

pollen with normal, C and S cytoplasms. The race O toxin has no differential effect on pollen germination.

This procedure can be accomplished in one to two hours and does not require sterile technique. Only small amounts of toxin are required for a test both because the pollen is very sensitive and because only a small volume of test medium is necessary. Plants are tested while they are still flowering and since the cells being assayed are reproductive cells, this procedure may be adapted for use as a selective device, for example, to select for toxin resistance. This procedure may also be of use in the isolation and purification of the race T pathotoxin since it permits the identification of the toxic fraction in a fractionation procedure.

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2. Nuclear restoration of fertility in S male-sterile cytoplasm in maize.

We reported previously (Genetics 60: 226, 1968; MGCNL 45: 30, 1971; Genetics 71: 607, 1972) on numbers of changes of the S cytoplasmic sterile condition to the fertile or semifertile level; they are not pollen transmissible. The fertility was assigned to changes in the cytoplasmic S element with the reservation that transmission of cytoplasm through the male gametophyte may be involved.

The present report deals with studies of four recent independent occurrences of mutations which restore fertility in S sterile cytoplasm and are pollen transmissible. These cases of mutation at restorer loci occurred in the progenies which were being searched for changes in the cytoplasm from male-sterile to male-fertile condition.

Male-sterile and male-fertile (maintainer) versions of five shrunken-2 inbred lines, R839, R851, R853, R853N and M825, were employed in these studies. The male-sterile cytoplasm incorporated into these lines traces to a Vg source which has been shown to be equivalent to the S (USDA) sterile cytoplasm.

Male-sterile plants from the sh₂ inbred lines were crossed by their corresponding maintainer inbred lines. The resulting progeny were searched for male-fertile exceptions, plants with entire tassels fertile or those

with fertile tassel chimeras, among otherwise male-sterile offspring. Over 300 such exceptional plants were identified during the 1971 summer growing season and were tested in the following manner. The exceptional male-fertile plants were either self pollinated or crossed as egg parents by the corresponding maintainer. These plants were also crossed as pollinators with sibling male-sterile individuals, and, in some instances, with male-sterile plants of other sh₂ inbred lines. A few exceptions were also crossed with an S male-sterile version of WF9. In all but four cases, the progenies of testcrosses with S male-sterile plants indicated that the male-fertile character of the exceptional plants was not transmitted through the pollen. These four cases do not fit the usual pattern of changed cytoplasm described above. In each instance, testcrosses of these exceptional male-fertile individuals with S male-sterile plants produced male-fertile offspring, suggesting a Mendelian, or nuclear, basis for the fertility. The four cases of restorer gene mutation are numbered I through IV and are discussed below.

Case I occurred in the R853N line. The restorer gene mutation occurred in an R853N maintainer strain which was represented in family 71-741. This mutation can not be traced to a single plant. The new gene restores WF9 cms S as well as R853N cms Vg.

Case II occurred in the R853 line and traces to a fertile tassel chimera borne on plant 71-737-16. The chimera involved one side of the main rachis and all florets of twelve lateral branches on that side of the tassel. In addition to R853 cms Vg, this new gene restores M825 cms Vg. Surprisingly, this gene does not restore R839 cms Vg which is ostensibly the same type of male-sterile cytoplasm as is carried by R853 and M825.

Case III also occurred in the R853 line and traces to a single plant, 71-739-37, with an entirely fertile tassel and a tiller that was also entirely fertile. In addition to R853 cms Vg, the case III mutation also restores the M825, R839 and R851 sources of Vg sterile cytoplasm as well as WF9 cms S.

Case IV occurred in the M825 line and traces to a fertile tassel chimera borne on plant 71-727-37. The main rachis and most lateral branches of the tassel of this plant were sterile. Eight contiguous lateral branches on one side of the tassel were fertile. In addition to

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