RNA-treated cell enzyme was 3.42 and 6.12 μg of glucose released, respectively, per milligram of cell homogenate protein over a period of 30 minutes (average of seven experiments).

The final question is whether the induced activity, 125 to 200 percent of the control, is due to glucose-6-phosphatase or to an acid phosphatase. The activity of glucose-6-phosphatase is optimum above *p*H 6 and near *p*H 6.8 (*5*). The activity of acid phosphatase is optimum close to *p*H 5.3 (*10*). Figure 3 shows the effect on enzyme activity of varying the *p*H. There are five points of special interest, namely: (i) The enzyme activity of calf liver is higher than that of mouse liver. (ii) The *p*H optimum of both calf and mouse liver enzyme is above 6, and maximal activity is obtained near *p*H 6.4 (iii) The activity of the control enzyme from the ascites tumor cell enzyme is optimum between *p*H 5.6 and 6. (iv) The activity of the RNA-treated enzyme from the tumor cell is optimum between *p*H 6 and 6.4. Thus RNA treatment has brought about a shift in the optimum *p*H of activity toward that of liver, with this enzymatic activity being attributable to glucose-6-phosphatase rather than to an acid phosphatase. (v) The enzyme activity of the treated cells is higher than that of the control cells. Thus, treatment of ascites cells with liver RNA leads to an increase in glucose-6-phosphatase activity.

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References and Notes

1. H. Amos, B. Askonas, R. Soeiro, *Nat. Cancer Inst. Monograph 13*, 155 (1964); E. P. Cohen and J. P. Parks, *Science* **144**, 1012 (1964); S. DeCarvalho, H. J. Rand, J. R. Uhrick, *Exp. Mol. Pathol.* **1**, 96 (1962); M. Fishman, *J. Exp. Med.* **114**, 837 (1961); H. Friedman, *Science* **146**, 934 (1964); J. A. Mannick and R. H. Egdahl, *ibid.* **137**, 976 (1962); D. Nathans, C. T. Notani, J. H. Schwartz, N. D. Zinder, *Proc. Nat. Acad. Sci. U.S.* **48**, 1424 (1962); M. C. Niu, *Symposium on Nucleic Acids and Biological Function* (Lombardo Institute, Academy of Science and Letters, Milan, Italy, 1964), p. 352; A. S. Weisberger, *Proc. Nat. Acad. Sci. U.S.* **48**, 68 (1962); E. Zimmermann and F. Turba, *Biochem. Z.* **339**, 469 (1964).
2. M. C. Niu, *Science* **131**, 1321 (1960); ———, C. C. Cordova, L. C. Niu, *Proc. Nat. Acad. Sci. U.S.* **47**, 1689 (1961); E. Zimmermann, E. Zoller, F. Turba, *Biochem. Z.* **339**, 53 (1963).
3. M. C. Niu, C. C. Cordova, L. C. Niu, C. L. Radbill, *Proc. Nat. Acad. Sci. U.S.* **48**, 1964 (1962).
4. V. S. Shapot, S. Ya. Davydova, G. A. Drozdova, *Federation Proc. Transl. Suppl.* **3**, T17 (1964).
5. B. T. Cori and C. F. Cori, *J. Biol. Chem.* **199**, 661 (1952).
6. J. Imsande and B. Ephrussi, *Science* **144**, 856 (1964).
7. M. C. Eaton, A. R. Scala, M. Jewell, *Cancer Res.* **19**, 945 (1959).
8. M. Rabinovitz, M. E. Olsen, D. M. Greenberg, *J. Biol. Chem.* **210**, 8379 (1954).
9. H. Borsook, E. M. Fischer, G. Keighley, *ibid.* **229**, 1059 (1957).
10. G. Schmidt, in *Methods in Enzymology*, S. P. Colowick and N. O. Kaplan, Eds. (Academic Press, New York, 1960), vol. 5, p. 523.
11. Supported by a grant from NSF (G-23644) and National Foundation #CRMS-213. I thank Drs. J. R. Fisher, E. L. Tatum and R. Wu for aid and discussions, and Y. H. Yuh for technical assistance.

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Cytological Basis of "Sex Ratio" in Drosophila pseudoobscura

Abstract. *Cytological investigations of both laboratory and wild "sex ratio" lines of* Drosophila pseudoobscura *reveal that, contrary to earlier reports, no extra replication of the X chromosome occurs in primary spermatocytes. Normal disjunction of the sex chromosomes at anaphase I leads to equal numbers of X-bearing and Y-bearing secondary spermatocytes. In the latter, the Y chromosome regularly shows a "degeneration" at second anaphase. The "sex ratio" effect can be explained in terms of regularly nonfunctional products of meiosis.*

