Variation in *Y* Chromosome Segregation in Natural Populations of *Drosophila melanogaster*

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Manuscript received July 8, 1986
Revised copy accepted October 15, 1986

ABSTRACT

Functional variation among *Y* chromosomes in natural populations of *Drosophila melanogaster* was assayed by a segregation study. A total of 36 *Y* chromosomes was extracted and ten generations of replacement backcrossing yielded stocks with *Y* chromosomes in two different genetic backgrounds. Eleven of the *Y* chromosomes were from diverse geographic origins, and the remaining 25 were from locally captured flies. Segregation of sexes in adult offspring was scored for the four possible crosses among the two backgrounds with each *Y* chromosome. Although the design confounds meiotic drive and effects on viability, statistical partitioning of these effects reveals significant variation among lines in *Y* chromosome segregation. Results are discussed in regards to models of *Y*-linked segregation and viability effects, which suggest that *Y*-linked adaptive polymorphism is unlikely.

GENETIC variation on the *Y* chromosome is subject to the same forces that are thought to lead to evolutionary change in autosomes, including mutation, natural selection, and genetic drift. Techniques that have been classically applied by population geneticists to study the significance of selection and drift can be applied to *Y*-linked variation. The unique feature of the *Y* chromosome is that, with few exceptions, it is isolated by a lack of recombination with other chromosomes. *X-Y* recombination occurs in the telomeric region of the short arm in mammals (COOKE, BROWN and RAPPOLD 1985; ROUYER *et al.* 1986), and at low frequency in *Drosophila melanogaster* (WILLIAMSON and PARKER 1976). The molecular evolution of the *Y* chromosome is therefore expected to be dominated by nonclassical exchanges, such as transposition, in addition to mutation and drift. The unique fatherson transmission of the *Y* chromosome also provides an unusual opportunity to examine evolutionary forces, particularly migration. Theoretical models indicate that in panmictic populations, *Y*-linked variation can show cyclical dynamics, and that the opportunity for polymorphism is low (CLARK 1987a).

In order to consider the opportunity for adaptive evolution of the *Y* chromosome, it is first essential to realize that the *Y* chromosome is not genetically inert. The *Y* chromosome of *Drosophila melanogaster* represents about 13% of the metaphase chromosome length in males (KENNISON 1983), and although it is entirely heterochromatic in larval salivary gland preparations, it has sites that are actively transcribed during spermiogenesis (HAREVEN, ZUCKERMAN and LIFSCHYTZ 1986). Mutagenesis and deletion mapping studies (HAZELRIGG, FORNILI and KAUFMAN 1982) have demonstrated six distinct genes on the *Y* that are necessary for male fertility. High resolution cytological techniques reveal 25 bands on the prometaphase *Y* chromosome of *D. melanogaster*, and deficiency mapping has been used to establish a correspondence between the fertility factors and cytological landmarks (GATTI and PIMPINELLI 1983).

Although *XO* males of *D. melanogaster* are phenotypically indistinguishable from *XY* males, they are sterile due to failure in spermatogenesis. All six of the *Y* chromosome fertility factors are necessary for male fertility, and there is evidence that at least three of these factors are transcriptionally active genes whose products are found in the sperm axoneme (GOLDSTEIN, HARDY and LINDSLEY 1982). It should be recognized that although the *Y*-linked fertility factors are necessary for fertility, their presence does not guarantee fertility. The chromosomal basis of male fertility is in fact quite complex, since a number of *X*;autosome translocations are male sterile, as are many *Y*-autosome translocations (LYTTLE 1984). There is at least one region of homology between the *X* and *Y* chromosome that is involved in spermatogenesis (LIVAK 1984), and by examining the fertility of interspecific hybrid males, COYNE (1985) found that male fertility required that the *X* and *Y* chromosome be conspecific.

