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.

Table 1 (contin.) Genotype	#Fertile males tested	#Progeny scored	Av. K.
+/Y; N-1/cn bw	37	1,408	0.528
mei 9^{A}/Y; "	40	3,107	0.446
mei 9^{D1}/Y; "	32	2,460	0.520
mei 41^{D1}/Y; "	36	1,918	0.183*
mei 41^{D5}/Y; "	35	2,210	0.140*
mus 101^{D1}/Y; "	21	1,962	0.426*
mus 101^{D2}/Y; "	19	1,716	0.409*
mus 102^{D1}/Y; "	59	3,853	0.453*
mei 9^{A}, mei 41^{A3}/Y; "	31	1,042	0.273*
mei 9^{A}, mei 41^{D5}/Y; "	34	880	0.248*
+/Y; OK1/cn bw	244	25,377	0.515
mei 9^{A}/Y; "	50	6,370	0.528
mei 41^{D1}/Y; "	25	2,412	0.293*
mei 41^{D5}/Y; "	15	667	0.225*
mus 101^{D1}/Y; "	37	2,236	0.387*
mus 101^{D2}/Y; "	37	2,336	0.437*
mus 102^{D1}/Y; "	19	1,216	0.469*
mei 9^{A}, mei 41^{A3}/Y; "	9	413	0.336*
+/Y; W8D/cn bw	149	15,770	0.522
mei 9^{A}/Y; "	50	5,838	0.528
mei 41^{D1}/Y; "	50	5,082	0.412*
mus 102^{D1}/Y; "	19	1,900	0.492*
mei 9^{A}, mei 41^{A3}/Y;	9	413	0.346*

* $P < 0.01$

Table 2. Genotype	#Fertile males tested	#Progeny scored	Av. K
A			
(1)+/Y; π_2/cn bw	19	1,719	0.467
(2) mei 41^{D1}/Y; π_2/cn bw	8	168	0.165
(3) mei 41^{D5}/Y; π_2/cn bw	6	136	0.207
B			
(2) mei 41^{D1}/Y; π_2/cn bw	8	349	0.473
(3) mei 41^{D5}/Y; π_2/cn bw	11	573	0.462

The k value drops significantly in dysgenic crosses A-2 and A-3 as compared to the dysgenic cross A-1, but in the B-2 and B-3 crosses, the k is restored to its normal A-1 value. Thus the effect of mei-41 alleles upon k is directly attributable to whatever genetic control is exhibited by the hybrid dysgenesis syndrome. Of further interest are three additional observations: First, the "control" k values for P chromosomes in the absence of any mus or mei mutants show that some P chromosomes have a reduced K value (e.g., T-007) (compared to the Canton-S control), whereas others do not (e.g., haifa^{12}, N-1, OK-1, W8D), and PRR mutants which reduce the K value, do so to approximately the same relative amount in all P strains. Mei 41 alleles have stronger affects than mus 101 alleles and mus 102 alleles. The magnitude of the reduction in the case of mei 41 T-007/ cn bw males is such that only 2-8% of the non-recombinant chromosomes which are present among the adult progeny are of the T-007 genotype, as opposed to almost 40% in the absence of the mei 41 allele.

Second, it may be suggested that the above results allow the easy identification of PRR mutants, in the absence of biochemical information. Mutants which reduce the k value may be PRR mutants, and based upon this suggestion, we propose that mus 102 is PRR defective.

Table 3.

Genotype (male)	# Adults/ # eggs	%Hatch	Genotype (male)	# Adults/ # eggs	%Hatch
+/Y; Canton-S/cn bw	3607/4671	77	+/Y; haifa^{12}/cn bw	1370/1888	73
mei 9^{A}/Y; "	142/173	82	mei 9^{A}/Y; "	425/616	69
mei 41^{D1}/Y; "	371/493	75	mus 102^{D1}/Y; "	485/652	74
mei 41^{D5}/Y; "	795/1104	72	mei 41^{D1}/Y; "	1766/3642	48
mei 9^{A}, mei 41^{A3}/Y; "	315/459	69	mei 41^{D5}/Y; "	1683/3422	49
mei 9^{A}, mei 41^{D5}/Y; "	140/220	63	mei 9^{A}, mei 41^{A3}/Y; "	344/705	49
mus 101^{D1}/Y; "	263/323	81	mei 9^{A}, mei 41^{D5}/Y; "	41/81	51
			mus 101^{D1}/Y; "	214/315	68
+/Y; T-007/cn bw	303/467	65	+/Y; N-1/cn bw	2336/4176	56
mei 9^{A}/Y; "	522/623	84	mei 9^{A}/Y; "	38/57	67
mus 102^{D1}/Y; "	465/536	87	mus 102^{D1}/Y; "	887/1857	48
mei 41^{D1}/Y; "	88/188	47	mei 41^{D1}/Y; "	1901/5160	37
mei 41^{D5}/Y; "	116/223	52	mei 41^{D5}/Y; "	1516/4491	34
mei 9^{A}, mei 41^{A3}/Y; "	60/122	49	mus 101^{D1}/Y; "	309/572	54
mus 101^{D1}/Y; "	128/217	59			

