mutant plastids which were restored to normal functioning by one or more restorer genes brought in by the pollen parent, the F₁ plants coming from these zygotes should possess mutant plastids whose expression would be realized in F₂ plants lacking the restorer genes. The ratio of green to white offspring would depend on the number of segregating restorer genes. The selfed F₁ plants which segregated whites in the F₂ would also be crossed as the pollen parent onto lines free of white alleles. None of the F₂'s from these outcrosses should segregate for white seedlings since normal plastids were contributed by the egg parent of the P₁ generation. If these results are obtained it follows that irreversible plastid mutations are produced by iojap and that, even though they may be restored to normal activity by genic interaction, their intrinsic mutant quality is retained and becomes evident when the restoring alleles are lost.

M. M. Rhoades

7. Disturbed ratios due to semi-lethality of etched kernels.

Ears segregating for the etched allele, which is 12 units distal to A in chromosome 3, often have a deficiency of homozygous etched kernels. Deviation from the expected percentage varies in different genetic backgrounds; in some, no marked discrepancy is found while in others there is a significant reduction in the number of etched kernels. Tests were made to determine if the deficiency of etched is gametophytic or zygotic in nature. Crosses of a Et/a Et x A Et/a et pollen gave 1: 1 ratios for the A:a pair so transmission of et pollen is normal. Crosses of A Et/a et by a Et pollen also gave 1: 1 ratios for A:a so et megaspores are fully viable. However, gave 1: 1 ratios for A:a so et megaspores are fully viable. However, the crosses of A Et/a et by a et showed that the deficiency of etched kernels is due to the deleterious effect of et on kernel development-i.e., etched acts as a semi-zygotic lethal. Etched kernels may abort early in development.

M. M. Rhoades

8. A test for recombination between the bt1 and sh3 alleles in chromo-

some 5.

Although the recessive mutants $\underline{bt_1}$ and $\underline{sh_3}$ differ markedly in their effect on kernel development, they are allelic. The compound $\underline{bt/sh}$ is similar in phenotype to \underline{sh} homozygotes. The phenotypes produced by the two mutants are so unlike that their allelism was unsuspected for some time and was accidentally revealed through a chance cross of the two mutant strains. Differing as they do in