SEGREGATION DISTORTION AND CROSSING OVER IN MALES OF *DROSOPHILA ANANASSAE*. I: PRELIMINARY GENETIC ANALYSIS

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SANDLER and NOVITSKI (1957) proposed the term "meiotic drive" (or segregation distortion) to describe a condition which leads to unequal segregation of the two alleles present in a heterozygote in consequence of certain aberrant mechanisms during meiosis. SANDLER, HIRAIZUMI and SANDLER (1959) and SANDLER and HIRAIZUMI (1959, 1960) have made a detailed analysis of this segregation distortion phenomenon in *Drosophila melanogaster* and they have shown that it may be attributable to one or more mutant gene (*SD* factor) located in the centromeric heterochromatin of the second chromosome. The principal properties of these factors, among others, are: (a) When a heterozygous SD/SD^+ male is crossed to any homozygous non-*SD* female, almost all progeny of this cross are SD/SD^+ and very few are SD^+/SD^+. (b) Heterozygous SD/SD^+ females, however, yield progeny in the expected 1:1 proportion. (c) Homozygous *SD/SD* males and females also produce no aberrant ratio of either chromosome among their progeny, although *SD/SD* males are most often sterile, an effect attributable to the mutual action of *SD* chromosomes (see HARTL 1969).

Several other cases of segregation distortion have been reported in Drosophila after the classic work of SANDLER, HIRAIZUMI and SANDLER (1959), in natural populations (e.g., HIRAIZUMI, SANDLER and CROW 1960; MANGE 1961) as well as in irradiated laboratory populations (HANKS 1965). The possible importance of this phenomenon as a evolutionary force has been investigated by SANDLER and NOVITSKI (1957) and by HIRAIZMI, SANDLER and CROW (1960).

Three hypotheses have been proposed to explain the underlying mechanism involved in the resulting distortion in segregation. The earliest of these, advocated by SANDLER and co-workers (1959), proposes an *SD*-induced break in the non-*SD* chromosome which then fails to survive. The second postulates a differential behavior of the anaphase poles for the differential yield of the *SD*- and non-*SD*-bearing chromosome complement (NOVITSKI and SANDLER 1957; PEACOCK and ERICKSON 1965). The third hypothesis assumes dysfunction of the non-*SD*-bearing sperms (HARTL, HIRAIZUMI and CROW 1967; HARTL 1969). Although ZIMMERING and FOWLER (1968) and ZIMMERING *et al.* (1970) have emphasized the role of the genotype of the parent females in causing aberrant segregation ratios, recent work by HARTL and co-workers (1967, 1969), has provided strong evidence favoring the sperm dysfunction concept. Yet another mechanism (not related to *SD* action) which may result in aberrant segregation ratios has been

postulated by NOVITSKI (1951, 1967). This requires the formation of an asymmetric dyad and is, at least theoretically, operative only in the female.

An interesting case of aberrant segregation has been observed in a laboratory strain of *Drosophila ananassae*. While it is not the purpose of this report to discriminate among the above postulated mechanisms for the observed distortion, some evidence from preliminary genetic analysis will be presented here to show that it is actually a case of segregation distortion and not the consequence of trivial causes like differential viability, zygotic mortality, or penetrance. Interestingly, in all studies on segregation distortion in Drosophila reported so far, with the exception of the cytoplasmic case studied by MINAMORI (1970), the phenomenon is associated with that sex in which crossing over is absent (SANDLER and HIRAIZUMI 1959; HANKS 1964; STALKER 1958, 1961; KATAOKA 1967). Also, segregation distortion is generally ineffective in the presence of inversions in its homologue (SANDLER, HIRAIZUMI and SANDLER 1959). Two outstanding features distinguish the present case from the others, viz., the distortion is associated with recombinants only and it is observed in both sexes.

MATERIALS AND METHODS

Three mutant strains, *px*, *pc*, and *px pc* (described below) and three wild-type strains (a 66+, a 77+, and a 99+) of *Drosophila ananassae* formed the materials for the present study. All these strains, except a 99+, have been maintained in this laboratory for more than 100 generations. Only strain a 99+ has been recently synthesized and made homozygous for the same salivary gland chromosome band sequence as *px pc*. Stock a 66+ was collected as a wild stock (originally strain a 6+) in 1954 from a suburban area of Calcutta, and strain a 77+ was collected from Behala, Calcutta, in 1965.

