Table 1. Variance ratio analyses for the 11 indicated mutagenesis experiments involving six chromosomal intervals in D. melanogaster.

Region analyzed	No. of lethal complementation groups	Mutagen	Mean no. of alleles per complementation group	Variance	Variance ratio	Reference
Chromosome 4	36	Spontaneous	0.806	1.190	1.477†	Hochman 1973
		X-rays	0.833	2.486	2.983***	
		EMS	3.111	18.273	5.873***	
		ICR-170	0.306	0.733	2.397***	
2R Heterochromatin	6	EMS	14.000	202.800	14.486**	Hilliker 1976
2L Heterochromatin	7	EMS	4.000	20.000	5.000**	Hilliker 1976
Df(3R)ry^{614}	9	EMS	3.778	23.444	6.206**	Hilliker et al. 1980
Df(3R)ry^{619}	15	EMS	4.933	20.210	4.097**	Hilliker et al. 1980
Zeste-white	15	EMS	5.533	53.552	9.678***	Lim & Snyder 1974
		TEM	1.800	10.029	5.571***	
		MMS	7.000	37.286	5.327***	Liu & Lim 1975

† $0.05 < P < 0.10$ ** $P < 0.01$ *** $P < 0.001$

Supported by a research grant, GM-09886, from the Public Health Service.

References: Cohen, A.C. Jr. 1960, Biometrics 16:203-211; Gilbert, N. 1973, Biometrical Interpretation, Oxford Univ. Press, London; Hilliker, A.J. 1976, Genetics 83:765-782; _____, S.H. Clark, A. Chovnick and W.M. Gelbart 1980, Genetics (in press); Hochman, B. 1973, Cold Spring Harbor Symp. Quant. Biol. 38:581-589; Lim, J.K. and L.A. Snyder 1974, Genet. Res. 24: 1-10; Liu, C.P. and J.K. Lim 1975, Genetics 79:601-611.

Hilliker, A.J.*, S.H. Clark, W.M. Gelbart** and A. Chovnick. University of Connecticut, Storrs, Connecticut. Cytogenetic analysis of the rosy micro-region, polytene chromosome interval 87D2-4; 87E12-F1, of D. melanogaster.

Figure 1 presents a summary of our cytogenetic analysis of the rosy micro-region. A total of 153 recessive lethals falling into this region were subdivided by inter se complementation, and complementation tests with rosy region deficiencies, into 20 lethal complementation groups. Adjacent complementation groups illustrated within parentheses in Fig. 1 have not been separated by deficiency from one another, hence their relative left-right order is unknown.

The recessive lethals employed in this study are listed in Table 1 according to complementation group (beginning with the most proximally located and continuing through to the most distal). Each listed recessive lethal mutation is accompanied by a description of its source, the mutagen used and, where possible, the isogenic third chromosome on which it was constructed (designated by the specific ry^{+} allele carried on that chromosome).

A majority of the 153 recessive lethals listed in Table 1 were synthesized by Hilliker and Clark (120) as lethal alleles of Df(3R)ry^{614} (34), Df(3R)ry^{619} (83) and Df(3R)ry^{75} (3) (see Table 2). These recessive lethals were recovered from the treatment of iso-3 males with either 0.025M EMS (Lewis and Bacher 1968) or gamma radiation (2000 to 4000 rads).

Since the majority of recessive lethals were synthesized as alleles of Df(3R)ry^{614} or Df(3R)ry^{619}, the region encompassed by these deficiencies, 87D2-4; 87E12-F1, defines the rosy micro-region.

*,**Present addresses: *CSIRO, Canberra City, ACT, Australia; **Harvard University, Cambridge, Massachusetts.

