plants with the expected 50% viable pollen. Thus, Mo2RF and K63 do not invariably prevent restoration in single crosses with restored steriles, and the high degree of sterility observed in the crosses 33-16 x Mo2RF and 33-16 x K63 must in some manner depend upon the genotypes peculiar to these hybrids.

Several investigators have pointed out that certain inbreds, including restorer lines, may lack one or more "modifier" genes which complement the "major" restorer genes in bringing about complete restoration. Sterility in the two exceptional single crosses could be explained by assuming that Mo2RF and K63 do not carry all of the necessary modifiers. However, these modifiers must be present in 33-16 since it contains S cytoplasm and is fully fertile (in Connecticut at least). Presumably, therefore, 33-16 would contribute the necessary modifiers to the single crosses with Mo2RF and K63. But it could be argued that the modifiers in 33-16 are recessive and that Mo2RF and K63 carry the dominant alleles. In other words, pollen fertility in S cytoplasm would require in addition to dominant restorer genes, one or more recessive modifiers, which are absent in Mo2RF and K63. If this is true, it is difficult to explain why Mo2RF and K63 did not also produce sterile offspring when crossed to the restored S sterile line A158SF (restorers from Ky21).

A possible, formal explanation for the observed results can be suggested. The restorer system in 33-16 may require recessive modifiers which are not essential for restoration in A158SF which has restorers from Ky21. The inbreds Mo2RF and K63 would carry the dominant alleles of these modifiers whose presence would prevent complete fertility in single crosses with 33-16, but would have no effect on F_1's with A158SF. The fact that 33-16 restores A158S and WF9S in F_1 would mean that the latter two inbreds carry the recessive modifiers. This is also indicated by the crosses 33-16 x A158 and 33-16 x WF9, both of which are fertile, and by the cross A158SF x 33-16 which is close to 100% fertile.

Evidence bearing on the above formal scheme can be obtained from the comparative behavior of A158SF with restorers from Ky21 and A158SF with restorers from 33-16. These two restored lines with a common A158 residual genotype might be expected to breed differently (when crossed by Mo2RF and K63, for example) if the S restorer systems in 33-16 and Ky21 differ in their requirements for modifier genes.

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3. The ms_1ms_1 genotype in T cytoplasm.

As pointed out in earlier notes all evidence indicates that genic and cytoplasmic male sterility are controlled by completely independent genetic systems. As part of this evidence we have previously cited the behavior of a ms_1ms_1 genotype in plants with S cytoplasm and S restorer

genes (MNL 1959, p. 14). Such plants were male sterile, thus demonstrating that the ms_1 gene operates in S cytoplasm and is not inhibited by S restorer genes. We have now obtained ms_1ms_1 individuals with T cytoplasm and T restorers.

The procedure by which this combination was produced is the same as that described earlier, and takes advantage of the close linkage between the ms_1 and y loci. C103T$Rf_1rf_1Rf_2Rf_2YYMs_1Ms_1$ plants were crossed as female by a WF9 stock heterozygous at the ms_1 locus, i.e. WF9$rf_1rf_1rf_2rf_2YyMs_1ms_1$, and several fertile F_1 plants were selfed. White kernels on 2 segregating ears were planted. Ignoring X-overs, these white kernels should be of the genotype $yyms_1ms_1$, and 9/16 of them should carry the Rf_1Rf_2 genes. If the ms_1 gene does not produce male sterility in T cytoplasm in the presence of the T restorer genes, 9/16 of the plants from white kernels would be expected to be fertile. The actual results in the two families were 38 sterile:1 fertile and 39 sterile:0 fertile. The ms_1ms_1 genotype, therefore, must be unaffected by T cytoplasm and T restorers.

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1. Cytogenetic changes induced by vegetable oils in Zea mays - a preliminary study.

Certain vegetable oils have been found to induce cytological and genetic changes in wheat.[1] Based on these findings, a similar project was initiated[2] to study the effects of castor and peanut oils on corn.

Procedure. Corn seeds were treated by soaking in castor and peanut oils for periods of 6, 12, 18 and 24 hours. Wiped dry, the seeds were germinated in petri dishes. Controls of untreated seeds were also set up. Excised root tips were fixed in fresh Carnoy's acetic-alcohol for

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1. Swaminathan, M. S. and Natarajan, A. T. Cytological and Genetic Changes Induced in Vegetable Oils in Triticum. Journal of Heredity July-August, 1959. pp. 177-187.

2. This study was made possible by a grant under the National Science Foundation Teacher Research Participation Program. The project was carried out under the direction of Dr. Margaret Thompson, Department of Plant Breeding, Cornell University.