Another important class of *Y*-linked genes are those for rDNA (BONCINELLI *et al.* 1983). These genes occur in multiple tandemly duplicated arrays on both the *X* and the *Y* chromosomes, and low copy number of rDNA genes is associated with *bobbed*, the first phenotypically distinguishable *Y*-linked trait described in Drosophila (STERN 1927). Variation in the nontranscribed spacer of rDNA is readily observed on South-

ern blots, and this has already proven to be a useful probe for multiple insemination in Drosophila (WILLIAMS and STROBECK 1986), as well as other inferences about molecular evolution of the *Y* chromosome (WILLIAMS, DESALLE and STROBECK 1985; WILLIAMS *et al.* 1986). In *Drosophila mercatorum* an insufficiency of rRNA is associated with the phenotype *abnormal abdomen*, characterized by a retention of juvenile cuticle in the adult (TEMPLETON, CREASE and SHAH 1985; DESALLE, SLIGHTOM and ZIMMER 1986). Classical genetic methods reveal *Y*-linked modification of *aa* expression, and molecular methods reveal variation in rDNA copy number on the *Y*. The *X*-linked *aa* occurs as a natural polymorphism, and its molecular basis is inactivation of the 28S subunit gene by insertion of *ins* elements (DESALLE, SLIGHTOM and ZIMMER 1986).

Besides the essential function of the *Y* chromosome in male fertility and rDNA, normal segregation with the *X* chromosome is important for the maintenance of the population sex ratio. Evolutionary models of sex ratio distortion by meiotic drive of sex chromosomes usually consider *X*-linked factors that distort segregation, but the *Y* chromosome also has segregation determinants (FALK, BAKER and RAHAT 1985). This study is concerned with *Y*-linked variation that is expressed in ways that could affect the evolutionary dynamics of *Y*-linked polymorphisms. In particular, using a direct scoring of progeny segregation, we assay the combined influence of *Y*-linked variation on viability and meiotic drive.

MATERIALS AND METHODS

The following strains of *D. melanogaster* were used:

Harwich: a strong *P* cytotype strain originally started by M. TRACEY from an isofemale line caught in Harwich, Massachusetts, and kindly supplied by M. KIDWELL. In the I-R system of hybrid dysgenesis, the Harwich line has the *I* cytotype (KIDWELL 1979). This line was made isogenic using balancer chromosomes and maintained by sib mating.

R15-83: an isofemale line started from a female caught in the Rothrock State Forest in the summer of 1983 and sib mated 10 generations just prior to use in these experiments.

The following are isofemale lines, with location, collector and date indicated:

AH41 and AH198: Apple Hill, California, J. COYNE, fall 1981.

Egaa-1: Egaa, Denmark, A. CLARK, summer 1982.

Fan6: St. Paul, Minnesota, D. FAN, summer 1980.

GB13 and GB41: Gundlach-Bundschu Winery, Sonoma, California, J. COYNE, spring 1980.

Hikone: Laboratory stock, Hikone, Japan.

Samarkand: Laboratory stock, USSR, *ca.* 1930.

St-4: Stillwater, Minnesota, J. CURTSINGER, summer 1980.

Wd-4 and Wd-7: Woodside, California, A. CLARK, summer 1979.

R1-85 - R25-85: 25 isofemale lines from Rothrock State Forest, Centre County, Pennsylvania, A. CLARK, summer 1985.

From each of the 36 isofemale lines an isomale line was begun by crossing a single male to virgin females. Male descendants within an isomale line are identical by descent for the *Y* chromosome. Isomale lines were tested for PM cytotype by test crossing to Canton-S and Harwich and F_1 female offspring were dissected and scored for gonadal dysgenesis (SCHAEFER, KIDWELL and FAUSTO-STERLING 1979). A minimum of 20 offspring were scored within each line. All lines were found to be moderately or strongly P cytotype, with the exception of Hikone and Samarkand, which were clear M cytotypes. Since P cytotype lines are generally found to be I cytotype in the I-R system, we can infer that most of the lines also have the I cytotype. Hybrid dysgenic effects would not be induced when these lines are crossed to other I cytotype lines.

