There were no return mutations at the yellow, white or forked loci. Considering the small number of offspring examined and the rarity of return mutations this is not a matter for surprise. There were eight cases of normal wings instead of the expected Beadex wings. These were either extreme overlaps of the beaded characters or somatic mutations. They did not breed true when mated to double-X yellow virgins, but gave again the beaded wings. There were four cases of reversion to full eye, and these bred true to full. Two were in the T-4 group and two in the T-8 group. This is a ratio of one reversion in 433 males in the T-8; one in 1,898 in the T-4. These are the results of the observations upon the five definite loci studied in the X-chromosome of the male offspring.

In addition, approximately one hundred other mutations, mosaics and abnormalities of various sorts were observed. These occurred in both males and females and affected all parts of the body. The strong temptation to preserve and breed everything that turned up was resisted and the original plan of rigidly concentrating on the five loci described above will be adhered to in further work now under way.

The four reversions to full eye at the bar locus are of considerable importance. Zeleny[2] has studied the bar-eye gene very extensively. He found that bar eye reverts to round eye about once in 1,600 times; and also discovered an allelomorph of bar, called ultra-bar, whose reversion rate is approximately the same. Zeleny considered the bar-eye character to be due to a gene mutation not different from other gene mutations in Drosophila. Upon evidence based on the sex ratio Zeleny concluded that mutations to full eye may occur in the male germ tract as well as in the female.

Sturtevant[3] assumes that Zeleny's data indicate that reversion occurs only in the female and reports extensive experiments which seem to show that reversions at the bar locus are due, not to gene changes at all, but rather to unequal crossing-over between the two X-chromosomes of the female. Hence no reversion in the male is possible, since no crossing-over occurs.

According to Sturtevant crossing-over in the bar region occurs in such a way that the respective points of interchange lie to the left of the bar locus in one chromosome, but to the right of it in the other one. Sturtevant made many crosses and all his data, compiled in nineteen tables, indicated that both reversion to round and to ultra-bar eye (called by him double-bar) was due to unequal crossing-over. He also made a count of 10,179 males derived from a cross with double-X yellow females, in which all males got their X-chromosome from the father, and in these males no rounds or double-bars were observed.

Furthermore, Muller and Dippel[4] counted 35,000 sons that had derived their X-chromosome from a bar-eyed father, and in this large number not a single case of reversion to full eye occurred. These results of Sturtevant and Muller and Dippel seemed to warrant the conclusion that mutations to round eye occurred exclusively in the female. And Sturtevant's experiments indicated that reversion to round eye was due to unequal crossing-over in the female and not to a gene mutation such as is responsible for character changes at other loci.

However, that reversions to round eye *are* possible without crossing-over seems proved by the work with X-rays. Muller (unpublished data) got two reversions to round eye in females in cases where there was no crossing-over near the bar locus (between forked and Beadex), one in control material and the other from lightly X-rayed flies. My results (described above) give one reversion to round in 433 males from heavily treated fathers and one in 1,898 males from fathers less heavily treated. These reversions were obtained under conditions which clearly rule out Sturtevant's theory of unequal crossing-over.

It is of interest that a mutation rate of one in 433 males is probably the highest rate of gene change yet reported in *Drosophila melanogaster*.

FRANK BLAIR HANSON

DEPARTMENT OF ZOOLOGY,
WASHINGTON UNIVERSITY

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A SPONTANEOUS MODIFICATION OF THE VISCOSITY OF FRESH BLOOD SERUM

IN order to study such unstable solutions as blood serum, it is important to dispose of methods which enable us to follow continuously the evolution of a phenomenon, as a function of time, or temperature, for example, without introducing uncontrollable or disturbing factors. The viscometer described in 1923 (based on the principle of coaxial cylinders) was devised in order to fulfil these requirements, and was used recently to determine the viscosity of fresh normal horse serum, as a function of time. It was found that such a serum, centrifuged immediately after separation from the clot and placed in the viscometer, behaved in the following way: At first, its viscosity, which is

rather high (relative viscosity $\frac{\eta}{\eta_0}$ *i.e.*, ratio of the absolute viscosity, of the solution, to that of the solvent, of the order of 3) generally increases for a short time —say ten minutes—then decreases rapidly, almost proportionally to the time, and finally reaches a stable value, considerably lower than the original one (order of magnitude $\frac{\eta}{\eta_0} = 1, 8$). Towards the end, the curve is logarithmic. The total phenomenon requires from one to two hours, as a rule, this time depending mainly, as far as we can see at present, on the amount of handling to which the serum has been submitted.

