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

the absence of any wild type males, indicating an induced lethal. Retests were performed from vials showing less than 20 progeny when feasible.

Results are presented in Table 1. Clusters of lethals were identified by the cumulative Poisson Distribution Test of Owen (1962). Clustered cases were counted as individual lethals and the data adjusted accordingly. The Kastenbaum-Bowman statistical tables were used to judge significance levels (Kastenbaum & Bowman 1970).

Table 1. Genotype	# Parental males tested	# SLRL tests (fertile)	% SLRL (#SLRL)
Canton-S	20	1,595	0.19(3)
w	234	11,710	0.15(17)
+Y; T-007/cn bw	118	4,471	1.21(54)
mei 9^{D1}/Y; " "	22	857	0.93(8)
mei 9^{A}/Y; " "	134	1,071	0.84(9)
mei 41^{D1}/Y; " "	196	1,883	1.17(22)
mei 41^{D5}/Y; " "	163	1,820	1.70(31)
mus 101^{D1}/Y; " "	179	2,129	1.22(26)
mus 101^{D2}/Y; " "	137	1,842	1.54(28)
mus 102^{D1}/Y; " "	23	1,086	0.55(6)
mei 9^{A}, mei 41^{D5}/Y; "	20	823	1.44(12)
mei 9^{A}, mei 41^{A3}/Y; "	31	1,149	1.22(14)
+Y; $haifa^{12}$/cn bw	107	4,507	0.93(42)
mei 9^{D1}/Y; " "	24	1,396	1.29(13)
mei 9^{A}/Y; " "	81	2,324	1,03(24)
mei 41^{D1}/Y; " "	118	2,075	0.72(15)
mei 41^{D5}/Y; " "	89	2,129	1.03(22)
mus 101^{D1}/Y; " "	80	2,198	1,05(23)
mus 101^{D2}/Y; " "	71	2,426	1.15(28)
mus 102^{D1}/Y; " "	32	1,547	1.23(19)
mei 9^{A}, mei 41^{D5}/Y; "	22	1,117	0.45(5)
mei 9^{A}, mei 41^{A3}/Y; "	28	1,357	1.25(17)
+Y; N-1/cn bw	104	4,381	1.23(54)
mei 9^{D1}/Y; " "	25	1,251	1.68(21)
mei 9^{A}/Y; " "	105	2,531	2.21(56)*
mei 41^{D1}/Y; " "	145	2,106	1.57(33)
mei 41^{D5}/Y; " "	77	2,399	1.67(64)*
mus 101^{D1}/Y; " "	55	2,224	2.07(46)+
mus 101^{D2}/Y; " "	83	2,234	1.66(37)
mus 102^{D1}/Y; " "	26	1,384	2.02(28)+
mei 9^{A} 41^{D5}/Y; " "	26	1,357	1.77(24)
mei 9^{A} 41^{A3}/Y; " "	23	980	1.73(17)

* $p < 0.01$ + $0.01 \leq p < 0.05$

It can be seen that most combinations of P-M dysgenesis and various mus(mei) mutants fail to display significant alterations in SLRL mutation frequencies from the frequencies observed from the P-M dysgenesis observed in the absence of the repair deficient strains. Exceptions exist for the P strain N-1, in combination with mei 9^{A} (but not with its allele mei 9^{D1}), mei 41^{D5} (but not with its allele mei 41^{D1}), mus 101^{D1} (but not with its allele mus 101^{D2}) and must 102^{D1}. None of the mus or mei mutants used in this study significantly increase or decrease the SLRL frequency by themselves (Mason 1980).

Overall, P-M dysgenic SLRL frequencies do not appear to be altered in the presence of the repair deficient strains, as a general rule. Supported by Williams College Discretionary Funds and Research Corporation Funds (BS) and NSF Grant DEB-7923007 and NIEHS Research Development Award KOA-ES 00087 (RCW).

References: Kastenbaum, M. & K. Bowman 1970, Mut. Research 9:527-549; Mason, J.M. 1980, Mut. Research 72:323-326; Owen, D. 1962, Handbook of Statistical Tables, Addison-Wesley Publ. Co., Reading MA. pp. 259-261.

Slatko,B.[1], S. Hanlon[1] & R.C.Woodruff[2].
1-Williams College, Williamstown, Massachusetts. 2-Bowling Green State University, Bowling Green, Ohio. P-M hybrid dysgenesis in D.melanogaster: Interaction with repair deficient mutants. III. Distorted transmission frequencies (K value) and unequal zygotic recovery.

