M825 cms Vg, this newly-arisen mutation restores WF9 cms S.

The male-fertile exceptions described here can be accounted for formally as mutations at one or more restorer gene loci in the nucleus. So far as we are aware, these are the first reported instances of mutations in restorer genes. That we should have encountered four such male-fertile exceptions seems highly coincidental. We think it may be significant, also, that these changes were encountered in the same strains in which we have identified numerous additional cases of male-fertile exceptions involving cytoplasmic "mutations". We suggest a common basis for the two kinds of events. According to this scheme, given the first appearance, by whatever process, of male-fertile elements in male-sterile cytoplasm, they may become established and continue to propagate either in the cytoplasm or in the nucleus. In the former case, the change registers as cytoplasmic and the new strain has the characteristics of a maintainer which transmits the male-fertile trait through the egg, but not the sperm. In the latter case, the change occurs in the nucleus and the new strain, now behaving as a restorer, transmits male fertility through both egg and sperm.

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1. Monogenic chlorotic-lesion resistance in corn to Helminthosporium maydis.

A source of resistance to race 0 of Helminthosporium maydis in an East African strain of corn tested in Nigeria (Jeweus Craig and J. M. Fajemisin, Plant Disease Reporter 53:742-743, 1969) was obtained from Dr. Craig. Corn Belt adapted resistant selections (RS) were developed through backcrossing, selfing and selection. Genetic studies in the field and in the greenhouse involving numerous susceptible U. S. inbreds reveal that the resistance in our selections is monogenic recessive in
inheritance. A portion of the seedling data is given in the following table:

<table>
<thead>
<tr>
<th>Cross</th>
<th>Observed No.</th>
<th>Expected No.</th>
<th>$\chi^2$</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R^a$</td>
<td>$S^b$</td>
<td>$R$</td>
<td>$S$</td>
</tr>
<tr>
<td>W64AxRS</td>
<td>0</td>
<td>20</td>
<td>256.5</td>
<td>256.5</td>
</tr>
<tr>
<td>(W64AxRS)xRS</td>
<td>262</td>
<td>251</td>
<td>256.5</td>
<td>256.5</td>
</tr>
<tr>
<td>(W64AxRS)$_F_2$</td>
<td>186</td>
<td>522</td>
<td>177.0</td>
<td>531.0</td>
</tr>
</tbody>
</table>

$^a$Resistant: small chlorotic lesions with limited fungus sporulation

$^b$Susceptible: large tan, oval to rectangular lesions with abundant fungus sporulation

The symbol *rnm* is proposed for the recessive gene conditioning this chlorotic-lesion resistance to *H. maydis*.

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1. The *Adh$_1$ FC8* Operon.

Earlier studies have established that the level of alcohol dehydrogenase activity in the plant is limited by the concentration of a specific factor which is essential for the activity of the *Adh* gene (Schwartz, 1971). Although various *Adh$_1$* alleles have been shown to differ in their ability to compete for the limited factor, enzyme level in segregating kernels and seedlings is constant and independent of *Adh$_1$* genotype as long as the *Adh$_2$* gene is not active. The *Adh$_2$* gene, which specifies a relatively inactive enzyme, competes with the *Adh$_1$* gene for the limited factor.