Virgin Harwich females were collected and crossed to males from each of the 36 female lines. Male offspring were again crossed to virgin Harwich females. This protocol was followed for ten consecutive generations, resulting in lines that were Harwich cytoplasm, Harwich autosomes and *X* chromosomes, and differing only in *Y* chromosomes. Another set of 36 *Y*-replacement lines was constructed in the R15-83 background. For brevity the 36 *Y*-replacement lines in the Harwich background will be referred to as the H lines, and the 36 *Y*-replacement lines in the R15-83 background will be denoted as R. In the absence of selection or drift, the expected probability of identity by descent of background alleles among the *Y* replacement lines is $1-0.5^{10}$ or 0.9990. Cellulose-acetate electrophoresis was used to check the lack of segregation among the *Y*-replacement lines (CLARK 1985). The original isofemale lines showed electrophoretic variation in the 6Pgd (1–0.6), Pgm (3–43.4), Idh (3–25.4), Est-C (3–47.7) and Est-6 (3–36.8), while samples of six flies per replacement line showed no variation. Cytotype tests, performed as described above, showed that all 72 replacement lines had strong *P* cytotypes. Replacement backcrossing should also result in all lines having the I cytotype of Harwich.

The 72 *Y* replacement lines were reared in half-pint milk bottles on Carolina 4–24 medium at 25° with a 12-hr light/dark cycle. Virgins were collected and for each *Y* chromosome, the following crosses were set up: H female × H male, H female × R male, R female × H male and R female × R male. Two virgin females and two males were placed into 95-mm shell vials to begin each cross. Mating and egglaying were allowed for four days, when the adult flies were transferred to a fresh duplicate vial for another four days of egg laying. In each of the four blocks, ten replicates were set up for each of the 144 crosses (36 *Y* chromosomes × four crosses). On the 17th or 18th day after flies were first introduced into a vial, the adult progeny were scored by sex. Subsequent scoring showed that over 96% of the progeny had emerged by this time.

RESULTS

The segregation data could be put into a table with (36 *Y* chromosomes) × (two maternal backgrounds) × (two paternal backgrounds) × (four blocks) × (five replicates) × (two duplicates) × (two sexes of offspring). A grand total of 328,132 flies were scored from 5760 vials, yielding an average productivity of 56.97 flies per vial. Each vial yielded an estimate of the sex ratio, defined as the fraction of the offspring that were males, and line means are reported in Table

TABLE 1

Progeny sex ratios (fraction male) pooled over crosses

Rothrock lines				Diverse lines			
Line	Sex ratio	SE	*N*	Line	Sex ratio	SE	*N*
R1	0.502	0.006	8812	AH41	0.489	0.006	10081
R2	0.498	0.007	9375	AH198	0.508	0.005	10207
R3	0.497	0.007	8743	Egaa-1	0.499	0.007	9326
R4	0.496	0.008	8378	Fan6	0.502	0.006	10685
R5	0.506	0.006	7644	GB13	0.500	0.008	7859
R6	0.475*	0.008	7662	GB41	0.514*	0.006	8842
R7	0.501	0.007	8032	Hikone	0.487	0.005	12853
R8	0.496	0.008	7826	Samarkand	0.484	0.007	8818
R9	0.506	0.006	8857	St-4	0.507	0.006	8263
R10	0.492	0.006	11147	Wd-4	0.510	0.006	9606
R11	0.502	0.006	9842	Wd-7	0.491	0.007	8270
R12	0.473*	0.007	7257				
R13	0.487	0.007	8649				
R14	0.495	0.007	8515				
R15	0.477*	0.006	9713				
R16	0.497	0.007	8573				
R17	0.501	0.007	9065				
R18	0.504	0.007	8916				
R19	0.500	0.006	9607				
R20	0.512*	0.006	9034				
R21	0.492	0.006	8029				
R22	0.500	0.007	9136				
R23	0.498	0.007	10397				
R24	0.502	0.007	11051				
R25	0.497	0.007	9062				

Asterisks indicate significant ($P < 0.05$) deviation from 0.5.

1. Figure 1 presents a histogram of the sex ratios of the entire sample, along with a histogram showing second chromosome segregation variation from a study of 28 nonlethal second chromosome replacement lines (A. G. CLARK and E. LYCKEGAARD, unpublished data). The sampling error in these two studies is expected to be similar because of the similarity in sampling effort and designs, yet the *Y* chromosome shows less variation in segregation than the second chromosomes ($F = 4.2$, $P < 0.001$). This suggests that the *Y* chromosomes have less variation in viability effects and/or meiotic drive than second chromosomes, an observation that is consistent with the theoretical expectation (see DISCUSSION).

