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

	mei-W5				mei-W22		
	chromosome 4				chromosome 4		
X chromosome	+	pol	ey	X chromosome	+	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, In(1)FM6, y^{31d} sc^{8} dm B/y^{+}Y; spa^{pol}/spa^{pol} males homozygous for mei-W5 by y pn/y pn; C(4) RM, ci ey ^{R}/O females, there were observed: y^{2} B ♀♀ = 213, pn ♂♂ = 217, B ♀♀ = 0, y pn ♂♂ = 9, y^{2} B; pol ♀♀ = 0, y^{2} B; ci ey ♀♀ = 33, pn; pol ♂♂ = 0, pn; ci ey ♂♂ = 28, B; pol ♀♀ = 0, B; ci, ey ♀♀ = 0, y pn; pol ♂♂ = 0, and y pn; ci ey ♂♂ = 0.