In 1957, Novitski and I. Sandler (*1*) proposed that in *Drosophila melanogaster* not all products of spermatogenesis are functional. Their argument was based on the observation that from a translocation heterozygote having two pairs of unequal homologs, the probability of obtaining the various gametic types could be predicted with remarkable mathematical exactitude. Multiplication of the probabilities of recovery of any two of the individual chromosomes provided a close estimate of their joint recovery. This rule could not apply if all four products of meiosis were functional, since each homolog, being present in two of the four cells, would have a probability of recovery of 50 percent. Implicit in their proposal were two points: that the nonfunctioning of some fraction of the products was a regular phenomenon, and that this was predetermined by some geometrical property of the meiotic divisions; the latter point followed from the observation that when two homologs were present the shorter was recovered more frequently. Zimmering (*2*) repeated and extended the original observations, showing that the treatment of recovery of unequal homologs as an exercise in probability is a procedure that can be repeated for a large number of situations.

The concept of a regular class of nonfunctional spermatozoa was recently verified by Peacock and Erickson (*3*). In their analysis of the phenomenon of "segregation distortion" (*4*) these authors showed, by comparing the numbers of sperms stored by females with the numbers of progeny obtained, that only half of the sperms produced by *D. melanogaster* males are capable of fertilizing an egg. This system also permitted the conclusion that in each primary spermatocyte one pole of the first-division spindle ultimately yielded the two functional gametes, the other pole giving rise to two nonfunctional sperms. Segregation distortion was seen to result from a preferential inclusion of the segregation distorter-bearing chromosome into the "functional" pole. Peacock (*5*) subsequently showed that in instances in which there appears to be meiotic loss of chromosomes (*6, 7*) no real cytological loss occurs; instead, the "lost" chromosomes preferentially segregate and migrate to the nonfunctional pole at the first meiotic division.

These observations cast new light on meiotic processes and provided a unified explanation of a number of meiotic abnormalities in *D. melanogaster*. On these grounds reexamination of the "sex ratio" phenomenon found in the *obscura* group of *Drosophila* seemed warranted. Analyses of "sex ratio" have been carried out by Gershenson (*7*), Sturtevant and Dobzhansky (*8*), Wallace (*9*), and others. In brief, these studies led to the following conclusions: (i) males carrying a certain X chromosome produced only daughters because all sperm were X-bearing; and (ii) all the sperm were X-bearing because at the first meiotic division the X chromosome underwent an extra replication and, at the same time, the Y chromosome degenerated. According to this scheme all four meiotic products would thus receive an X chromatid.

In the study reported here, "sex ratio" males were obtained from two different sources (*10*). All the lines we used exhibited a typical "sex ratio" effect; for example, a male of line Borrego Springs-3 yielded progeny consisting of 292 females and no males.

Cytological examinations of spermatogenesis were made for both "sex ratio" males and their wild-type sibs. In the latter males the meiotic divisions were completely regular, but in "sex ratio" males there was a striking de-

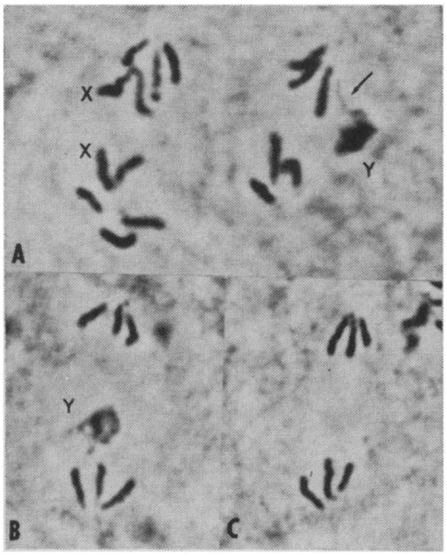

Fig. 1. (*A*) Two anaphase-II divisions, possibly in sister cells. The X-bearing cell (left) shows normal anaphase separation of the V-shaped X chromosome and of the three rod-shaped autosomes; the dot-like autosome is also visible at both poles. In the Y-bearing cell (right) the autosomes have undergone normal anaphase disjunction, but the Y chromosome exhibits the "degenerate" condition characteristic of "sex ratio." Evidence of some centromeric activity of the Y chromosome in this cell is indicated by the arrow. (*B*) A Y-bearing cell in anaphase II showing normal anaphase behavior of the autosomes and the "degeneration" of the Y chromosome. (*C*) A cell in anaphase II; no sex chromosomes are present. The material at upper right is a portion of the chromosome complement of another cell.

parture from normality. The course of meiosis is usually as follows: the X and Y chromosomes pair and separate normally in the first division, but in the second division the Y chromosome loses its characteristic appearance and appears as a chromatin mass which usually shows no centromeric activity at anaphase (Fig. 1, *A* and *B*). In some individuals this "degeneration" of the Y occurs early in metaphase II, while in others it is not seen until the autosomes have initiated their anaphase movement. Occasionally the aberrant behavior of the Y is seen as early as anaphase I. In accord with this observation, some second-division cells lack a sex chromosome (Fig. 1*C*) and their sister cells contain both a normal X chromosome and a Y chromatin mass. These cells would result from inclusion of the Y into the cytoplasm of the X daughter cell at the end of the first division. In three males, analyses of complete anaphase-II cysts were made; in each case the 32 X-bearing cells were normal and the 32 Y-bearing cells had only autosomes at the poles, with the Y-mass remaining at the equator. In none of the males examined was there any indication that all second-division cells contained an X chromosome.

