeding conditions throughout the entire time of the experiment, an adequate number of containers were filled with the isotope solution and at once frozen on dry ice and stored at  $-30^{\circ}\text{C}$  until used. The larvae (ten per point), collected from a synchronized culture, were thoroughly washed in Ephrussi's Ringer solution containing antibiotics and then incubated in 1 ml. of tritiated yeast suspension for 8 hours at  $24^{\circ}\text{C}$ . After incubation the larvae, washed in Ringer solution containing excess unlabelled Thymidine, were transferred to normal food (Bakker, 1962) for growth until the late third instar.

Autoradiography of squashed salivary chromosomes were made using Kodak NTB 2 bulk emulsion, exposed for 2 weeks, developed in Kodak D 19, fixed and then stained with acetic orcein or toluidine blue.

The figures 1 and 2 show two microphotographs of these preparations.

References: Arnold, G. 1965, *J. Morph.*, 116:65; Bakker, K. 1962, *Archiv. Néerl. Zool.* tome 14:200; McMaster-Kaye, R. J. 1957, *Bioch. Bioph. Cytol.*, 4:5; IDEM 1959 *ibidem*, 5:461; IDEM 1960 *ibidem*, 8:56; Plaut, W. et al. 1966, *J. Mol. Biol.*, 16:85; Taylor, J. H. et al. 1955, *Exptl. Cell Res.*, 9:460; Woods, P. S. et al. 1961, *Proc. Nat. Acad. Sci.*, 46:1486.

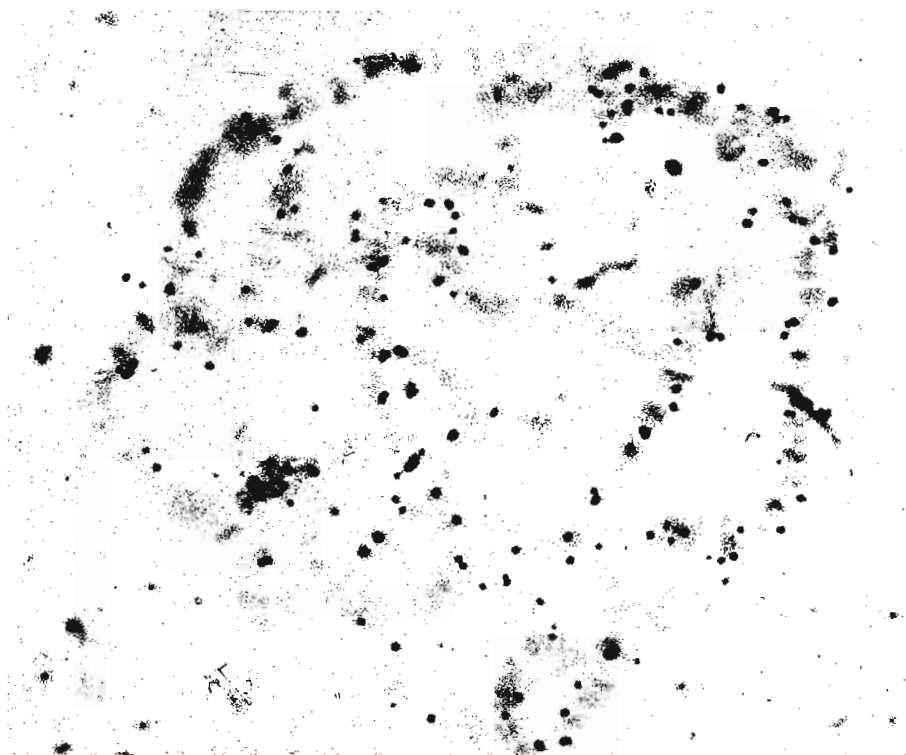


Fig. 1: Autoradiography of squashed chromosomes of *D. hydei* stained with toluidine blue - Magnification 600x

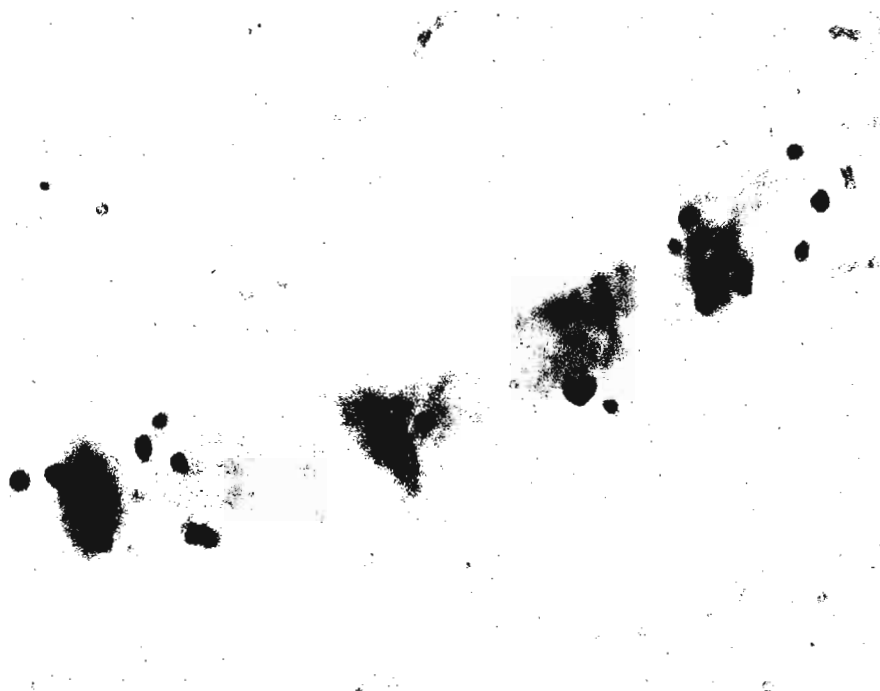


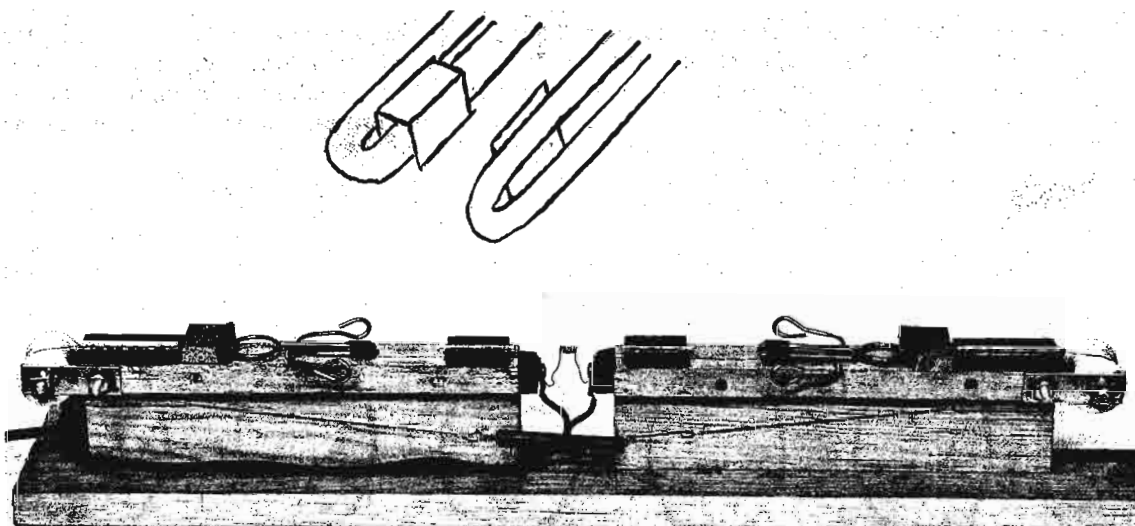
Fig. 2: Higher magnification of a chromosome, stained with acetic orcein, immersion, 2500x (note the very low background level)

Seecof, R. L. City of Hope Medical Center, Duarte, California. An apparatus for drawing micropipettes.