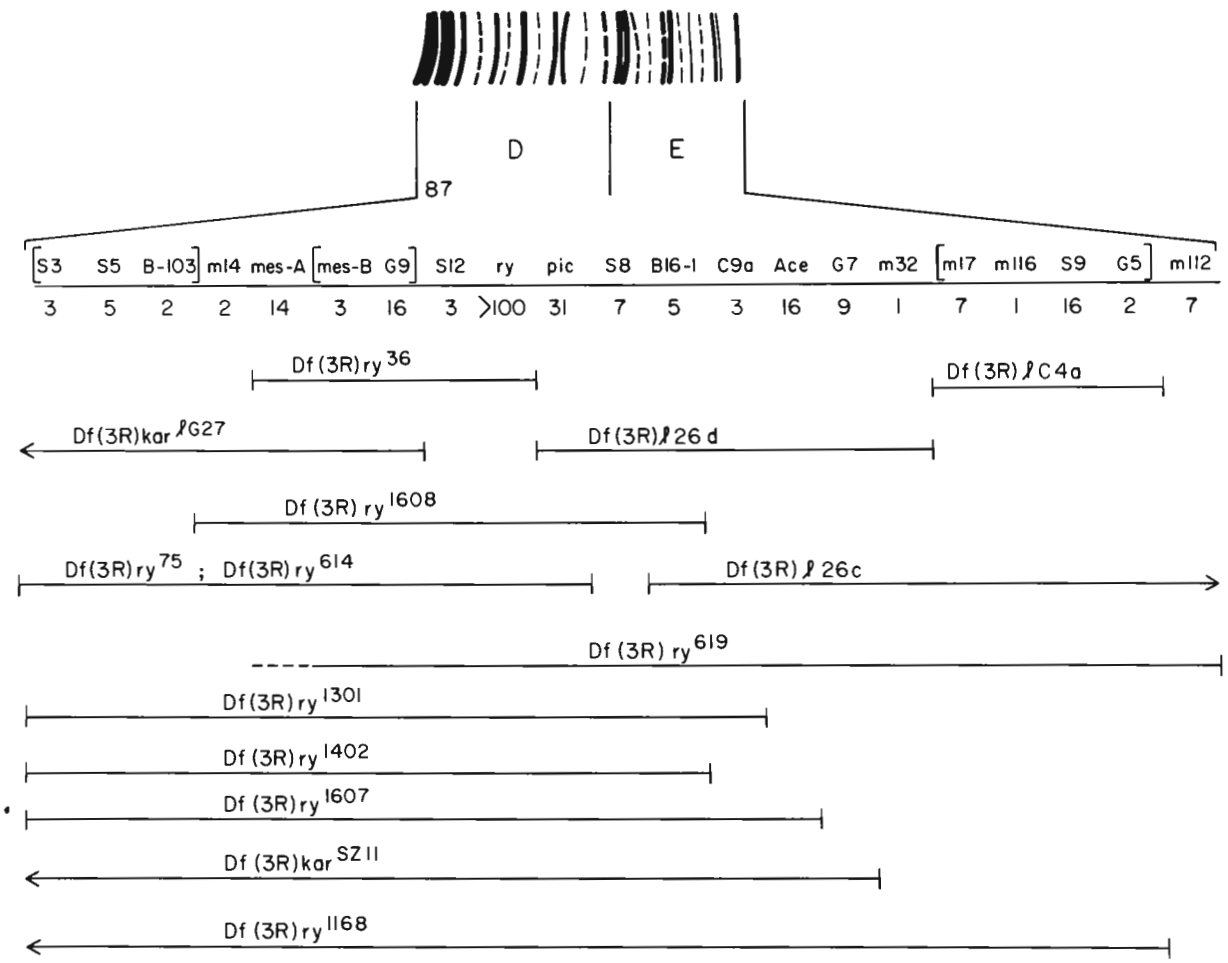

Figure 1. Summary of the cytogenetic analysis of polytene chromosome region 87DE.

The rosy region recessive lethals synthesized by Gelbart, by Schalet and by Deland (Table 1) were selected as lethal alleles of larger deficiencies which encompassed the entire 87D2-4; 87E12-F1 interval. Further details on the synthesis of the recessive lethals provided by Schalet and by Deland may be found in Schalet, Kernaghan and Chovnick (1964) and Deland (1971).

Each rosy region recessive lethal was subsequently tested for complementation with the rosy region deficiencies listed in Table 2.

On the basis of complementation tests with rosy region deficiencies, the recessive lethals fell into 14 clusters. Within each cluster, ALL inter se combinations of recessive lethals were examined for complementation. Further, when the recessive lethals within each cluster were resolved into complementation groups, each recessive lethal within each cluster was tested for complementation with a representative allele of each of the complementation groups defining the immediately adjacent clusters. Finally, most recessive lethals were tested for complementation with a representative allele of each complementation group within the rosy micro-region.

Let us now consider, briefly, each functional group in terms of phenotype and complementation pattern, beginning with the most proximally located.

