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Mariner, *Mos* and associated aberrant traits in *Drosophila mauritiana*

JAMES W. JACOBSON*
Department of Biology, University of Houston, Houston, Texas 77204-5513

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Summary

A suite of aberrant genetic traits, including increased mutation rate, sex-limited mutation and distorted transmission ratios, was produced among progeny of genetic crosses between two strains of *Drosophila mauritiana* when a paternally contributed *Mos* excision factor is placed into a non-*Mos* genetic background. In the reciprocal cross, involving maternally contributed *Mos* and *Mos* associated cytoplasm, the same genetic abnormalities are not observed. Differential effects on *mariner* excision in germ-line versus somatic tissue are apparent. Because *Mos* is known to influence the mobility of the *mariner* transposable element, these traits may be associated with *mariner* excision and/or transposition.

1. Introduction

Transposable elements are ubiquitous components of the eukaryotic genome (Shapiro, 1983; Berg & Howe, 1989) and are potentially significant sources of genetic variation (MacKay, 1986, 1987; Fitzpatrick & Sved, 1986; Torkamanzehi *et al.* 1988). Consequently, factors that regulate the patterns and processes of transposition and excision are important determinants of the impact these mobile genetic elements have on their hosts.

In efforts to understand the nature and characteristics of these regulatory factors, much research interest has focused on the phenomena of 'hybrid dysgenesis' in *Drosophila melanogaster*, a syndrome of correlated genetic abnormalities including sterility, male recombination, increased mutation rate, chromosomal rearrangements and distorted transmission ratios (Kidwell *et al.* 1977; Bregliano & Kidwell, 1983; Engels, 1983), that occurs when members of certain transposable element families are mobilized upon introduction into genetic backgrounds lacking specific regulatory factors. At least two distinct types of transposons in *D. melanogaster*, the *P* and *I* elements (Bregliano & Kidwell, 1983; Engels, 1983), are involved in these phenomena. A third transposon, *hobo*, produces remarkably similar events (Blackman *et al.* 1987; Yannopoulos *et al.* 1987).

The manifestation of 'hybrid dysgenesis' appears to depend upon both chromosomal and cytoplasmic components and, although widely investigated, the nature of these regulatory factors is not yet known in detail (Engels, 1984; Simmons & Bucholz, 1985). What is known is that over time, strains that contain multiple copies of these transposons undergo a change to a state where transposition is rare, presumably because the appropriate regulatory framework has arisen. In the P–M system this change is described as a switch from the 'M cytotype' where transposition occurs at high frequency to the 'P cytotype' where transposition is repressed (Engels, 1983).

The *P* element also exhibits dramatic tissue specificity. Transposition/excision events are largely limited to the germ-line with somatic events only rarely being observed. Restriction of transposition to the germ line [a characteristic common among *D. melanogaster* transposons (Engels, 1983; Rubin, 1983; Berg & Howe, 1989)] is achieved for the *P* element through differential processing of the RNA (Laski *et al.* 1986). It is not known if differential RNA processing is a general mechanism for restricting the mobility of other transposons to the germ-line tissue.

In contrast to the tissue specific pattern of *P*-element transposition in *D. melanogaster*, the *mariner* transposon is mobile in both germ-line and somatic tissue in *D. mauritiana*, a sibling species of *D. melanogaster*. *Mariner* has superficial structural similarities with the *P* element of *D. melanogaster*. It is relatively small, 1286 base pairs, has short inverted terminal repeats, contains a single open reading frame, and is present in the *D. mauritiana* genome in ~15–30 copies. An insertion of *mariner* in the *X*-chromosome-

* To whom offprint requests should be addressed.

linked *white* gene is the cause of the *white-peach* (w^{pch}) mutation in *D. mauritiana* (Jacobson & Hartl, 1985; Haymer & Marsh, 1986; Jacobson *et al.* 1986). Germ-line reversion of w^{pch} to wild type occurs by excision of the inserted *mariner* element and *mariner* excision in somatic tissue results in patches of wild-type pigmented tissue in an otherwise mutant eye. Consequently, the appearance of wild-type-eyed flies and/or animals with mosaic eyes in the w^{pch} background is a convenient and reliable assay for *mariner* excision in both germ-line and somatic tissue.