The mutant plexus (*px*) causes formation of a network of venation on wings between the 2nd and 3rd longitudinal veins. In peacock (*pc*), the wings are curved sharply, usually at a 90° angle, but the angle can vary between 30° and 90° to the dorsal surface of the body. Both *px* and *pc* are in the 3rd linkage group with a map distance of 22.55 units between them (RAY-CHAUDHURI *et al.* 1962; MUKHERJEE 1957, unpublished). The linkage group and map distance were determined from original 3- and 4-point recessive stocks in which no distortion from normal disjunction was observed except in some cultures which were scored, but not considered for determination of map distance since at that time no explanation could be given for those exceptional cases. Both *px* and *pc* also showed independent segregation from the markers in linkage groups 1, 2, and 4 (MUKHERJEE 1957, unpublished).

Penetrance of the mutants px *and* pc: The penetrance of *px* and *pc* has been checked by examining the number of nonmutant flies emerging in the culture as well as by testing the transmission of the mutant phenotypes from the recombinant and nonrecombinant *px*, *pc*, and *px pc* flies obtained from testcrosses. In none of the cultures of the three mutant strains was a fly obtained in which *both* its wings were of nonmutant phenotype. In all except two groups of such progeny tests, *px* and *pc* were transmitted unequivocally in expected proportions. In these two groups *px pc* nonrecombinant or recombinant males and females were crossed to *px pc* flies. Some unexpected *px* flies were obtained from these crosses: in 19 out of 5134 in one case and in 22 out of 8350 in another case, *pc* was not expressed. 41 females in the former case and 75 *px pc* males in the latter case were tested by pair mating. It is therefore clear that the penetrance of both mutants is nearly 100%.

Variable expressivity and method of scoring the mutant phenotypes: Both mutants *px* and *pc* show considerable variability in expression. In homozygous *px/px* three different categories of wing venation are generally observed; viz., extreme network of venation on the region between the 2nd and 3rd longitudinal wing veins, a delta-like pattern of venation in that region, or just a

streak of vein. In heterozygous *px*/+ none of these patterns are observed. In about 15 to 17% heterozygous progeny of a cross *px*/*px* × +/+, however, a dot-like structure is found at the same site where the plexus-like effect is seen in a *px*/*px* fly. Such flies having the dot on *both* wings, whenever they appeared in testcross progeny, were considered heterozygotes and were not included among *px*/*px* or *px pc*/*px pc*.

With regard to *pc*, at least one wing shows a sharp bend at an angle greater than 60°; flies with both wings more or less normal are extremely rare. Even when one or both wings are only slightly bent, a fold or crease across the wing in the region of the junction of the alula with the main body of the wing (toward the anterior cross-vein) sharply distinguishes the peacock (*pc*) character from normal. The heterozygous *pc*/+ flies are clearly normal, indicating the complete recessiveness of *pc* to *pc*+. All testcross data were taken from pair matings. Flies were examined from the 12th day and scored until no more flies emerged for two consecutive days. The Drosophila stocks were raised on medium containing agar, cornmeal, brown sugar, yeast, and Nipagin. All stocks were reared throughout the developmental stages in an air-conditioned room maintained at 24° ± 1°C.

RESULTS

Preliminary analysis of the testcross data with *px pc*/++ males yielded a high degree of inequality among the reciprocally recombinant classes in pair matings as well as mass cultures. The testcross progeny from pair matings further showed a great range of individual variation (MUKHERJEE 1961). These facts were at first attributed to crossing over during early spermatogonial or apical cell stages according to the model of WHITTINGHILL (1955). It was realized later that, except in certain early lots of testcrosses and especially in those with unselected lines of the mutant strains, the inequality was due to the appearance among the recombinant progeny of only one and the same recombinant class. The data presented in Table 1 reveal that in the first batch of experiments (done 1958–1959), there was a very low number of *px* recombinant flies and also a low number of the nonrecombinant double recessives as compared to their reciprocal classes (Expt. I in Table 1). The simplest explanation for such inequality, on the basis of the data from this series of experiments may be that the mutant *px* is less viable, both in *px*/*px* and *px pc*/*px pc* condition.