The 1(3)S3 complementation group is associated with two additional, semi-lethal alleles, 1(3)A34-1 and 1(3)A46-1. The 1(3)A46-1 allele is associated with a mean viability of approximately 15% when heterozygous with 1(3)S3. Both alleles, when heterozygous with 1(3)S3, are associated with variation in dorsocentral bristle number and length. Further, among heterozygotes for the semi-lethal alleles and 1(3)S3, females greatly outnumber males. The 1(3)A34-1/1(3)A46-1 heterozygotes are associated with about 75% viability and normal bristle morphology and sex ratio.

Table 1. Recessive lethals listed according to complementation group.

Lethal allele	Isogenic third chromosome	Mutagen	Source
1(3)S3	--	X-rays	Schalet
1(3)A34-1	ry+4	EMS	Hilliker, Clark
1(3)A46-1	ry+4	EMS	Hilliker, Clark
1(3)S5	--	X-rays	Schalet
1(3)C8a	--	X-rays	Chovnick
1(3)E4a	--	X-rays	Chovnick
1(3)G12	ry+2	EMS	Gelbart
1(3)9-13	ry+11	EMS	Hilliker, Clark
1(3)B-103	ry+11	EMS	Hilliker, Clark
1(3)A6-1	ry+4	EMS	Hilliker, Clark
1(3)m14	--	EMS	Deland
1(3)10-194	ry+11	EMS	Hilliker, Clark
1(3)mes-1A	--	X-rays	Schalet
1(3)G2	ry+2	EMS	Gelbart
1(3)G3	ry+2	EMS	Gelbart
1(3)G8	ry+2	EMS	Gelbart
1(3)G19	ry+2	EMS	Gelbart
1(3)A27-2	ry+4	EMS	Hilliker, Clark
1(3)10-140	ry+11	EMS	Hilliker, Clark
1(3)2-34	ry+11	EMS	Hilliker, Clark
1(3)8-9	ry+11	EMS	Hilliker, Clark
1(3)A13-1	ry+4	EMS	Hilliker, Clark
1(3)13-62	ry+11	EMS	Hilliker, Clark
1(3)4-22	ry+11	EMS	Hilliker, Clark
1(3)B26-1	ry+11	EMS	Hilliker, Clark
1(3)A12-2	ry+4	EMS	Hilliker, Clark
1(3)mes-4B	--	X-rays	Schalet
1(3)34-2	ry+4	EMS	Hilliker, Clark
1(3)B14-1	ry+11	EMS	Hilliker, Clark
1(3)G9	ry+2	EMS	Gelbart
1(3)G15	ry+2	EMS	Gelbart
1(3)G21	ry+2	EMS	Gelbart
1(3)6-120	ry+11	EMS	Hilliker, Clark
1(3)2-228	ry+11	EMS	Hilliker, Clark
1(3)11-147	ry+11	EMS	Hilliker, Clark
1(3)A39-2	ry+4	EMS	Hilliker, Clark
1(3)B10-1	ry+11	EMS	Hilliker, Clark
1(3)B13-2	ry+11	EMS	Hilliker, Clark
1(3)B13-3	ry+11	EMS	Hilliker, Clark
1(3)B23-1	ry+11	EMS	Hilliker, Clark
1(3)B25-1	ry+11	EMS	Hilliker, Clark
1(3)H37	ry+11	EMS	Hilliker, Clark
1(3)H73	ry+11	EMS	Hilliker, Clark
1(3)H2	ry+11	Gamma	Hilliker, Clark
1(3)H23	ry+11	Gamma	Hilliker, Clark
1(3)S12	ry+11	X-rays	Schalet
1(3)G1	ry+2	EMS	Gelbart
1(3)B21-4	ry+11	EMS	Hilliker, Clark
1(3)pic^{21}	--	X-rays	Schalet
1(3)m10	--	EMS	Deland
1(3)G23	ry+4	EMS	Gelbart
1(3)G26	ps612	Gamma	Gelbart
1(3)8-107	ry+11	EMS	Hilliker, Clark
1(3)12-196	ry+11	EMS	Hilliker, Clark
1(3)A34-3	ry+4	EMS	Hilliker, Clark
1(3)33-1	ry+4	EMS	Hilliker, Clark
1(3)D-64	ry+11	EMS	Hilliker, Clark
1(3)A3-3	ry+4	EMS	Hilliker, Clark
1(3)A112	ry+11	EMS	Hilliker, Clark
1(3)A80	ry+11	EMS	Hilliker, Clark
1(3)A19-1	ry+4	EMS	Hilliker, Clark
1(3)A42-1	ry+4	EMS	Hilliker, Clark
1(3)3-119	ry+11	EMS	Hilliker, Clark
1(3)A12-3	ry+4	EMS	Hilliker, Clark
1(3)A19-2	ry+4	EMS	Hilliker, Clark
1(3)8-181	ry+11	EMS	Hilliker, Clark
1(3)A111	ry+11	EMS	Hilliker, Clark
1(3)B2-4	ry+11	EMS	Hilliker, Clark
1(3)H10	ry+11	EMS	Hilliker, Clark
1(3)H49	ry+11	EMS	Hilliker, Clark
1(3)H51	ry+11	EMS	Hilliker, Clark
1(3)H59	ry+11	EMS	Hilliker, Clark
1(3)H72	ry+11	EMS	Hilliker, Clark