In the w^{pch} strain, reversion to wild type takes place at a frequency of approximately 10^{-3} per gene per generation and somatic mosaics are produced at similar frequencies (Jacobson & Hartl, 1985). E25H is derived from the w^{pch} strain and carries the same unstable eye-colour allele. In addition, E25H carries, in the third chromosome, the dominant *Mos* excision factor (Bryan *et al.* 1987) which is itself a particular copy of the *mariner* transposon (Medhora *et al.* 1988). The reversion rate of the w^{pch} allele in the presence of *Mos* is approximately tenfold greater than in the original w^{pch} strain, i.e. $\sim 10^{-2}$ per gene per generation. In fact, the somatic excision of *mariner* in E25H is so greatly enhanced that virtually every animal is an eye-colour mosaic and this somatic mosaicism is diagnostic of the presence of *Mos* (Bryan *et al.* 1987).

The somatic instability of *mariner* is unique among *Drosophila* transposons and an understanding of the mechanism by which *Mos* affects *mariner* mobility is an important first step in elucidating factors regulating *mariner* transposition. In this report, I present results of experimental crosses designed to examine the relative importance of cytoplasmic and chromosomal determinants in the regulation of *mariner* mobility by the *Mos* factor in *D. mauritiana*.

2. Materials and methods

(i) *Strains and crosses*

Drosophila strains and crosses described in the text were reared at 25 °C on Formula 4-24 Instant Drosophila Medium (Carolina Biological Supply, Burlington, NC). Two strains of flies were used in this analysis: w^{pch} (w^{pch}/w^{pch}; +/+; +/+; +/+) and E25H (w^{pch}/w^{pch}; +/+; *Mos*/*Mos*; +/+). Both strains are maintained in my laboratory and have been cultured separately for about 7 years.

(ii) *Data collection*

Anaesthetized flies were sexed and scored for eye-colour phenotype through a dissecting microscope. Three phenotypic classes for each sex were scored, namely: non-mosaic (*white peach*), mosaic, and wild type. The possibility of an incorrect phenotypic classification for an animal in this analysis is an important consideration in the data collection. To obviate such an error, in scoring for mosaic- *vs.* non-mosaic-eyed flies, any abnormal pigmentation was closely examined at high magnification. If a spot could not be unambiguously identified as wild-type pigment, the animal was scored as a non-mosaic. Previous analysis of the w^{pch} (Jacobson & Hartl, 1985) and E25H strains (Bryan *et al.* 1987; Medhora *et al.* 1988) have shown that the presence of *Mos* is readily diagnosed by the production of eye-colour mosaics in the w^{pch} background. Even extensively mosaic-eyed animals are readily distinguished from true wild-type revertants thereby eliminating erroneous phenotypic assignments. Furthermore, progeny testing of numerous wild-type revertants of w^{pch} (Jacobson & Hartl, 1985; Bryan *et al.* 1987) has revealed that most, if not all, wild-type eye-colour revertants of w^{pch} are true germ-line revertants and not just extensive somatic mosaics.

(iii) *Statistical analyses*

The overall results of the genetic crosses were analysed for independence and significance using a fully saturated hierarchical log-linear model and SPSS/PC+ software (SPSS Inc., 444 N. Michigan Ave., Chicago, Illinois, 60611). Additional statistical testing was carried out by standard procedures (Sokal & Rolf, 1987).

