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which stimulate the mutability of the miniature gene. Our stock has not been tested for such possible modifiers.

Any case of a new mutable gene should be carefully tested and analyzed, for by so doing it may be possible to find a satisfactory explanation for this unique type of inheritance. The pressure of other work has not made this possible in the present case. I have therefore given all material to Mr. C. P. Oliver, who plans to make such a study.

J. T. PATTERSON

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A CASE DEMONSTRATING THE ABSENCE OF SOMATIC INDUCTION IN DROSOPHILA

IN a culture obtained from the cross of an X-rayed apricot male (w^a = apricot eye, an allelomorph of w , white eye) by a \overline{yy} female (“ \overline{yy} ” indicates attached X-chromosomes homozygous for y , the gene for yellow body), all the males appeared phenotypically indistinguishable from white, and the females were \overline{yy} . There were 92 males and 103 females in all in this culture, and among them not a single exception to the above results was observed. In order to exclude the supposition of a possible contamination of the culture, or of the \overline{yy} female taken for the cross with the X-rayed apricot male not having been virgin, this same male was crossed again with three other virgin \overline{yy} females. All the 212 males obtained from these crosses also appeared to be phenotypically white, and the females \overline{yy} . Some of these males were again crossed with \overline{yy} females, giving a second generation consisting of phenotypically white males and \overline{yy} females. All this showed that in the apricot culture we had obtained a mutation phenotypically identical with the mutation “white.”

Two unusual facts appeared in this connection. The first fact was that in a cross of this new mutation with homozygous white females, instead of our obtaining the expected pure line of white, that is, a line in which all males and females were white, we actually obtained white males, but all the females, contrary to expectation, appeared to be phenotypically like apricot. This showed that a transgene (“ x ”) had arisen which was not white, but some other kind of recessive. This recessive modified the gene w^a so as to produce the white

phenotype. It lay in the X-chromosome in some other position than apricot, and lost its modifying effect when in the heterozygous state. This is why females of the composition $\frac{w^a x}{w +}$, obtained from the cross of $w^a \times$ males by females homozygous for white $\left(\frac{w}{w}\right)$ produced apricot-like instead of white females $\left(\frac{w^a}{w}\right)$ females are similar in eye color to $\frac{w^a}{w^a}$.

A further investigation showed that the character "white" in our case was a complicated phenomenon, being produced by the joint action of two genes, the old gene apricot and a newly produced gene "ruby" (allelomorphic or identical with the previously known mutant of that name lying at locus 7.5 in the old standard map of the X-chromosome). The determination of this gene was made by the method of finding the per cent. of crossing-over between the gene for apricot and the gene in question. Among a total of 798 observed flies derived from females of the composition $\frac{w^a x}{cv\ ct\ f}$, 49 crossovers between w^a and x were obtained, or 6.1 per cent. From this we may conclude that the gene modifying the character apricot to white must lie at locus 7.8 ($6.1 + 1.7$, the latter being the locus of the gene apricot). This locus is very close to the previously determined locus of the known gene ruby (*rb*). The cross of the male carrying the new gene " x " (ruby?) with $\frac{rb}{rb}$ females, homozygous for the previously known ruby, gave a pure line of ruby. This definitely confirms the supposition of the origin, in this case, of the gene *rb*.

The second unusual fact is that, in the culture obtained from the cross of the male w^a by \overline{yy} , not only one male offspring was a mutant, as ordinarily is found to be the case at the first appearance of a mutant gene, but *all* the males of the culture at once appeared as mutants. In other words, *all* the germ cells of the X-rayed apricot male, without exception, seemed to have mutated at once, and in one direction—a case quite unthinkable from the point of view of modern genetics. Evidently the cause of this phenomenon is to be found not in simultaneous and identical mutations of all the germ cells, but in some other kind of principle. It is necessary to assume that the hereditary change in question appeared not at the time of the X-raying

of the sexually mature apricot male, whose germ cells were already differentiated, but much earlier, and precisely at that stage of its embryonic development, after the time when the cell—or rather, chromatids—destined to form the optic anlage had become separated from the germ tract, but before any cells—or rather, chromatids—of the germ tract had become separated from each other. This was probably at the first or second zygotic cell division, or at the prior chromatid division corresponding to that. In that single germ cell the new transgenation ruby must have arisen. Further development then led to the result that all germ cells had, in addition to the gene apricot, the gene ruby as well, but that the somatic cells did not have the latter. In consequence a male was obtained which carried in all his somatic cells the gene apricot, and possessed genotypically yet another additional factor—ruby.