Table 1. [continued]

Lethal allele	Isogenic third chromosome	Mutagen	Source
1(3)H19	ry+11	Gamma	Hilliker, Clark
1(3)H22	ry+11	Gamma	Hilliker, Clark
1(3)C-9-2	ry+11	Gamma	Hilliker, Clark
1(3)C-17-3	ry+11	Gamma	Hilliker, Clark
1(3)C-18-1	ry+11	Gamma	Hilliker, Clark
1(3)H54	ry+11	EMS	Hilliker, Clark
1(3)S8	--	X-rays	Schalet
1(3)B21-2	ry+11	EMS	Hilliker, Clark
1(3)B30-1	ry+11	EMS	Hilliker, Clark
1(3)H79	ry+11	EMS	Hilliker, Clark
1(3)B13-4	ry+11	EMS	Hilliker, Clark
1(3)H66	ry+11	EMS	Hilliker, Clark
1(3)H9	ry+11	Gamma	Hilliker, Clark
1(3)B16-1	ry+11	EMS	Hilliker, Clark
1(3)B16-4	ry+11	EMS	Hilliker, Clark
1(3)B27-2	ry+11	EMS	Hilliker, Clark
1(3)H13	ry+11	EMS	Hilliker, Clark
1(3)H69	ry+11	EMS	Hilliker, Clark
1(3)C9a	--	X-rays	Chovnick
1(3)B2-6	ry+11	EMS	Hilliker, Clark
1(3)B26-2	ry+11	EMS	Hilliker, Clark
1(3)26	--	X-rays	Schalet
1(3)m15	--	EMS	Deland
1(3)B2-5	ry+11	EMS	Hilliker, Clark
1(3)B4-2	ry+11	EMS	Hilliker, Clark
1(3)B8-2	ry+11	EMS	Hilliker, Clark
1(3)B15-2	ry+11	EMS	Hilliker, Clark
1(3)B22-1	ry+11	EMS	Hilliker, Clark
1(3)B22-2	ry+11	EMS	Hilliker, Clark
1(3)B27-1	ry+11	EMS	Hilliker, Clark
1(3)B29-1	ry+11	EMS	Hilliker, Clark
1(3)B29-2	ry+11	EMS	Hilliker, Clark
1(3)H36	ry+11	EMS	Hilliker, Clark
1(3)H41	ry+11	EMS	Hilliker, Clark
1(3)H89	ry+11	EMS	Hilliker, Clark
1(3)B21-5	ry+11	EMS	Hilliker, Clark
1(3)H15	ry+11	EMS	Hilliker, Clark
1(3)G7	ry+2	EMS	Gelbart
1(3)B1-3	ry+11	EMS	Hilliker, Clark
1(3)B9-1	ry+11	EMS	Hilliker, Clark
1(3)B13-1	ry+11	EMS	Hilliker, Clark
1(3)B30-2	ry+11	EMS	Hilliker, Clark
1(3)H34	ry+11	EMS	Hilliker, Clark
1(3)H91	ry+11	EMS	Hilliker, Clark
1(3)H75	ry+11	EMS	Hilliker, Clark
1(3)H20	ry+11	Gamma	Hilliker, Clark
1(3)m32	--	EMS	Deland
1(3)m17	--	EMS	Deland
1(3)B1-1	ry+11	EMS	Hilliker, Clark
1(3)B11-1	ry+11	EMS	Hilliker, Clark
1(3)B16-3	ry+11	EMS	Hilliker, Clark
1(3)B26-3	ry+11	EMS	Hilliker, Clark
1(3)H45	ry+11	EMS	Hilliker, Clark
1(3)H77	ry+11	EMS	Hilliker, Clark
1(3)m116	--	EMS	Deland
1(3)S9	--	X-rays	Schalet
1(3)m102	--	EMS	Deland
1(3)B1-2	ry+11	EMS	Hilliker, Clark
1(3)B1-5	ry+11	EMS	Hilliker, Clark
1(3)B2-3	ry+11	EMS	Hilliker, Clark
1(3)B8-1	ry+11	EMS	Hilliker, Clark
1(3)B8-4	ry+11	EMS	Hilliker, Clark
1(3)B12-2	ry+11	EMS	Hilliker, Clark
1(3)B15-1	ry+11	EMS	Hilliker, Clark
1(3)21-3	ry+11	EMS	Hilliker, Clark
1(3)B26-4	ry+11	EMS	Hilliker, Clark
1(3)B28-1	ry+11	EMS	Hilliker, Clark
1(3)H9	ry+11	EMS	Hilliker, Clark
1(3)H30	ry+11	EMS	Hilliker, Clark
1(3)H32	ry+11	EMS	Hilliker, Clark
1(3)H57	ry+11	EMS	Hilliker, Clark
1(3)G5	ry+2	EMS	Gelbart
1(3)B4-1	ry+11	EMS	Hilliker, Clark