With this apparatus micropipettes can be fashioned which are suitable for making injections into *Drosophila*. The apparatus is very inexpensive to build and 100 pipettes per hour can readily be made.

These pipettes can be used in the injection apparatus described by Seecof (DIS 41:185).

To make pipettes, a piece of capillary tubing (O.D. 0.7 - 1.0 mm) about 18 cm long is inserted into the two 1.5 in. guides and the heating element coil at the top-center of the apparatus. The two Day pinchcock clamps are then each pulled forward 1.25 in. to grasp the capillary tubing where it protrudes from the guides. This motion also stretches the spring because each clamp is attached to the spring by a cord passing around the spool at each end of the apparatus. The heating element is then energized by a switch (not shown), the capillary tubing softens, and two pipettes are simultaneously drawn by the spring tension. The element is de-energized immediately afterwards to avoid excessive heat. The pipette tip can later be broken to a proper-sized opening with forceps.



Most of the dimensions of the apparatus are not critical because the shape of the pipette tip can be controlled by the spring tension (adjustable by changing the length of the cord), the size and shape of the heating coil, and the temperature of the coil (adjustable by changing the voltage). An apparatus of the dimensions given here operates well at 7.5 volts and 2.72 lbs. of spring tension.

Each piece of cord passes from the spring, through a 2 in. guide, around a spool, through a 2 in. guide, through a 0.75 in. wood block and a 0.25 in. foam-rubber bumper glued to the block, and is attached to the clamp. All six guides are 3 mm pyrex tubing and are glued in place with Duco cement. Guides and heating coil are mounted in the same vertical plane. The four guides on the top of the apparatus are raised  $\frac{3}{16}$  in. on blocks so as to be in the same horizontal plane as the clamp tips and the heating coil. The heating coil is  $\frac{3}{8}$  in. long and was fashioned by winding 9 turns of wire around a piece of 3 mm tubing. The heating element and connecting wires are connected at bakelite terminal strips. The strips and element are mounted in a gap 2 in. wide. The sewing-thread spools are  $\frac{1}{16}$  in. long and ride on 0.25 in. bolts. The top of the apparatus is 1.25 in. wide and its overall length, including spools, is 22.5 in.

The tips of the clamps are modified with pieces of metal as shown in the close-up, to give a grasping area about 0.5 in. long and  $\frac{3}{16}$  in. high. The metal pieces and clamp tips are covered with pieces of 0.5 in. long latex tubing ( $\frac{1}{8}$  in. bore,  $\frac{1}{16}$  in. wall). The metal added to the tips was obtained by cutting pieces from Hunt paper clips (metal about 0.5 mm thick).

This investigation was supported by Public Health Service Grant AI-05038-04 and the Norman D. Denny Research Fellowship fund.

Erk, Frank C. State University of New York, Stony Brook, N.Y. An expressivity scale for melanotic tumors.

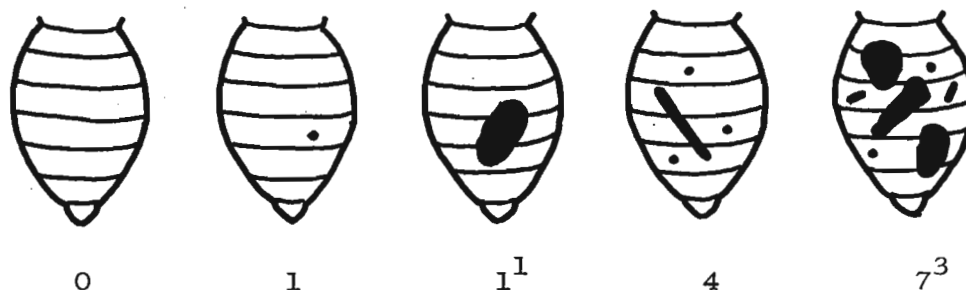
Melanotic tumors occur or can be induced in many strains of *Drosophila*, but penetrance varies widely and is affected by both genetic and environmental factors.

There may be also a wide range of expressivity in certain strains, although it has been difficult to take this factor into account in most studies. Because of the range in number and size of tumors found among flies in a given strain, presumably sensitive to both genetic and environmental factors, the melanotic tumor strains offer excellent material for the study of expressivity, surely a perplexing problem in physiological genetics. Tumors occupying fixed locations (head, spiracles, mouthparts) present another range of problems, but are not considered here.

There is no fixed site of free-floating tumors, which can be moved easily by external manipulation. Probably the tumors tend to migrate posteriorly during larval development and movement; they are usually found in the abdomen, although they occasionally appear also in the thorax, head, and legs. The variations in number and size appear to be independent of location.

The study of the effects of various factors on expressivity in tumor strains depends on a useful expressivity scale that will characterize flies according to both number and size of tumors present. In the scale I have used, a base number represents the number of distinct tumors - those that do not separate upon dissection. A superscript is applied to the base number to indicate how many (if any) of the tumors are "large," that is, greater than the width of one abdominal segment in their largest dimension. This scale provides a convenient measuring unit within the fly itself, while at the same time compensating for differences in size of flies which may be due to sex, genotype, or nutritional state. Thus, the presence of "large" tumors means, among other things, that a significant proportion of the fly's mass is made up of melanized inclusions.

Sample applications of the scale are shown below:



Expressivity scoring is a tedious process when it is done by dissection, but it can be eased considerably by using Sang's fructose method (DIS 41:200) and by using a multi-key counter, with one channel assigned to each point on the expressivity scale.

Carlson, J. H. Fairleigh Dickinson University at Madison, Wisconsin. A source of paper for *Drosophila culutres*.

For the past year the author has been using Kimwipes (Type 900-S) Disposable Wipers (Kimberly-Clark Corporation, Neenah, Wisconsin) as the paper inserts in *Drosophila* food bottles. These are

absorbent, non-linting and will absorb up to 15 times their weight in liquids. They are chemically inert and come in various sizes. The size I find most convenient is 5 x 9 inches. They come in a sealed cardboard box with a punch out hole on top. They are of the "pop-up" type which makes dispensing easy. There are 240 sheets to the box and a carton of 72 boxes costs \$20.88 (Fisher Catalogue) which works out to 29¢ per box. I have also used regular Kleenex type tissues cut into thirds, but I find that the Kimwipes are more convenient. When folded in half and used in a standard half pint bottle they make a nice paper funnel into which anesthetized flies may be dropped and allowed to revive.

Jost, P. and W. Sistrom. University of Oregon, Eugene, Oregon. Effects of humidity on increasing the yield from vial pair matings.

In a series of experiments in which less than 25% of the zygotes were expected to be viable, difficulties were encountered with bacterial contamination and drying of food when pair matings (either in vials or in 1/2 pint bottles) were

attempted under the conditions in an ordinary incubator, humidified by evaporation from open pans of water.

Failure of as high as 70% of all pair matings to produce adult progeny was found in several experiments, even though over 90% of the vials contained some larval stages. Addition of extra yeast and a few drops of distilled water to the vials in the first week of egg-laying did not appreciably increase the yield. However, raising the humidity in the vials up to a point at which condensation appeared, markedly increased both the number of vials yielding adults and the adults obtained per vial. In addition, bacterial growth was much less apparent. When these conditions of high humidity were maintained for the first few days after mating, approximately 70 - 85% of the matings gave adult progeny, and the average yield per vial was increased several-fold.

Conditions of high humidity were achieved in an ordinary incubator by using aluminum foil wrapping. Wire trays were lined with foil on which were placed five to six layers of paper towels saturated with a dilute solution of copper sulphate to inhibit growth of mold.

The vials were placed upright in the tray on the wet paper towels, and pieces of foil-wrapped cardboard one inch higher than the vials were placed along the sides of the tray before the foil was closed and sealed. This gave a closed chamber with an inch or so between the top of the vials and the foil cover. After five to six days the foil cover was torn open and the excess humidity allowed to dissipate before the first pupation occurred. Maintenance of conditions of high humidity for as long as ten days delayed pupation and did not give a maximum yield. All experiments were conducted at 25°C, and the medium used was a standard cornmeal, molasses, agar, yeast and propionic acid, but with added antibiotic (Hendrix and Ehrlich, DIS 40:99).