3. Results

In order to separate chromosomal and cytoplasmic components affecting *mariner* behaviour, genetic crosses were designed to produce animals that receive a single Mos^+ or *Mos*-bearing chromosome into a common w^{pch} background where *mariner* excision is readily detected. Flies with wild-type eyes in this background are the result of germ-line excision of *mariner* while mosaic-eyed flies result from somatic excision. Animals heterozygous for *Mos* and hemi- or homozygous for w^{pch} were produced following the mating scheme outlined in Fig. 1. The *Mos* heterozygotes were then back-crossed to homozygous Mos^+ males (cross A) or females (cross B). For each experimental cross, replicate matings were carried out with ~ 50 pairs of adults in each of 2–4 one-half pint milk bottles containing 30 ml of media. Sequential broods from each set of crosses were obtained by removing the mated adults to a fresh media bottle 5–6 days following the initial introduction and again 5–6 days later. Progeny from experimental crosses were scored through the 20th day following initial introduction into the media bottle. Flies were immobilized with CO_2 anaesthesia. Two sets of experimental crosses were conducted. Experiment 1 (A1, B1) was carried out in November 1988 and Experiment 2 (A2, B2) in May 1989.

Overall results of the experimental crosses are summarized in Table 1. Results from crosses designated A1 and B1 are totals of two replicate matings.

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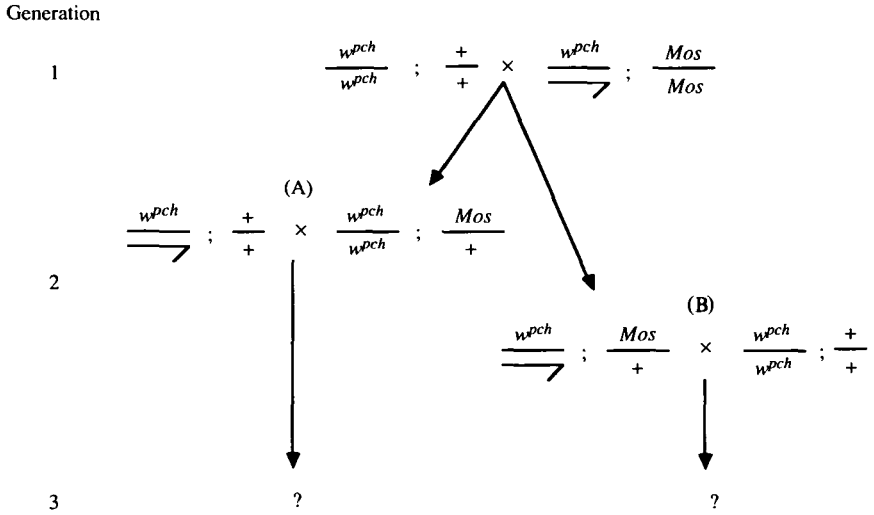

Fig. 1. Mating scheme to produce animals heterozygous for *Mos* and hemi- or homozygous for the unstable w^{pch} allele. Males used in cross A and females used in cross B were from the *white peach* (w^{pch}; Mos^{+}) strain described in the text. Progeny of cross A and cross B were scored by sex for three phenotypic classes: non-mosaic, mosaic, and wild type.

Table 1. *Summary of genetic cross results*

		Phenotype				
Cross	Sex	Non-mosaic	Mosaic	Wild type	Reversion frequency	Totals
A1	F	295	481	9	0·0115	785
	M	348	446	3	0·0038	797
	Total	643	927	12		1582
B1	F	193	188	14	0·0354	395
	M	200	252	0	—	452
	Total	393	440	14		847
A2	F	1189	1492	13	0·0048	2694
	M	1250	1413	10	0·0037	2673
	Total	2439	2905	23		5367
B2	F	665	689	32	0·0231	1386
	M	700	814	0	—	1514
	Total	1365	1503	32		2900

Four replicate matings are summarized as cross A2 and three replicate matings produced results summarized as cross B2. No significant differences in outcome were observed among replicates within a particular set of crosses and therefore only the summary data are presented here.