TABLE I

SPONTANEOUS DECREASE OF THE VISCOSITY OF FRESH BLOOD SERUM (HORSE)

(Exp. N° 10 Ser. N° 3) (Relative viscosity, η) Time minutes	η
0	2.640
5	2.730
7	2.640
10	2.580
15	2.440
20	2.140
26	1.905
30	1.870
35	1.845
40	1.835
45	1.830
60	1.824
70	1.824

Important fluctuations are sometimes observed at the beginning of the experiment. The spot moves jerkily, as if the bob (inside suspended cylinder of the viscometer) were attached to the outside moving cup and suddenly released.

When the spot is allowed to come back to the zero, that is, when the constant speed motor is stopped, during the first minutes of an experiment, it does *not* come back all the way to the zero, but stops at a certain distance—say thirty or fifty divisions of the scale. As the experiment progresses, however, the distance becomes shorter and shorter, and when the stable value of the viscosity is attained, the zero checks perfectly.

It seems to us that the aforesaid facts can readily be accounted for in the following way: the chemical evolution of the serum begins the minute it is separated from the fibrinogen. It has lost its power of coagulating rapidly, but the splitting, if we may be allowed to say so, of the "plasma molecule" leaves a "serum molecule," which does not immediately reach a stable state. The amputation of the fibrinogen exposes certain unstable chemical groups, which rearrange themselves in time by shifting or otherwise. While they are exposed, however, they still retain to a small extent the power of adhering to one another and to form reticular structures in the solution. These structures are invisible even under the ultra-microscope. But they are strong enough to apparently increase the viscosity of the serum, and, when the rotation of the cup is stopped, to prevent the spot from returning to the zero. As time elapses, the number of molecules capable of adhering to one another decreases, and when the transformation is complete, the zero checks, and the *true* viscosity is measured. Before that, what was measured was really viscosity *plus* a kind of plasticity, and the curves obtained express the progressive passage from one state to the other.

This hypothesis seems to account for the observed phenomena. The increase which takes place at the beginning may be due to the fact that handling of the serum (aspirated and poured into the cup by means of a pipette) breaks down the existing structures, which tend to rebuild themselves up as soon as the serum is in the cup; this would also account for the fluctuations described at the beginning of this paper.

This phenomenon is not without precedent in the colloidal world. Certain iron sols will coagulate, and become fluid again on mere shaking. It is known that the so-called "stabchen-sole" studied by Siedentopf, Szegvari and Zocher show the tendency to form structures by sticking to one another. This phenomenon is different from adsorption, as only certain parts of the particle seem capable of sticking, usually the extremities: Orientation of colloidal molecules or particles is one phase of this phenomenon.

We may now understand why, when studying living cells with dark-field illumination, certain particles can be seen in what seems to be a perfectly structureless protoplasm, obviously agitated with brownian movement, but apparently limited in all direction, as a bird in a cage. The bars of the cage are not visible: yet they are there; this explains how difficult it is to measure the "viscosity" of protoplasm, and also why the protoplasm does not flow out of fibroblasts which usually show at the end of one of their arms an "opening" which is not limited by any visible line. The protoplasm is not coagulated, yet it is maintained, not by surface tension, but by these reticular structures.

In finishing this preliminary note, I wish to express many thanks to Dr. Simon Flexner, director of the Rockefeller Institute, for having allowed me to take over the instruments and apparatus which made this work possible.

LECOMTE DU NOÜY

INSTITUT PASTEUR, PARIS