DISTORTED SEX RATIOS DUE TO *SEGREGATION DISTORTER* IN *DROSOPHILA MELANOGASTER*[1]

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SANDLER and NOVITSKI (1957) have defined "meiotic drive" as a force resulting from the mechanics of the meiotic divisions which is potentially capable of altering gene frequencies. One of the best studied cases of meiotic drive is *segregation distorter* (symbolized *SD*) in *Drosophila melanogaster*. *SD* is probably genic in nature and located in the centromere region of chromosome 2. When an *SD*-bearing chromosome is heterozygous with an SD^+-bearing chromosome in a male, the resulting *SD* sperm fertilize a greater number of eggs than do the SD^+ sperm (SANDLER, HIRAIZUMI, and SANDLER 1959).

In addition, there are two modifiers of *SD* located on the second chromosome. *Stabilizer of SD* (symbolized *St(SD)*) is located in the distal portion of the right arm (DENELL and JUDD 1968). When it is present, *SD* action is stable and the segregation ratio (defined as k: the frequency of *SD* among total second chromosomes recovered in the F_1 progeny) is consistently high from male to male; when it is absent k shows much more variation between males resulting in a reduced value for the mean k (SANDLER and HIRAIZUMI 1960a). Also, very closely linked and to the right of *SD* is *activator of SD* (symbolized *Ac(SD)*) which, in coupling with *SD*, is necessary for distortion to occur (SANDLER and HIRAIZUMI 1960b; HIRAIZUMI and NAKAZIMA 1967).

Segregation distorter chromosomes collected in the wild and carrying all three elements are referred to as "original-*SD*" lines, while *SD Ac(SD)* chromosomes derived by recombination are referred to as "recombinant-*SD*" lines.

HIRAIZUMI and NAKAZIMA (1967) showed that *SD* causes a distortion of sex chromosome recovery in addition to its effect on the second chromosome. Since their *SD* stocks had been repeatedly backcrossed to a *cn bw* stock to yield a uniform *cn bw* genetic background, these authors chose the sex ratio (defined as the proportion males recovered) of this *cn bw* stock as a standard value. When males carrying different combinations of the three components of the *SD* system heterozygous with a *cn bw* second chromosome were test crossed to *cn bw* females, the sex ratio in the F_1 classes representing the recovery of one or the other paternal second chromosome were often different from each other and from the control sex ratio. These authors further noted that the level of distorted sex chromosome recovery appears positively correlated with the value of k both within and be-

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tween male lines. HIRAIZUMI and NAKAZIMA suggested that during meiosis I there is a nonrandom segregation of the second and sex chromosomes into presumptive functional and nonfunctional sperm.

The purpose of this study is to further examine the relationship between k and sex ratio in the progeny of *SD* males. Evidence will be presented suggesting that first and second chromosome segregation occur at random with respect to each other at the first meiotic division. It will be argued that there is distorted sex chromosome recovery only from SD^+-bearing gametes.

MATERIALS AND METHODS

SD-72 is an "original-*SD*" chromosome collected from a wild population in Madison, Wisconsin (SANDLER, HIRAIZUMI, and SANDLER 1959). *R(SD-36)-1* is a "recombinant-*SD*" chromosome derived from *SD-36*, another "original-*SD*" chromosome collected in Madison (SANDLER and HIRAIZUMI 1959). These *SD* chromosomes were maintained heterozygous with a *cn bw*-bearing second chromosome (*cn*: cinnabar eye, 2-57.5; *bw*; brown eye, 2-104.5) in males. Such males were backcrossed every generation to *cn bw* females to provide a standard genetic background. All males used in these experiments were heterozygous for *cn bw*, so that references to *SD* males are understood to mean *SD/cn bw* males.

Experiments were performed in shell vials containing standard Drosophila cornmeal medium. Except under specified conditions, all flies were maintained at the laboratory temperature, 23°C.

The analysis of untreated lines: Over several years, standard crosses of *SD-72/cn bw* males individually to *cn bw* females were performed, most often as controls for various experimental treatments. These experiments did vary somewhat in the age of males when crosses were initiated (1 to 5 days), in the number of *cn bw* tester females used (2 to 5), and in the time over which mating was allowed (2 to 5 days). Females were either discarded after the mating interval or were transferred several additional times to yield a greater number of progeny. Since any aging effect on *SD-72* males over such a time period would be small and since mated females show no change in recovery ratios over such a time interval, these crosses are pooled for the purpose of the analysis to be described.