(i) *Statistical test for interactions*

The data set presented in Table 1 was examined for interactions using a fully saturated hierarchical log linear model that includes: cross (A *vs.* B), block (1 *vs.* 2), sex (male *vs.* female) and phenotype (non-mosaic *vs.* mosaic). Results of this analysis are summarized in Table 2. The analysis is robust to the order of entry of variables and a non-significant result never became significant due to the order of entry. No significant four-way interactions were obtained from the analysis (D.F. = 1, $\chi^2 = 2{\cdot}107$, $P = 0{\cdot}1467$) and, as can be seen in Table 2, only one three-way interaction is significant (Cross * Sex * Phenotype). Significant two-way interactions were observed for Cross * Sex, Cross * Phenotype, and Block * Phenotype. From these analyses, it appears that the most important determinant of both sex and phenotype (non-mosaic or mosaic) of an individual animal is the type of the parental cross (A or B) that produced it. The significant Block * Phenotype interaction is discussed below.

(ii) *Rate and pattern of* w^{pch} *reversion*

Although data summarized in Table 1 reveal that wild-type revertants result among progeny of each type of cross, it is apparent that both the frequency

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Table 2. *Results of hierarchical log-linear analysis for interactions within genetic data*

Effect name	D.F.	Partial χ^2	P
Cross * Block * Sex	1	0·062	0·8040
Cross * Block * Phenotype	1	3·105	0·0780
Cross * Sex * Phenotype	1	11·416	0·0007
Block * Sex * Phenotype	1	0·211	0·6463
Cross * Block	1	0·019	0·8914
Cross * Sex	1	8·835	0·0030
Block * Sex	1	0·685	0·4079
Cross * Phenotype	1	8·505	0·0035
Block * Phenotype	1	7·484	0·0062
Sex * Phenotype	1	0·700	0·4028

and pattern of this process differ. Both male and female revertants were recovered in cross A progeny, but only female revertants were recovered in cross B progeny. The absence of male revertants among cross B progeny could result from small sample sizes. However, if the probability of recovering a male revertant is 0·0038 (Table 1, cross A1 and A2), then the probability of not recovering a male revertant among the males scored in cross B is $P = (0{\cdot}9962)^{(452+1514)} = 0{\cdot}00056$. Therefore, the lack of male revertants in cross B is unlikely to be due to sampling error alone since the sample size is sufficient to eliminate missing male revertants merely by chance. Among cross B progeny, germ-line reversion of w^{pch} appears to be limited to females.

In addition to differences in the pattern of reversion, the rates of reversion also differ markedly between the two types of crosses (Table 1). Results of statistical testing for differences in germ-line reversion rate among crosses are summarized in Table 3. As can be seen, the difference in reversion rates for A1 and A2 females are marginally significant while rates in B1 and B2 are not. Even within cross A1, the female reversion rate appears to be elevated above that for males. However, this difference is not statistically significant (2×2 contingency $\chi^2 = 3{\cdot}124$, $P > 0{\cdot}05$) suggesting that slightly higher reversion rates occurred for both females and males in cross A1 with respect to cross A2. Closer inspection of A1 reveals a possible source of the significant Block * Phenotype interaction shown in Table 2. Ratios of mosaic to non-mosaic animals for each of the cross A progeny classes are: A1 females (1·63), A1 males (1·28), A2 females (1·26), A2 males (1·13) suggesting that the higher germ-line reversion rate in A1 female progeny is accompanied by a similar increase in somatic excision rate leading to a significant effect of block on phenotype.

In comparing reversion rates between crosses A and B, only female progeny results are used since these occur in both cross types. From Tables 1 and 3, it is evident that the germ-line reversion rate among female progeny of cross B is three- to fourfold higher than in cross A. Reversion rates presented here for A1 and A2 are similar to the reversion rate of $\sim 10^{-2}$ per gene per generation for the *Mos*-containing E25H strain reported previously by Bryan *et al.* (1987).