Maintenance of extremely high humidity during the first few days of egg laying may prove to be generally useful in increasing the yield of adults from pair matings where lowered fertility or high zygotic mortality reduces the number of larvae to such an extent that early drying of the food occurs, in spite of the presence of pans of water in the usual cabinet-type incubator.

#### TEACHING NOTE

Lifschytz, Eliezer & Raphael Falk Hebrew University, Jerusalem, Israel. "Complementation map" in *Drosophila*.

Beginning students often have difficulties in grasping the method of genetic mapping by means of "deficiencies", as applied e.g., by Benzer for the rII locus of T4.

The system of the X-chromosome lethals covered by Y.w<sup>+</sup> described in this issue (Lifschytz, E.) proved to be very useful in clarifying this genetic method. The students were given all combinations of matings between the lethals, as well as the mating between a white (non-lethal) stock and the lethals. The results were plotted in a matrix form, and the students constructed the "complementation map" by themselves.

Compiled in part by Irwin H. Herskowitz

D. = *Drosophila*

D.m. = *D. melanogaster*

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- 1964 Meiotic X-Y exchange and nondisjunction induced by irradiation in the *D.* male. *Genetics*, 50: 633-638.
- 1964 X-Y nondisjunction and exchange induced by X rays in primary spermatocytes of the adult *D.* *Genetics*, 49: 499-504.

J. A. Beardmore of the Genetical Institute, University of Groningen, Netherlands, is moving to head a new Department of Genetics, January 1st, 1967, in the University College of Swansea, Wales.

Francis M. Butterworth, formerly of Department of Biology, University of Virginia, has joined the faculty of Oakland University, Rochester, Michigan, as an Assistant Professor, where he will continue to work on the development of adipose tissue.

Ove Frydenberg of the University of Copenhagen will be Visiting Professor at the Universidad de Sao Paulo, Brazil from July 1966 until mid 1967. Dr. Frydenberg will give post-graduate courses on Population Genetics and Biostatistics and will work on population genetics problems.

John K. Lim, Associate Professor of Biology, Wisconsin State University, Eau Claire is spending a sabbatical leave within the Department of Biology, City of Hope Medical Center, Duarte, California.

Yung Sun Kang, Seoul National University, Korea, was elected the chairman of the Board of Directors, the Korean Commission for Conservation of Nature and Natural Resources, and from this year ( 1966) an ecological research on DMZ will be performed by using the research grant from the Smithsonian Institution in the United States.

M. Kawabe, Biological Laboratory, Kobe University, Nada, Kobe, Japan, has just come back from a one year stay with the Department of Human Genetics, Michigan State University.

Ingerid Kvelland, University of Oslo, Norway, will return to Oslo August 1st, 1967, after her stay as Research Associate at the laboratory of Dr. Doermann, University of Washington, Seattle, Washington.

J. D. Mohler has left the Department of Zoology, Oregon State University and has joined the Department of Zoology, University of Iowa, Iowa City, Iowa, 52240.

Öistein Strömnaes, University of Oslo, Norway, is on leave of absence to work with Professor R. K. Mortimer, Donner Laboratories, University of California, Berkeley, one year from September 1st, 1966.

Eliot B. Spiess, has joined the Department of Biological Sciences at the University of Illinois at Chicago Circle where he will serve to develop a genetics section.

Peter A. Parsons has moved to the School of Biological Sciences of the new La Trobe University in Melbourne, Victoria, Australia.

Sarah B. Pipkin, formerly Research Associate at The Johns Hopkins University, has joined the faculty of Howard University as Associate Professor of Zoology, beginning September 1st, 1966.

T. Taira, formerly of the National Institute of Genetics, Misima, Shizuoka-ken, has moved to the Department of Biology, School of Education, Waseda University, Tokyo, where he is constructing a new Drosophila Laboratory and is continuing work on biochemical genetics. He is also collecting the morphological mutants of Drosophila melanogaster.

Bungo Sakaguchi has joined the faculty of Agriculture, Kyushu University, Fukuoka, Japan as an Associate Professor. He is establishing a new Drosophila Laboratory of physiological genetics and continuing studies on biochemical genetics and hereditary infections in Drosophila. He formerly was with the National Institute of Genetics, Misima, Japan.

James H. Carlson, Department of Biology, Fairleigh Dickinson University, Madison, New Jersey would like to receive genetic reprints (general and *Drosophila*) in order to build up the departmental library.

M. Levitan, Department of Biology, George Mason College of the University of Virginia, Fairfax, Virginia, would appreciate exchanging reprints.

David Williamson, Anatomy Department, Woman's Medical College of Pennsylvania, Philadelphia, Pennsylvania, would appreciate exchanging reprints.

J. E. Watson, Department of Zoology-Entomology, School of Agriculture and Agricultural Experiment Station System, Auburn University, Auburn, Alabama, would appreciate reprints to build up the library in his newly established *Drosophila* Laboratory.

#### ANNOUNCEMENT

The National Science Foundation has had translated from the Russian the Transactions of the Institute of Cytology, Histology, and Embryology, Vol. II, No. 1, 1948, containing the following articles:

I.A. Rapoport. Phenogenetic analysis of independent and dependent differentiation.  
pp. 1-140.

A.A. Malinovskii. Elementary correlations and variation in the human organism.  
pp. 141-216.

L. V. Lugovaya. On the significance of giant polymorph cells in the development of *Flavobacter* sp. n. cultures. pp. 217 - 248.

Copies of these translations are available on loan and may be requested from the Genetic Biology Program.

#### ANNOUNCEMENT

Dr. M. Demerec died of a heart attack on April 12th, 1966, at the age of 71. Dr. Demerec's reprint collection has joined the reprint collections donated by other prominent geneticists in the Library, Cold Spring Harbor Laboratory of Quantitative Biology, Cold Spring Harbor, New York 11724. It is requested that subscribers of DIS send reprints to the library to maintain there the availability of an up-to-date and complete collection. There are still available reprints of most of Dr. Demerec's own publications and publications emanating from his laboratory. Blanket requests for reprints will be honored only to libraries interested in binding the collection. Requests for individual titles may be made to:

Miss Agnes Fisher,  
Genetics Research Unit,  
Box 200,  
Cold Spring Harbor,  
Long Island, New York 11724

Carlson, E. A. University of California, Los Angeles, California. Public lectures, the scientist and the press.

In April 1966, I presented a public lecture on genetics and human values. This was one of six lectures on future advances in biology and medicine. The series, The Human Agenda, was the Spring

1966 Faculty Lecture series at UCLA. These lectures are geared to a lay audience and they are selected by a faculty committee on public lectures.

The popular response to this series was unexpectedly large and overflow crowds attended all six of the lectures. Each of the participants had reservations about giving a public lecture on this theme but the coordinator of the series was successful in obtaining our consent. The format of these lectures was unusual because we were asked to speculate freely as far into the future as we could project the developments in our fields. Each scientist was paired with a non-scientist who responded with his reactions to these future developments.

The Los Angeles Times sent a science reporter, about five days before the series began, to interview the coordinator and me since I was to present the first lecture. The UCLA publicity director also attended this conference so that he would be informed in case there was a response to the article in the Los Angeles Times. I gave the reporter a typed abstract since I do not write out my lectures or speeches. In about 30 minutes he questioned me about the meaning of some terms that I had used (genetic load, electrophoretic diaphragm, necrogenetics) and both the series coordinator and the UCLA publicity director supplied their own examples and paraphrase, which I had to qualify at times because of the inaccuracies of over-simplification. Most of this conference and virtually all of my public talk was concerned with Muller's famous problem of the "genetic load" of mutations and various eugenic programs that have been suggested or which might arise in the future. The reporter never showed me his draft nor did I know what he wrote until I saw the April 12, 1966 edition of the L.A. Times. U.P.I. took one half of the L.A. Times story and sent it across the world. This was the portion carrying a highly sensational and inaccurate account of a process (necrogenetics) that I predicted would be used about two and a half centuries from now. My actual lecture never mentioned King Tut, Joan of Arc, or Mozart but the U.P.I. dispatch did. Further, I took pains to describe the process as artificial twinning, but the press assumed wrongly that a necrogenetic twin would have the personality (or even the memory) of deceased individuals.

