

denum. Neither chromium nor molybdenum was found in the spleen of E2902.

Nothing is known of the rôle of either chromium or molybdenum in the animal economy. Chromium and molybdenum belong to the oxidation reduction type of element and although their significance is unknown in human tumors yet it is more than possible that they have a definite and active rôle in the metabolism of the tumor cell.

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*THE ABSENCE OF AUTONOMY IN THE DEVELOPMENT OF
THE EFFECTS OF CERTAIN DEFICIENCIES IN DROSOPHILA
MELANOGASTER*

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In a recent paper presented at the Sixth International Congress of Genetics, Sturtevant² indicated the significance of mosaics for the study of the developmental effects of genes.

One of the important questions in this field is that of the autonomous or non-autonomous development of characters. It has been known, since the work of Morgan and Bridges,³ that in gynandromorphs of *Drosophila melanogaster* which result from elimination of one of the X chromosomes, the development of sex and certain sex-linked characters is perfectly autonomous. It has been found since that this autonomy of the differentiation of characters is not a general rule. Sturtevant² indicates a series of cases in which the differentiation of the character is influenced

by the genotypic constitution of the surrounding tissues. This lack of autonomy of development of a character has been observed, for example, in the case of the genes *scute*, *Bar*, *vermilion*, etc.

The question of the autonomous differentiation of the effects of deficiencies has recently been examined by Demerec.⁴ This author has shown that if in a female of *D. melanogaster*, one *X* chromosome is eliminated during somatic cell division (as an effect of a *Minute* gene), then if the other *X* chromosome carries a deficiency, the cells in which this elimination has taken place are not viable. This result indicates that the lethal effect of the deficiency is not influenced by the viability of the surrounding cells and seems to show that the lethal effect is autonomous in development.

At the suggestion of Professor A. H. Sturtevant I have made a similar study of two deficiencies in the *X* chromosome, the *scute-8* deficiency which involves the yellow to *achaete* interval with the remainder of the chromosome inverted, and the deficiency 100 (obtained by Mrs. L. V. Morgan) in which the yellow, *achaete* and *scute* genes are lost. A female heterozygous for the deficiency mated to a normal male produces daughters which, since they receive a normal *X* from the father, are all viable; among the males, which receive only a *Y* chromosome from the father, there is a class which is inviable because it carried no section homologous to the deficient region. This class can be saved by the introduction of a duplication covering the deficient region.

The duplication *scute-10-2* was used in these experiments because it was observed by Sturtevant (unpublished) that it is frequently eliminated during somatic divisions. This elimination leads to the production of mosaics which are visible if the *X* chromosome contains genes the manifestation of which is suppressed by the allelomorphs carried in the duplication.

Females heterozygous for the deficiency were crossed with *scute-10-2* duplication males, that is, males which carry, in addition to a normal *X* and *Y* chromosome, a fragment containing the normal allelomorphs of the genes yellow and silver and the mutant genes *achaete* and *scute*; the *X* chromosome of the male was marked by the gene *apricot* and the normal *X* of the female by the genes yellow and *Hairy wing*. Under these conditions the males carrying the deficient *X* chromosome were identifiable by the genes used as markers (w^a , $+^y$ and *ac*).

In my experiments the duplicating fragment insured the viability of the males carrying the deficient *X* chromosome but the fragment was eliminated in certain cells which consequently were of a genotypic constitution unattainable in a whole fly. But in this case these groups of cells were surrounded by viable cells.

The spots always appeared on the fifth, sixth and seventh abdominal

segments and were visible as yellow patches on a black background (loss of the normal allelomorph of yellow). The size of the patches indicated immediately that the cells constituting them were able to divide. Studies of the chitin showed, moreover, that these spots were covered with hairs and bristles of normal size and number. This fact constitutes sufficient proof of the viability of the hypodermal cells which form the hairs and bristles. These results, identical in the case of the two deficiencies studied, show that the lethal effect of the deficiency is suppressed by the viable surrounding tissues.

It should be pointed out that the absence of the $+^y$ locus in the cells of the spots provokes the appearance of yellow color. Some of the bristles in spots, the cells of which have no yellow locus, are yellow; but it was not possible to establish any relation between color and position of the bristles in the spots.

In order definitely to establish that the spots were the result of the elimination of the fragment, crosses of duplication males were made with silver females. In agreement with expectation, it was found that half of the males showed silver spots, the silver color of the remainder of the fly being suppressed by the normal allelomorph in the duplication. These silver spots differed from the yellow spots in that they were much larger, a fact which shows that in this cross elimination of the fragment occurs at an earlier stage of development.

It is concluded that groups of deficiency-carrying cells surrounded by cells of normal constitution are viable in the case of the two *X* chromosome deficiencies studied.

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