Effect of the Nuclear Genome of Corn on Sensitivity to Helminthosporium maydis race T-toxin and on Susceptibility to H. maydis race T

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ABSTRACT

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Differences in susceptibility of corn genotypes (containing Texas male-sterile cytoplasm) to *Helminthosporium maydis* (Nisikado and Miyake) race T and in sensitivity to *H. maydis* race T-toxin were determined using quantitative bioassays. Evaluation of a wide range of genotypes indicated that both the level of susceptibility of corn to the fungus and the degree of sensitivity to the toxin were influenced by the nuclear genome. However, differences in susceptibility to the fungus were not correlated with differences in sensitivity to the toxin. Nuclear genes that influence cytoplasmic sensitivity to the toxin appear to play no role in disease development, whereas cytoplasmic sensitivity to toxin is known to cause increased susceptibility to *H. maydis* race T. These observations, and those of previous studies on the genetics of *H. maydis*, support the hypothesis that this toxin is not required for the establishment of a compatible host-parasite relationship but that it does contribute to the virulence of *H. maydis* race T on corn containing Texas male-sterile cytoplasm. This role in disease differs from those that have been described for host-specific toxins produced by several other fungal plant pathogens.

Additional key words: southern corn leaf blight, host-specific toxin.

Susceptibility of corn to *Helminthosporium maydis* Nisikado and Miyake race T is conditioned by both cytoplasmic and nuclear genes. Certain inbreds and hybrids are more susceptible than others (1, 7, 13, 14), but any genotype containing a male-sterile cytoplasm from the Texas (T) group is more susceptible than its counterpart in non-Texas or nonsterile (N) cytoplasm (1, 9, 10, 14, 20). Efforts to elucidate the molecular basis of these selectivities have centered on the host-specific toxin (HMT-toxin) produced by *H. maydis* race T. Although the toxin is selective for corn with T-type cytoplasm (3, 4, 5, 6, 10, 22, 25), its role as an essential factor in pathogenicity has not been established.

Evaluation of the biological significance of HMT-toxin is facilitated by comparison with other host-specific toxins. Toxins produced by *H. victoriae, H. carbonum* race 1, and *Periconia circinata* which are pathogens of oats, corn, and sorghum, respectively, appear to be required for disease initiation and development (19). The most convincing evidence for the roles of these three toxins in disease has come from genetic studies of both host and pathogen. In each of the three hosts, susceptibility to both the pathogen and its toxin is controlled by a single genetic locus. In each case, plants susceptible to the fungus are highly sensitive to the toxin, plants intermediate in susceptibility to the fungus are intermediate in sensitivity to the toxin, and plants resistant to the fungus are highly insensitive to the toxin (8, 12, 16, 21). This correlation between susceptibility to the fungus and sensitivity to the toxin in each case suggests that susceptibility is a direct result of the action of toxin on the plant.

In two of the pathogens, *H. victoriae* and *H. carbonum* race 1, both specific pathogenicity and toxin production are controlled by the same genetic locus (genetic analysis of *P. circinata* has not been done). When *H. victoriae* and *H. carbonum* race 1 are crossed sexually, the progeny segregate 1:1:1:1 for ability to produce the oat-specific toxin, the corn-specific toxin, both toxins, and neither toxin (19). Isolates producing oat-specific toxin are pathogenic only to susceptible oats, isolates producing corn-specific toxin are pathogenic only to susceptible corn, isolates producing both toxins are pathogenic to both susceptible oats and susceptible corn, and isolates producing neither toxin are nonpathogenic. The appearance of nonparental types in the progeny indicates that neigher fungus possesses the other's gene for specific pathogenicity; i.e., each fungus has a unique gene for specific pathogenicity and the product of gene action in each case is the host-specific toxin. Thus, each host-specific toxin is required by the toxin-producing fungus for specific pathogenicity. In addition to these cases, there is much evidence that the host-specific toxin produced by *Alternaria kikuchiana* is required for specific pathogenicity in the black spot disease of Japanese pear (17).

