

chromosomes apparently result from chromosome unbalance. Two types of control experiments indicate that the female-sterile host is not responsible for this behavior: Wild type diploid ovaries grown in *fes* hosts give rise to fully viable eggs, and from many normal diploid females into which superfemale ovaries had been implanted the offspring obtained were entirely from the normal ovaries of the host, suggesting that, under these conditions, superfemale ovaries are functionally similar to those grown in *fes* hosts. Since there are no marked irregularities in somatic development of a superfemale individual that would indicate disturbances in mitotic cell division, we can conclude that the particular chromosome unbalance characteristic of such individuals has a specific effect on the meiotic mechanism. A simple interpretation follows the assumption that the difficulty lies in the mechanism normally responsible for crossing-over.

¹ Bridges, C. B., *Science*, **54**, 252-254 (1921).

² Morgan, L. V., *Genetics*, **10**, 148-178 (1925).

³ Ephrussi, B., and Beadle, G. W., *Amer. Nat.*, **70**, 218-225 (1936).

⁴ Ephrussi, B., and Beadle, G. W., *Bull. Biol. Fr. Belg.*, **69**, 492-502 (1935).

⁵ Beadle, G. W., and Ephrussi, B., *Proc. Nat. Acad. Sci.*, **21**, 642-646 (1935).

⁶ Beadle, G. W., *Genetics*, **20**, 179-191 (1935).

⁷ Beadle, G. W., *Amer. Nat.*, **71**, 277-279 (1937).

⁸ Dobzhansky, Th., *Bull. Bureau Genet. Acad. Sci. Leningrad*, **8**, 91-158 (1930).

⁹ Clancy, C. W., and Beadle, G. W., *Biol. Bull.*, **72**, 47-56 (1937).

¹⁰ Beadle, G. W., *Jour. Genet.*, **29**, 277-309 (1934).

¹¹ Warwick, B. L., *Tex. Agr. Exp. Sta. Bull.*, **463**, 1-28 (1932).

¹² Darlington, C. D., *Recent Advances in Cytology*. Ed. 2, Churchill, London (1937).

¹³ Gowen, J. W., *Jour. Exptl. Zool.* **65**, 83-106 (1933).

AN EFFECT OF THE Y-CHROMOSOME ON THE SEX-RATIO OF INTERRACIAL HYBRIDS OF *DROSOPHILA PSEUDOÖBSCURA*

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It was shown by Lancefield (1929) that *D. pseudoöbscura* *A-B* hybrid females, when back-crossed either to *A* or to *B* males, give sex-ratios among their offspring that may deviate widely (in either direction) from 1:1. My own experience confirms this result; I have, however, never found a significant excess of male offspring unless the father was descended from certain of the older mutant stocks. Back-crossing to males from wild stocks or to males whose ancestry could be traced wholly to recently collected wild stocks has never resulted in significantly more than 50 per cent male offspring.

It appears that a given combination of stocks usually gives approximately the same proportion of males in separate cultures, whether these are reared simultaneously or not. Some stocks appear to be heterogeneous, but the observations establish that the major causes of the variable sex ratio are genetic.

The analysis of the genetic basis involved is incomplete, but certain of its elements can now be stated. (1) The source of the *A* parent of the F_1 female tested is very influential, that of the *B* parent less so—possibly of no significance. (2) The source of the male to which the F_1 female is mated strongly influences the result; part but not all of this influence is due to the *Y*-chromosome carried by such a male.

Since the last-mentioned point involves a new type of effect of the *Y*-chromosome, it seems desirable to record it, even though the analysis is still unfinished.

Several strains of Race *B* from the mountains of California (Sequoia National Park, Lassen National Park, Dunsmuir) give, when crossed to *A-B* hybrids, many fewer males than do other strains (including some from the same localities). If certain types of F_1 females are used, these strains give from 0 to 5 per cent males. Certain Race *A* strains from Mexico give similar results.

In order to study the nature of such differences, reciprocal matings were made between "high" and "low" strains within a race, and the F_1 males were mated to *A-B* females. Table 1 shows the results of a few such experiments. These results show that the F_1 males differed, and were in every case more like the paternal than the maternal stock—a result that can only be interpreted as being dependent on the *Y*-chromosome. Corroborative evidence is available, extremely low ratios having been found to be characteristic of the males from complex experiments in which the *Y* came from Sequoia-8, another "low" strain of Race *B*. In Race *A*, experiments analogous to those here reported have indicated a *Y* difference between the Georgetown and LaGrande-4 (Washington) stocks; here, however, the difference between the parental strains is much less marked, and the experiments are correspondingly less conclusive.

On the other hand, such experiments involving the Seattle-4 and Shelter Cove-5 (California) strains have indicated that the *Y*'s of these strains do not differ in their effects, though the ratios given by the two stocks are clearly and consistently different. This result shows that there must be other (evidently autosomal) genes affecting the potentialities of the males used in these experiments.

Table 1 is itself in agreement with this conclusion, since in every case the F_1 males carrying the "low" *Y* give a higher proportion of sons than did the males of the pure "low" strain.

TABLE 1

MALES	F_1 ♀ FROM BARE (A) ♀ BY CINNABAR (B) ♂			F_1 ♀ FROM ORANGE (B) ♀ BY BARE (A) ♂		
	♀ ♀	♂ ♂	♂ ♂ 100 ♀ ♀	♀ ♀	♂ ♂	♂ ♂ 100 ♀ ♀
Race A						
Georgetown, Texas	245	134	55	120	86	72
Oaxaca-4, Mexico	426	38	9	338	102	30
F_1 , Oax.-4 × Texas	213	96	45	315	241	77
F_1 , Texas × Oax. 4	322	68	21	330	161	49
Race B						
Seattle-4	238	79	33	238	184	77
Sequoia-17	237	10	4	353	35	10
F_1 , Seq. 17 × Seat. 4	360	206	57	281	223	79
F_1 , Seat. 4 × Seq. 17	453	90	20	593	108	18

Two further points may be noted. First, the aberrant sex ratios are evidently due to the death of more males than females, and these males receive the paternal Y. The chromosome in question is therefore present in the individuals whose survival-rate determines the observed ratios. Second, all the extreme "low" Y's known belong to Dobzhansky's (1937) Type I, which is cytologically the largest Y known in the species. It is yet to be determined if all Type I Y's are alike, or if (as now seems more likely) some of them are of the "high" type.

Finally, it should be stated that F_1 cultures ($A \times B$ or $B \times A$) consistently give 1:1 sex ratios, as do matings within either race, in the absence of sex-linked lethals and of the sex-ratio gene (Sturtevant and Dobzhansky 1936), which were certainly not present in the A - B experiments here described.

Dobzhansky, T., *Genetics*, 22, 340-346 (1937).

Lancefield, D. E., *Zeits. ind. Abst. Vererb.*, 52, 287-317 (1929).

Sturtevant, A. H., and T. Dobzhansky, *Genetics*, 21, 473-490 (1936).

THE EFFECT OF X-RAYS ON CHROMOSOMES IN MITOSIS

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Studies have been made of the frequency of x-ray-induced chromosome abnormalities in anaphases in the mouse sarcoma C R 180, the rat carcinoma, Walker 256 and in the root tips of the seedlings of *Vicia faba*, *Pisum sativum*, *Allium cepa* and *Lycopersicum esculentum*. In all organisms the frequency of chromosome abnormalities (induced attachment and frag-