Linear models: A number of preliminary tests were done to be sure that the data satisfied the assumptions for analysis of variance. The Kolmogorov D test indicated that the segregation data adequately fitted a normal distribution (STEPHENS 1974). Variances were found to be homogeneous across treatments [$F_{max} = 1.54$, not significant (NS)]. Means and variances in sex ratios were not significantly correlated ($r = -0.0034$), and there was no effect of vial density (count of progeny) on the sex ratio ($r = 0.02917$). Variances were also found to be independent of mean sex ratio, but there was a trend in that the more productive vials showed a lower sampling variance (Figure 2). This was treated by arcsine transforming the segregation data, although analyses with and without this transformation gave the same qualitative results. Nested analysis of variance indicated that the four blocks were homogeneous, so blocks were not included in the classifications used in the final model.

Environmental or rearing effects can be quantified by testing homogeneity of duplicates, replicates and blocks. If the parents in a particular vial became infected or were otherwise affected by an environmental agent that resulted in a skewed offspring sex ratio, then this effect might be carried over into the duplicate vial when they are transferred. Such an effect would result in a correlation among duplicate pairs. When segregations of duplicate pairs are adjusted by group means and plotted (Figure 3), we find no significant correlation ($r = 0.0402$, NS).

The linear model that was fitted to the data could be written as:

$$s_{ijkl} = u + y_i + m_j + p_k + (ym)_{ij} + (yp)_{ik} + (ymp)_{ijk} + e_{ijkl}$$

where s_{ijkl} is the segregation of the ith *Y* chromosome with maternal background j and paternal background k, in replicate l; u represents the grand mean; m_j the maternal background (H or R); p_k the paternal background (H or R) and e_{ijkl} is the error term. The

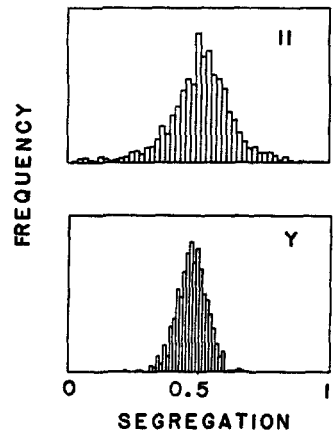

FIGURE 1.—Histograms of segregations of wild extracted second and *Y* chromosomes. The top figure is for 28 extracted second chromosomes, where the segregation is the fraction wild phenotype progeny from the cross +/SM5 × +/+ (A. G. CLARK and E. LYCKEGAARD, unpublished data). The lower figure is for the *Y* lines studied here, where the segregation parameter is the fraction of progeny that are male.

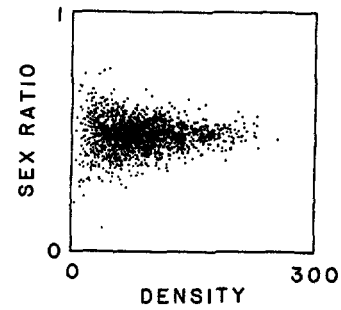

FIGURE 2.—Scattergram of sex ratio (fraction male) against density for the 5760 vials scored.

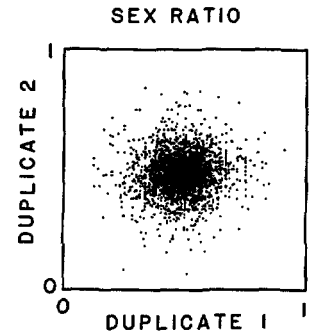

FIGURE 3.—Scattergram of sex ratios in progeny from duplicate pairs of vials, where duplicates shared parental flies.

interaction terms *ym* and *yp* account for the possibility of differences in segregation of a particular *Y* chromosome due to maternal or paternal backgrounds. The *Y* chromosomes are considered to come from a population sample, and are treated as a random effect, while the maternal and paternal backgrounds are treated as fixed effects. Note that throughout the analysis of the linear model, there is no attempt to separate the effects of meiotic drive and viability as causes of what are referred to as variation in segregation.