Third, males whose genotypes contain mei 9, mei 41 and a P chromosome are largely sterile. Less than 10% of males of this genotype are fertile, and among the fertile males, fertility is low. These effects are not observed with P mei 41, P mei 9 or mei 9 mei 41 males. It is unclear why the P mei 9 mei 41 combination leads to male sterility, especially when mei 9 appears to have no effect on k.

As to the mechanism of the k value reduction in P males containing PRR mutants, light microscopy reveals no obvious structural defects in the testis and there appear to be as numerous an amount of motile sperm as in P males themselves. Electron microscopy of spermiogenesis is in progress, but a series of "egg hatch" experiments (Table 3) reveals that the cause of the reduction in k may be due to zygote mortality, rather than a spermiogenic defect. It may be recalled that Matthews (1981) has shown that 71% of the reduction in k in T-007/ cn bw males is due to spermiogenic defects and 29% is due to dominant lethality of eggs. In the presence of PRR mutants, this "egg hatch" is drastically reduced, even in P combinations where there was little or no original k reduction. This dominant lethality defines a new hybrid dysgenesis phenotype. (Supported by Williams College Discretionary Funds and Research Corporation Funds(BS) and NSF Grant DEB-7923007 and NIEHS research development award K04-ES00087 (RCW)).

Smirnova, S.G. & E.M. Khovanova. Institute of Molecular Genetics, USSR Academy of Sciences, Moscow, USSR. Temperature effects on the activity of H-factor in Drosophila simulans.

A factor of instability, termed the H factor, has been discovered in D.simulans (Khovanova 1977). H selectively raises the somatic recombination rate in the X chromosomes of dorsal prothoracal disc cells by a factor of 5 to 10, while the frequency of somatic mosaicism in the derivatives of the eye-antennal and dorsal mesothoracal disks remains at a low level.

The H factor is localized in the X chromosome. Its activity in dorsal prothoracal disk cells rises sharply in the presence of live yeast in the cultural medium. Studies of H-carrying stocks have suggested that the activity of H may be influenced by the temperature at which the culture grows. To test this supposition, the following crosses were effected:

1) ♀♀ yw(H^-) x ♂♂ +/Y (H^-) 2) ♀♀ yw(H^-) x ♂♂ v/y (H^+)

(the H-carrying stocks are marked H^+); those without the H factor are marked H^-). Eggs laid in 4-5 hours on a medium containing live yeast were placed in a thermostat at 25°C (A series) and at 16°C (B series). Macrochaetae of the head and thorax were analyzed in F_1 females. The results are shown in the Table.

In cross (1) the rate of mosaic spots is low in the humeral region and other regions, it changes insignificantly with the temperature downshift from 25° to 15°C in the humeral region and remains practically unchanged in the other regions tested.

In cross (2) the rate of mosaic spots in the humeral region at 25°C is five times as high as in cross (1) at the same temperature. The spot rate in the other regions is no different from that in cross (1). At 16°C in cross (2) the spot rate increases significantly in the humeral regions, while in the eye-antennal and dorsal mesothoracal disk derivatives it grows 10 to 20-fold.

Table 1.

Type of cross	Series	Number of ♀♀F_1	Somatic mosaicism in humeral region		Somatic mosaicism in other regions	
			# spots	%	# spots	%
1) ♀♀yw(H^-)x ♂♂+/Y(H^-)	A(25°C)	1669	8	0.47	6	0.35
	B(16°C)	2085	22	1.05	9	0.43
2) ♀♀yw(H^-)x ♂♂v/y(H^+)	A(25°C)	1629	37	2.27	4	0.24
	B(16°C)	2477	124	5.00	107	4.32