(iii) *Maternal effect*

Animals that are heterozygous for *Mos* are expected to transmit the factor to only one-half of their offspring and therefore in crosses involving the w^{pch} allele, non-mosaic and mosaic progeny should occur in equal proportions. In strains of *D. simulans* into which both the unstable w^{pch} allele and *Mos* from *D. mauritiana* have been introduced through repeated backcrossing, Bryan & Hartl (1988) have reported a dramatic maternal effect in the pattern of *mariner* somatic excision with excess numbers of mosaic progeny resulting from crosses where the female parent is a *Mos* heterozygote. A similar effect is evident in the results presented here for *D. mauritiana* and produces the significant Cross by Phenotype interaction presented in Table 2. As can be seen in Table 1 for crosses A1 and A2, where the female parents are *Mos* heterozygotes (see Fig. 1), mosaic progeny were produced in excess of non-mosaics and this is true for both male and female progeny. In B1 and B2, where the male parents are the *Mos* heterozygotes, equal numbers of mosaic and non-mosaic female progeny are produced and this is the expected outcome of transmission of a single dominant genetic factor. However, in male progeny from B1 and B2 significantly more mosaic than non-mosaic animals are produced suggesting increased rates of somatic excision in male progeny of these crosses. This is particularly intriguing since it appears that germ-line excision is decreased in cross B males relative to the rate in females (Table 1).

(iv) *Sex ratios*

Results presented in Table 2 demonstrate a significant effect of cross on sex of subsequent progeny. In attempting to explain the excess numbers of mosaic male offspring of the B crosses shown in Table 1, an analysis of sex ratio for each of the crosses was carried out and the results are presented in Table 4. In A1 and A2, numbers of females to males is 1:1 while in B1 and B2 more male progeny than female progeny are produced. Close inspection of data in Table 1 shows

Table 3. *Two by two χ^2 contingency test for differences in germ-line reversion frequency of* w^{pch} *among female progeny of genetic crosses*

Test	D.F.	χ^2	P
A1 × A2	1	4·196	0·0405
B1 × B2	1	1·759	0·1847
A1 × B1	1	7·545	0·0060
A2 × B2	1	27·216	0·0001

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Table 4. *Test for distortion in sex ratio among progeny of genetic crosses*

Cross	Females	Males	χ^2	*P*
A1	785	797	0·09	N.S.
B1	395	452	3·84	$\leqslant 0{\cdot}05$
A2	2694	2673	0·082	N.S.
B2	1386	1514	5·650	$<0{\cdot}025$

N.S., not significant.

that mosaic males in particular are found in excess. Given that the ratio of mosaics : non-mosaics is 1 : 1 in B female progeny, the expected number of non-mosaic and mosaic males in crosses B1 and B2 is one-half the total number of females for each male phenotype, i.e. 197·5 and 693 respectively which are essentially the numbers of non-mosaic male progeny produced in each cross. It appears that not only is there an excess number of males in the B crosses, but that these are mosaic males carrying *Mos*.

4. Discussion

In the w^{pch} background, the behaviour of the *mariner* transposon can be followed genetically in the germ line, through the production of wild-type eye-colour revertants, and in the soma, through the production of eye-colour mosaics. By the examination of sibling progeny that have received either a Mos^+- or a *Mos*-containing chromosome into a common w^{pch} background, details of the interaction between *Mos* and *mariner* in each of these tissue types can be assessed. The crosses diagrammed in Fig. 1 produce offspring that are hemi- or homozygous for the w^{pch} allele, either heterozygous Mos/Mos^+ or homozygous Mos^+/Mos^+ and differ mainly in the source of egg cytoplasm.

Cross A progeny undergo development with cytoplasm that has come from Mos/Mos^+ heterozygous mothers. In each case, the A crosses produce results close to expected values for sex ratio (1 : 1), pattern and frequency of w^{pch} reversion ($\sim 10^{-2}$ in males and females), and are exceptional only in the production of excess mosaic male and female progeny, a result that is readily explained by a maternal effect similar to that already reported for *D. simulans* w^{pch}; *Mos* (Bryan & Hartl, 1988). The most likely cause of this phenomenon is the transmission of a factor (nucleic acid or protein) through the cytoplasm from mother to offspring that enhances *mariner* excision in the developing somatic tissue resulting in an excess of eye-colour mosaics. This factor may be a product of *Mos* itself or a result of interactions with other genetic determinants. Additional inquiry will be required to reveal its specific characteristics.