It is important to determine whether or not HMT-toxin plays a similar role in disease. Genetic analysis of *H. maydis* suggests that HMT-toxin is not required for

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pathogenicity. When *H. maydis* race T is crossed sexually with *H. maydis* race O (pathogenic to corn but produces no detectable HMT-toxin), only parental types appear in the progeny (26, 27). Thus, the two races appear to share genes for specific pathogenicity, but they differ in one or more genes for virulence on T-cytoplasm corn. The gene(s) for virulence apparently code for production of HMT-toxin, because all isolates that produce HMT-toxin have increased virulence on T-cytoplasm corn, and all isolates that do not produce HMT-toxin do not have such increased virulence (27).

Genetic analysis of the host is the topic of this report. Since studies of the pathogen indicate that HMT-toxin plays a fundamentally different role in disease than do the toxins from *H. victoriae* and *H. carbonum* race 1, it is important to know whether or not genetic analysis of the host supports this interpretation. Previous work has established that all male-sterile cytoplasms which confer increased susceptibility to *H. maydis* race T also are sensitive to HMT-toxin (3, 4, 5, 6, 10, 22, 25). However, there are conflicting reports as to whether or not nuclear genes (in T-cytoplasm) that confer increased susceptibility to *H. maydis* race T also condition sensitivity to HMT-toxin. Corn genotypes containing T-cytoplasm which differ in susceptibility to *H. maydis* race T are reported to either (i) show no differences in sensitivity to HMT-toxin (3, 24, 25), (ii) show differences in sensitivity to HMT-toxin which are not correlated with differences in susceptibility to *H. maydis* race T (2, 18), or (iii) show differences in sensitivity to HMT-toxin which are correlated with differences in susceptibility to *H. maydis* race T (22, 23). The conflicting reports may be explained by differences in procedures used by various workers. In all cases, experiments were done with either a small number of corn genotypes, toxin preparations containing low activity, or bioassays that are insensitive or nonquantitative (28).

The objective of this study was to reexamine the possibility that the same nuclear genes condition both susceptibility to *H. maydis* and sensitivity to HMT-toxin. Experiments were performed using two quantitative bioassays, a partially purified toxin preparation with relatively high specific activity, and a substantial number of corn genotypes including susceptible, intermediate, and resistant inbreds and F_1 hybrids derived from them.

MATERIALS AND METHODS

Corn inbreds in T cytoplasm that were susceptible, intermediate, or resistant to *H. maydis* race T were maintained by increasing them each summer in field plots at Ithaca, N.Y. Poor vigor of inbreds and nonuniformity among seed lots can cause misleading results in bioassays with HMT-toxin (2, 24). To reduce these problems, hybrid seeds were produced. A range of reactions to *H. maydis* race T was obtained by making crosses among susceptible inbreds, among intermediate inbreds, and among resistant inbreds; all experiments were done using a single lot of seed from each of the 24 inbreds and hybrids compared in this study. Plants used for inoculation or for dark CO_2-fixation experiments were grown for 16 days in a controlled environment chamber at 23 C, 80% relative humidity, and with a 16-hr photoperiod (17.2 klx).

Helminthosporium maydis race T (isolate NY9), preserved in infected corn leaf tissue, was freshly reisolated from the leaf tissue for each experiment and grown on potato-dextrose agar in fluorescent light for 7 days. Spores for inoculation were dislodged from cultures with sterile water containing 0.05% Triton X-100 and the concentration was adjusted to 500 spores/ml. Sixteen-day-old corn plants (20/genotype) were misted with the spore suspension until the leaves were uniformly wet. Inoculated plants were held in a fog chamber for 24 hr at 30 C and then incubated for 5 days in the 23-C chamber. The lengths and widths of 45 discrete lesions on the third leaves of plants of each genotype were measured. Lesion areas were calculated using the formula for an ellipse. The set of 24 inbreds and hybrids was tested for susceptibility three times; in each experiment the rank of the genotypes was the same and the statistically significant differences were similar to those shown in Table 1.