The data presented in Expts. II and III in Table 1 are the results of testcrosses from heterozygotes for the distorting *px pc* and *px* chromosomes, using inbred laboratory strains of *px pc* and *px*. Unlike the first experiment, in these crosses the *pc* (or non-*px*, as in Expt. III)-containing recombinant class was either lacking or reduced (depending on the F_1 heterozygote's sex) in comparison with *px* type. Apart from the opposite effect in the early and later experiments, the proportions of the four classes of progeny in the three experiments considered together rule out the possibility that lack of normal viability of the mutant strain is the cause of their distorted ratios. For example, if the lower number of *pc* among recombinants in Expt. II were due to lower viability of *pc*, the number of *px pc* and *pc* in Expt. III should also have been proportionately lower in comparison with ++ and *px*, respectively. This is not actually realized. The data on viability provide further evidence against differential viability. It may be pointed out here that while the recombination frequencies in experiments with strains a 66+ and a 77+ are similar in both sexes, those with strain a 99+ in Expts. II and III are somewhat lower, especially in the male.

TABLE 1

Testcross progeny, recombination frequencies, and degree of distortion among the recombinants and nonrecombinants in px pc *of* Drosophila ananassae

Heterozygous parent		Number	Testcross progeny						$\bar{K}$*	
Genotype (strain)	Sex	of pair matings	++ *a*	*px pc* *b*	*px* *c*	*pc* *d*	Total number	Percent recombination	NR	R
Experiment I:										
+ +/*px pc*	♂	44	4831	2805	81	1262	8979	15.0	...	...
Experiment II:										
+ +/*px pc*	♀	45	1892	1694	1028	323	4937	27.1	0.48	0.76
(a 99+)									(0.01)	(0.02)
+ +/*px pc*	♀	13	460	448	345	75	1328	31.6	0.49	0.83
(a 66+)									(0.02)	(0.04)
+ +/*px pc*	♀	19	681	750	631	84	2146	33.3	0.52	0.88
(a 77+)									(0.02)	(0.01)
+ +/*px pc*	♂	29	1091	979	399	1	2470	16.2	0.46	0.99
(a 99+)									(0.02)	(0.002)
+ +/*px pc*	♂	20	709	746	405	4	1864	21.6	0.51	0.99
(a 66+)									(0.07)	(0.005)
+ +/*px pc*	♂	12	370	451	215	2	1038	21.1	0.55	0.99
(a 77+)									(0.04)	(0.004)
Experiment III:										
+ *pc*/*px* +	♀	21	219	355	859	740	2173	26.4	0.53	0.62
									(0.02)	(0.03)
+ *pc*/*px* +	♂	26	38	147	982	931	2098	8.8	0.53	0.80
									(0.01)	(0.06)

* $\bar{K}$ = mean segregation ratio, $b/(a+b)$ or $c/(c+d)$. NR = nonrecombinant. R = recombinant. Results of Experiment I are from high crossover lines, data from MUKHERJEE (unpublished). High yield in this set is because data were pooled from several broods. Values given in parentheses are ± standard errors of mean K values.

It is evident from the data in Table 1 that in all crosses and in both sets (Expts. II & III) there is a disproportionately higher number of one and the same particular class despite the fact that the two reciprocally noncrossover classes are more or less similar in size: In the crosses of Expt. II, the *px* crossover class exceeds the *pc* class; and in the crosses of Expt. III, the *px pc* recombinant class is greater than ++. The K values are the proportions of the majority class of recombinants or nonrecombinants among the total number of recombinants or nonrecombinants, respectively. This definition is modified after SANDLER, HIRAIZUMI and SANDLER (1959). Mean K values in the last two columns of Table 1 clearly show that while recovery of the reciprocally nonrecombinant classes is almost normal, recovery of reciprocal recombinants is far from the expected 1:1. The ratio is far more aberrant in the case of heterozygous males than in the case of heterozygous females. A detailed analysis of the K values for individual heterozygous males further reveals that the K values in the females vary considerably but those in the males are highly homogeneous, consistent, and unambiguous (Figures 1 to 4). This is true for both *px pc*/++ as well as *px* +/+ *pc* heterozygotes. Interestingly, the mean K values in the testcross progeny using the latter