The *R(SD-36)-1* males analyzed in this section are the control group in the temperature experiment, for which details are supplied below.

The effect of aging on sex ratio: One day old *SD-72/cn bw* males were mated individually with four *cn bw* females. The males were then transferred without anesthetization to four new females every two days until the end of the eighth brood. The females within each brood were then transferred three times over a total of ten days after the initial mating. The progeny of each of these eight broods, representing sperm sampled for two days intervals, were counted; these broods are denoted as A through H. Data are presented only for those twenty males which remained fertile through brood H.

Low temperature treatment: SD-72/cn bw males were crossed to *cn bw* females. After several days the parents were discarded and the cultures were either maintained at the laboratory temperature or placed at 18°C. Young *SD-72/cn bw* males were chosen from the early hatch of each group and test crossed individually with 5 *cn bw* females. The males were discarded after three days, and the females were transferred three times over a total of eleven days and discarded. All of the F_1 progeny were scored.

R(SD-36)-1/cn bw males were also crossed to *cn bw* females. After several days the parents were discarded and the culture vials were either maintained at the laboratory temperature or transferred to 18°C. Young *R(SD-36)-1/cn bw* males were chosen from the early hatching progeny of each group and mated individually with three *cn bw* females. All parents were cleared after four days, and the resulting progeny were scored.

RESULTS

Variation within untreated lines; tests of SD-72/cn bw *males:* As described above, eight experiments were performed involving in part the cross of *SD-72/cn bw* males individually with *cn bw* females. The data from these experiments are summarized in Table 1. The relative recovery of paternal chromosomes is measured by three ratios: k, the proportion of *SD* among total paternal second chromosomes recovered; *SD* sex ratio, the proportion of males among progeny receiving the paternal *SD* second chromosome; and *cn bw* sex ratio, the proportion of males among progeny receiving the paternal *cn bw* second chromosome. For the values in Table 1, the three ratios were calculated for each of the males tested (except, of course, in the case of males producing no *cn bw* progeny, for which no *cn bw* sex ratio can be calculated). The mean k and sex ratio values in Table 1 are calculated from the equally weighted values for each male tested.

Relationships among k, *SD* sex ratio, and *cn bw* sex ratio may be ascertained from these data. For the 428 *SD-72* males tested, a linear regression was calculated showing the change in *SD* sex ratio with respect to k. Similarly, for the 160 of these males which yielded *cn bw* offspring, regressions were calculated for the *cn bw* sex ratio with respect to k, and for the *cn bw* sex ratio with respect to *SD* sex ratio. The regression coefficients from these comparisons are presented in Table 2.

It is evident from the regression coefficient found that the *SD* sex ratio of a male is independent of the k value it shows. However, the regression coefficient showing the change in *cn bw* sex ratio with respect to k is significantly different from zero ($P < .02$). Thus as second chromosome distortion (k) decreases, the proportion of males among *cn bw* offspring increases (which also represents a decrease

TABLE 1

A summary of recovery ratios from a series of experiments in which SD-72/cn bw *(AI–VIII) or* R(SD-36)–1/cn bw *(BI) males were mated individually to* cn bw *females*

Experiment	Number of males tested	Average number of progeny/♂	F_1 sex ratio		k
			SD	*cn bw*	
AI	19	193	.538	.298	.994
AII	23	235	.512	.156	.990
AIII	94	216	.541	.256	.992
AIV	94	217	.528	.222	.990
AV	50	514	.511	.158	.994
AVI	64	168	.503	.080	.995
AVII	28	397	.508	.105	.987
AVIII	56	209	.534	.223	.994
A Total	428	255	.524	.190	.992
BI	96	131	.527	.399	.789

(k = the proportion *SD* among total F_1 progeny; *SD* and *cn bw* sex ratios = the proportion males among progeny receiving the *SD* and *cn bw* paternal second chromosome respectively.)