The HMT-toxin was produced on modified Fries' medium as described previously (5). The culture filtrate was adjusted to pH 4, filtered, mixed with two volumes of methanol, and held at 5 C for 24 hr. The resulting precipitate was removed by filtration, and the methanol was removed in vacuo at 40 C. The remaining solution was restored to its original volume with water, and toxin was extracted by partitioning three times with equal volumes of chloroform. The chloroform phases were pooled, dehydrated with sodium sulfate, filtered, and evaporated to dryness in vacuo at 40 C. The water-soluble portion of the residue was resuspended in a volume of water 200-fold less than that of the original culture filtrate, layered on a 1.6×80 cm Bio-Gel P-2 column, and eluted with water at a flow rate of 10 ml/hr. The toxin-containing fractions (detected by the dark CO_2 fixation assay) were pooled, concentrated to a volume 100-fold less than that of the original filtrate, and stored at 4 C. This stock preparation had a dry weight of 4.37 mg/ml and was considered to have relatively high specific activity because it inhibited dark CO_2 fixation (2) in corn inbred W64A T (but not N) by 50% at 0.15 μg dry wt/ml. Toxin prepared in this way is active and host-specific in all bioassay systems (28) used to identify HMT-toxin activity in culture filtrate or in partially purified preparations. Host-specific activity in the preparation could be resolved into two or more bands by thin-layer chromatography, but the migration rates of these bands were not reproducible between experiments (G. A. Payne and O. C. Yoder, *unpbulished*). Multiple toxins have been reported previously for *H. maydis* race T (11) but since the biological significance of multiple components is uncertain (28), the total recoverable toxin was used for the experiments reported here.

Seedling root growth and dark CO_2-fixation bioassays were used to quantify sensitivity of corn genotypes to HMT-toxin. Dark CO_2-fixation was selected because it accurately quantifies toxin activity and is one of the most sensitive bioassays available; i.e., 5 to 10 times more sensitive than seedling root growth (2, 28). Seedling root growth was selected because it has been used frequently in previous comparisons of corn genotypes (3, 10, 15, 23, 24, 25) and because it is semi-quantitative (28); however, its use was limited to genotypes with uniform seed germination and a high level of seedling vigor. Both

bioassays were performed as described previously (28) except that 16-day-old corn plants were used for the dark CO_2-fixation bioassay. Sensitivity of each corn genotype to toxin was quantified for both bioassays by plotting percentage inhibition versus the logarithm of toxin concentration. Data were analyzed by linear regression, a best-fit line was established for each genotype, and genotypes were compared at various points on the linear portions of their dosage-response curves. Using the dark CO_2-fixation assay, the complete set of 24 genotypes (Table 1) was tested three times; a subset including inbreds CO109, W64A, B37, C103, and MO17 was tested seven additional times. Using the seedling rot growth bioassay, the subset of ten genotypes with good seedling vigor (Table 2) was tested twice; a subset including inbreds W64A, B37, and MO17 was tested four times. The relative toxin-sensitivity of each genotype was consistent among experiments.

RESULTS

The inbreds and single-cross hybrids in T-cytoplasm had a range of susceptibilities to *H. maydis* race T (Table 1). The lesion area of the most susceptible inbred was 13 times greater than that of the most resistant inbred. Some of the inbreds and hybrids used in this comparison were tested previously for susceptibility to *H. maydis* race T by other workers (7, 13, 14); their relative rankings were similar to ours (Table 1).

Genotypes in T-cytoplasm also differed in sensitivity to HMT-toxin in both the seedling root-growth assay and the dark CO_2-fixation assay. Table 1 shows the relative sensitivities of the genotypes at a toxin concentration for each assay which inhibited W64A (an inbred intermediate in sensitivity to the toxin) approximately 50%. At these toxin concentrations, differences among the genotypes were small when the seedling root growth assay was used, but large when the dark CO_2-fixation assay was used. The rank of genotypes (from most susceptible to resistant) by the seedling root growth assay did not correlate with the rank according to the dark CO_2 fixation assay ($r = 0.2194$, $P = 0.50$). In both assays, the order of sensitivity to the toxin was different from the order of susceptibility to the fungus (Table 1). It is interesting that analysis of 10 commercial hybrids in T-cytoplasm revealed no apparent correlation between sensitivity to HMT-toxin and susceptibility to the fungus (25).