This model was tested by a mixed model analysis of variance using the data from 22 stocks with *Y* chromosomes of diverse geographic origin, the 50 Rothrock stocks, and the entire set of 72 lines considered together. Results appear in Table 2. The partitioning of variance was similar for all three of these analyses, indicating that the within- and among-population levels of variation are comparable. The *Y* chromosome main effect was significant in two of the three analyses. Simple one-way analysis of variance comparing *Y* segregation within backgrounds or pooled across backgrounds also showed significant heterogeneity. The maternal background was also a significant main effect in the analysis of diverse lines and in the pooled analysis, perhaps reflecting viability differences between the Harwich and Rothrock X chromosomes. Further discussion of the biological interpretation of these terms appears in the Discussion.

Log-linear models: The sex ratio scored from the progeny of a cross can be distorted by either sex-specific differences in viability or due to meiotic drive. Differences among *Y* replacement lines in progeny sex ratio may likewise be due to *Y* chromosome mediated viability or meiotic effects. To some extent these effects can be examined by using the different genetic backgrounds, and testing interactions between the *Y* chromosomes and the backgrounds. Table 3 indicates a parameterization of the offspring counts, allowing viability differences among the three female genotypes, a viability effect of each of the 36 *Y* chromosomes in each of the two backgrounds, and a segregation parameter for each of the 36 *Y* chromosomes with each of the two background *X* chromosomes.

As described below, the model parameters coincide with the terms in a log-linear analysis. The significance of these terms was tested by fitting the data to a hierarchical series of log-linear models (BISHOP, FIENBERG and HOLLAND 1975). The data, when pooled over blocks, replicates and duplicates, can be considered as a contingency table with 36 *Y* chromosomes × two maternal backgrounds × two paternal backgrounds × two sexes of offspring. The first three of these dimensions are constrained by the design of the experiment, and it is only the last variable that is measured as a response variable. Hence all log-linear models have a fixed configuration of terms including:

$$C_{YMP} = u + u_Y + u_M + u_P + u_{YM} + u_{YP} + u_{MP} + u_{YMP}.$$

The count of males and females that emerge from each vial serve as the response variable, and we are interested in seeing how this response is affected by the design variables *Y* chromosome, *M*aternal background and *P*aternal background. Define the variable R as the response. A hierarchical subset of the following model was tested for goodness-of-fit to the data

TABLE 2

Analysis of variance of *Y* chromosome segregations

	Diverse lines			Rothrock lines			Pooled		
	d.f.	SS	*F*	d.f.	SS	*F*	d.f.	SS	F
Y	10	0.165	1.11	24	0.409	1.88**	35	0.588	1.56*
M	1	0.088	13.73***	1	0.027	1.91	1	0.091	16.93***
Y × M	10	0.104	1.51	24	0.214	0.68	35	0.344	0.87
P	1	0.004	0.52	1	0.003	0.19	1	0.007	0.89
Y × P	10	0.112	1.63	24	0.314	1.01	35	0.427	1.08
M × P	1	0.003	0.42	1	0.018	1.40	1	0.007	0.60
Y × M × P	10	0.069	0.82	24	0.312	1.55	35	0.395	1.34
Error	1716	14.977		3900	32.760		5616	49.049	

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

TABLE 3

Parameters of viability and meiotic segregation

Cross	Progeny
H × H	$K_{Hi}V_{HF}$
	$(1\text{-}K_{Hi})V_{HM}$
H × R	$K_{Ri}V_{HRF}$
	$(1\text{-}K_{Ri})V_{HM}$
R × H	$K_{Hi}V_{HRF}$
	$(1\text{-}K_{Hi})V_{RM}$
R × R	$K_{Ri}V_{RF}$
	$(1\text{-}K_{Ri})V_{RM}$

using the BMDP routine P4F (BROWN 1983):

$$\log(m_{YMPR}) = C_{YMP} + u_R + u_{YR} + u_{MR} + u_{PR} + u_{YMR} + u_{YPR} + u_{MPR} + u_{YMPR}.$$

The goodness-of-fit tests for these models were scaled by the among-replicate heterogeneity chi-square in order to account for the cluster sampling of these data (ANDERSON *et al.* 1986; BRIER 1980; CLARK 1985). Model fits are presented in Table 4, and the significance of individual terms are given in Table 5. The log-linear analysis confirms the significance of the variation in segregation among the *Y* chromosomes, as indicated by the significant *YR* term and the necessity in incorporating this term in the model.