TABLE 2

Regression coefficients (b) *for comparisons of the recovery ratios indicated from* SD-72/cn bw *or* R(SD-36)–1/cn bw *males test crossed to* cn bw *females, with the associated Student's* t *values and degrees of freedom from a test with null hypothesis* b = 0

	SD-72/cn bw			*R(SD-36)-1/cn bw*		
	b	df	*t*	*b*	df	*t*
SD sex ratio *vs. k*	0.011	427	0.13	—0.021	95	—0.50
cn bw sex ratio *vs. k*	—1.782	159	—2.45*	—0.371	95	—2.89**
cn bw sex ratio *vs. SD* sex ratio	—0.296	159	—0.54	0.035	95	0.10

*, ** indicate a significant difference at the .05 and .01 levels, respectively.

in distortion). Finally, as is expected from the previous two results, *cn bw* sex ratio is independent of *SD* sex ratio.

Test of R(SD-36)-1/cn bw *males:* Ninety-six males from this "recombinant-*SD*" line were test crossed individually to *cn bw* females, and the resulting mean recovery ratios are also given in Table 1. Relationships between these recovery ratios were analyzed by the same methods already described for *SD-72* males, and the resultant regression coefficients are presented in Table 2. The results are equivalent to those found for the "original-*SD*" line: the *cn bw* sex ratio is related to *k* and independent of the *SD* sex ratio, while the *SD* sex ratio is independent of *k*.

The effect of aging on sex ratio: SANDLER and HIRAIZUMI (1961b) noted that both "original-" and "recombinant-*SD*" chromosomes tested showed a decrease of the mean *k* value with increasing age of the parental male. Each line studied approached a lower limit at its own rate, but this limit was reached at about the same time (17 days).

SD-72/cn bw males were collected over several hours, aged one day, and mated individually with four *cn bw* females. They were then transferred without anesthetization to four new females every two days until the end of the eighth brood. These eight broods, representing sperm sampled for two day intervals, are denoted as A through H. The resulting recovery ratios are presented in Figure 1.

Figure 1 shows the relationship between the recovery ratios and the age of the males tested. The mean *k* value of these males decreases with respect to time with a slope $b_k = -0.0107$, which is significantly different from zero at the 1%-level ($t = -5.35$, df = 7). The data are consistent with those of SANDLER and HIRAIZUMI (1961b) for *SD-5*, another "original-*SD*" line.

The *SD* sex ratio shows no change with respect to time over the period examined ($b_{SD} = 0.0006$, $t = .005$, df = 7). However, the *cn bw* sex ratio increases with time ($b_{cn\ bw} = 0.0430$). This estimate of the rate of increase is significantly different from zero at the 1%-level ($t = 4.13$, df = 7).

The effect of low temperature treatment: Experiments were performed to test for a temperature effect on the recovery of the first and second chromosomes in the progeny of *SD-72* and *R(SD-36)-1* males. As described previously, the control

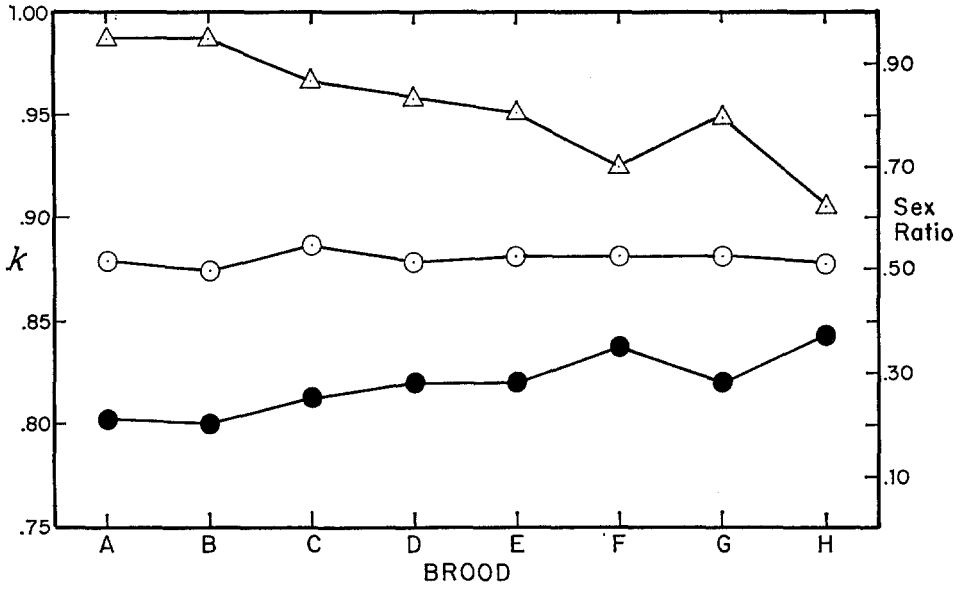

FIGURE 1.—Mean values of k (△), *SD* sex ratio (○), and *cn bw* sex ratio (●) are depicted for *SD-72/cn bw* males brooded every two days with new *cn bw* females. The left vertical axis designates values of k, and sex ratio values are presented on the right vertical axis.