The slopes of the toxin dosage-response curves varied among genotypes, which made data inconclusive when a single toxin concentration was used. Therefore, the toxin concentrations required for 20%, 50%, and 80%inhibition were calculated from the best-fit regression line of the dosage-response data for each genotype. Using either the seedling root growth assay (Table 2) or the dark CO_2-

TABLE 1. Susceptibility to *Helminthosporium maydis* race T and sensitivity to HMT-toxin of corn genotypes in T-cytoplasm

Genotype	Susceptibility		Sensitivity to toxin			
			Dark CO_2-fixation assay		Seedling root growth assay[x]	
	Rank	Lesion area (mm^2)[y]	Rank	Inhibition (%)[yz]	Rank	Inhibition (%)[yz]
CO109	1	40 a	2	72 a		...
AY191	2	35 a	24	25 i		...
B14A	3	34 a	20	41 fgh		...
AY191 × W64A	4	30 b	16	54 cde	8	51 cde
W64A	5	24 c	11	58 cd	6	52 bcd
CO109 × W64A	6	23 c	1	75 a		...
A239	7	23 c	14	55 cd		...
A97	8	22 c	17	52 de		...
W64A × B14A	9	22 c	15	54 cd	9	49 de
AY191 × CO109	10	21 cd	13	57 cd		...
CI64	11	20 cd	3	71 ab		...
WF9	12	20 cd	23	29 i		...
A97 × SD10	13	18 de	9	58 cd	2	60 a
B37	14	17 def	19	44 efgh		...
WF9 × W64A	15	17 def	12	57 cd	1	61 a
AY499	16	16 def	7	61 bcd		...
B37 × A239	17	14 efg	18	51 defg	4	59 ab
C103 × CI64	18	14 fgh	21	41 gh	3	60 a
AY499 × MO17	19	11 ghi	8	60 bcd	5	58 abc
AY499 × C103	20	10 hi	6	61 bcd		...
MO17 × CI64	21	9 i	22	34 hi	10	44 e
C103	22	5 j	5	65 abc		...
MO17	23	3 j	10	58 cd		...
MO17 × C103	24	3 j	4	71 ab	7	51 cde

[x]Blanks indicate genotypes which could not be used in the seedling root growth assay because of poor seedling vigor (designated inbreds) or an inadequate supply of seeds (designated hybrids).

[y]Values within columns followed by the same letter are not significantly different from each other ($P = 0.05$) according to Duncan's multiple range test.

[z]Inhibition caused by toxin (146 ng/ml for the dark CO_2-fixation assay; 1,460 ng/ml for the seedling root growth assay) compared to a value for a control not treated with toxin. Values were within the linear portion of the dosage-response curve for each genotype.

fixation assay (Table 3), differences in sensitivity of genotypes to toxin were found at each toxin concentration. However, there was no correlation between susceptibility of corn genotypes to the fungus and their sensitivities to the toxin in either the root-growth assay (Fig. 1) or the CO_2-fixation assay (Fig. 2) at any toxin concentration tested. Similarly, there was no correlation between susceptibilities of the genotypes to the fungus and slopes of the toxin dosage-response curves for the same genotypes in either assay (Fig. 3).