In this way there has been obtained in *Drosophila*, in a natural way, a result which Castle, working on guinea pigs, Guthrie, Davenport, Shultz on chickens, Magnus on rabbits, and Kaltenbach on ducks had tried to obtain artificially. That is, we obtained a fly whose “outer case” of somatic tissue differed in its hereditary content from the germinal genotype contained within it. It is noteworthy that this “somatic case” did not exercise any detectable influence upon the germinal genotype, despite the fact that the offspring of this fly were rather numerous. The phenomenon described points to the presence of a “germinal tract” (*Keimbahn*) in the embryogeny of *Drosophila*.

Since the work of Johannsen, the ideas “phenotype” and “genotype” have been generally accepted in biology. Without these ideas modern genetics would never have reached the high level on which it is found at the present time. Nevertheless, these ideas are not always used in the same sense and at times they are given an erroneous interpretation, which leads to wrong formulations and to confusion.

Ordinarily we understand by the “phenotype” of an organism its genotype as realized under certain particular conditions. As the “genotype” we have in view a collection of genes in each germ cell. The “genotype” is understood to be the hereditary content of the organism. The term “genotypic” is considered equivalent to the term “hereditary.” Very often an indissoluble unity of phenotype and genotype is implied, con-

noting a unity of the non-hereditary manifestation of a character, and the hereditary content of an organism.

It appears to us that these concepts are not quite accurate or valid, but are in need of definite correction. Above all it is necessary to point out that we can speak of the phenotype as of a realized hereditary genotype only in the sense of its connection with time, but not with space, that is, historically but not physiologically. The genotype of the germ cells of the apricot male, in the case above described, contained, in addition to the other genes, the gene ruby as well. This gene was realized in the offspring; it was not realized at all in our male, but existed in a latent state, hidden within its "somatic case." The male apricot did not realize the genotype of his own germ cells, but only that genotype of the *parents*, which he had received in his *somatic* cells. The genotype of the germ cells themselves is never realized in the organism possessing these germ cells, it manifests itself only in the offspring, and does so only insofar as it goes over into the somatic cells of these offspring. Therefore the connection between the phenotype of an organism and the hereditary genotype of its germ cells, in other words, the connection between the characters of the organism and its material carriers of heredity, is an historical connection but not at all an existing biological one. It is self-understood that this historical connection does not exclude physiological connections and reciprocal actions between the somatic and the germ cells in each given organism. But these connections have nothing in common with heredity, any more than, for instance, the physiological connections between any of the somatic organs and parts of the organism have anything in common with heredity. From the fact that we may discover a very close physiological connection between the lungs and the heart, it surely will never occur to any one to make the deduction of a *hereditary* influence of these organs on each other.

One can not consider the ideas "genotypic" and "hereditary" as identical. In each organism, as is known, there are in principle as many genotypes as there are cells, and we should in general distinguish the inherited genotype of the germ cells from the inherited genotype of the somatic cells. For, although the latter coincides with the former in an overwhelmingly great number of cases on account of the shortness of the historical path from parents to children, the two must be dis-

tinguished on principle, if only for the reason that we often have to do not only with ordinary phenotypic changes of the organism, in reaction to influences of the environment, but with definite changes in the genotype. And only in this sense can one properly speak of a unity of phenotype and genotype.

The genotype of the germ cells never manifests itself in its carrier. *It has no phenotype at all*, as long as it exists in a hidden state, as long as it does not appear. We judge the genotype of the germ cells of an observed organism only by the subsequent analysis of the phenotype of its *offspring*. The developments of the somatic and of the germ cells do not depend *genotypically* upon each other, although both come from the same root, the same fertilized egg cell, and must exist in permanent, physiological connection with one another. A genotypic change of the "root" leads to a change in both branches growing out of it; a change, however, in some part of one of these branches is not reflected correspondingly in the other.

All these facts show once more how far modern biology has gone beyond the reasonings of the Lamareckians concerning so-called "somatic induction," beyond their naïve faith in the inheritance of acquired characters.

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CHANGE IN COLOR PATTERN IN A CAPTIVE RED SQUIRREL

AN immature male red squirrel (*Sciurus hudsonicus loquax*), one of a litter of five born in captivity on August 20, 1927, showed a marked deviation from the normal coloration of this species. The mother of this litter was captured alive and uninjured at Holland, Michigan, on August 5, 1927.

The unusual coloration consisted of a number of light gray spots on the dark gray and reddish-brown body. The locations of the spots were as follows: on the neck, a small area back of each ear; a large triangular patch on the back, extending from the shoulders to a point half-way down the back of the animal; a circular spot on each side of the body, midway between the belly and the dorsal line, just anterior to the hind legs, and one on each side of the rump at the base of the tail, the latter being totally gray (Fig. 1).