sterility was causing the sterility. Also a "state" of the cytoplasm (dauermodification) may have been induced by the mutagen treatment. Another possibility under investigation is that cytoplasmic mutations for sterility were induced but when they were crossed to the untreated controls restorer genes were brought in leading to fertility. This assumes that the inbred line is segregating for restorer genes. To examine this possibility, remnant seed from each treatment that showed male sterility was planted and outcrossed with one of two unrelated inbred lines. These F_1's have been self-pollinated and will be planted in order to examine this theory. This approach may be feasible since Edwardson (3) reported that genes which restore fertility to cytoplasmic male sterile corn occurred in 59.6% of Latin American varieties and that the frequency of such genes in U.S. inbreds is 10.5% and that 2.81% were segregating for restorer genes. Also the variety Golden June, the source of Texas male sterile cytoplasm, was segregating for restorer genes (4).

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1. Unusual reaction of N and T cytoplasms to H. maydis, Race T.

One maize hybrid with N cytoplasm in a 1971 experiment segregated into a 3 resistant:1 susceptible phenotypic ratio. The same hybrid with T cms had one resistant plant. Paired entries were planted with hand planters. It is possible, but not likely, that this plant resulted from a kernel intended for the adjacent plot.

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