DISCUSSION

Results of this study indicate that the nuclear genes in corn which condition susceptibility to *H. maydis* race T have no apparent effect on sensitivity to HMT-toxin, which is controlled by the cytoplasm. Thus, HMT-toxin is not a host-specific toxin when nuclear genes are considered; it is host-specific only when cytoplasms are compared. This conclusion is based on our observation that there was no correlation between susceptibilities of

TABLE 2. Inhibition of seedling root growth of corn genotypes in T-cytoplasm by *Helminthosporium maydis* race T-toxin

Genotype	Toxin (ng/ml) required for inhibition of:[a]			Correlation coefficient[b]	Slope of regression line
	20%	50%	80%		
W64A	340	1,621	7,721	0.96	44
W64A × B14A	114	1,833	8,138	0.99	46
AY191 × W64A	279	2,123	16,195	0.98	34
WF9 × W64A	122	1,199	11,779	0.88	30
B37 × A239	298	1,533	7,902	0.93	33
A97 × SD10	126	839	5,567	0.99	36
C103 × CI64	247	1,162	5,462	0.94	45
AY499 × MO17	138	1,120	9,066	0.94	33
MO17 × CI64	668	2,286	7,818	0.97	56
MO17 × C103	165	1,401	11,907	0.88	32

[a]Toxin concentrations required for 20, 50, and 80% inhibition were calculated from a best-fit regression line through the linear par of the dosage-response curve for each genotype.

[b]The correlation coefficient for each genotype indicated a significant ($P = 0.01$) fit of the points to the regression line.

TABLE 3. Inhibition of dark CO_2-fixation of corn genotypes in T-cytoplasm by *Helminthosporium maydis* race T-toxin

Genotype	Toxin (ng/ml) required for inhibition of:[a]			Correlation coefficient[b]	Slope of regression line
	20%	50%	80%		
CO109	23	72	229	0.91	60
AY191	126	340	919	0.94	69
B14A	46	230	1,159	0.96	43
AY191 × W64A	49	125	315	0.99	74
W64A	17	98	549	0.95	40
CO109 × W64A	30	72	170	0.98	80
A239	27	116	491	0.88	48
A97	25	135	724	0.93	41
W64A × B14A	24	89	331	0.97	53
AY191 × CO109	45	130	372	0.89	66
CI64	29	92	287	0.89	61
WF9	82	335	1,376	0.93	49
A97 × SD10	22	104	479	0.97	45
B37	42	235	1,320	0.82	40
WF9 × W64A	35	144	594	0.92	49
AY499	28	96	332	0.88	56
B37 × A239	30	160	857	0.97	41
C103 × CI64	81	194	467	0.97	79
AY499 × MO17	19	87	388	0.94	46
AY499 × C103	31	114	421	0.96	53
MO17 × CI64	31	164	883	0.94	41
C103	27	82	243	0.96	63
MO17	24	109	498	0.88	46
MO17 × C103	32	78	191	0.96	77

[a]Toxin concentrations required for 20, 50, and 80% inhibition were calculated from a best fit regression line through the linear part of the dosage-response curve for each genotype.

[b]The correlation coefficient for each genotype indicated a significant ($P = 0.01$) fit of the points to the regression line.

corn genotypes to the fungus and their sensitivities to the toxin. The effect of nuclear genes on toxin-sensitivity is apparently indirect; i.e., the variation among genotypes in sensitivity to toxin may be caused by effects of these nuclear genes on aspects of metabolism which are only indirectly related to toxin-sensitivity. This is supported by the observation that sensitivity of genotypes to toxin was a function of the bioassay used; differences in toxin-sensitivity detected in vitro may be artifacts and have no biological significance. The products of the nuclear genes which can influence toxin-sensitivity under laboratory conditions play no apparent role in determining the outcome of the host-parasite encounter.