Table 1. [continued]

Lethal allele	Isogenic third chromosome	Mutagen	Source
1(3)m112	--	EMS	Deland
1(3)B9-2	ry+11	EMS	Hilliker, Clark
1(3)B16-2	ry+11	EMS	Hilliker, Clark
1(3)B17-1	ry+11	EMS	Hilliker, Clark
1(3)H21	ry+11	EMS	Hilliker, Clark
1(3)H24	ry+11	Gamma	Hilliker, Clark
1(3)H25	ry+11	Gamma	Hilliker, Clark

Table 2. Deficiencies employed in this analysis and their breakpoints.

Deficiency	Breakpoints	Synthesis	Cytology
$Df(3R)ry^{36}$	No visible deletion	Schalet	Gelbart, Hilliker, Lefevre
$Df(3R)ry^{75}$	Df(3R)87D1-2; 87D14-E1	Schalet	Gelbart, Lefevre
$Df(3R)ry^{81}$	Df(3R)87C1-3; 87D14-E2	Schalet	Gelbart, Lefevre
$Df(3R)ry^{614}$	Df(3R)87D2-4; 87D11-14	Gelbart	Gelbart, Hilliker
$Df(3R)ry^{619}$	Df(3R)87D7-9; 87E12-F1	Gelbart	Gelbart, Hilliker
$Df(3R)ry^{1168}$	Df(3R)87B15-C1; 87E9-12	Hilliker	Gelbart
$Df(3R)ry^{1301}$	Df(3R)87D2-4; 87E1-2	Gelbart	Gelbart
$Df(3R)ry^{1402}$	Df(3R)87D2-4; 87D14-E2	Gelbart	Gelbart
$Df(3R)ry^{1607}$	Df(3R)87D3-4; 87E2-3	O'Donnell	Gelbart
$Df(3R)ry^{1608}$	Df(3R)87D4-6; 87E1-2	O'Donnell	Gelbart
$Df(3R)kar^{1G27}$	Df(3R)87B3-5; 87D6-12 + In(3R)87B-D, 99E1-F1	Gelbart	Gelbart, Hilliker
$Df(3R)kar^{SZ-11}$	Df(3R)87C7-8; 87E5-6	Gausz et al. (1980)	
Df(3R)1C4a	Df(3R)87E5-7; 87E11-F1	Chovnick	Gelbart
Df(3R)126c	Df(3R)87E1-2; 87F11-12	Chovnick	Gelbart, Lefevre
Df(3R)126d	Df(3R)87D11-13; 87E3-5	Chovnick	Gelbart, Hilliker, Lefevre

The 1(3)S5 associated complementation group is represented by four other alleles (Table 1). All allele combinations are completely lethal.

The 1(3)B-103 complementation group is associated with two alleles (Table 1).

The foregoing three lethal complementation groups have not been separated from one another by deficiency or by recombination analysis, thus their relative left/right order is not defined.

The next complementation group, 1(3)ml4 (Fig. 1), has two alleles and is separable by deficiency from adjacent complementation groups. Df(3R)ry^{1608} separates 1(3)ml4 from the complementation groups to its left while Df(3R)ry^{36} separates it from mes-A.