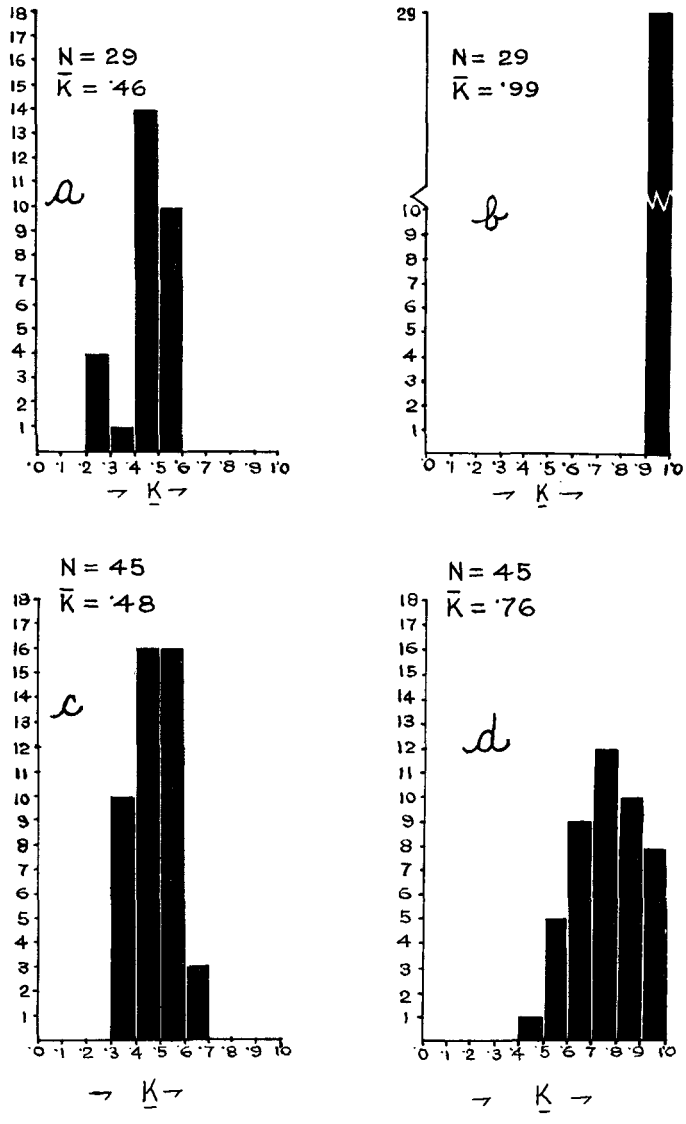

FIGURE 1.—Histograms of the K values for nonrecombinant and recombinant flies obtained in the testcrosses using $++/px\ pc$ heterozygotes with strain a 99+. a and b: Values for nonrecombinants and recombinants, respectively, in the progeny of the heterozygote males. c and d: Respective values in the progeny of the heterozygote females.

group of parents as well as the recombination frequencies in this set are lower than K values and recombination frequencies in the experiments using $px\ pc/++$ parents. Whether this is simply coincidence or there is a relation between K and amount of recombination is not clear from the present data.

Viability and zygotic mortality tests: The possibility that a subvital effect of the mutant *pc* causes its absence in the testcross progeny can be ruled out by

