RELATIONS BETWEEN CHROMOSOME SIZE AND EFFECTS OF INVERSIONS ON CROSSING-OVER IN DROSOPHILA MELANOGASTER

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Schultz and Redfield[1,2] have shown that inversions in chromosomes I and II cause an increase in crossing-over in chromosome III and that inversions in chromosomes I and III have similar effect on crossing-over in chromosome II. Bridges[3] showed that inversions in chromosomes I, II and III when present simultaneously have no effect on crossing-over in chromosome IV. Steinberg[5] showed that inversions in chromosomes II and III cause an increase in crossing-over in chromosome I. It was found both by Schultz and Redfield and by Steinberg that different inversions caused different amounts of increase in crossing-over in a given chromosome.

Table 1, which is a combination of data of Schultz and Redfield (unpublished) for chromosomes II and III and Steinberg[5] for chromosome I, shows that a given inversion causes a different average increment in crossing-over per unit map length in different chromosomes. For example, the C1B inversion causes a thirty-eight per cent increase in crossing-over per unit map length in chromosome II while in chromosome III it causes an increase of forty-six per cent per unit map length (table 1). The question arises as to what causes this differential quantitative effect of inversions on crossing-over in non-homologous chromosomes. While no complete answer to this question can be given at the present time, reference to tables 2 and 3 will show a partial answer.

TABLE 1

SHOWING A SUMMARY OF THE INCREASES IN CROSSING-OVER CAUSED IN NON-HOMOLOGOUS CHROMOSOMES BY INVERSIONS (VALUES EXPRESSED AS DECIMAL FRACTIONS)

INVERSIONS	CHROMOSOME I	CHROMOSOME II	CHROMOSOME III	TOTALS
C1B	....	0.38	0.46	0.84
Curly	0.22	....	0.49	0.71
Payne	0.33	0.55	....	0.88

Table 2 shows a comparison of the ratios of the relative lengths of metaphase chromosomes[4] with the ratios of the interchromosomal effects of an inversion or pair of inversions on crossing-over in these chromosomes. Thus chromosome I is sixty-nine per cent as long as chromosome II and the effect of the Payne inversions on I is sixty per cent of the effect of the same inversions on II, likewise chromosome I is fifty-seven per cent as

long as III while the effect of the Curly inversions on the former is forty-five per cent of that on the latter; and finally chromosome II is eighty-three per cent of III and the effect of the C1B inversion on chromosome II is also eighty-three per cent of that on III. Within the limits of error which are probably large because:

(1) of the difficulties involved in measuring *Drosophila* chromosomes,

(2) the interchromosomal effects of the inversions were measured in three different experiments, and

(3) the first chromosome was not completely marked,

these pairs of ratios may be considered very similar.

TABLE 2

SHOWING THE RATIOS OF THE RELATIVE METAPHASE LENGTHS OF THE GONIAL CHROMOSOMES AND THE CORRESPONDING RATIOS OF THE INTERCHROMOSOMAL EFFECT OF INVERSIONS

CHROMOSOMES	I/II	I/III	II/III
Ratio of sizes	100/145 = 0.69	100/175 = 0.57	145/175 = 0.83
Ratio of effects	0.33/0.55 = 0.60	0.22/0.49 = 0.45	0.38/0.46 = 0.83

TABLE 3

SHOWING THE PERCENTAGE OF THE TOTAL CHROMATIN LENGTH CONTAINED IN EACH OF THE CHROMOSOMES AND THE CORRESPONDING PERCENTAGE OF THE TOTAL INTERCHROMOSOMAL EFFECT OF INVERSIONS SHOWN BY EACH CHROMOSOME

CHROMOSOMES	I	II	III	IV
Percentage of total chromatin	23.3	33.7	40.7	2.3
Percentage of total effect	22.6	38.3	39.1	0.0

Table 3 shows a comparison of the percentage length of a given chromosome (i.e., the length of the chromosome divided by the sum of the lengths of all four chromosomes and multiplied by 100) with the per cent of the total increase in crossing-over shown in this chromosome. For example, the increases in crossing-over in chromosome I are .22 + .33 = .55 (table 1), the total increase in crossing-over is .84 + .71 + .88 = 2.43 (table 1); the percentage of the total effect which is shown by chromosome I is $.55/2.43 \times 100 = 22.6$. These figures (table 3) show even more clearly than those in table 2 that the effect of an inversion on crossing-over in a non-homologous chromosome is directly proportional to the length of the chromosome affected. In addition table 3 shows that the proportion of the total effect which goes to a given chromosome is approximately equal to that proportion of the total length of all the chromosomes which the affected chromosome constitutes. Therefore, the effect of an inversion on crossing-over in a non-homologous chromosome is a function of both the inversion itself and of the length of the chromosome affected. This fact explains Bridges'[3] observation that inversions do not affect crossing-over in chromosome IV. Chromosome IV, according to the above, would be ex-

pected to show about two per cent of the total increase in crossing-over (table 3), i.e., crossing-over in chromosome IV should be increased by approximately five per cent. Such an increase in crossing-over in chromosome IV is so small as to be detectable only in extremely large test populations.

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[1] Morgan, T. H., Bridges, C. B., and Schultz, J., *Yearb. Carnegie Instn.*, **31**, 303 (1932).

[2] Morgan, T. H., Bridges, C. B., and Schultz, J., *Yearb. Carnegie Instn.*, **32**, 298 (1933).

[3] Morgan, T. H., Bridges, C. B., and Schultz, J., *Yearb. Carnegie Instn.*, **33**, 274 (1934).

[4] Morgan, T. H., Bridges, C. B., and Sturtevant, A. H., *Bibliog. Genet.*, **1** (1925).

[5] Steinberg, A. G., *Genetics*, **21**, 615 (1936).

LINKAGE STUDIES OF THE RAT (RATTUS NORVEGICUS). II[1]

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King[2] has recently described the occurrence and inheritance of waltzing in an inbred race of rats reared at the Wistar Institute. Concerning the mode of inheritance this statement was made: "Available evidence seems to indicate that waltzing is due to a single gene, but that the extent to which this gene expresses itself depends upon modifying factors which favor or inhibit its action." Selection of the most pronounced waltzers through a series of generations increased the incidence of waltzing. In the F_6 generation the percentage of waltzers produced by waltzing parents had risen to 93 per cent.

The waltzing mutation made its first appearance in albino individuals and though it has since been introduced into colored individuals, it consistently occurs in a higher percentage of the albino than of the colored individuals in litters containing both sorts. This indicates that it is linked with albinism and that its gene is borne in the same chromosome as the albino gene. The evidence for this conclusion is as follows:

Crosses were made between albino waltzers and colored non-waltzers of the varieties known as hairless, kinky and blue. All F_1 individuals were, as expected, colored non-waltzers. The F_2 population obtained from each