The next complementation group is that associated with mes-1A. The deficiency, Df(3R)ry^{36} (Fig. 1, Table 1) separates mes-1A from 1(3)ml4, the proximally flanking complementation group. Further, 1(3)mes-1A was separated from the distally flanking complementation group associated with 1(3)mes-4B by Df(3R)ry^{74} (Schalet, Kernaghan and Chovnick 1964), a deletion which has been lost. All of the 14 mes-A alleles are semi-lethals associated with a visible mutant phenotype when hemizygous and in mutant allele heterozygous combinations. The visible phenotype is characterized by extra head and thoracic bristles; especially marked is duplication of the anterior scutellar bristles. Surviving mutant allele heterozygotes uniformly express the mes-A phenotype. Although mes-A alleles were tested for complementation with 1(3)m14, all mes-B alleles and three alleles of the 1(3)G9 group [1(3)G9, 1(3)G15 and 1(3)G21]. All heterozygotes exhibited full complementation.

The next complementation group, mes-B (Fig. 1), has three alleles and is similar to the mes-A group in that alleles are semi-lethal and associated with a recessive visible phenotype; namely, outspread wings, held at about a 45° angle to the body, and a dark trident-like crown on the dorsal thorax. The penetrance of this phenotype is excellent. (Occasionally, thoracic bristle duplication reminiscent of that associated with mes-A alleles is observed.)

The adjacent lethal complementation group, the 1(3)G9 complex, is associated with 16 alleles (Fig. 1), one of which, 1(3)H23, is semi-lethal. Although the 1(3)G9 complementation group has not been separated by deficiency from the mes-B group, all combinations of mes-B alleles with 1(3)G9 alleles complement fully. Thus, we conclude that these two complementation groups represent separate gene loci. The order of 1(3)G9 and mes-B is unclear. We infer that 1(3)G9 is to the right of mes-A from the following observations: Df(3R)ry^{619} appears to have its left breakpoint in the immediate vicinity of mes-A for although heterozygotes for Df(3R) ry^{619} and alleles of the mes-A complementation group exhibit a mes-A phenotype, they have much greater viability than is ordinarily the case for mes-A allele hemizygotes, suggesting that the mes-A locus on the Df(3R)ry^{619} chromosome is partially functional; whereas, Df(3R)ry^{619} is completely lethal in combination with 1(3)G9 alleles.

The next complementation group (Fig. 1), that associated with 1(3)S12, consists of three alleles and is separated from 1(3)G9 and mes-B by Df(3R)kar^{1G}27. We observed that 1(3)S12 fully complemented with 1(3)G1. However, neither allele complemented with 1(3)B21-4. Since all three recessive lethals mapped by deficiency analysis immediately adjacent to the rosy locus we concluded that the complementation map observed was a function of allele complementation. Although 1(3)S12 and 1(3)B21-4 are completely lethal when hemizygous, 1(3)G1 is associated with low hemizygous viability. Surviving hemizygotes for 1(3)G1 uniformly express a phenotype of very thin and short thoracic bristles. Although 1(3)S12 has not been separated by deficiency from the rosy locus, recombination experiments have demonstrated that 1(3)S12 maps to the left of all rosy locus variants which have been assigned positions in the rosy locus genetic map (Chovnick et al. 1976; McCarron et al. 1979).

The next complementation group is that associated with the rosy locus (ry: 3-52.0), a genetic unit containing the xanthine dehydrogenase structural element and adjacent cis-acting regulatory sequences (Chovnick et al. 1976; McCarron et al. 1979).

The next complementation group is that associated with the previously described piccolo (pic) locus, which has been separated from the rosy locus by deficiencies and recombination (Schalet, Kernaghan and Chovnick 1964; Fig. 1). Df(3R)ry^{36} serves to place pic to the right of ry; Df(3R)ry^{614}, to place pic to the left of 1(3)S8. A total of 32 recessive lethal and semi-lethal alleles of this locus were available for analysis. Unlike all other complementation groups, not all inter se allele combinations of pic variants were tested for complementation (185 of 496 possible allele combinations were tested). Of the pic alleles listed in Table 1, all allele combinations involving the 19 alleles from 1(3)pic^{21} to 1(3)A111 inclusive were tested for complementation. Of the 19 alleles extensively analyzed, 6 alleles exhibited, in combination, heterozygous surviving progeny--1(3)pic^{21}, 1(3)m10, 1(3)A42-1,

1(3)A12-3, 1(3)A33-1 and 1(3)A3-3. The heterozygous surviving progeny were of reduced viability and uniformly exhibited a pic phenotype, namely, short, fine bristles and abnormal tergite morphology. Thus interallelic complementation extending to the visible phenotype was not observed among alleles of the pic locus.

