

Miklos, G. L. G. University of California, San Diego, California. SD distributions and the measurement of distortion.

In Segregation-Distorter (SD) experiments, K value distributions are often encountered which have seemingly unusual shapes, and a distribution can appear to be a composite of two different histograms.

It is sometimes found in such an experiment that many or most SD/SD⁺ males exhibit very high distortion, whilst others show greatly reduced distortion or none at all. The interpretation of the shape of SD histograms can be approached using the make analysis of Miklos and Smith-White (Genetics 67:305-317, 1971). This communication presents the results of an experiment in which an unusual SD distribution is interpreted as having a high variance on the make scale. It further gives other odd shapes which can be generated by the make method, and the following examples extend the spectrum of histogram shapes initiated by the two high variance examples of Miklos and Smith-White (1971). The problems associated with the use of K value as a measure of distortion will also be clear from the examples.

A. During a series of experiments designed to investigate the phenomenon of conditional distortion, (Sandler and Hiraizumi, Genetics 46:585-604, 1961) an SD-72 chromosome was passed through females, and the SD-72/cn bw sons of the SD-72/cn bw mothers were tested for distortion in the standard way. Instead of obtaining the characteristic high K value distribution, in which all males yield values near 1.00, it was found that approximately 25 per cent of the males exhibited markedly reduced distortion. These results are shown in histogram A, in which the 65 tested males yielded 9,509 progeny. This type of histogram is frequently encountered in the SD literature, and it is due to a high level of distortion, together with large between-male variability.

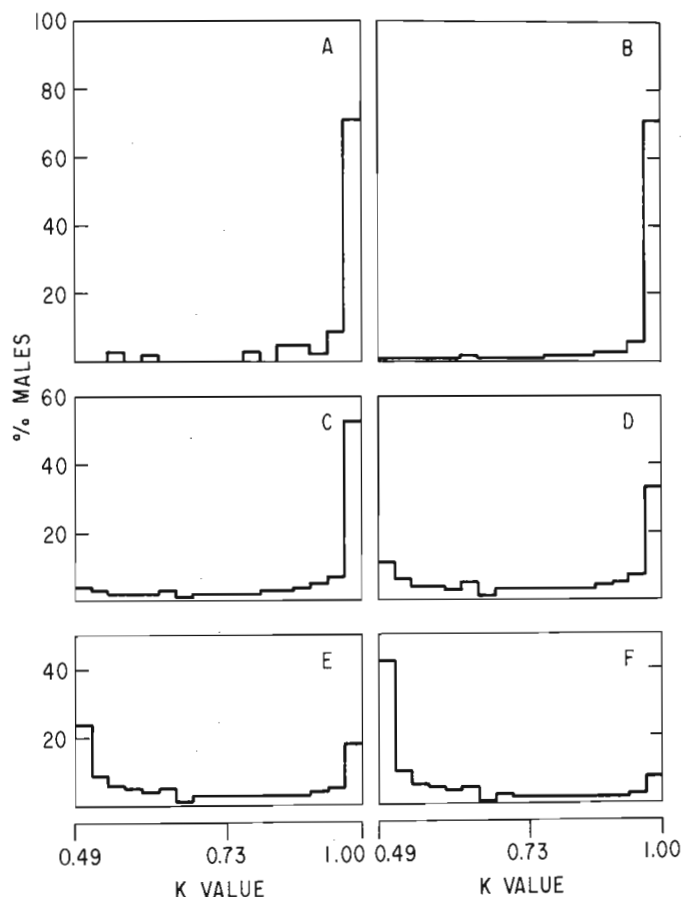
The theoretical histograms B,C,D,E and F have been derived from normal distributions

on the make scale. They all have the same high variance ($V_M = 4.0$), and differ only in their means; B,C,D,E and F have means of 8,7,6,5 and 4 Probits respectively, corresponding to K values of 0.999, 0.98, 0.86, 0.67 and 0.54. It can be seen that the experimental histogram A corresponds to a theoretical one such as B, which has a high mean and a large underlying variance. The level of distortion of the SD-72 stock has thus remained unaltered during the passage of the SD-72 chromosome through females, however the between-male variance has increased. If the level of distortion is lowered, but a high between-male variance is retained, then SD distributions such as B,C,D,E and F are obtained. Histograms such as these are relatively abundant in the SD literature, and most probably occur when some form of heterogeneity is introduced into the SD stock.

It should be pointed out that if an SD distribution is highly skewed, its mean and median can differ by a large amount, and the mean will provide an inappropriate measure of distortion because it is influenced by the skewness. The median is unaffected and should be utilized in such cases.