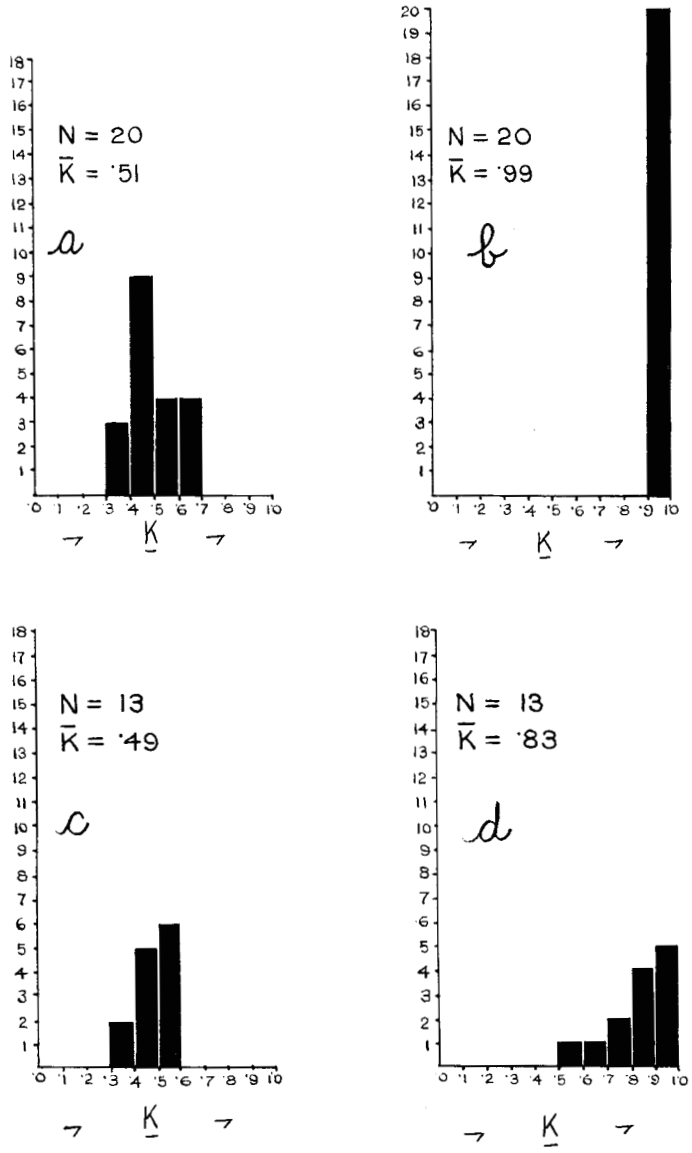

FIGURE 2.—Histograms of the K values for nonrecombinant and recombinant flies obtained in testcrosses using ++/*px pc* heterozygotes with strain a 66+. a, b, c, and d same as in Figure 1.

comparing the egg-to-adult ratios of the mutants with those of wild type grown in the same environment. For this purpose, 20 eggs each of *px*, *pc*, and *px pc* were separately placed with 20 eggs of the wild-type strain (a 99+) in a vial, and the numbers of each type of eclosed adult were scored. The data in Table 2 clearly show that with respect to viability, *pc* as well as *px pc* have the same relationship with + as *px* has with +.