The next complementation group, that associated with 1(3)S8, consists of 7 alleles (Fig. 1; Table 1), all combinations of which exhibit complete lethality. Df(3R)126c places 1(3)S8 to the left of 1(3)B16-1.

The next complementation group, associated with 1(3)B16-1, consists of 5 alleles (Table 1) and on the basis of its localization by complementation with rosy region deficiencies almost certainly corresponds to the previously described 1(3)S6 locus (Schalet, Kernaghan and Chovnick 1964), the sole representative allele of which was lost prior to the present analysis. All allele combinations were lethal save two. Heterozygotes for 1(3)H69/1(3)B16-1 and for 1(3)H69/1(3)B27-2 were of 4% and 15% viability, respectively. Surviving heterozygous progeny were of normal phenotype.

The 1(3)C9a complementation group consists of three hemizygous lethal alleles which exhibit limited allele complementation. The heterozygotes, 1(3)C9a/1(3)B2-6, 1(3)C9a/1(3)B26-2 and 1(3)B2-6/1(3)B26-2, were of 8%, 14% and 2% mean viability, respectively. Surviving heterozygous progeny are somewhat reduced in size relative to wild type. Df(3R)ry^{1402} and Df(3R)ry^{1608} place 1(3)C9a to the right of 1(3)B16-1.

The next complementation group, Ace, associated with 1(3)26 (Schalet, Kernaghan and Chovnick 1964), consists of 16 alleles (Table 1). All allele combinations are lethal save two, both involving 1(3)B15-2. The heterozygotes 1(3)m15/1(3)B15-2 and 1(3)B15-2/1(3)B22-1 are of 5% and 12% viability, respectively. This locus has been the focus of a recent analysis (Hall and Kankel 1976) which presents strong evidence that the locus associated with the 1(3)26 complementation group is the site of the structural gene for acetylcholin-esterase. Hence, following the suggestion of Hall and Kankel (loc. cit.) it is renamed Ace, although 1(3)26 must remain a synonym. Df(3R)ry^{1607} places Ace to the left of 1(3)G7, the next complementation group.

The 1(3)G7 complementation group is represented by 9 alleles (Table 1); all allele combinations exhibit complete lethality.

The next complementation group is associated with only one allele, 1(3)m32. A second allele, 1(3)J38, has been generated by J. Hall. The two alleles are completely noncomplementary. Df(3R)kar^{SZ11} places 1(3)m32 to the right of 1(3)G7 and Df(3R)126d separates 1(3)m32 from the cluster of lethal loci to the right (Fig. 1).

The next four complementation groups, associated with 1(3)m116, 1(3)m17, 1(3)S9 and 1(3)G5 have not been separated by deficiency.

The 1(3)m17 group includes 7 alleles and exhibits limited allele complementation as outlined in Fig. 2. One allele combination shown as noncomplementary in Fig. 2 does exhibit weak viability. This heterozygote, 1(3)H45/1(3)H77, is associated with a mean viability of 11%.

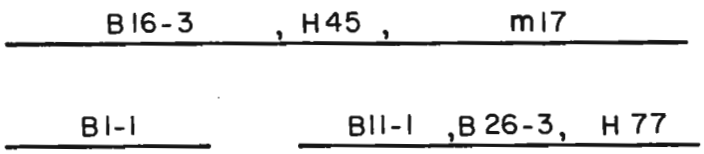

Fig. 2. Complementation map of the 1(3)m17 group.

The 1(3)m116 complementation group is associated with only one allele.

The 1(3)S9 complementation group is associated with 16 alleles and exhibits limited allele complementation as indicated in Fig. 3. All complementing allele combinations involve 1(3)B8-4. Three allele combinations indicated as noncomplementary in Fig. 3 do, in fact, exhibit weak complementation, all involving 1(3)B8-4. The heterozygotes 1(3)B8-4/1(3)B2-3, 1(3)B8-4/1(3)B15-1 and 1(3)B8-4/1(3)B12-2 are of 14%, 13% and 22% mean viability, respectively.