I received a number of letters in response to the U.P.I. dispatch, including queries from Australia and France. Most of the letters were uncomplimentary ("If you were my son I'd beat you with a broom handle and teach you who to respect"). Some of my colleagues were interviewed by their student papers and I was surprised to see the remark attributed to one professor at Santa Barbara ("he's nuttier than a fruit cake; are you sure he's at UCLA?").

The Washington Post asked me if I would write a 2500 word essay on my talk. I consented to do so, and they published it (June 5, 1966) with the title "Science Promises Custom Child." The same article was sent through their syndicate to the Providence (R.I.) Sunday Journal and published (June 5, 1966) as "When Man Seeks to Control Heredity." The Victoria Daily Times (June 10, 1966) used the title "Genetics and the Future of Man."

Sometimes scientists refuse to give public lectures because they find the press coverage too puerile. I believe there should be some reform in the press, especially in headline writing, but it is wrong for the scientist to put his blame exclusively on the press. After all, who should a reporter, in one or two hours, be expected to know the complexities that take us twelve years or more to acquire? A newspaper is not a professional journal and its content is ephemeral rather than permanent. Few of the readers will carry away detail from such articles but they will carry away a few impressions about science.

What surprises me, however, is the ready acceptance with which scientists believe what they see in a newspaper about science. I have raised my eyebrows more than once at poor science writing, obvious errors, and incredible oversimplification. Why do many scientists accept, uncritically, the poor impressions they gain from reading about a colleague's work in the newspapers?

I hope that this note will convey to my colleagues that they should hold their pessimistic feelings in reserve until they have had a chance to see what their colleague did write or say. Second, I hope the graduate students reading this article will not feel discouraged if they receive bad press coverage in their first public lecture. It is, I believe, important for scientists to express their concern to the public about scientific issues which affect human values. If we elect only to do so from the safety of professional journals we weaken the strength of our concern. The press will never "grow up" unless we are willing to take some abuse and misrepresentation as part of this responsibility to convey our ideas to the public. Similarly, we need to improve our own attitude towards the press and we need more care in judging our colleagues or their work as reported by the press.

#### ANNOUNCEMENT

The University of Alberta at Edmonton invites applications for the Headship of the Department of Genetics in the Faculty of Science. At present the department has 8 academic staff members and this number should rise to at least 12 in the next few years. In 1968, the Department of Genetics, together with the Departments of Botany, Microbiology and Zoology will move into new quarters in the Biological Sciences complex. The new Head will be expected to provide scientific and personal leadership for the department. Applicants should submit statements giving biographical details and outlining research in progress and future plans, together with a list of publications and the names of three persons who can be approached for supporting references. The salary for the Headship, which will be at the rank of Full-Professor will be not less than \$17,000 per annum. Applications or requests for further information should be sent to:

The Dean,  
The Faculty of Science,  
University of Alberta,  
Edmonton, Alberta,  
CANADA

#### NINTH ANNUAL

#### DROSOPHILA RESEARCH CONFERENCE

The Ninth Annual Drosophila Research Conference will be held at the University of Texas on April 7th to 9th, 1967.

Arrangements have been made to hold the general sessions of the Conference in the new Academic Center Auditorium. All of the conferees can be housed in the Villa Capri Motor Hotel a few blocks from campus.

The tentative schedule is to hold the opening session Friday evening, April 7th followed by a short reception. The Sunday session will end at noon at allow you to leave that afternoon if you wish.

Details of the Conference schedule and housing will be mailed out in January. This mailing list has been compiled from the list of participants at past conferences and the list of investigators in D.I.S. If you do not receive further information about this Conference, get in touch with Burke H. Judd, Department of Zoology, University of Texas, Austin, Texas, 78712.

## Geographical Supplement

This list includes only laboratories not previously listed in DIS and individuals in previously listed laboratories who were not named in DIS #41; a complete list will be included in a future edition.

ARGENTINA

Buenos Aires: Argentine Atomic Energy Commission, Department of Radiobiology, Tel. No. 70-7711, Ext. 59.

Spirito, Sonia Elena (Miss) Undergraduate research fellow.

AUSTRALIA

Adelaide, South Australia: Flinders University, School of Biological Sciences, Tel. No. 76-0511, Ext. 2229.

Morgan, M. (Miss) Laboratory Assistant.

Melbourne, Victoria: Le Trobe University, School of Biological Sciences, Tel. No. 26-3308

Hosgood, Sally M. W. (Miss) B.Sc. Senior Demonstrator. Ecological and behaviour genetics  
MacBean, I. T. B.Sc. Demonstrator. Behaviour genetics  
Parsons, P. A. Ph.D. Professor. Population and behaviour genetics

Sydney, New South Wales: University of Sydney, Department of Animal Husbandry, Tel. No. 68-0522, Ext. 2184.

Carrick, M. C. B.Sc.Agr., Research Assistant. Population genetics

AUSTRIA

Vienna: University of Vienna, Department of General Biology, Tel. No. 422767

Enzenhofer, Rita (Miss) Research Student. Subobscura, inversion polymorphism  
Feuerbach, Heidemarie (Miss) Research Student. Linkage disequilibrium

BRAZIL

São Paulo, São Paulo: Faculdade de Filosofia, Ciências e Letras da USP, Departamento de Biologia Geral, Caixa Postal 8.105, Tel No. 8-2111.

Brito da Cunha, A. Ph.D. Associate Professor. Population genetics; Drosophila infections with micro-organisms  
França, Z. M. Postgraduate Student. Analysis of recombination in Drosophila  
Frydenberg, Ove, Ph.D. Visiting Professor from the University of Copenhagen. Population genetics; biostatistics  
Magalhães, L. E. de Ph.D. Assistant Professor. Population genetics

Pavan, C. Ph.D. Head of Department. Cytogenetics of Rhynchosciara; Drosophila infections with micro-organisms  
Santos, E. P. dos B.Sc. Instructor. Population dynamics  
Sene, F. M. Graduate Student. Ecology and taxonomy of Drosophila  
Souza, H. M. L. de B.Sc. Instructor. Population genetics  
Targa, H. J. B.Sc. Instructor. Ecology and taxonomy of Drosophila  
Tedeschi, M. V. Postgraduate Student. Population genetics  
Toledo, J. S. de B.Sc. Instructor. Population genetics  
Toledo F<sup>o</sup>, S. A. Ph.D. Assistant Professor. Population genetics  
Zanardi, S. M. M. Postgraduate Student. Population genetics.

#### CANADA

Vancouver, British Columbia: University of British Columbia, Department of Zoology, Tel. No. 228-2131, Ext. 54, Code: 604.

Chabun, Paul B.Sc. honors Student. Biochemical genetics  
Duck, Peter B.Sc. honors Student. Cytogenetics  
Erasmus, Udo B.Sc. honors Graduate Student. Mutation  
Procunier, Douglas B.Sc. honors Graduate Student. Radiation genetics  
Tarasoff, Mary (Mrs.) B.Sc. honors Graduate Student Chromosome mechanics

#### CHILE

Santiago: Universidad de Chile, Departamento de Genética, Instituto de Biología "Juan Noé" Av. Zañartu 1042

Fernández, R. Graduate Student. (University of Trujillo, Perú). Population genetics  
Lamborot, M. (Miss) Research Assistant. (at present, University of Bruxelles, Belgium).  
Population genetics; isolating mechanisms

#### COLOMBIA

Bogotá D. E.: Universidad de Los Andes, Institute de Genética.