The absence of a significant MPR interaction implies a lack of significance of variation among the female viabilities, and this makes the biological interpretation of the log-linear terms very direct. The *YR* term reflects differences among the *Y* chromosomes (in either viability or meiotic segregation) that are not influenced by the genetic background. The *YMR* term is associated with a viability effect, where the different *Y* chromosomes confer different viabilities of the males that bear them, and this viability depends on the parental origin of the *X* chromosome. If there was

TABLE 4

Log-linear analysis of *Y* chromosome segregations

Model	Degrees of freedom	Chi square
1. *YMP*, R	143	245.67***
2. *YMP*, *YR*	108	176.29***
3. *YMP*, MR	142	229.42***
4. *YMP*, PR	142	251.23***
5. *YMP*, *YR*, MR	107	151.80**
6. *YMP*, *YMR*	72	103.13**
7. *YMP*, *YR*, PR	107	172.94***
8. *YMP*, *YPR*	72	124.44***
9. *YMP*, MR, PR	141	228.72***
10. *YMP*, MPR	140	230.17***
11. *YMP*, *YR*, MR, PR	106	154.65**
12. *YMP*, *YR*, MPR	105	153.15**
13. *YMP*, *YPR*, MR	71	98.97*
14. *YMP*, *YMR*, MR	71	99.47*
15. *YMP*, *YPR*, MPR	70	101.05**
16. *YMP*, *YMR*, MPR	70	101.50**
17. *YMP*, *YMR*, *YPR*	36	50.89
18. *YMP*, *YMR*, *YPR*, MPR	35	48.76

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

TABLE 5

Significance of terms in log-linear models

Term	Models	Degrees of freedom	Chi square
YR	1–2	35	69.38**
MR	1–3	1	16.25***
PR	1–4	1	0
YMR	5–6	35	48.67[a]
YPR	7–8	35	48.50[a]
MPR	9–10	1	0

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.
[a] $\chi^2_{(35\text{d.f.})}(0.05) = 49.8$.

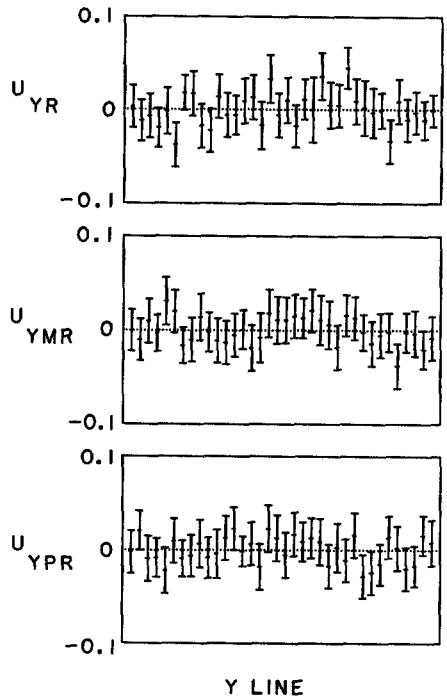

FIGURE 4.—Plots of the log-linear terms for each of the 36 *Y* chromosome lines, obtained from fits of the saturated model to the data. Model estimates ± 2 SE values are plotted on the vertical axis. Position on the *X* axis conveys no statistical information, but indicates the *Y* line identity. The 11 diverse lines are indicated first, with the 25 *Rothrock* lines to the right.

significant variation among the *Y* chromosomes in meiotic behavior, and it depended on whether the males have an H or R *X* chromosome, this would be revealed by a significant *Y*PR term. Models that adequately fit the data (Table 4) require the presence of both of *Y*MR and *Y*PR, even though these terms just miss significance at the 5% level.

Selected log-linear terms are displayed graphically in Figure 4. The figures show the model estimates of the terms for the 36 *Y* chromosomes with error bars showing ± two standard errors. There are five *Y* chromosomes whose segregation term (*Y*R) is significantly different from zero, two show a significant *Y*MR term and one shows a significant *Y*PR term. The deviant lines are identified in Table 1.

Another way to examine the acceptability of log-linear models is to plot the ratio of the parameter estimates to their standard errors (λ/SE) on a normal probability scale against their rank order. If the null hypothesis were true, then these points would fall on a straight line (DANIEL and WOOD 1980, CLARK 1987b). Figure 5 shows the results of this procedure. The *Y*R terms show five lines that deviate from straight lines, there are two *Y*MR terms that deviate and one *Y*PR term that deviates. The overall lack of *Y*MPR term significance can be judged by the linearity of the points in the last panel of Figure 5, which shows an excellent fit.