males in each case were maintained at laboratory temperature (23°C) throughout development, while the treated males were placed at 18°C from larval stages through eclosion. All males were test crossed to *cn bw* females. The resulting data are presented in Table 3. The associated statistical analysis utilized an analysis of variance, with k values from *SD-72* males transformed to $\sin^{-1}\sqrt{k}$. Note that the two control groups were included in Table 1 (AV and BI).

The k values of both types of *SD* males were affected by the cold temperature treatment ($P < .01$), but in opposite directions. For the *SD-72* chromosome, the decrease in second chromosome distortion (k) is accompanied by an increase in *cn bw* sex ratio ($P < .05$), which also represents a decrease in distortion. The

TABLE 3

Recovery ratios among offspring from the control and cold treated males mated to cn bw/cn bw *females*

Parental males	Treatment	Number of males tested	k	F_1 sex ratios	
				SD	*cn bw*
SD-72/cn bw	23°C	50	.994	.511	.158
	18°C	16	.901**	.498	.333*
R(SD-36)-1/cn mw	23°C	96	.789	.527	.399
	18°C	105	.885**	.524	.373

*, ** indicate a significant difference from the control at the .05 and .01 levels, respectively.

sex ratio of *SD* offspring remains unchanged. In the case of the *R(SD-36)-1* chromosome, neither the *SD* nor the *cn bw* sex ratios show a statistically significant change with temperature shock, despite the increase in *k*.

DISCUSSION

In early work on segregation distortion SANDLER, HIRAIZUMI, and SANDLER (1959) suggested a model in which, at some stage of meiosis, *SD* causes a misreplication (breakage) of the SD^+-bearing chromosome which is then eliminated before fertilization, presumably due to a bridge formation at anaphase II. CROW, THOMAS and SANDLER (1962) provided confirmatory evidence that breakage is involved. However, in a cytological examination of spermatogenesis in *SD* males, PEACOCK and ERICKSON (1965) found no evidence for chromosome breakage or bridge formation.

PEACOCK and ERICKSON (1965) counted stored sperm and progeny recovered in females inseminated by young *SD* males, and found that only about one-half of the stored sperm successfully fertilized eggs. This result was paralleled when wild-type males were used. PEACOCK and ERICKSON suggested that as a regular aspect of meiosis in *D. melanogaster* an inequality of the spindle poles exists at the first meiotic division such that two functional and two nonfunctional sperm are eventually produced. They further suggested that segregation distortion acts by the differential segregation of the *SD*-bearing second chromosome to the functional pole.

HIRAIZUMI and NAKAZIMA (1967) noted an effect of segregation distortion on sex chromosome recovery. They concluded that *SD* causes a severely distorted sex ratio among progeny receiving the paternal SD^+ chromosome, and a slightly distorted sex ratio when the paternal *SD* chromosome is recovered. They proposed a hypothesis, based on the functional pole model, in which *SD* has some sort of homology with some part of the *X* chromosome such that they compete to reach the functional pole. They further proposed that *Ac(SD)* and *St(SD)* "inactivate" the *X* chromosome in such a way that the probability of the *X* reaching the functional pole is reduced and that of the *SD* second chromosome increased.

The PEACOCK and ERICKSON "functional pole" hypothesis for the mechanism of segregation distortion clearly predicts that actively distorting *SD* males will produce the same number of progeny as less actively distorting males or equivalent wild-type males under conditions of optimal recovery of zygotes. However, HARTL, HIRAIZUMI, and CROW (1967) have reported a significant negative correlation between the number of offspring and the degree of distortion when sperm are exhaustively sampled from young *SD* males. This decrease in fertility was interpreted in terms of a mechanism in which the sperm receiving the non-*SD* chromosome are somehow rendered unable to follow the normal course leading ultimately to fertilization. This model is denoted the "dysfunctional sperm" hypothesis.

