

with a single crossover in region 1 will give $\frac{++Y}{++Y} / \frac{++Y}{++Y}$. The combined expectation of these two genotypes is 22% of selfed $\underline{Y} \underline{Y}$ ears. The observed frequency is 1/19 or about 5%.

Thus both classes 1 and 5 in Table 2 as well as the recombination percentages in Table 3 agree in placing rgd distal to po on the short arm.

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1. Synthesis of hybrid esterase enzymes in E heterozygotes.

By investigating newly synthesized enzymes that are still associated with the template on which they were synthesized, we have been able to establish that in the heterozygotes the new hybrid enzymes are synthesized as such on the ribosomes and do not result from random dimerization of previously synthesized monomers. We have been able to rule out the possibility that the particle-bound enzymes represent nonspecific adsorption of free enzymes to the ribosomes. The hybrid enzymes very probably result from interaction between two messenger RNA molecules specified by the two alleles, each of which contributes some information to the specificity of the hybrid enzyme.

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2. Regulatory mutant at the E locus.

The esterase enzymes specified by the E alleles are normally synthesized in the maternal tissue, endosperm, and embryo of the developing kernel as well as in the young seedling. A mutant has been found which affects the distribution of the enzyme in these tissues. Normal amounts of enzyme are synthesized in the diploid tissue but synthesis of the enzyme in the endosperm is almost completely blocked. The mutant, designated $\underline{E}^{F'}$, controls the synthesis of an F type enzyme that shows the same electrophoretic mobility as the F type enzyme produced by the normally behaving \underline{E}^F allele. In heterozygotes the $\underline{E}^{F'}$ allele is not influenced by and does not affect the homologous allele so that, for example, in $\underline{E}^{F'}/\underline{E}^{F'}/\underline{E}^N$ endosperm only N type enzyme is detected electrophoretically and in the amount expected from a single dose of the gene.

Since the E gene is not active throughout the life cycle of the plant, we propose that the E locus is compounded of a regulatory and structural gene. According to this hypothesis, $\underline{E}^{F'}$ has a mutant regulatory gene which fails to "turn on" the structural gene in the endosperm tissue. The regulatory gene is similar to the operon in the β -galactosidase case described by Jacob and Monod in that it operates only in the cis condition controlling the structural gene on the same chromosome, and is very closely linked to the structural gene. No crossovers have been found in over 2000 tested endosperms. Since the E alleles can be distinguished only by the electrophoretic migration rate of the esterase enzymes which they specify, the test involves individual electrophoresis of single, immature endosperms.

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