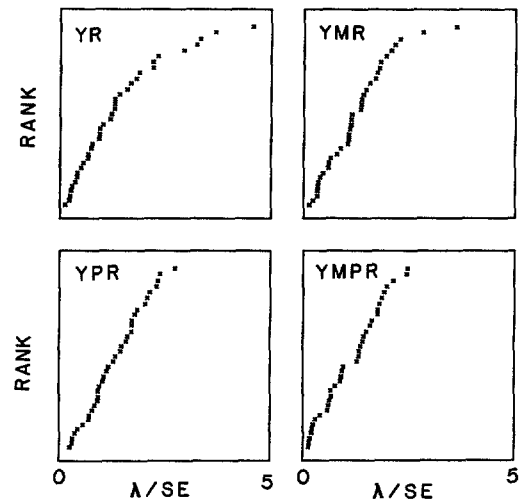

FIGURE 5.—The ratios of the log-linear terms to their standard errors are plotted on a normal probability scale against their rank order. Under the null hypothesis these should fall on a straight line. Deviations indicate lines with significant departures.

DISCUSSION

This study represents an assay of *Y* chromosome mediated variation in sex chromosome segregation in *D. melanogaster*, and comparison with the classical second chromosome extraction results shows that the *Y* chromosome is much less variable than the second chromosome. Despite the low level of *Y* chromosome segregation variation, the sampling effort was sufficient to detect subtle differences with statistical significance. Interpretation of this result requires consideration of the strengths and weaknesses of this type of segregation study. Ideally it would be possible to score male gametes to have a direct assessment of meiotic products. Cytological techniques that reveal mature sperm bundles have been useful in identifying a cause of segregation distortion in the *SD* system (TOKUYASU, PEACOCK and HARDY 1977; HARTL and HIRAIZUMI 1976), but for such slight deviations from Mendelian segregation, such techniques are not practical. The confounding of meiotic drive and viability effects precludes an understanding of a precise mechanism, but the ascertainment of variation in segregation reveals naturally occurring *Y* chromosome variation that affects transmission dynamics.

A potential problem with segregation studies is the artifactual influence of hybrid dysgenesis induced transposition of transposable elements. Dysgenic crosses could lead to gross distortions of segregation in association with the syndrome of abnormalities caused by hybrid dysgenesis (BREGLIANO and KIDWELL 1983). In addition to the direct segregation effects, dysgenic crosses would cause mobilization of P and/or I element transposition, with the result that genetic backgrounds and extracted chromosomes would undergo rapid mutational divergence. In these studies attempts were made to minimize the problems

of hybrid dysgenesis by testing cytotypes of initial stocks, and performing replacement backcrossing in the same P cytotype. It should be appreciated however that even if all stocks are P cytotype, P element transposition may still occur, but the rate of transposition is about 1/23 that under dysgenic crosses (ENGELS 1983). There was some variation in the strength of P cytotypes in the lines both before and after the replacement backcrossing, and it is not clear whether there is a low level of P element transposition and hybrid dysgenesis under conditions of varying P cytotype frequency. Since the differences observed among lines were subtle, it is not possible to rule out a subtle dysgenic effect. However, if there is such a subtle dysgenic effect, it is likely to be occurring in natural populations as well.

Segregation studies such as this have several distinct advantages over other assays of variation. Perhaps most important is that the variation being assayed is functional: the parameters of transmission that are being estimated have direct bearing on the evolutionary dynamics of the polymorphisms. The genetic background is well controlled, so that the effects that are seen can be safely ascribed to the varying *Y* chromosomes. Each cross resulted in progeny with female genotypes in common for all vials, so that the segregations are relative to a genotype with a standardized viability, analogous to the Cy/Pm technique (as opposed to the Cy technique of second chromosome segregation assay, which assumes dominance of the balancer chromosome). Finally, environmental effects are well accounted for by the design.