It should be clearly noted that although the explanation for deviant sex ratios proposed by HIRAIZUMI and NAKAZIMA (1967) was based on the functional pole

model, HIRAIZUMI, as one of the proponents of the dysfunctional sperm model (HARTL, HIRAIZUMI, and CROW 1967), later argued against an explanation for segregation distortion on the basis of a functional pole.

A mechanism for distorted sex ratio: The investigations reported in this paper are not entirely consistent with the conclusions of HIRAIZUMI and NAKAZIMA (1967). When recovery ratios of males heterozygous for either an "original-" or "recombinant-*SD*" chromosome are examined, the sex ratio of offspring receiving the SD^+ paternal chromosome (the *cn bw* sex ratio) varies proportional to the value of k. Further, when *SD-72* males are brooded, both the *cn bw* sex ratio and k show a significant decrease in distortion with age, while the *SD* sex ratio shows no change. Finally, when the two *SD* lines are treated at 18°C, k values are changed, though in opposite directions. For *SD-72* parental males the decrease in k is accompanied by a statistically significant increase in *cn bw* sex ratio. The increase in k of treated *R(SD-36)-1* males is mirrored by a decrease in *cn bw* sex ratio (which is also an increase in distortion), although the effect is not statistically significant in this case. There is no detectable effect of cold shock on the *SD* sex ratio for either line.

These data show that the *SD* sex ratio is not affected by the action of segregation distortion, a result more consistent with the dysfunctional sperm model than with the functional pole model. The following mechanism, based on the dysfunctional sperm model, is proposed to account for both first and second chromosome distortion in *SD* males: (1) At the first meiotic division, the sex chromosomes segregate to the spindle poles at random with respect to the other chromosomes. (2) Due to the action of *segregation distorter* some or all of the sperm receiving the non-*SD* second chromosome do not function in fertilization. (3) The degree to which these presumptive SD^+-bearing sperm are rendered nonfunctional is affected by the sex chromosome carried by that gamete. This sex chromosome effect is stronger as the level of distortion increases. (4) Since segregation distortion does not usually affect the functionality of *SD*-bearing sperm, there is no sex chromosome effect on the recovery of such sperm.

The well established ability of sperm to function irrespective of their genome (MULLER and SETTLES 1927; MCCLOSKEY 1966) and the lack of influence of the sex chromosome on recovery in the *SD* progeny class make it apparent that the effect of the sex chromosomes on the recovery of SD^+ sperm is due to a modification of the action of segregation distortion. A stronger effect of the sex chromosomes with increasing distortion supports this conclusion. The mechanism involved is left an open question, however, and the model suggested does not distinguish between the possibilities that the X chromosome protects the presumptive SD^+ sperm more effectively than the Y or that the X chromosome somehow facilitates the recovery of such sperm from the action of distortion.

The relationship of sex ratio and k: HIRAIZUMI and NAKAZIMA (1967) concluded that *segregation distorter* causes first as well as second chromosome distortion. When sex ratios of progeny receiving the *SD-* or SD^+-bearing paternal chromosomes were compared to the standard sex ratio of the *cn bw* stock, the *SD* sex ratio was slightly higher than the standard and the SD^+ sex ratio was often

much lower. They utilized, as a measure of the overall degree of first chromosome distortion, the parameter Δ_2, which is the *SD* sex ratio minus the SD^+ sex ratio. They concluded that k is positively correlated with Δ_2 both within and between lines.

The present study confirms the interdependence of *cn bw* sex ratio distortion and second chromosome distortion within lines, but the *SD* sex ratio is independent of k. Thus the correlation of Δ_2 with k noted by HIRAIZUMI and NAKAZIMA appears due to the subtraction of *cn bw* sex ratio, a quantity inversely proportional with k, from *SD* sex ratio, which is constant with respect to k.

If the *SD* sex ratio is not affected by distortion, why does it differ from the standard value? The assumption that the expected sex ratio is that of the *cn bw* stock providing the genetic background on which *SD* chromosomes are tested may not be valid, for it does not take into account the possibility of F_1 zygotic selection based on genetic differences within the tested *SD* chromosome.

HIRAIZUMI and NAKAZIMA (1967) concluded that k is also positively correlated with Δ_2 when different lines are compared. For the lines studied here the *SD* sex ratios are virtually the same (Table 1). However, since the *cn bw* sex ratios are quite different for the two lines, Δ_2 does decrease with a decrease in k: $\Delta_2 = .524 - .190 = .334$ for *SD-72* males; $\Delta_2 = .527 - .399 = .128$ for *R(SD-36)-1* males.

