Table 1. Observed and expected associations between 3L and 3R karyotypes in laboratory strains of D.ananassae.

Karyotypes 3L / 3R		ST/ST	ST/DE	DE/DE	Total
A - Calcutta strain					
ST/ST	obs.	70	73	3	146
	exp.	79.78	63.87	2.39	
ST/ET	obs.	30	7	0	37
	exp.	20.22	16.17	0.61	
Total		100	80	3	183
χ^2 = 13.22 d.f. = 2 P < 0.005					

Karyotypes 3L / 3R		ST/ST	ST/DE	DE/DE	Total
B - Shillong strain					
ST/ST	obs.	22	16	4	42
	exp.	7.32	19.73	14.95	
ST/ET	obs.	1	46	20	67
	exp.	11.67	31.47	23.86	
ET/ET	obs.	0	0	23	23
	exp.	4.01	10.80	8.19	
Total		23	62	47	132
χ^2 = 96.84 d.f. = 4 P < 0.001					

The occurrence of non-random association of delta and eta inversions in the third chromosome of D.ananassae is probably due to differential selection involving interaction between widely separated loci which supports the hypothesis postulated by Levitan (1958).

References: Brncic,D. 1961, Genetics 46:401-406; Futch, D.G. 1966, Univ.Texas Publ. 6615:79-120; Levitan, M. 1958, Cold Spring Harb.Symp.Quant.Biol. 23:252-268; ____ 1961, Science 134:1617-1619; ____ 1973, Evolution 27:215-225; ____ 1978, Genetics 89:751-763; Levitan, M. & F.M. Salzano 1959, Heredity 13:243-248; Prakash, S. 1967, Genetics 57:385-400; Shirai, M. & D. Moriwaki 1952, DIS 26:120-121; Singh, B.N. 1970, Ind.Biol. 2:78-81; ____ 1973, Genetica 44:602-607; ____ 1974a, Cytologia 39:309-314; ____ 1974b, Caryologia 27:285-292; ____ 1982, Genetica 59:151-156; ____ 1983a, Experientia 39:99-100; ____ 1983b, Genetica (in press); Sperlich, D. & H. Feuerbach-Mravlag 1974, Evolution 28:67-75.

Slatko, B., L. Fritts, M. Parker, S. Hanlon & S. Carperos. Williams College, Williamstown, Massachusetts.

P-M hybrid dysgenesis in D. melanogaster: Interaction with repair deficient mutants. I. Male recombination induction.

P-M hybrid dysgenesis is known to at least partially involve transposable P elements which can be activated in appropriate hybrid individuals. One attempt to ascertain the biochemical basis of P element control of hybrid dysgenic function involves the construction of appropriate P-M hybrids which contain mutants defective in various pathways of repair of induced genetic damage (mei and mus mutants). F_1 males containing an X-linked mei or mus mutant and containing second chromosomes heterozygous for a P chromosome and a cn bw chromosome were produced in such fashion as to be a dysgenic genotype (e.g., a P chromosome from the P stock and the cn bw and mei or mus mutant chromosomes from the female parent.

One dysgenic trait common to all P-M systems is the presence of recombinants among the progeny of dysgenic males, albeit at lower frequencies than in females. An assay of male recombination activity in dysgenic P-M males containing an X-chromosome repair deficient mutant was performed by crossing mei (or mus)/Y; P/cn bw males (constructed as above) to cn bw females and assaying for recombination among the progeny.

Three P chromosomes from diverse natural populations--T-007 (Texas), haifa[12] (Israel), N-1 (California) were tested, in addition to a control (non-P) strain, Canton-S. In addition, where feasible, two alleles at each mus/mei locus were also tested. Mei-9 defines an excursion repair defect, mei-41 and mus-101 define post-replication repair defects and mus-102 is, as yet, undefined.

Results from these crosses are presented in Table 1. None of the mei or mus mutants utilized in this report showed significant frequence of male recombination over the Canton-S control. In addition, none of the tested combinations showed a statistically significant increase or decrease in the frequency of male recombination induction over the P chromosome control for each set. It should be noted that as crosses were performed with individual males, it was possible to account for clusters (premeiotic events) in the data set. Clusters

Table 1.

