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The Interaction of Genes in Development.

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Since it has been shown that the transmission of most hereditary traits can best be explained on the theory of the gene, it is important to try to trace the relation between particular genes in the fertilized egg and the correlated somatic manifestations that appear during development. That such correlations do exist has been abundantly demonstrated, but just how the gene exerts its effect on the soma is still unknown. As a first step towards an analysis of this problem one may inquire into the interrelation between the genes themselves. Here there are two possibilities to be considered: (1) that the genes interact with each other to give the cell its structural and functional characteristics; (2) that each individual gene separately exerts its own specific influence.

These alternatives may be illustrated by the following schemata, in which A and B represent the genes in a cell whose contribution to the soma may be either S or S' (depending on such factors

$$(1)\ \left.\begin{array}{c} A \\ \\ B \end{array}\right\} \text{—}N\text{—} \left\{\begin{array}{l} S \\ \\ S' \end{array}\right. \qquad (2)\ \begin{array}{l} A\text{—}n\text{ —}S \\ \quad >n'\text{—}S' \\ B\text{—}n'' \end{array}$$

as topographical relations, stimuli, etc.). In (1) all the interaction between A and B is in the nucleus and N is the single resultant effect—a particular type of protoplasm. This would seem to correspond to a common conception among experimental biologists. In (2) A and B are more or less independent, n, n' and n'' representing distinct elements in the total of cytoplasmic potentialities. In this case it is not the genes themselves, but certain of their secondary products that interact, so that while *A* ultimately affects both *S* and *S'*, *B* affects only one of them. The determination of which of these alternatives is in accord with the true situation is of considerable importance to the understanding of protoplasmic reactions in general.

Data bearing on this question have been obtained from experiments with the *Y* gene in mice. Individuals with the genetic formula *CCSSYy* show two very different traits; they have yellow fur and they become decidedly adipose after reaching maturity. When *SS* (self color) is replaced by *ss* (spotting) the amount of yellow in the pelage is greatly restricted, and it might be expected, if the interaction were according to the first scheme, that the effect of *Y* on adiposity would also be limited. But a considerable series of controlled observations has shown that mice of the genetic constitution *ssYy* become fully as adipose as those that are *SSYy* and greatly exceed those that are *ssyy* or *SSyy*. This may be explained on the basis of the second scheme if *Yy* and *ss* be substituted for *A* and *B* respectively. *S* in the schema now represents adiposity, *S'* the amount of yellow fur. In this connection it might be objected that spotting is not a modifier of yellow in the usual sense of the term. However, there is some evidence that it really is such, and if it were not, it would still furnish evidence of another kind in favor of the second alternative.

Likewise, when *CC* (color) is replaced by *cc* (albinism) yellow is entirely suppressed but the adiposity is uninfluenced as determined by tests in which, as before, litter mates were used for controls. The following table shows the genetic formula, the color, and the weight at seven months of three pairs of littermate sisters.

TABLE I.

Genetic formula	Color of fur	Weight at seven months
CCS-Yy	Pure yellow	44 grams
CCS-yy	Pure black	24 ”
CCssYy	Spotted yellow	54 ”
CCssyy	Spotted brown	35 ”
ccSSYy	Pure white	52 ”
ccSSyy	Pure white	24 ”

A further effect of the *Y* gene is that it acts as a lethal when homozygous. Consequently a series of albino mice carrying *Y* were tested to see if a combination of genes which suppresses its effect on color would also suppress its lethal action. Among the forty-four individuals tested none was found in which *YY* had failed to act as a lethal, which indicates that *cc* had been without influence in this respect. The chances against the observations being purely fortuitous are about thirty thousand to one.

The three known effects of the *Y* gene occur at about six, twenty-five, and ninety days after fertilization of the egg, and the evidence cited indicates that a complex which modifies one of these effects may have no necessary influence on either of the others. This points to the conclusion that the genes do not mutually interact in the beginning to produce a certain individual type of protoplasm, but that, on the contrary, they behave as separate dynamic factors in development.

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Correlation of Protozoan Infections of Human Mouth with Extent of Certain Lesions in Pyorrhea Alveolaris.

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Gingival exudate or interproximal debris has been obtained from 350 individuals, displaying a wide variety of peridental conditions, and has been cultivated for 48 hours at 37° C. in Boeck's medium. This technique reveals many infections of *Endamoeba gingivalis* and *Trichomonas buccalis* in the gums that are negative to other means of diagnosis.

In a quantitative survey of 100 dental students in the University of California we have found that infection with *Endamoeba gingivalis* is closely correlated with the extent of gingival pocket formation (Table I).