There is convincing evidence that the cytoplasmic factor(s) which conditions sensitivity of corn to HMT-toxin is the same factor(s) which causes increased susceptibility to *H. maydis* race T (3, 4, 5, 6, 10, 22, 25). However, there is no apparent interaction between the cytoplasmic factor for sensitivity to toxin and the nuclear genes that control reaction to the fungus. This is suggested by the observation of a close correlation between susceptibility of corn genotypes in T-cytoplasm

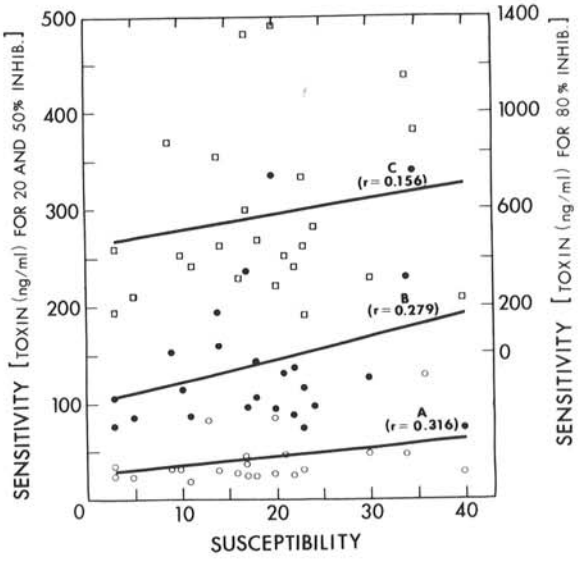

Fig. 2. Regression of sensitivity of corn genotypes to *Helminthosporium maydis* race T-toxin (as determined by the dark CO_2-fixation assay) on susceptibility of the same genotypes to *H. maydis* race T. Data for susceptibility (lesion area) and sensitivity [toxin concentration (ng/ml) required for 20% (line A), 50% (line B), and 80% (line C) inhibition] are given in Tables 1 and 3. None of the r values is significant ($P = 0.1$).

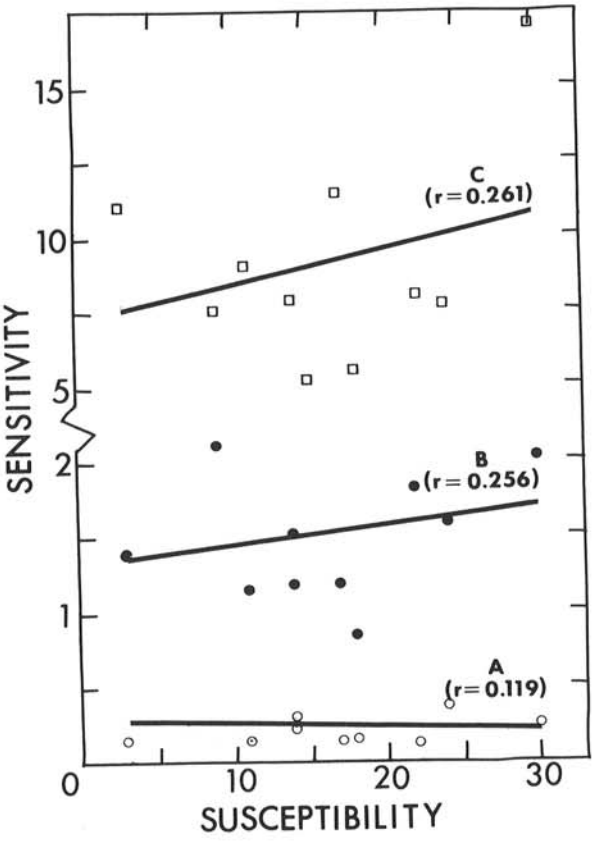

Fig. 1. Regression of sensitivity of corn genotypes to *Helminthosporium maydis* race T-toxin (as determined by the seedling root growth assay) on susceptibility of the same genotypes to *H. maydis* race T. Data for susceptibility (lesion area) and sensitivity [toxin concentration (μg/ml) required for 20% (line A), 50% (line B), and 80% (line C) inhibition] are given in Tables 1 and 2. None of the r values is significant ($P = 0.5$).