In the two preceding reports, Slatko, Hanlon & Carperos (DIS, this issue) and Slatko, Hanlon, Carperos, Woodruff & Mason (DIS, this issue) used male recombination induction and mutation induction (sex-linked recessive lethals) to assay for effects of repair defective mutants (mus, mei) upon the P-M hybrid dysgenesis syndrome. We have utilized a third parameter of hybrid dysgenesis, distorted transmission frequencies (k), as a further assay. The k value is defined as the frequency of progeny containing the wild (+) phenotype second chromosome among all non-recombinant progeny of the cross +/cn bw ♂ x cn bw ♀ . The expected Mendelian k is 0.50 (e.g., ½ the non-recombinant progeny receive the + chromosome from the male parent and] receive the cn bw chromosome).

Results from crosses used to generate dysgenic F_1 males in the presence (or absence) of various repair deficient mutations [e.g., +/Y; P (or Canton-S)/ca bw male x (mei, mus, or +/Basc; cn bw female] are presented in Table 1. Five P chromosomes from diverse geographic natural populations [T-007 (Texas), haifa^{12} (Israel), N-1 (California), OK1 (Oklahoma) and W8D (Georgia)] were utilized, in addition to a control series utilizing the Canton-S second chromosome.

Table 1

Genotype	#Fertile males tested	#Progeny scored	Av. K
+/Y; Canton-S/cn bw	176	13,213	0.539
mei 9^{A}/Y; "	20	3,437	0.546
mei 9^{D1}/Y; "	20	1,212	0.522
mei 41^{D1}/Y; "	38	2,778	0.581
mei 41^{D5}/Y; "	23	1,602	0.513
mus 101^{D1}/Y; "	16	1,687	0.590
mus 101^{D2}/Y; "	20	2,256	0.599
mus 102^{D1}/Y; "	34	2,767	0.549
mei 9^{A}, mei 41^{A3}/Y; "	32	1,909	0.429
mei 9^{A}, mei 41^{D5}/Y; "	15	942	0.413
+/Y; T-007/cn bw	99	5,141	0.385
mei 9^{A}/Y; "	26	1,268	0.353
mei 41^{D1}/Y; "	27	1,483	0.026*
mei 41^{D5}/Y; "	17	1,057	0.083*
mus 101^{D1}/Y; "	20	1,648	0.197*
mus 101^{D2}/Y; "	22	2,144	0.288*
mus 102^{D1}/Y; "	51	1,429	0.304*
mei 9^{A}, mei 41^{A3}/Y; "	30	1,661	0.153*
mei 9^{A}, mei 41^{D5}/Y; "	14	557	0.072*
+/Y; haifa^{12}/cn bw	41	4,102	0.529
mei 9^{A}/Y; "	16	1,284	0.523
mei 9^{D1}/Y; "	19	1,147	0.555
mei 41^{D1}/Y; "	44	3,138	0.374*
mei 41^{D5}/Y; "	29	1,396	0.228*
mus 101^{D1}/Y; "	21	2,006	0.465*
mus 101^{D2}/Y; "	26	2,296	0.463*
mus 102^{D1}/Y; "	36	1,870	0.459*
mei 9^{A}, mei 41^{A3}/Y; "	45	1,703	0.299*
mei 9^{A}, mei 41^{D5}/Y; "	36	1,809	0.266*

It can be seen from the data that for each P chromosome set, mei-9 alleles had no effect on K values, whereas all 41, 101, 102 alleles reduced the k value significantly (as judged by 2-Factor F tests).

Mei-9 mutants define defects in wild type excision repair, whereas mei 41 and mus 101 mutants define defects in post-replication repair (PRR). Mus 102 has not yet been characterized.

To verify that these results followed the pattern observed for other phenotypes associated with hybrid dysgenesis, two sets of additional crosses were performed utilizing a P chromosome stock isolated from Wisconsin and kindly supplied by Bill Engles, π_2. In the set A crosses, π_2 males were crossed to cn bw ♀♀ (A-1), mei 41^{D1}; cn bw ♀♀ (A-12) and mei 41^{D5}; cn bw ♀♀ (A-3).

In the set B crosses, C(1)DX, y, f; π_2 ♀♀ were crossed to mei 41^{D1}; cn bw males (B-2) or mei 41^{D5}; cn bw males (B-3). These crosses should generate non-dysgenic F_1 males. From these five crosses, males were collected (the genotypes are shown in Table 2), and backcrossed to cn bw females.