The regression coefficients expressing the relationship between *cn bw* sex ratio and k for the two lines studied (Table 2) are significantly different ($P < .01$): *cn bw* sex ratio varies more strongly with respect to k in the offspring of *SD-72* than of *R(SD-36)-1* males. This discrepancy could be due to inadequacies in the use of linear regressions. The majority of *SD-72* males give k values above 0.95, while most of the *R(SD-36)-1* males tested give k values below this level. It seems possible that a higher order equation might be constructed to account for both the rates of change of *cn bw* sex ratio with k in terms of different portions of the same curve. A single curve does not appear valid, however. If one compares the mean *cn bw* sex ratios for those males of each genotype in the region $k < 0.95$, the *cn bw* sex ratio from *R(SD-36)-1* males is still higher ($P < .01$) than that of *SD-72* males (.363 and .170, respectively).

Thus it can be concluded that the rate of change of *cn bw* sex ratio with respect to k is determined by the genotype of the males tested. It is attractive to suggest that this difference is due to the presence or absence of *St(SD)* in the two chromosomes tested, but any critical argument requires the testing of additional lines.

HIRAIZUMI and NAKAZIMA (1967) characterized each *SD* line studied by recovery ratios calculated from pooled data, summed over all males, of the four progeny classes (*SD* and *cn bw* males and females). In the present study recovery ratios were calculated for each male, and these values were equally weighted to derive means characterizing the line as a whole. There are disadvantages to each of these methods of measuring relative recovery of paternal chromosomes. When mean recovery ratios are calculated from equally weighted values for each male tested, the small number of *cn bw* offspring often yielded by a single male introduces a large sampling error, especially for the *cn bw* sex ratio. On the other hand, when ratios are calculated from results summed over all males, the few

males with relatively large numbers of *cn bw* offspring contribute disproportionately to the overall recovery ratios. Since the *cn bw* sex ratio changes with k, and a lower k reflects a larger number of *cn bw* offspring, the method used in this study seems to depict recovery from these *SD* males in the most meaningful way.

The focus of treatment effects: The data presented in Table 2 indicate that within untreated lines the degrees of first and second chromosome distortion vary proportionally. Cause and effect relationships cannot be distinguished from these data. However, it is possible to ask whether the temperature or aging effects on segregation distortion affect k and *cn bw* sex ratio differentially. To consider this question it will arbitrarily be assumed that for males studied the k value determines the *cn bw* sex ratio in accordance with the linear regressions relating these two parameters.

For both lines in which a temperature effect on distortion was studied, the k value is significantly changed by the treatment. For the mean k from treated males of each line the *cn bw* sex ratio expected was calculated using the regression coefficients in Table 2. On this basis treated *SD-72* males are expected to yield a *cn bw* sex ratio of .330, which is very close to the observed value of .333. For *R(SD-36)-1* males the expected value, .373, and the observed value, .364, also show good agreement.

An explanation for the statistically significant effect of cold shock on the *cn bw* sex ratio in the case of the *SD-72* chromosome and not the *R(SD-36)-1* chromosome may now be provided. The higher rate of change of *cn bw* sex ratio with respect to k in the former case yields a significantly changed sex ratio, while the observed value of the *R(SD-36)-1 cn bw* sex ratio is consistent with that expected, but not statistically different from the control, due to a lower rate of change between these two parameters.

It is also interesting to consider the question of a differential effect of age on k and *cn bw* sex ratio of brooded *SD-72* males. The pertinent relationship is that between the rates of change of k and *cn bw* sex ratio with respect to age: b_k and $b_{cn\ bw}$. When the absolute values of b_k and $b_{cn\ bw}$ are compared, they are not the same ($P < .01$; $t = 3.05$ with $df = 12$). However, since *cn bw* sex ratio varies only proportionally with k, these two slopes would not be expected to be equal. On the assumption that k determines the *cn bw* sex ratio, $b_{cn\ bw}$ should be adjusted by dividing it by 1.782, the regression coefficient relating these parameters. The resulting adjusted value is -0.0241. Further, this adjusted $b_{cn\ bw}$ should be compared to an adjusted b_k, equal to -0.0140, calculated only from those males yielding *cn bw* offspring. When such a comparison is made, the adjusted values of b_k and $b_{cn\ bw}$ do not differ significantly ($P < 0.3$, $t = 0.95$ with $df = 12$). For this test of significance, the additional variance involved in the value of 1.782 is not taken into account. This simplification appears valid since no significant difference is noted in its absence.