It should be emphasized that the features of meiosis just described were found in both the laboratory and wild stocks. All the lines had three inversions in the right arm of the "sex ratio" X chromosome, these inversions corresponding to those figured by Dobzhansky and Epling (*11*). Counts were made on sperm bundles at spermatid and mature sperm stages in "sex ratio" and control males; "sex ratio" males had a full complement of 128 sperms in each bundle, with no indications of degeneration or abnormal development.

These observations lead to a simple explanation for the "sex ratio" effect in terms of the regular nonfunctioning of two of the products of meiosis: preferential movement of the X chromosome to the functional pole at anaphase I ensures an all-female progeny. Although it remains to be proved that the abnormal behavior of the Y chromosome is not responsible for the inactivation of the non-X-bearing sperm, it is possible to present a strong argument against a direct role of the degenerating Y chromosome. Schultz (*12*) has shown that sperms bearing neither an X nor a Y chromosome are functional, producing viable but sterile XO males. In our material the frequency of nondisjunction of the sex chromosomes at the first division is sometimes unusually high (up to 10 percent), but the progeny class which would correspond to this event, the XO males, does not appear with any appreciable frequency. It seems preferable to regard the occasional males that occur in some cultures as resulting from the rare failure of the X chromosome to become oriented toward the functional pole.

In connection with this study we also examined "sex ratio" in *D. athabasca* (*13*), another species of the *obscura* group. The features of meiosis were identical with those described for *D. pseudoobscura*, with the X and Y chromosomes disjoining regularly at first anaphase and the Y "degenerating" in the second division. Novitski (*14*) had previously shown that the "sex ratio" X chromosome of *D. athabasca* carries three inversions which bear some resemblance to those of *D. pseudoobscura*. Apparently "sex ratio" has enjoyed long-term involvement in the genetic systems of the *obscura* complex.

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References and Notes

1. E. Novitski and I. Sandler, *Proc. Nat. Acad. Sci. U.S.* **43**, 318 (1957).
2. S. Zimmering, *Genetics* **45**, 1253 (1960).
3. W. J. Peacock and J. Erickson, *ibid.*, in press.
4. L. Sandler, Y. Hiraizumi, I. Sandler, *ibid.* **44**, 233 (1959).
5. W. J. Peacock, *ibid.*, in press.
6. S. Gershenson, *J. Genet.* **28**, 297 (1933); L. Sandler and G. Braver, *Genetics* **39**, 365 (1954).
7. Gershenson, *Genetics* **13**, 488 (1928).
8. A. H. Sturtevant and Th. Dobzhansky, *ibid.* **21**, 473 (1936).
9. B. Wallace, *Evolution* **2**, 189 (1948).
10. We thank Th. Dobzhansky for a stock from his laboratory and A. H. Sturtevant who accompanied E. N. on a hazardous trip to Borrego Springs and Mexicali to capture additional strains.
11. Th. Dobzhansky, *Carnegie Inst. Wash. Publ.* *554* (1944), p. 49.
12. J. Schultz, *Genetics* **18**, 284 (1933).
13. Stock provided by Dwight Miller.
14. E. Novitski, *Genetics* **31**, 508 (1946).
15. Supported by PHS training grant 5TIGM373-05 and NSF research grant GB1332.

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Seed Dispersal Velocity in Four Dwarfmistletoes

Abstract. *By means of a high-speed photographic technique, the initial velocities of seeds were studied as they were expelled from the fruits of four Colorado dwarfmistletoes:* Arceuthobium douglasii, A. campylopodum *f.* cyanocarpum, A. vaginatum *f.* cryptopodum, *and* A. americanum. *Velocities of the seeds of the latter two species averaged 2600 centimeters per second and were significantly greater than those of the first two, which averaged 2200 centimeters per second. The initial velocity of 526 seeds of the four dwarfmistletoes averaged 2400 centimeters per second.*

The dwarfmistletoes (*Arceuthobium* spp.), which are one of the most serious agents of disease of western coniferous forests, have explosive fruits for seed dissemination. Local dispersal of the plants is almost exclusively due to this mechanical seed expulsion. As part of our studies of the biology of *Arceuthobium*, we measured the velocities of seeds immediately after they were expelled from the fruit.

Each *Arceuthobium* fruit contains a