2. Genic analysis of some mutant characters.

a. Giant plant (gi)

This simple Mendelian recessive mutant (gi) with white endosperm was crossed to a normal plant with yellow endosperm. The $F_1$ plant was normal as expected and the data on the segregation in 128 $F_2$ plants are listed in Table 2.

Table 2. Data on the segregation of the giant and yellow endosperm characters in the $F_2$ progeny.

| Character | + | gi | Total |
|---|---|---|---|
| Y | 59 | 22 | 81 |
| y | 44 | 3 | 47 |
| Total | 103 | 25 | 128 |

From this table, it seems highly probable that the two characters, giant and yellow endosperm, are linked in coupling phase ($x^2 = 7.856$). The present data indicate that yellow endosperm is caused by two or more genes. It is impossible to ascertain which of the genes governing yellow endosperm is linked with gi, although the gene gi may be on chromosome 6.

b. Recessive old gold stripe ($og_r$).

The phenotypic appearance of this mutant was exactly similar to that of the dominant old gold stripe reported by Lindstrom (1935). However, the genetic behavior differs as follows:

(i) The character is controlled by a recessive gene $og_r$, probably located on the right side of R in the linkage map ($li$-16-$g$-20-$og_r$) instead of to the left of g as was found for the dominant Og.

(ii) This character is considered to show cytoplasmic inheritance. Accordingly, the variegation appears not only in the homozygous recessive condition but also in the heterozygous $F_1$. The frequency of cytoplasmic transmission was low when the $og_r$ stripe was used as the male parent, while it was high when used as the female parent. Consequently, the disturbance in segregation ratios was more pronounced in the latter case than in the former. In this stripe, the yellow variegation on the leaf-blade is most conspicuous in the upper leaves and in the flag leaves of the plant in the first generation of the homozygous $og_r$ combination in the $F_2$ and backcross populations. On further selfing of the homozygous $og_r$ plants, the appearance of striping shows a trend to decrease progressively. The striping usually disappears entirely in about the 6th or 7th generation of selfing, in spite of the homozygous recessive $og_r$ constitution. Nevertheless, this apparently normal plant behaves genetically just like the homozygous $og_r$-stripe plant when crossed.

Table 3. Recombination percents of the three genes, $og_r$, g and li, on chromosome 10.

| Phase | No. of fam. | Total plants | $og_r$-g | g-li | $og_r$-li |
|---|---|---|---|---|---|
| $F_2$ * | 1 | 506 | 40.0 | 17.0 | 51.5 |
| B * | 1 | 206 | 53.7 | 16.5 | 60.2 |
| $F_2$ 1) | 3 | 766 | 21.3 | 15.0 | 30.4 |
| B 2) | 4 | 1303 | 26.5 | 17.9 | 44.7 |
| B 3) | 5 | 852 | 25.6 | 17.7 | 43.3 |

* came from the female parent of the $F_1$ plants with the cytoplasmic stripe.

1) came from the $F_1$ normal plant without any striping.
2) indicates the use of the female heterozygous parent with the stripe.
3) indicates the use of the female heterozygous parent without the stripe.

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1. The inheritance of resistance to brown spot of corn.

A study was designed to investigate the inheritance of brown spot of corn (Physoderma maydis). A susceptible inbred (NC7) was crossed with a resistant inbred (GT154) and the following generations derived for study: $F_1$, $F_2$, $F_3$, $B_1$, $B_2$, $B_{11}$, $B_{12}$, $B_{22}$. Individual plants were examined for brown spot symptoms and given a rating from 0 to 5; 0 being no symptoms and 5 being very badly infected. Data were collected at one location in 1954 and at each of two locations in 1955. The data were analysed using the methods proposed by Powers, Locke and Garrett 1950, and Powers, 1955.

It was found that four or more gene pairs differentiate the parents with respect to brown spot resistance. Several genetic models involving four loci and five loci, each defining the disease reaction of every genotype, were found which were compatible with the $F_2$ data obtained at each year-location. All of the models which were found to be compatible with the data involved epistasis; i.e., non-additivity between genotypes at certain of the loci. Further evidence of epistasis was