Terres, E. M. General Help and Collector  
Vega Barve, D. B.Sc. Instructor. General genetics

#### CZECHOSLOVAKIA

Prague 6: Institute of Experimental Botany, Flemming's Square 2

Prokopová, Alena Research Student. Chemical mutagenesis  
Šrám, R. Assistant. Chemical mutagenesis

FRANCE

Gif-sur-Yvette, 91 - Essonne: Centre National de la Recherche Scientifique, Laboratoire de Génétique évolutive et de Biométrie.

Bocquet, C. Professor, Head of Department. Population genetics  
 Bösiger, E. Ph.D. Maître de recherches. Heterosis, sexual selection  
 Chassagnard, M. -T. (Mrs.) Technician.  
 Devaux, J. (Mrs.) Technician.  
 Frey, F. (Mrs.) Technician.  
 Label, E. (Mrs.) Technician.  
 L'Hélias, C. (Miss) Ph.D. Maître de recherches. Cytoplasmic DNA studies of *Drosophila*  
 Louis, M. (Mrs.) Technician.  
 Teissier, G. Professor. Population genetics, quantitative inheritance, biometry  
 Tsacas, L. Ph.D. Chargé de recherches. Systematics of *Drosophilidae*

Orsay: Faculté des Sciences, Laboratoire de Zoologie-Entomologie. Tel. No. 928-57-30

Lauge, G. (Miss) Maître Assistant. Triploid intersexuality in *Drosophila melanogaster*

Paris: Faculté des Sciences, Laboratoire de Biologie Animale 3 C.P.E.M. 7, quai Saint-Bernard Paris 5ème, Tel. No. DAN 07 25 poste 3121

Faugeres, Annie (Mrs.) Graduate Student. Sexual isolation  
 Periquet, Georges Assistant. Populations genetics  
 Petit, Claudine (Mrs.) Maître de Conférences. Populations genetics; sexual isolation

Strasbourg (Bas-Rhin): Faculté des Sciences, Laboratoire de Zoologie.

Sigot, André Professeur sans chaire. CO<sub>2</sub> sensitivity in *Drosophila*

GERMANY

Berlin-Buch: Deutsche Akademie der Wissenschaften Institut für experimentelle Krebsforschung Lindenberger Weg, Tel. No. 56 98 51

Magdon, Erwin Ph.D. Head of Department Radiation Biology. *melanogaster*: radiation genetics  
 Pasternak, Luise (Mrs.) Dr. D. *melanogaster*: chemical mutagens  
 Winterfeld, Gisela Ph.D. Curator of stocks. *melanogaster*: radiation genetics

1 Berlin 33 (Dahlem): Institut für Genetik der Freien Universität Berlin, Rudeloffweg 9, Tel. No. 76 90 640

Bartsch, Hartmut Graduate Student. Cytogenetics  
 Oltze, Almut (Miss) Technical Assistant. Curator of stocks. *melanogaster*: radiation genetics

Munich 2: Zoologisches Institut der Universität, Luisenstrasse 14, Tel. No. 5902359.

Davis, Catherine W. C. (Miss) Puffing  
 Dési, Ildikó (Miss) Technician  
 Illmensee, Karl Developmental genetics  
 Kasten, Renate (Miss) Technician  
 Kress, Horst Puffing  
 Plathner, Irmela (Miss) Curator of Stocks

Münster (Westf.): Institut für Strahlenbiologie der Universität, 17 Hittorfstrasse, Tel. No. 40711/179

Kuhlmann, Dieter Dr. Cytology  
Traut, Anneliese (Mrs.) Technician

Tübingen: Max-Planck-Institut für Biologie, Abteilung Beermann, Spemannstr. 34, Tel. No. 32 47.

Dawid, Igor Biochemistry of DNA  
Gretzmacher, Ilse (Mrs.) Function of the Y chromosome in different *Drosophila* species

Tübingen: Max-Planck-Institut für Biologie, Abteilung Reichardt, 74 Tübingen, Spemannstr. 38 Tel. No. 82393

Franceschini, Nicolas Graduate Student. Quantitative behaviour and neural integration  
Götz, K. G. Dr. Professor. Behavior genetics  
Hengstenberg, Roland Graduate Student. Electrophysiology of the visual system; behavioural genetics  
Stroebel, Charlotte (Miss) Stockkeeper

#### GREAT BRITAIN

Aberdeen, Scotland: University of Aberdeen, Department of Genetics, Tel. No. Aberdeen 40241, Ext. 209

Evans, H. J. B.Sc. Ph.D. Cytogenetics, radiation genetics  
Fox, D. P. B.Sc. M.Sc. Cytogenetics, radiation genetics  
Watson, W. A. F. B.Sc. Ph.D. Mutagenesis

Brighton, Sussex, England: University of Sussex, School of Biology.

Dingley, Fay (Miss) B.Sc. Research Assistant. Physiology of aging

Chalfont St. Giles, Bucks, England: Chester Beatty Research Institute, Institute of Cancer Research, Pollards Wood, Department of Genetics, Tel. No. Little Chalfont 2530.

Coggle, P. R. (Mrs.) B.Sc. Research Assistant  
Parsons, R. (Miss) B.Sc. Research Assistant  
Skinner, J. P. (Miss) B.Sc. Research Assistant

Durham, England: University of Durham, Department of Zoology, Tel No. 3541, Ext. 253.

Bowler, K. Dr. Temperature physiology and ageing in *Drosophila*

London, England: Birkbeck College, University of London, Department of Zoology, Tel. No. LAN 6622.

Lamb, Marion J. (Miss) Ph.D. Radiation and aging; radiation genetics

Sheffield, England: The University, Department of Genetics.

Belt, A. L. (Miss) B.Sc. Graduate Student. Physiological genetics

Sheffield, England: The University, Department of Psychology.

Connolly, K. J. B.Sc. Lecturer. Behaviour genetics

GREECEThessaloniki: The University, Department of General Biology.

Marakis, S. B.Sc. Assistant

Triantaphyllidis, K. B.Sc. Assistant

INDIACalcutta 19: University of Calcutta, Department of Zoology, Cytogenetics Laboratory,  
35 Ballygunge Circular Road, Tel. No. 47-5144

Das, Asoke K. M.Sc. Developmental genetics and crossing over - ananassae

Joardar, Bhabani M.Sc. Systematics and Cytotaxonomy

Mitra, Nivedita B.Sc.(Hons) Developmental genetics

Pal, Tapan K. B.Sc.(Hons) Dosage compensation

Rai Chaudhuri, Anjana M.Sc. Developmental genetics

Sen, Lipi B.Sc.(Hons) Gene physiology

Kalyani, W. Bengal: University of Kalyani, Department of Zoology, Genetics Laboratory.  
Tel. No. 286.

Bhunya, Sankar Prasad M.Sc. Research Scholar. Chemically induced chromosome breakage of  
Drosophila

Das, Anil Kumar B.Sc. Technical Assistant.

New Delhi: Indian Agricultural Research Institute, Botany Division. Tel. No. 51138

Sushil Kumar Ph.D. Genetics, radiation biology

ISRAELJerusalem: The Hebrew University, Laboratory of Genetics. Tel. No. 35291

Braha, Moshe B.Sc. Student. Y suppressed lethals

Cohen, Arnon B.Sc. Student. Induced crossing-over

Goldberg, Susan (Miss) Laboratory Assistant

Itin, Ahuva (Miss) Laboratory Assistant

Lifschytz, Eliezer M.Sc. Research Assistant. Fine structure and function of prune

Ronen, Rachel (Miss) Laboratory Assistant

ITALY

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Danieli, G. A. D.Sc. Research Assistant. Salivary glands  
Matessi, C. Research Student. Population genetics  
Rodino, E. Research Student. Salivary glands

JAPAN

Hiroshima: Hiroshima University, Faculty of Science, Zoological Laboratory

Fujioka, N. Undergraduate. Population genetics

Hiroshima: Hiroshima University, Research Institute for Nuclear Medicine and Biology, Laboratory of Radiation Genetics. Tel. No. 51-1111, Ext. 461.