Cross B progeny undergo development with cytoplasm from Mos^+/Mos^+ homozygous mothers and produce exceptional results in several categories. Wild-type revertants are recovered at elevated frequencies only among female progeny, sex ratios are distorted with a male bias, and an excess number of mosaic males is produced. Since the cross B males receive the *Mos* excision factor from their fathers and cytoplasm that has not been associated with *Mos* from their mothers, the excess mosaic males are not expected to be the result of a maternal effect like that seen in cross A. Additionally, if a maternal effect were involved it should be evident in both male and female progeny as observed in cross A.

It is tempting to regard these diverse genetic phenomena (increased mutation rate, sex-limited mutation, distorted transmission and sex ratios) as 'hybrid dysgenesis-like'. The experimental crosses employed provide a mechanistic basis for these phenomena that is similar to 'hybrid dysgenesis' in that asymmetrical results follow inheritance of a specific cytoplasm type, and a factor (*Mos*) is involved that has been shown to mobilize the *mariner* element in the w^{pch} allele and other sites in the genome (Medhora *et al.* 1988). The germ-line and somatic reversion of the w^{pch} allele are readily explained by the mobilization of the inserted *mariner* element in the presence of the *Mos* excision factor. The increased rate of germ-line reversion in cross B females could result from an interaction (or lack thereof) between chromosomal copies of *Mos* and some specific determinant associated with the cytoplasm in animals carrying *Mos*. In this aspect, the system appears analogous to the P and M cytotype of *D. melanogaster*. Why the germ-line reversion would be limited to one sex in cross B is uncertain since revertants of both sexes are recovered among cross A progeny.

Any explanatory model must also account for the male-biased sex ratios observed among cross B progeny. Although transmission distortion has been reported in 'hybrid dysgenic' crosses in *D. melanogaster* (Hiraizumi, 1977; Bregliano & Kidwell, 1983), the sex-ratio distortion in the *D. mauritiana* crosses appears to result from an excess of a particular phenotypic class of males, namely, eye-colour mosaics. Interestingly, the animals present in excess demonstrate movement of *mariner* in the somatic tissue and absence of movement in the germ-line tissue that produced them. If the sex ratio distortion is a result of mobilization of *mariner*, new insertions could be disrupting the activity of genetic factors involved in sexual differentiation. However, since the two replicates of cross B produce such consistent results, this explanation requires that new insertions occur in similar genomic regions in each experiment. It is more likely that *mariner* mobilization results in the excision of transposon copies persisting in the w^{pch} and E25H strains used in these analyses. Excision of *mariner*, like that of the *P* element (Engels, 1983), often generates deletions (Jacobson, unpublished; Bryan *et al.* 1987) potentially affecting the genetic activity of flanking regions. Additional molecular analyses are required to

determine the degree to which mobilized *mariner* elements excise and/or transpose to other sites in the genome in these animals.

Because they occur in association with the *Mos* excision factor, the phenomena described above may involve mobilization of the *mariner* transposon. Whether the phenotypic consequences result entirely from new excision/insertion mutations or some other phenomenon is not clear and will require more detailed examination. Previous studies (Jacobson & Hartl, 1985; Haymer & Marsh, 1986; Bryan *et al.* 1987) have interpreted the somatic instability of *mariner* as an evolutionary paradox in that, unlike other *Drosophila* transposons (Engels, 1983; Rubin, 1983; Berg & Howe, 1989), its movement has not become restricted to the germ-line. An alternative hypothesis is suggested by the present results. *Mariner* excision is regulated in both tissues but, the regulatory mechanism(s) differ between the two tissue types. Additional investigation of the interactions between *Mos* and *mariner* will help to elucidate the details of these regulatory differences.

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References

Berg, D. E. & Howe, M. M. (1989). *Mobile DNA*. American Society for Microbiology, Washington, DC.

