2. Behavior of Ind 33-16.

Josephson and Jenkins showed (Jour. Amer. Soc. Agron. 1948) that male sterility in certain double cross hybrids involving the inbred 33-16 was due to the presence of 33-16 cytoplasm. Cytoplasm from 33-16 is represented in our collection by source H, which, as shown above, is identical with S type cytoplasm by all genetic tests. Since the inbred 33-16 is fertile, it presumably has a full complement of all necessary S restorer genes. However, certain findings of Josephson and Jenkins raise questions about the nature of fertility restoration in 33-16. Of the five single crosses involving 33-16, three were listed as fertile with only 1-15% sterile plants. But two single crosses, 33-16 x K63 and 33-16 x Mo2 RF, were reported to give 95% and 99% sterile plants respectively. The latter results are unexpected, and suggest either that all, or some, of the restorer genes in 33-16 are recessive, or that K63 and Mo2 RF possess dominant genes which prevent normal expression of the 33-16 restorers.

Crosses recently grown at this Station provide some information on the behavior of 33-16. First, it is clear that 33-16 does carry restorer genes for S type cytoplasm. Moreover, like S restorers from other sources, the restorers in 33-16 are dominant. The single crosses A158S13 x 33-16, WF9S13 x 33-16, as well as A158 steriles A-I x 33-16 and WF9 steriles A-I x 33-16 were fertile. Further, pollen fertility appeared to be approximately 50% in the plants of these single crosses examined with a hand microscope in the field, and an actual count of pollen stained with IKI in the hybrid WF9S12 x 33-16 revealed 55% well filled grains. Thus, when heterozygous, the S restorers in 33-16 appear to behave like typical S restorers in their effects on pollen viability. Preliminary evidence for allelism between the S restorers from Ky21 and 33-16 was found in the cross A158SF4 (homozygous for S restorers from Ky21) x 33-16, where the three plants examined appeared to be nearly 100% restored. If the restorer genes from the two sources were non-allelic and completely independent, 25% of the pollen grains would lack restorers and abort, fertility thus being only 75%.

The cross WF9T11 x 33-16 was completely sterile; 33-16 does not, therefore, have the two genes needed to restore WF9T steriles.

Since the restorer genes in 33-16 are apparently dominant like other known S restorers, attention was also directed toward the inbreds K63 and Mo2RF, the two inbreds Josephson and Jenkins reported to give sterile F_1 hybrids with 33-16 as seed parent. When grown in Connecticut the hybrid 33-16 x Mo2RF was almost completely sterile; anthers were extruded in an irregular pattern, and little or no pollen was shed. The cross 33-16 x K63 was more fertile, but was not fully normal, the anthers frequently failing to open.

These inhibitory effects of Mo2RF and K63 on pollen restoration were not expressed on S restorer genes from Ky21. Crossed to A158SF lines homozygous for Ky21 restorers, both Mo2RF and K63 gave fertile

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.

Harry T. Stinson, Jr.

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