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Kyoto: Kyoto University, Faculty of Science, Department of Zoology

Imaizumi, Tadashi Dr. Assistant. Physiological genetics and embryology  
Kato, Masaru Dr. Assistant Professor. Biological genetics  
Nakamura, Kenji Dr. Professor. Cytogenetics and physiology  
Okuda, Chizuko (Miss) Technical Assistant. Curator of Stocks

Mitaka: International Christian University, Department of Biology, Division of Natural Science.

Sinotô, Yosito D.Sc. Professor. Cytological studies of salivary chromosomes  
Shoji, Taro Assistant Professor. Cytogenetical studies

Osaka: Osaka University Medical School, Department of Genetics. Tel. No. 443-5531, Ext. 269-271

Ishii, K. Graduate Student. Embryology and Musca genetics

Sakai: University of Osaka Prefecture, Department of Biology

Nakashima-Tanaka, E. Assistant. Developmental genetics; vestigial mutant, genetics of resistance  
Ogaki, M. Dr. Assistant Professor. Genetics of resistance

Sapporo: Hokkaido University, Faculty of Science, Department of Zoology, Tel. No. 71-2111 Ext. 3522 and 2752.

Kaneko, A. Research Assistant. Geographical distribution; cytogenetics; taxonomy; population genetics  
Makino, S. Dr. Professor. Cytogenetics; population genetics  
Momma, E. Dr. Assistant Professor. Geographical distribution; cytogenetics; population genetics  
Shima, T. Research Assistant. Geographical distribution; cytogenetics  
Tokumitsu, T. Graduate Student. Geographical distribution; cytogenetics

Tokyo: Tokyo Metropolitan University, Department of Biology. Tel. No. 717-0111, Ext. 360, 369 and 371

Itô, S. (Miss) Graduate Student. Salivary gland chromosomes  
Oguma, Y. Undergraduate student  
Yamazaki (Ichida), H. (Mrs.) Graduate Student. Biochemical genetics, biochemistry

Tokyo: St. Paul's (Rikkyo University), Department of Physics, Tel. No. (983) 0111

Amagasa, A. Research Assistant. Molecular genetics  
Domon, M. Research Assistant (On leave 1965, Univ. of Toronto, Canada)  
Ishizaka, S. Dr. Lecturer. Radiation biology  
Matsudaira, Y. Dr. Lecturer. Radiation genetics  
Yamasaki, T. Lecturer. Genetics of micro-organisms

Tokyo: Waseda University, School of Education, Department of Biology. Tel. No. (203) 4141, Ext. 258

Ishii, S. Dr. Assistant Professor. Endocrinology  
Iwata, T. Research Assistant. Microbiology  
Nakano, R. (Miss) Research Assistant. Pharmacology  
Numanai, H. Dr. Professor, Head of Department. Physiology and embryology  
Taira, T. Dr. Assistant Professor. Biochemical genetics  
Terada, M. (Miss) Research Assistant. Ecology  
Uda, F. M.S. Research Associate. Biochemical genetics and tissue culture  
Yasumasu, I. Dr. Assistant Professor. Biochemical embryology

NETHERLANDSHaren (Gr.): University of Groningen, Genetical Institute. Tel. No. 05900-45947

Emmens-Pieters, J. (Mrs.) Technical Assistant  
Geertsema, S. P. B.Sc. Graduate Student. Isoenzymes in *D. melanogaster*  
Olthoff, H. M. Technical Assistant  
Veeman, D. B.Sc. Graduate Student. Lethals in laboratory populations

Utrecht: Genetisch Instituut der Rijksuniversiteit, Opaalweg 20. Tel. No. 22541

Hasselaar, J. B.Sc. Demonstrator

NORWAY

Bergen: University of Bergen, Institute of Anatomy

Abro, Arnold Assistant Professor. Spermatogenesis, radiation effects, studied with electron microscope and fluorescence

Blindern: University of Oslo, Institute of General Genetics, P.O.Box 1031

Föyn, Björn Professor. Chairman

Oslo: Norsk Hydro's Cancer Research Institute, Laboratory for Genetics.

Brigteen, A. K. (Miss) Research Assistant

Kralheim, S. (Miss) Research Assistant

Kumar Sushil Ph.D. Cytology

Pearce, S. (Miss) Research Assistant

RHODESIA

Salisbury: University College of Rhodesia, Department of Biological Science.

Paterson, H. E. Population genetics, speciation, cytology

SOUTH AFRICA

Johannesburg: University of the Witwatersrand, Department of Zoology. Tel. No. 835-8181.

Nolte, D. J. D.Sc. Reader in Genetics. Polygenes

Scott, Doreen (Miss) Curator of Stocks

Stellenbosch: University of Stellenbosch, Department of Genetics. Tel No. 2211

Allan, J. S. Ph.D. Quantitative genetics

Louw, J. H. Ph.D. Quantitative genetics

SWEDEN

Uppsala: University of Uppsala, Department of Genetics

Johansson, V. Assistant. *Melanogaster*; mutations

Tengblad, E. Technical Assistant. Curator of Stocks

SWITZERLANDZürich: Zoologisches Institut u. Museum der Universität.

Furrer, Robert Graduate Student. Imaginal blastemas  
Klenk, Karl Graduate Student. Imaginal blastemas  
Weideli, Hansjörg Graduate Student. Amino acids  
Widmer, Bruno Graduate Student. Amino acids  
Winzeler, Tobias Graduate Student. Fat body

USSRObninsk, Kaluga District: Institute of Medical Radiology, Department of General Radiobiology and Genetics.

Timofeeff-Ressovsky, N. W. Head of Department

Besides Dr. Timofeeff-Ressovsky and his wife, the *Drosophila* group includes half a dozen young scientists. The group is interested in microevolution, population genetics, mutability of single loci in *Drosophila*, the mechanisms of chromosome non-disjunction in *Drosophila*, radiation effects on non-disjunction and crossing over, and genetic aspects of onthogenesis in *Drosophila*.

UNITED STATESAda, Ohio: Ohio Northern University, Department of Biology, Tel. No. (419) 634-3015.

Butler, Daniel R. Ph.D. Associate Professor. General biology, genetics

Albany, New York, 12203: State University of New York at Albany, Department of Biology, Tel. No. (518) 457-3300.

Muckenthaler, Florian A. Ph.D. Assistant Professor. Oogenesis and development

Albuquerque, New Mexico, 87106: University of New Mexico, Department of Biology, Tel. No. (505) 277-0111, Ext. 5140.

Johnson, William W. Ph.D. Assistant Professor. Experimental population genetics  
Pyati, Jagadeesh B.Sc. Graduate Student. Experimental population genetics

Amherst, Massachusetts, 01002: Amherst College, Department of Biology, Tel. No. (413) 253-2561

Casey, Lucy (Mrs.) Curator of Stocks. Research Assistant.  
Hexter, W. M. Ph.D. Professor. Genetic fine structure and crossing over  
Ives, P. T. Ph.D. Research Associate. Radiation and population genetics

Plough, H. H. Professor Emeritus. Mutation and environmental effects  
Russell, Phyllis (Mrs.) Research Assistant  
Tiffany, Barbara (Miss) Technical Assistant  
Yost, H. T., Jr. Ph.D. Professor. Cell particulates, radiation effects, crossing over

Ann Arbor, Michigan, 48104: University of Michigan, Department of Zoology, Cytogenetics  
Laboratory of Carnegie Institution of Washington, Tel. No. (313) 764-1454