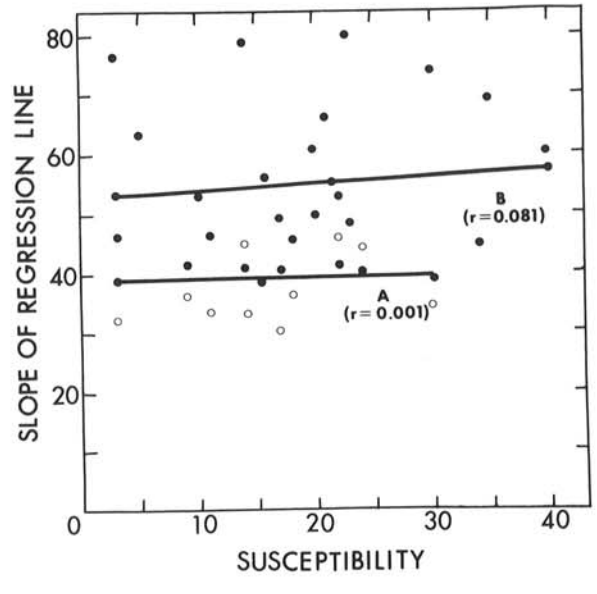

Fig. 3. Regression of slopes of toxin dosage-response curves for corn genotypes on susceptibility of the same genotypes to *Helminthosporium maydis* race T. Data for susceptibility (lesion area) are given in Table 1; data for slopes of dosage-response curves are given in Table 2 (for the seedling root growth assay, represented here by line A) and Table 3 (for the dark CO_2-fixation assay, represented here by line B). Neither of the r values is significant ($P = 0.5$).

to race T and their corresponding susceptibilities in N cytoplasm (14). The T-cytoplasm seems to confer a systematic, predictable increase in susceptibility of corn genotypes to race T. The apparent independence of the effects of nuclear genes and the cytoplasm on disease reaction indicates that the effects are additive, and implies that HMT-toxin interacts with cytoplasmic factors but not with a combination of factors coded for by both nuclear and cytoplasmic genes.

The foregoing observations support the suggestion that HMT-toxin is not involved in establishment of compatibility between *H. maydis* race T and susceptible corn genotypes, but that it does cause increased levels of disease if the corn contains cytoplasm which is sensitive to the toxin. The role of HMT-toxin in disease appears to differ from the roles of *H. victoriae* toxin, *P. circinata* toxin, *A. kikuchiana* toxin, and *H. carbonum* race 1 toxin, each of which is thought to condition compatibility between host and parasite.

The inability of HMT-toxin to distinguish accurately between susceptible and resistant corn genotypes in T-cytoplasm indicates that caution must be exercised if the toxin is used to screen for disease resistance in corn breeding programs. There is good evidence that the toxin accurately identifies cytoplasms susceptible to *H. maydis* race T (3, 4, 5, 6, 10, 22, 25). Nevertheless, since nuclear genes for susceptibility do not appear to condition sensitivity to toxin, a plant could be insensitive to toxin and yet highly susceptible to the fungus.

Inconsistencies among reports (2, 3, 18, 22, 23, 24, 25) on the effect of nuclear genes on sensitivity of corn to HMT-toxin may be due to differences in procedures used. In cases where no effect of nuclear genes was observed (3, 24, 25) the seedling root growth bioassay was used; its relative insensitivity to toxin does not permit accurate quantification (28), although it did show some small differences among genotypes in our experiments (Table 1). Previous indications that differences in sensitivity of corn genotypes to toxin are not correlated with sensitivity to toxin were based on observations of too few genotypes to be convincing (2, 18). One report (22) of a correlation between sensitivity of corn genotypes to toxin and susceptibility to the fungus is inconclusive because it is based on the use of only three genotypes, which were tested with a bioassay reported (28) to be highly insensitive to HMT-toxin. The other such report (23) concludes that nuclear genes for restoration of fertility condition both increased resistance to *H. maydis* race T and decreased sensitivity to HMT-toxin. However, the data in that report (23) show differences that were small (statistical analyses were insufficient) and inconsistent among corn genotypes in both reaction to the fungus and reaction of root growth and mitochondrial respiration to the toxin. To date, there is no well-documented evidence that the same nuclear genes condition reaction to both fungus and toxin.

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