A measurement of the relative zygotic mortality of the testcross progeny has

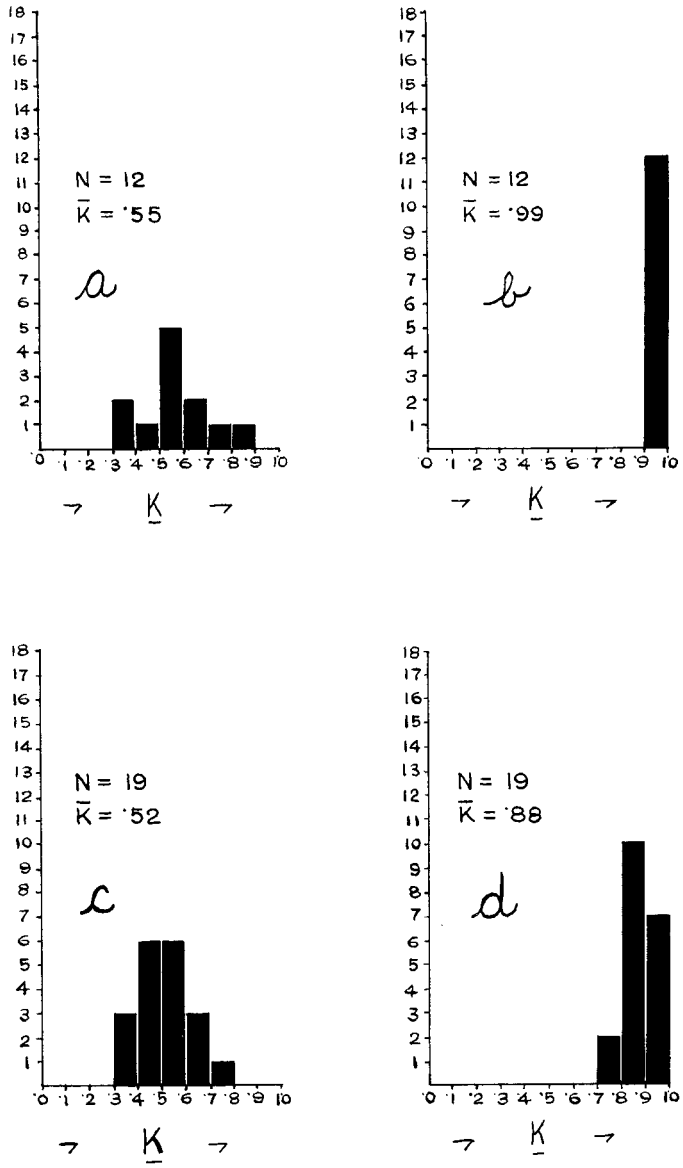

FIGURE 3.—Histograms of the K values for nonrecombinant and recombinant flies obtained in the testcrosses using $++/px\ pc$ heterozygotes with strain a *77*+. a, b, c, and d same as in Figure 1.

been carried out. For this purpose, heterozygous $px\ pc/++$ males or females were crossed to $px\ pc$ homozygotes in pairs for a short period of time and the flies were transferred to several consecutive sets of cultures for at least four days. Pair matings were made in 5 × 2.5 cm containers. All eggs were carefully counted under a dissecting binocular microscope with a total magnification of about 40×.

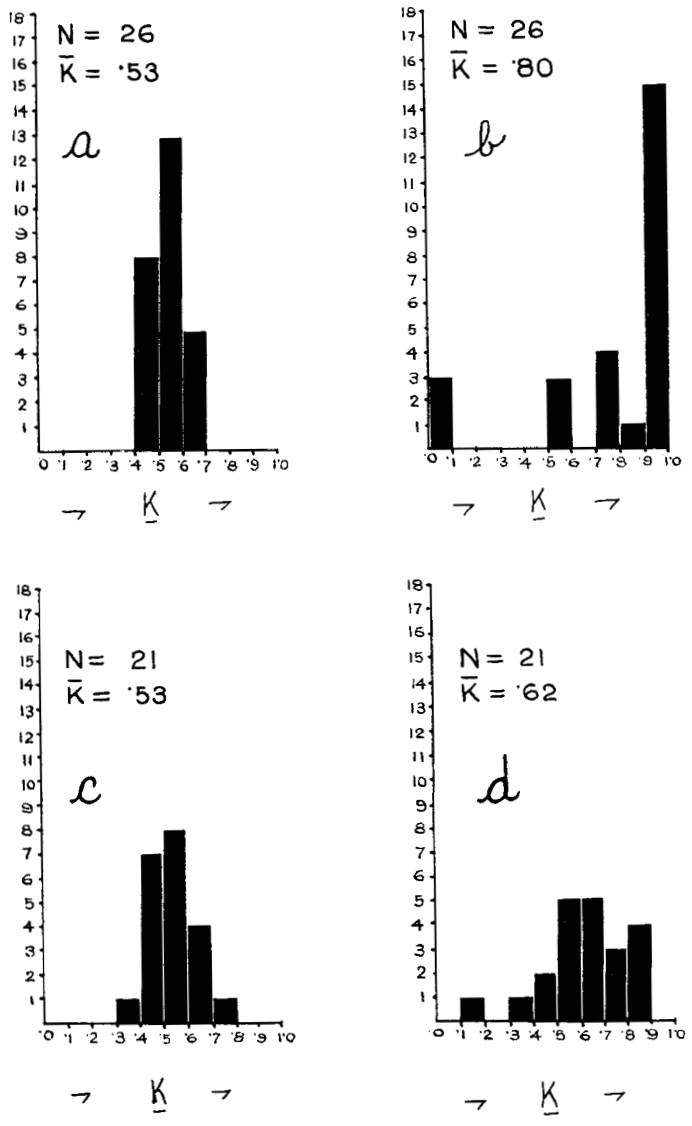

FIGURE 4.—Histograms of the K values for nonrecombinant and recombinant flies obtained in the testcrosses using + *pc*/*px* + heterozygotes; a, b, c, and d same as in Figure 1.