Colot, Hildur Part-time Technical Assistant  
Dick, Carolyn Part-time Technical Assistant and Stockkeeper  
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 Devaux, J. France, Gif-sur-Yvette  
 Diamantis, B. St. Paul, Minnesota  
 Dick, C. Ann Arbor, Michigan  
 Dingley, F. England, Brighton  
 Domon, M. Japan, Tokyo  
 Duck, P. Canada, Vancouver  
 Duffy, J. P. Jamaica, New York  
 Edenberg, H. New York, New York  
 Edington, C. W. Washington, D. C.  
 Edwards, J. W. Winston-Salem, North Carolina  
 Eggert, R. Davis, California  
 Eichenberger, E. Pasadena, California  
 Einspruch, M. College Park, Maryland  
 Eisenstein, L. I. Tempe, Arizona  
 Emmens-Pieters, J. Netherlands, Haren  
 Enzenhofer, R. Austria, Vienna  
 Epstein, A. Fayetteville, Arkansas  
 Erasmus, U. Canada, Vancouver  
 Evans, H. J. Scotland, Aberdeen  
 Faugeres, A. France, Paris  
 Fernández, R. Chile, Santiago  
 Feuerbach, H. Austria, Vienna  
 Fischetti, T. College Park, Maryland  
 Flippen, D. Houston, Texas  
 Forbes, C. Moscow, Idaho  
 Fowler, G. Providence, Rhode Island  
 Fox, D. P. Scotland, Aberdeen  
 Föyn, B. Norway, Blindern  
 França, Z. M. Brazil, São Paulo  
 Franklin, I. D. Philadelphia, Pennsylvania  
 Franklin, S. Houston, Texas  
 Frey, F. France, Gif-sur-Yvette  
 Frye, M. E. Irvine, Kentucky  
 Frye, S. H. Irvine, Kentucky  
 Fujioka, N. Japan, Hiroshima  
 Furrer, R. Switzerland, Zürich  
 Gabay, S. J. Urbana, Illinois  
 Garcia, M. Bowling Green, Ohio  
 Garner, I. Bowling Green, Ohio  
 Gearhart, J. Ithica, New York  
 Geertsema, S. P. Netherlands, Haren  
 Geoghegan, J. L. Lexington, Kentucky  
 Gerdes, R. A. College Station, Texas  
 Goldberg, S. Israel, Jerusalem  
 Gottardi, A. Italy, Milan  
 Götz, K. G. Germany, Tübingen  
 Grahn, D. T. Pullman, Washington  
 Green, R. C. Providence, Rhode Island  
 Gretzmacher, I. Germany, Tübingen

- Haas, F. Houston, Texas  
Halyard, A. Philadelphia, Pennsylvania  
Hardjosworo, P. Ann Arbor, Michigan  
Hasselaar, J. Netherlands, Utrecht  
Hatch, M. Fairfax, Virginia  
Hayes, N. Duarte, California  
Heller, F. Ithaca, New York  
Hexter, W. M. Amherst, Massachusetts  
Hiatt, V. S. St. Paul, Minnesota  
Homyk, T. Chapel Hill, North Carolina  
Hooper, G. B. Poughkeepsie, New York  
Hosgood, S. M. W. Australia, Melbourne  
Hubbard, W. B. Chapel Hill, North Carolina  
Hughes, J. Bowling Green, Ohio  
Hunt, P. Berkeley, California  
Illmensee, K. Germany, Munich  
Imaizumi, T. Japan, Kyoto  
Irvine, S. R. Logan, Utah  
Ishii, K. Japan, Osaka  
Ishii, S. Japan, Tokyo  
Ishizaka, S. Japan, Tokyo  
Itin, A. Israel, Jerusalem  
Ito, R. Ann Arbor, Michigan  
Itô, S. Japan, Tokyo  
Ives, P. T. Amherst, Massachusetts  
Iwata, T. Japan, Tokyo  
Jacobs, J. L. Pullman, Washington  
Jewell, J. A. Wilmington, Ohio  
Joardar, B. India, Calcutta  
Johansson, V. Sweden, Uppsala  
Johnsen, R. C. Providence, Rhode Island  
Johnson, W. W. Albuquerque, New Mexico  
Johnston, H. H. Wilmington, Ohio  
Kalicki, H. G. Garden City, L.I., New York  
Kaneko, A. Japan, Sapporo  
Kasten, R. Germany, Munich  
Kato, M. Japan, Kyoto  
Kidera, K. Lake Forest, Illinois  
Kieso, R. A. Tempe, Arizona  
Kim, M. S. Providence, Rhode Island  
King, P. Houston, Texas  
Kishimoto, Y. Japan, Kobe  
Kitagawa, O. New York, New York  
Klenck, K. Switzerland, Zürich  
Klug, W. S. Evanston, Illinois  
Koenig, P. Chapel Hill, North Carolina  
Komasa, N. J. Davenport, Iowa  
Kralheim, S. Norway, Oslo  
Kress, H. Germany, Munich  
Krivshenko, E. D. Rochester, New York  
Krivshenko, J. D. Rochester, New York  
Kuhlmann, D. Germany, Münster  
Kuhn, R. A. Tempe, Arizona  
Kumar, S. Norway, Oslo  
Label, E. France, Gif-sur-Yvette  
Lamb, M. T. England, London  
Lamborot, M. Chile, Santiago  
Lauge, G. France, Orsay  
Leahy, M. G. Los Angeles, California  
Lesnik, L. Philadelphia, Pennsylvania  
Levitan, M. Fairfax, Virginia  
L'Hélias, C. France, Gif-sur-Yvette  
Lifschytz, E. Israel, Jerusalem  
Lott, R. G. Tempe, Arizona  
Louis, M. France, Gif-sur-Yvette  
Louw, J. H. South Africa, Stellenbosch  
Luttrell, G. Bowling Green, Ohio  
Lyon, M. Ann Arbor, Michigan  
MacBean, I. T., Australia, Melbourne  
Magalhães, L. E. de Brazil, São Paulo  
Magdon, E. Germany, Berlin  
Maquire, J. Galesburg, Illinois  
Mahowald, A. P. Milwaukee, Wisconsin  
Makino, S. Japan, Sapporo  
Malich, C. W. Moffett Field, California  
Momma, E. Japan, Sapporo  
Marakis, S. Greece, Thessaloniki  
Marzluf, G. Milwaukee, Wisconsin  
Matessi, C. Italy, Padova  
Matsudaira, Y. Japan, Tokyo  
McArthur, E. Auburn, Alabama  
McMahon, J. A. Dayton, Ohio  
Merrell, D. J. Minneapolis, Minnesota  
Miller, M. A. Bowling Green, Ohio  
Mitra, N. India, Calcutta  
Montgomery, R. Houston, Texas  
Moore, L. Northridge, California  
Morgan, M. Australia, Adelaide  
Moseman, M. L. Knoxville, Tennessee  
Muckenthaler, A. Albany, New York  
Mukerjee, D. Houston, Texas  
Murakami, A. Bowling Green, Ohio  
Musser, P. Bowling Green, Ohio  
Mylar, C. Houston, Texas  
Mylar, P. Houston, Texas  
Nakamura, K. Japan, Kyoto  
Nakano, R. Japan, Tokyo  
Nakashima-Tanaka, E. Japan, Sakai  
Nagle, C. College Park, Maryland  
Nagy, D. Bowling Green, Ohio  
Narayanan, Y. St. Louis, Missouri  
Neeley, J. C. Atchison, Kansas  
Nolte, D. J. South Africa, Johannesburg  
Norman, S. D. DeKalb, Illinois  
Nova, M. Stony Brook, New York  
Numanoi, H. Japan, Tokyo  
Obasun, D. Medford, Massachusetts  
O'Brien, S. J. Ithaca, New York  
Ogaki, M. Japan, Sakai  
Oguma, Y. Japan, Tokyo  
Ohata, F. Japan, Hiroshima  
Ohh, B. K. Australia, Sydney  
Okuda, C. Japan, Kyoto  
Olander, R. Galesburg, Illinois  
Olthoff, H. M. Netherlands, Haren  
Oltze, A. Germany, Berlin  
Osborn, A. F. Wilmington, Ohio  
Owuor, M. Ithaca, New York  
Pal, T. K. India, Calcutta  
Paley, J. P. Urbana, Illinois  
Parker, M. Houston, Texas  
Parry, D. Seattle, Washington  
Parsons, P. A. Australia, Melbourne  
Parsons, R. England, Chalfont St. Giles  
Pasternak, L. Germany, Berlin  
Pateron, H. E. Rhodesia, Salisbury  
Pavan, C. Brazil, São Paulo  
Pearce, S. Norway, Oslo