Despite the differences in the assumptions of the linear and log-linear statistical tests, both methods reveal significant differences in *Y* chromosome segregation. Analysis of variance makes use of the approximately normal distribution of the data to determine the probability that samples were drawn from the same distribution. The observed variance in segregation is partitioned into components, and the significance of effects is determined by the magnitudes of these components. Log-linear analyses are perhaps more specifically designed for the count data that we have here. The assumption of multinomial sampling is known to be violated, but it is violated in a way that is fairly well understood, and correction for cluster sampling is easily done. The biological interpretation of terms of the log-linear model may be more direct than that of linear models. Finally, the test of goodness-of-fit allows an additional assessment of the model's ability to describe the data.

Another approach to ascertain genetic variation in sex ratio is to consider it as a quantitative trait. TORO and CHARLESWORTH (1982) applied directional selection and sib analysis to a sample of the Ives population of *D. melanogaster*. No heterogeneity in sex ratio was detected by either technique. Although this method should detect genetic variation in sex ratio attributable to any chromosome, the sampling effort was somewhat smaller and fewer initial lines were used. In addition, the high degree of homozygosity of extracted lines used in our study may accentuate the expression of variation among lines.

The finding of very small levels of *Y* chromosome segregation variation is not surprising in light of theoretical models. HAMILTON (1967) showed that *Y* chromosome meiotic drive would result in very rapid fixation of the favored chromosome. *Y* chromosome drive has been described in the mosquito *Aedes aegypti* in an apparently balanced polymorphism (HASTINGS and WOOD 1978). MAFFI and JAYAKAR (1981) analyzed a two-locus modifier model of the *Y* drive system in *Aedes*, and found restrictive conditions of linkage and drive under which *Y* chromosome polymorphism could be maintained. Opposing viability effects can maintain polymorphism of meiotically driven autosomal and *X*-linked alleles, but there are no conditions on autonomous *Y*-linked variation that can maintain a balanced polymorphism (CLARK 1987a). With *X-Y* interactions in drive and viability effects, *Y* polymorphism can be maintained, but only a very small portion of the parameter space admits polymorphism. The theoretical conclusion is that the opportunity for adaptive *Y* polymorphism is very small, and the small effects that were detected are most likely transient.

A significant point raised by CURTSINGER (1984) and supported by the findings here is that subtle effects of meiotic distortion may be fairly common in natural populations, and the consequences of even subtle meiotic distortions can be very significant to the evolution of a genetic element. The biology of chromosomal segregation suggests meiotic mechanisms for slight distortion from Mendelian segregation. MCKEE (1984) observed *X-Y* chromosome segregation in male *D. melanogaster* that were deficient in *X* heterochromatin (Xh^-), and found that *Y* and *XY* bearing spermatids failed in the individualization process in spermiogenesis, analogous to the degeneration of the non-*SD* homolog on the second chromosome. This raises the question of whether there may be *Rsp*-like sites on the *Y* chromosome that may modulate meiotic behavior. MCKEE's (1984) results discredit a competitive model of meiotic drive, where chromosome binding sites are thought to be limiting, but are consistent with mechanisms that yield a continuum of meiotic drive parameters.

BAKER and CARPENTER (1972) observed sex-specific differences in first division meiotic mutants, and argued that meiotic drive in males may be an evolutionarily favored mechanism for removing nondisjunc-

tional meiotic products. The problem with this argument is that the conditions for invasion of a meiotically driven allele depend on its own transmission properties, not on indirect benefits to the population. But if the driven allele increases male fertility by selectively removing nondisjunctional gametes, it may have a direct selective advantage. The observation of subtle segregation variation among *Y* chromosomes is consistent with the quantitative nature of segregation determinants. Families of repeated DNA sequences appear to be important in the determination of male fertility as well (HAREVEN, ZUCKERMAN and LIFSCHYTZ 1986), with dramatic differences occurring among even closely related species. By careful examination of the mechanism of sex chromosome dysjunction, the molecular basis of spermiogenesis, and patterns of *Y*-DNA sequence variation, a rich picture of *Y* chromosome evolution is emerging.

I thank LISA KEITH, JESSICA JONES, ERIK KEITH, and EVA LYCKEGAARD for technical help. This work was supported in part by National Institutes of Health grant HD18379 and National Science Foundation grant BSR-8515952.

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Communicating editor: B. S. WEIR