The eggs were allowed to develop, and the adults eclosing from these eggs were classified and counted. The data from this test are in Table 3. It is evident that about 95 to 97% of the eggs hatch and develop to adulthood. Independent tests carried out similarly with *px pc*/++ (♂ or ♀) × ++ show that in this case also about 95% of the eggs develop into adults. Table 3 reveals further that the contribution of the different types of progeny from the reciprocal crosses to the total number of adults is also of the same order as that in Table 1 (cf. data on

TABLE 2

Relative proportions of adults of the wild-type strain (a 99+) and px, pc, *and* px pc *eclosed from a given number of fertilized eggs (N = 20) in different pair-culture experiments and their homogeneity*

Exptl. culture sets	Number of progeny hatched		Mean ratio	Total		Summed data		Homogeneity	
	Wild	Mutant		χ^2	df	χ^2	df	χ^2	P
Set I									
$++$ *vs.* $+$ *px*	198	205	0.97	0.82	12	0.11	1	0.72	0.4
Set II									
$++$ *vs.* $+$ *pc*	167	169	0.99	0.58	10	0.01	1	0.57	0.5
Set III									
$++$ *vs. px pc*	181	181	1.00	0.35	11	0.00	1	0.35	0.7

strain a 99+). Therefore, these data are sufficient to rule out the possibility that the loss of *pc* in the progeny of these crosses was due to an effect occurring after fertilization.

DISCUSSION

A case of aberrant segregation in *Drosophila ananassae* has been presented. It resembles segregation distortion in *Drosophila melanogaster* in more than one way. Like *SD* in *D. melanogaster*, there is distortion of only one particular chromosome (viz., *px* or *px pc*) against its homologue (viz., *pc* or ++), and recovery is more distorted from males than from females. However, several important points distinguish the present case from *SD*. The unusual but interesting aspects of this aberrant segregation are that (1) it is associated with a laboratory mutant strain; (2) it occurs in both sexes; and finally, (3) it affects only the recovery of the recombinants without any influence on the nonrecombinants. Furthermore,

TABLE 3

Zygotic mortality test : egg-to-adult ratio and corresponding segregation ratio in px pc/++ *heterozygotes*

Cross*	Total eggs in all cultures	Total flies emerged	Number of cultures	Mean egg-to-adult ratio	Progeny classes				$\bar{K}_1$† ±SE	$\bar{K}_2$† ±SE
					++	*px pc*	*px*	*pc*		
A	3028	2890	25	1.04±0.005	1105	1051	539	195	0.48 (±.005)	0.74 (±.015)
B	2386	2322	18	1.03±0.006	956	934	431	1	0.49 (±.007)	1.00 (±0.00)
C	1456	1381	10	1.05±0.005	1381	....	...	...	...	...
D	1359	1275	10	1.06±0.001	1275	....	...	...	...	...

* A: ♀ *px pc*/++ × *px pc* ♂
B: ♀ *px pc* × *px pc*/++ ♂
C: ♀ *px pc*/++ × ++ ♂
D: ♀ ++ × *px pc*/++ ♂

† $\bar{K}_1$ and $\bar{K}_2$ are mean segregation ratios for nonrecombinants and recombinants, respectively.

although the proportion of the more frequent of the two reciprocally recombinant classes in males is invariably nearly 1.0, that proportion in females is highly variable (K varying from 0.65 to 0.9). Since viability, rate of development (tested independently, not presented here), and penetrance have been established as normal upon careful analysis, the reality of such aberrant segregation must be taken seriously. The phenomenon is especially interesting, because it appears to be confined to recombinants and because it has been discovered in a species which shows a high frequency of crossing over in males (MORIWAKI 1937; RAY-CHAUDHURI *et al.* 1962; MUKHERJEE 1961; KALE 1969; HINTON 1970).