- Periquet, G. France, Paris  
Petit, C. France, Paris  
Pettit, B. Milwaukee, Wisconsin  
Pipkin, S. B. Washington, D.C.  
Pittendrigh, C. S. Princeton, New Jersey  
Plathner, I. Germany, Munich  
Plough, H. H. Amherst, Massachusetts  
Pontecorvo, G. Scotland, Glasgow  
Potter, P. R. DeKalb, Illinois  
Powers, L. M. Washington, D.C.  
Prinz, R. Storrs, Connecticut  
Procunier, D. Canada, Vancouver  
Prokopová, A. Czechoslovakia, Prague  
Pyati, J. Albuquerque, New Mexico  
Pyle, A. Iowa City, Iowa  
Quamme, H. Madison, Wisconsin  
Rai Chaudhuri, A. India, Calcutta  
Rangaswamy, V. S. Bowling Green, Ohio  
Rasch, E. M. Milwaukee, Wisconsin  
Rasch, R. W. Milwaukee, Wisconsin  
Reeves, C. R. Davis, California  
Reid, C. Salt Lake City, Utah  
Rice, C. S. Davenport, Iowa  
Riker, K. C. Providence, Rhode Island  
Robbins, L. G. Seattle, Washington  
Rodino, E. Italy, Padova  
Rodman, T. New York, New York  
Rollins, H. O. Knoxville, Tennessee  
Ronen, R. Israel, Jerusalem  
Rose, R. W. Philadelphia, Pennsylvania  
Rounds, S. St. Louis, Missouri  
Rowan, M. J. Houston, Texas  
Rowan, M. J. Rochester, New York  
Ruffin, V. Auburn, Alabama  
Runge, R. Lake Forest, Illinois  
Russell, P. Amherst, Massachusetts  
Samuel, E. M. Boston, Massachusetts  
Santos, E. P. dos Brazil, São Paulo  
Sauza, H. M. L. de Brazil, São Paulo  
Sayers, E. R. Tuscaloosa, Alabama  
Scandlyn, B. J. Knoxville, Tennessee  
Schneider, I. Washington, D.C.  
Scott, J. Providence, Rhode Island  
Schlesinger, K. Boulder, Colorado  
Scott, D. South Africa, Johannesburg  
Sekura, R. Madison, New Jersey  
Sen, L. India, Calcutta  
Sena, E. Madison, Wisconsin  
Sene, F. M. Brazil, São Paulo  
Sergio, D. Washington, D.C.  
Shapin, S. A. Madison, Wisconsin  
Sherald, A. College Park, Maryland  
Sherwin, R. Chicago, Illinois  
Shima, T. Japan, Sapporo  
Shoji, T. Japan, Mitaka  
Shull, K. Tuscaloosa, Alabama  
Sigot, A. France, Strasbourg  
Sinotô, Yosito Japan, Mitaka  
Skinner, J. P. England, Chalfont St. Giles  
Slutzky, K. Stony Brook, New York  
Smit, I. Buarte, California  
Smith, J. D. College Station, Texas  
Snyder, L. A. St. Paul, Minnesota  
Snyder, S. St. Louis, Missouri  
Sori, A. College Park, Maryland  
Spiess, E. B. Chicago, Illinois  
Spiess, L. D. Chicago, Illinois  
Spirito, S. E. Argentina, Buenos Aires  
Sram, R. Czechoslovakia, Prague  
Stables, R. Chapel Hill, North Carolina  
Stewart, D. J. Tempe, Arizona  
Storm, L. W. Whitewater, Wisconsin  
Stroebe, C. C. Germany, Tübingen  
Sulerud, R. L. Minneapolis, Minnesota  
Sushil, K. India, New Delhi  
Sutton, A. Salt Lake City, Utah  
Szendy, S. Jamaica, New York  
Taira, T. Japan, Tokyo  
Tarasoff, M. Canada, Vancouver  
Targa, H. J. Brazil, São Paulo  
Taylor, D. Dayton, Ohio  
Tedeschi, M. V. Brazil, São Paulo  
Teissier, G. France, Gif-sur-Yvette  
Tengblad, E. Sweden, Uppsala  
Terada, M. Japan, Tokyo  
Terres, E. M. Colombia, Bogotá  
Thompson, J. Jr. Norman, Oklahoma  
Thompson, M. Bowling Green, Ohio  
Tiffany, B. Amherst, Massachusetts  
Timofeeff-Ressovsky, N. W. USSR, Obninsk  
Tokumitsu, T. Japan, Sapporo  
Toledo, F. S. A. Brazil, São Paulo  
Toledo, J. S. de Brazil, São Paulo  
Tonetti, A. M. DeKalb, Illinois  
Torgerson, R. Salt Lake City, Utah  
Traut, A. Germany, Münster  
Triantaphyllidis, K. Greece, Thessaloniki  
Trzaski, T. J. Garden City, L.I., New York  
Tsacas, L. France, Gif-sur-Yvette  
U, R. New York, New York  
Uda, F. Japan, Tokyo  
Valencia, R. Argentina, Buenos Aires  
van Arkel, G. A. Netherlands, Utrecht  
Van Valen, L. Chicago, Illinois  
Veeman, D. Netherlands, Haren  
Vega Barve, D. Colombia, Bogotá  
Vinje, M. M. Davenport, Iowa  
Voigtlander, J. A. Madison, Wisconsin  
Vollmer, C. Chapel Hill, North Carolina  
Walkup, B. E. Bellingham, Washington  
Wallis, E. T. Bowling Green, Ohio  
Watson, J. E. Auburn, Alabama  
Watson, W. A. F. Scotland, Aberdeen  
Webster, R. A. Argonne, Illinois  
Weideli, H. Switzerland, Zürich  
Westenhaver, R. Auburn, Alabama  
Whitney, J. B. Chapel Hill, North Carolina  
Widmer, B. Switzerland, Zürich  
Wiedmeier, V. T. Davenport, Iowa  
Williams, G. Berkeley, California  
Williams, S. Philadelphia, Pennsylvania  
Williamson, D. L. Philadelphia, Pennsylvania  
Williamson, J. H. Oak Ridge, Tennessee  
Wilson, K. Bethesda, Maryland  
Winterfeld, G. Germany, Berlin  
Winzeler, T. Switzerland, Zürich  
Woolf, C. M. Tempe, Arizona  
Yamasaki, T. Japan, Tokyo

Yamazaki (Ichida), H. Japan, Tokyo  
Yasumasu, I. Japan, Tokyo  
Yoon, J. S. Houston, Texas  
Yoon, K. S. Houston, Texas  
Yost, H. T. Jr. Amherst, Massachusetts

Young, S. Ann Arbor, Michigan  
Zanardi, S. M. M. Brazil, São Paulo  
Zasada, C. Bowling Green, Ohio  
Zimmering, S. Providence, Rhode Island  
Zucker, N. New York, New York

For a lark, I submit a parody of Shelley's "To A Lark".

To a Puff

Hail to thee, puffed region!  
Inert thou never wert,  
That from helix or near it,  
Unravelled is thy heart  
Of double strains, epigenetisch' Art.

Looser still and looser  
From the core thou springest  
Like a test tube primer;  
The program at its purest,  
Unravelling still more, instructions ever singest.

In the phaséd lighting  
Of the contrasted sun,  
In sequence, we are finding,  
Thou dost emote and run  
To hide as bashful coils, when thy bidding's been done.

We do verge on madness  
As we strain to know  
What may be the cues  
By which you come and go -  
A thing wherein we feel there is some hidden want.

H. J. Barr,  
Lecturer in Zoology,  
The University of Wisconsin