The data from temperature and aging experiments suggest that there is no differential effect of these treatments on k and sex ratio. It seems likely that these treatments affect the action of the *SD* locus itself, and that k and *cn bw* sex ratio are in turn determined by the degree of *SD* action.

The temperature effect on segregation distortion: ERICKSON (1965) described a temperature effect on the *SD* system. This approach was extended by MANGE (1968), who noted an effect on recovery from various *SD* lines of both high (30°C) and low (19°C) temperature treatments. She utilized *SD-72* as well as "original-*SD*" chromosomes and recombinant derivatives from a wild population collected in Baja California. Heat shock resulted in a lowering of *k* in the Baja recombinant lines, but had no effect on *SD-72*. Treatment at 19°C caused a decrease in distortion, of varying magnitude, in all *SD* lines examined. From brooding experiments she concluded that meiosis is the time of *SD* temperature sensitivity. Her results vary, then, from the work presented above in which cold shock causes a decrease in second chromosome distortion for *SD-72*, but an increase for *R(SD-36)-1*.

Several explanations for the difference in behavior for the recombinant chromosomes in the two experiments appear plausible. The first is that different "original-*SD*" chromosomes may show different behavior with respect to cold treatment, with loss of *SD* modifiers in the resultant recombinant chromosomes affecting only the magnitude of this effect. However, it is also possible that the elements of the segregation distortion system other than *SD* itself may determine the direction as well as the magnitude of a temperature effect on *k*. The elements of the *SD* chromosomes used by MANGE (1968) were not characterized, and the degree of genetic change in forming the recombinant chromosomes cannot be ascertained. In the case of *R(SD-36)-1*, the loss of *St(SD)* with respect to its *SD-36* predecessor might account for the increase rather than decrease in distortion, as compared to *SD-72* which still carries the stabilizer.

The relationship between temperature sensitivity and the elements of the *SD* system cannot be determined at this time. However, the control mechanisms involved should be elucidated by an experimental analysis utilizing temperature treatment of "original-*SD*" chromosomes of several origins and the recombinant chromosomes derived from them.

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SUMMARY

The offspring of a male heterozygous for a second chromosome bearing *segregation distorter* are predominantly *SD*. From such SD/SD^+ fathers, SD^+ offspring are largely daughters, while there is some tendency for *SD* offspring to be sons.—The present study examines the relationship between *k* (the frequency with which a heterozygous male transmits *SD* to his progeny) and sex ratio in the progeny of *SD-72/cn bw* and of *R(SD-36)-1/cn bw* males. The sex ratio (proportion of males) of progeny receiving the SD^+ paternal chromosome (designated the *cn bw* sex ratio) varies proportionally with the value of *k* among males of each line, while the sex ratio of progeny receiving the *SD* paternal chromosome (designated the *SD* sex ratio) is independent of *k*. This result is supported by an aging experiment with the *SD-72* line and by temperature shock experi-

ments with both lines: a treatment effect on k is accompanied in each case by an effect on the *cn bw* sex ratio, while the *SD* sex ratio is apparently not affected by treatment.—These results indicate that only the sex ratio of offspring receiving the SD^+ paternal second chromosome is affected by the action of *segregation distorter*. The data do not support the conclusions of HIRAIZUMI and NAKAZIMA (1967), who postulated nonrandom meiotic I segregation such that the *X* chromosome and *SD* tend to segregate to opposite poles. The following mechanism, based on the dysfunctional sperm model (HARTL, HIRAIZUMI, and CROW 1967), is proposed to account for the distorted recovery of both first and second chromosomes from *SD* males: (1) At the first meiotic division, the sex chromosomes segregate to the spindle poles at random with respect to the other chromosomes. (2) Due to the action of *segregation distorter*, some or all of the sperm receiving the SD^+ second chromosome do not function in fertilization. (3) The degree to which these presumptive SD^+-bearing sperm are rendered nonfunctional is affected by the sex chromosome carried by that gamete. This sex chromosome effect is stronger as second chromosome distortion increases. (4) The sex chromosome constitution has no differential effect on the functionality of an *SD*-bearing sperm.

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