Blackman, R. K., Grimaila, R., Koehler, M. M. D. & Gelbart, W. M. (1987). Mobilization of *hobo* elements residing within the *Decapentaplegic* gene complex: suggestion of a new hybrid dysgenesis system in *Drosophila melanogaster*. *Cell* **49**, 497–505.

Bregliano, J. C. & Kidwell, M. G. (1983). Hybrid dysgenesis determinants. In *Mobile Genetic Elements* (ed. J. A. Shapiro), pp. 363–410. New York: Academic Press.

Bryan, G. J. & Hartl, D. L. (1988). Maternally inherited transposon excision in *Drosophila simulans*. *Science* **240**, 215–217.

Bryan, G. J., Jacobson, J. W. & Hartl, D. L. (1987). Heritable somatic excision of a Drosophila transposon. *Science* **235**, 1636–1638.

Engels, W. R. (1983). The P family of transposable elements in *Drosophila*. *Annual Review of Genetics* **17**, 315–344.

Engels, W. R. (1984). A *trans*-acting product needed for P-factor transposition in *Drosophila*. *Science* **226**, 1194–1196.

Fitzpatrick, G. J. & Sved, J. A. (1986). High levels of fitness modifiers induced by hybrid dysgenesis in *Drosophila melanogaster*. *Genetical Research* **48**, 89–94.

Haymer, D. S. & Marsh, J. L. (1986). Germ line and somatic instability of a *white* mutation in *Drosophila mauritiana* due to a transposable genetic element. *Developmental Genetics* **6**, 281–291.

Hiraizuma, Y. (1977). The relationship among transmission frequency, male recombination and progeny production in *Drosophila melanogaster*. *Genetics* **87**, 83–93.

Jacobson, J. W. & Hartl, D. L. (1985). Coupled instability of two *X*-linked genes in *Drosophila mauritiana*: germinal and somatic mutability. *Genetics* **111**, 57–65.

Jacobson, J. W., Medhora, M. M. & Hartl, D. L. (1986). Molecular structure of a somatically unstable transposable element in *Drosophila*. *Proceedings of the National Academy of Sciences, U S.A.* **83**, 8684–8688.

Kidwell, M. G., Kidwell, J. F. & Sved, J. A. (1977). Hybrid dysgenesis in *Drosophila melanogaster*: a syndrome of aberrant traits including mutation, sterility and male recombination. *Genetics* **86**, 813–833.

Mackay, T. F. C. (1986). Transposable element-induced fitness mutations in *Drosophila melanogaster*. *Genetical Research* **48**, 77–87.

Mackay, T. F. C. (1987). Transposable element-induced polygenic mutations in *Drosophila melanogaster*. *Genetical Research* **49**, 225–233.

Medhora, M. M., Macpeek, A. H. & Hartl, D. L. (1988). Excision of the *Drosophila* transposable element *mariner*: identification and characterization of the *Mos* factor. *EMBO Journal* **7**, 2185–2189.

Shapiro, J. A. (ed.) (1983). *Mobile Genetic Elements*. New York: Academic Press.

Simmons, M. J. & Bucholz, L. M. (1985). Transposase titration in *Drosophila melanogaster*: a model for cytotype in the P–M system of hybrid dysgenesis. *Proceedings of the National Academy of Sciences, U.S.A.* **82**, 8119–8123.

Sokal, R. R. & Rolf, F. J. (1987). *Introduction to Biostatistics*. New York: W. H. Freeman & Co.

Torkamanzehi, A., Moran, C. & Nicholas, F. W. (1988). P-element-induced mutation and quantitative variation in *Drosophila melanogaster*: lack of enhanced response to selection in lines derived from dysgenic crosses. *Genetical Research* **51**, 231–238.

Yannopoulos, G., Stamatis, N., Monastirioti, M., Hatzopoulos, P. & Louis, C. (1987). *Hobo* is responsible for the induction of hybrid dysgenesis by strains of *Drosophila melanogaster* bearing the male recombination factor *23.5MRF*. *Cell* **49**, 487–495.