All postulated explanations of *SD* entail the failure of SD^+ sperm to function in SD/SD^+ males. However, while NOVITSKI and SANDLER (1957) as well as PEACOCK and ERICKSON (1965) postulate preferential inclusion of SD^+ in half of the products of all meiotic divisions that normally fail to function, irrespective of whether the male carries *SD* or not, HARTL, HIRAIZUMI and CROW (1967) and HARTL (1969) have presented evidence which strongly suggests that the presence of *SD* in the primary spermatocyte causes the SD^+ sperm to fail to function, and that in the absence of *SD* all products of meiosis develop into functional sperm. Most of the workers on *SD* have now accepted the latter explanation, although ZIMMERING and FOWLER (1968) and ZIMMERING *et al.* (1970) have shown that the progeny-to-sperm ratios among the crosses involving SD/SD^+ males and different non-*SD* females deviate greatly from the expected 0.5, are in the range of 0.7–0.9, and depend upon the genotype of the female parent.

While such a mechanism may also be operative in the present case of segregation distortion in *D. ananassae*, the association of the distortion with only the recombinant class and its presence in both sexes leads us to contend that additional explanations must be sought in order to fulfil these criteria. Analysis in light of an alternative hypothesis of nonrandom disjunction, proposed by NOVITSKI (1951) and explained in more detail in NOVITSKI (1967), reveals that at least two facts encountered in the present case of aberrant segregation in *D. ananassae* fulfil the requirement for the operation of the phenomenon of nonrandom disjunction: first, the phenomenon is associated with genetic exchange; and secondly, it is operative also in the female. However, nonrandom disjunction fails to explain the presence of aberrant segregation in the male. Furthermore, it requires both a distinct difference in size of the two homologues (whose segregation is to be distorted) and formation of asymmetrical dyads following exchange. Cytological examination of the polytene chromosomes and mitotic configurations (from larval neural ganglion) of homozygous ++/++, *px pc/px pc*, and heterozygous *px pc*/++ as well as of their different classes of progeny, fails to reveal any size difference between the homologues, although such examination does not preclude the possibility of a minor size difference. One may, however, suspect that an associated inversion could result in such asymmetric dyad formation. This presumption is also ruled out by the use of the wild-type stock a 99+ which does not show any inversion loop in that chromosome when made heterozygous with *px pc*; nevertheless, such heterozygotes exhibit a high degree of distortion in the segregation of recombinant classes.

It seems clear, therefore, that any one of these hypotheses alone fails to explain all the properties of the aberrant segregation in *D. ananassae*. Future work will emphasize this aspect. It may be added that the results reported here, although still preliminary, open up a new line of thought concerning the high frequency of spontaneous crossing over in males of *Drosophila ananassae*, in contrast to its complete absence in other species of the genus, and its relation to segregation distortion.

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SUMMARY

A case of aberrant segregation in a laboratory strain (*px pc*) of *Drosophila ananassae* has been presented. The distortion is observed in both sexes (i.e., in heterozygous males as well as females) but only in the segregation of recombinants and not in the segregation of nonrecombinants. That this disproportionate recovery of the two reciprocally recombinant classes should not be attributed to differential viability, zygotic mortality, or lack of penetrance of the single mutant has been demonstrated by careful experimental analysis. The phenomenon observed in *px pc*/++ heterozygotes has been compared with that in *SD*/*SD*$^+$ in *D. melanogaster*. Although certain features are common to both, the present case of segregation distortion is distinguished from *SD* effects by certain unique features, viz., its association only with recombinants and its occurrence in both sexes. The distortion is, however, more extreme and less variable in males than in females heterozygous for *px pc* or *px* (*K* values varying from 0.61 to 0.88 in females and from 0.80 to 1.00 in males). —Results of testcrosses with three wild-type strains of different origin show that the distortion is not due to any effect of the genotypic background of the wild-type strains which differ from each other mainly by the presence and absence of an inversion.

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