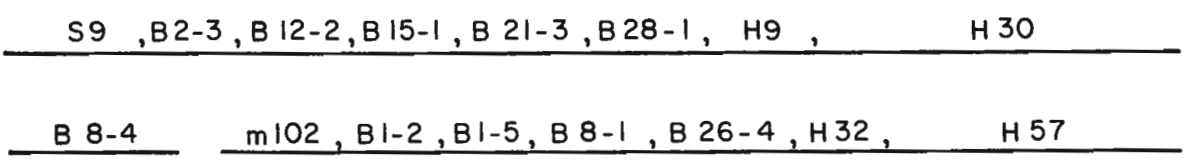

Fig. 3. Complementation map of the 1(3)S9 group.

The fourth member of this cluster of complementation groups is that associated with 1(3)G5 which has two alleles (Table 1).

The most distal complementation group in the 87D2-4-87E12-F1 interval is that associated with 1(3)ml12. The complementation map of the alleles of this group is presented in Fig. 4. Although 1(3)H24 and 1(3)H25 are indicated as noncomplementary to 1(3)ml12, they do, in fact, weakly complement. The heterozygotes 1(3)H24/1(3)ml12 and 1(3)H25/1(3)ml12 are of 40% and 24% mean viability, respectively; however, unlike other complementing allele combinations of this locus, surviving heterozygotes are associated with a mutant visible phenotype of short, very thin bristles and irregularly arranged ommatidia. Df(3R)ry^{1168} separates 1(3)ml12 from the complementation groups to the left.

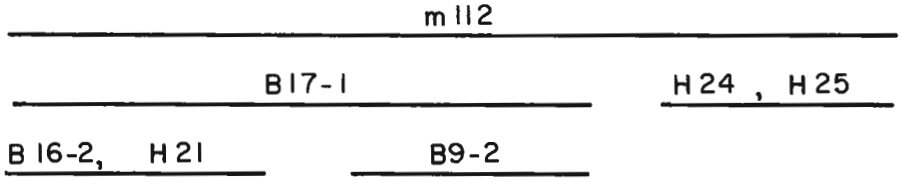

Fig. 4. Complementation map of the 1(3)ml12 group.

Overall, we have observed 21 complementation groups within the 87D2-4 to 87E12-F1 interval, a polytene chromosome segment of 23 or 24 chromomeres.

Supported by a research grant, GM-09886, from the Public Health Service.

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Hilliker, A.J. Univ. of British Columbia, Vancouver, British Columbia. Meiotic effects of second chromosome heterochromatic deletions.

It has been suggested by a number of authors (reviewed in Yunis and Yasmineh 1971) that centromeric heterochromatin may promote the initiation of meiotic pairing of homolgous chromosomes and, further, protect the centromere from the "rigors of meiosis"--presumably the terminalization of chiasmata and subsequent reductional segregation of homologous dyads. These hypotheses suggested the following experiments, as they predict that heterozygosity for second chromosome heterochromatic deficiencies may result in appreciable second chromosome nondisjunction and/or chromosome loss. The proximal deficiencies studied were Df(2R)M-S2^{10}, which is deficient for the 2R heterochromatic block and Df(2L)C', which is undoubtedly deficient for much of the 2L proximal heterochromatin (Hilliker 1976).

Df(2R)M-S2^{10}: Virgin females heterozygous for Df(2R)M-S2^{10} and b pr cn were crossed, singly, in vials, to C(2L)VH1,lt; C(2R)P,px males and brooded for six days. As these compound-second autosome bearing males produce nearly equal frequencies of the four classes of C(2L); C(2R); nullo-2; and diplo-2 sperm (Gibson 1977); nullo-2 and diplo-2 female gametes, the consequence of second chromosome nondisjunction or chromosome loss, may be recovered as viable zygotes with 25% efficiency. Thus by the use of control (multiplier) crosses to estimate the total number of fertilized eggs one may assay second chromosome loss and nondisjunction in Drosophila females by mating them with compound second autosome bearing males.

From an estimated 4844 fertilized eggs, no nondisjunctional progeny were recovered. Thus neither chromosome loss or nondisjunction of chromosome two is associated with heterozygosity for Df(2R)M-S2^{10}. The absence of chromosome loss is in contradiction to the theory of the protection of centromeres by flanking heterochromatin. No newly induced isochromosome bearing exceptions (a possible consequence of chromosome "breakage") or patroclinous progeny were observed. Clearly the M(2)S10 chromosome is stable despite the absence of the 2R heterochro-

*Present address: CSIRO, Canberra City, ACT, Australia.