B. These results demonstrate the deceptive nature of K value distributions. Another property is that K is

not uniform over its range of measurement. The difference in distortion between K's of 0.95 and 0.90 is not the same as between 0.70 and 0.65 for example. It takes the same amount of "work" to go from K = 0.67 to K = 0.86 (ie, 5 Probits to 6 Probits), as it does to go from



$K = 0.98$ to $K = 0.999$ (7 Probits to 8 Probits). Thus changes in distortion in terms of K value can be misleading, and an unambiguous description of an SD experiment involves describing means, variances and changes in Probits.

The difficulties are increased further when tests of significance are involved. Owing to the nature of the K scale, two distributions may appear to be significantly different under standard statistical tests, but in reality, are not significantly different when compared in Probits. This is not due to a deficiency in the test itself, but rather to the unsuitable analytical properties of the K scale.

These results on the shape of SD histograms and the nonuniform changes in K over the range of distortion, further show that the phenomenon of Segregation-Distortion is best considered in terms of the make analysis.

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Sandler, L. University of Washington, Seattle, Washington. Induction of autosomal meiotic mutants by EMS in *D. melanogaster*.

A scheme for the detection and isolation of autosomal meiotic mutants (i.e. mutants on either chromosome 2 or 3 that affect disjunction of either the sex chromosomes or chromosome 4 in either

sex or X chromosome recombination in females) has been described by Sandler et al. (Genetics 60:525-558, 1968). They examined autosomes collected from natural populations.

This scheme has now been applied to mutagenized major autosomes. Chromosomes 2 and 3 were recovered from Canton-S males treated with EMS according to the method of Lewis and Bacher (DIS 43:193, 1968) using a treatment that produced about 10% (54/545) sex-linked recessive lethals after one additional backcross generation to resolve mosaics. The scheme of Sandler et al. for examining autosomes was also modified to resolve mosaics.

There were 35 lethal-free 2-3 complements examined for meiotic mutants -- 24 in both sexes, 8 in females only (the males were sterile) and 3 in males only (the females were sterile). Among these, two meiotic mutants were recovered: (1) mei-W5, a second chromosome recessive that causes the production, in homozygous males, of sperm lacking paternal chromosomes and has no obvious effects in females, and (2) mei-W22, a third chromosome recessive that eliminates recombination and increases nondisjunction in homozygous females and is sterile in males (for reasons not yet investigated).

From the cross, $\text{In}(1\text{LR})\text{sc}^{\text{VI}}$, $y \text{ pn } v \cdot y^+/y$; $\text{spa}^{\text{pol}}/\text{spa}^{\text{pol}}$ females homozygous for the indicated meiotic mutant by $Y^{\text{S}}X \cdot Y^{\text{L}}$, $\text{In}(1)\text{EN}$, $v \text{ f } B/O$; $C(4) \text{ RM}$, $ci \text{ ey } R/O$ males, there were observed:

mei-W5				mei-W22			
X chromosome	chromosome 4			X chromosome	chromosome 4		
	+	pol	ey		+	pol	ey
(v,v ⁺)B/+♀♀	97	0	0	(v,v ⁺)B/+♀♀	51	2	5
B ⁺ ♀♀	0	0	0	B ⁺ ♀♀	17	4	3
v f B ♂♂	0	0	0	v f B ♂♂	14	7	9
pn v ♂♂	26	0	0	pn v ♂♂	32	3	8
y ♂♂	20	0	0	y ♂♂	37	2	3
y pn ♂♂	6	0	0	y pn ♂♂	0	0	0
v ♂♂	9	0	0	v ♂♂	0	0	0
y pn v ♂♂	11	0	0	y pn v ♂♂	0	0	0
+ ♂♂	11	0	0	+ ♂♂	0	0	0
pn ♂♂	2	0	0	pn ♂♂	0	0	0
y v ♂♂	2	0	0	y v ♂♂	0	0	0

From the cross, $\text{In}(1)\text{FM6}$, $y^{\text{3ld}} \text{ sc}^8 \text{ dm } B/y^+Y$; $\text{spa}^{\text{pol}}/\text{spa}^{\text{pol}}$ males homozygous for mei-W5 by $y \text{ pn}/y \text{ pn}$; $C(4) \text{ RM}$, $ci \text{ ey } R/O$ females, there were observed: $y^2 B \text{ ♀♀} = 213$, $\text{pn } \text{ ♂♂} = 217$, $B \text{ ♀♀} = 0$, $y \text{ pn } \text{ ♂♂} = 9$, $y^2 B$; $\text{pol } \text{ ♀♀} = 0$, $y^2 B$; $ci \text{ ey } \text{ ♀♀} = 33$, pn ; $\text{pol } \text{ ♂♂} = 0$, pn ; $ci \text{ ey } \text{ ♂♂} = 28$, B ; $\text{pol } \text{ ♀♀} = 0$, B ; $ci \text{ ey } \text{ ♀♀} = 0$, $y \text{ pn}$; $\text{pol } \text{ ♂♂} = 0$, and $y \text{ pn}$; $ci \text{ ey } \text{ ♂♂} = 0$.