Genotype		#F_1 males tested	#Progeny scored	%Recomb.
+Y;	Canton-S/cn bw	176	13,305	0.02
mei 9^{A}/Y;	" "	20	1,677	0
mei 9^{D1}/Y;	" "	15	1,212	0
mus 101^{D1}/Y;	" "	16	1,687	0
mus 101^{D2}/Y;	" "	20	2,256	0
mus 102^{D1}/Y;	" "	45	2,767	0
mei 41^{D1}/Y;	" "	38	2,778	0
mei 41^{D5}/Y;	" "	23	1,602	0.12
mei 9^{A}, mei 41^{A3}/Y;	"	32	1,909	0
mei 9^{A}, mei 41^{D5}/Y;	"	29	1,219	0
+Y;	T-007/cn bw	99	5,175	0.66
mei 9^{A}/Y;	" "	26	1,272	0.31
mus 101^{D1}/Y;	" "	20	1,677	0.66
mus 101^{D2}/Y;	" "	22	2,149	0.23
mus 102^{D1}/Y;	" "	51	1,447	0.55
mei 41^{D1}/Y;	" "	27	3,145	0.20
mei 41^{D5}/Y;	" "	17	1,060	0.28
mei 9^{A}, mei 41^{A3}/Y;	"	30	1,676	0.89
mei 9^{A}, mei 41^{D5}/Y;	"	14	558	0.18
+/Y;	haifa^{12}/cn bw	41	4,124	0.41
mei 9^{A}/Y;	" "	16	1,289	0.39
mei 9^{D1}/Y;	" "	19	1,150	0.26
mus 101^{D1}/Y;	" "	21	2,009	0.15
mus 101^{D2}/Y;	" "	26	2,202	0.20
mus 102^{D1}/Y;	" "	36	1,880	0.43
mei 41^{D1}/Y;	" "	44	3,147	0.29
mei 41^{D5}/Y;	" "	29	1,402	0.43
mei 9^{A}, mei 41^{A3}/Y;	"	45	1,711	0.31
mei 9^{A}, mei 41^{D5}/Y;	"	36	1,818	0.44
+Y/;	N-1/cn bw	37	1,421	0.86
mei 9^{A}/Y;	" "	40	3,114	0.19
mei 9^{D1}/Y;	" "	32	2,475	0.61
mus 101^{D1}/Y;	" "	21	1,970	0.41
mus 101^{D2}/Y;	" "	19	1,729	0.75
mus 102^{D1}/Y;	" "	59	3,868	0.39
mei 41^{D1}/Y;	" "	36	1,955	1.89
mei 41^{D5}/Y;	" "	35	2,222	0.54
mei 9^{A}, mei 41^{A3}/Y;	"	31	1,052	0.95
mei 9^{A}, mei 41^{D5}/Y;	"	34	888	0.90

were counted as single recombinants and the data adjusted accordingly. Insignificance was ascertained by the methods of Kastenbaum-Bowman (1970). No evidence of increased clustering was apparent in any one particular genotype.

Therefore, it appears from the data that these repair deficient mutant males, tested for the influence upon P-M dysgenesis via a male recombination assay, fail to show such an influence. (Supported by Williams College Discretionary Funds and Research Corporation Funds.)

References: Kastenbaum, M. and K. Bowman 1970, Tables for determining the statistical significance of mutation frequencies, Mut. Research 9:527-549; Owen, D. 1962, Handbook of Statistical Tables, Addison-Wesley Publ. Co., Inc. (Reading, MA), pp. 259-261.

Slatko,B.[1], S.Hanlon[1], S.Carperos[1], R.C. Woodruff[2] & J.Mason.[3] 1-Wiliams College, Williamstown, Massachusetts. 2-Bowling Green State University, Bowling Green, Ohio. 3- N.I.E.H.S., Research Triangle Park, North Carolina. P-M hybrid dysgenesis in D.melanogaster: Interaction with repair deficient mutants. II. Recessive lethal induction.

In the preceding report, Slatko, Hanlon & Carperos used male recombination induction as an assay for increased or decreased P-M hybrid dysgenesis activity in males in the presence of a variety of X-linked repair deficient mutants. In this report, sex-linked recessive lethal (SLRL) tests have been utilized to assay P-M activity. Similar to the previous report, F_1 dysgenic males were produced from crosses of P strain fathers to M strain cn bw mothers who also contained an X-linked repair deficient mutant (mei or mus). These F_1 males were individually crossed to Basc females and individual F_2 heterozygous Basc females were crossed to Basc males. These crosses were scored for