Expression of Monogenic Chlorotic-Lesion Resistance to Helminthosporium maydis in Corn

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ABSTRACT

The recessive gene *rhm* conditions resistance in corn to Illinois isolates of *Helminthosporium maydis* races O and T. Its expression, in terms of lesion type, lesion size, and extent of sporulation, depended upon the level of resistance contributed by other genetic systems, plant cytoplasm, and race of *H. maydis*. Monogenic-resistant plants with normal or *cms*-T cytoplasm, infected with race O, formed small chlorotic lesions with reduced sporulation, in contrast to large necrotic lesions with abundant sporulation on susceptible plants. The increase in resistance to race O due to *rhm* was less in corn plants having other genetic systems for resistance, than in more susceptible plants. These effects of *rhm* against race O were consistent in both greenhouse- and field-grown plants. The expression of *rhm* against race T depended upon plant cytoplasm. Plants with normal cytoplasm exhibited chlorotic lesions. Fewer conidia of race T formed in necrotic lesions of normal cytoplasm plants than of race O. Seedling plants with *cms*-T cytoplasm homozygous for *rhm*, and infected with race T, expressed chlorotic-shot-hole-type lesions in the greenhouse. Gene *rhm* did not appear to affect sporulation of race T in lesions on plants having *cms*-T cytoplasm.

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The increased susceptibility to *Helminthosporium maydis* Nisikado and Miyake (*Cochliobolus heterostrophus* Drechs.), the cause of southern leaf blight of corn (*Zea mays* L.) having the *cms*-T (Texas) source of cytoplasmic male sterility was first reported in the Philippine Islands in 1961 (7). In 1969 this relationship was observed in the USA (9). In 1970 an epiphytotic of southern leaf blight occurred in the southern, central, and eastern corn-growing regions of the USA. This outbreak was caused by a previously undescribed race of *H. maydis* now designated as race T (5, 11). Of 30 sources of cytoplasmic male-sterile corn, 26 are resistant and four are susceptible to race T (12). Race O of *H. maydis*, which was prevalent before 1970, does not exhibit specialized

pathogenicity to different corn cytoplasms. The specificity of race T to *cms*-T corn is due to a cytoplasm-specific pathotoxin produced by the fungus, and is controlled by one gene in the fungus. The amount of pathotoxin produced is probably controlled by many genes (6).

Resistance to race O in corn is both oligogenic (2) and polygenic (4, 8). Oligogenic resistance to West African isolates of *H. maydis* was reported to be controlled by two linked recessive genes (2). This resistance has been termed chlorotic-lesion resistance (1). Resistant corn stocks with resistance coming from Nigerian introductions supplied by J. Craig have been developed in Illinois, and these are adapted to the Corn Belt. Genetic studies with these stocks have shown that resistance to an Illinois isolate of race O is inherited as a single recessive gene. The gene has been designated *rhm* (10).

This paper describes the effect of gene *rhm* in plants of different inbred backgrounds, and having either normal (nonsterile) or *cms*-T cytoplasm, on the expression of resistance to an Illinois isolate of *H. maydis* race O and of race T.

MATERIALS AND METHODS.—*Pathogen isolates.*—Two isolates of *H. maydis* were used. Race O came from bulk inoculum maintained and used in Illinois disease research plots in 1969 and prior years. Race T was obtained from infected *cms*-T corn leaf tissue in 1970 Illinois test plots inoculated with race T collected from central Illinois in 1969. Both isolates were highly pathogenic and representative of those described previously (11).

Corn stocks.—In experiment 1, backcross populations segregating both resistant and susceptible plants were used in studies of lesion types and sporulation within lesions. They were produced by crossing a resistant genetic stock homozygous for *rhm* (10) onto inbreds W64A, Mo940, W64A *cms*-T, and CI64 *cms*-T. The F_1's were then backcrossed to the genetic stock. This yielded backcross populations that had 75% of the germplasm contributed by the genetic stock and 25% contributed by the corn inbred lines.

In experiment 2, five inbred lines in normal cytoplasm and their *cms*-T counterparts were selected for a study of the effect of genetic systems that determine quantitative differences in lesion size and percentage of leaf tissue blighted on the expression of gene *rhm*. The lines, chosen according to their disease reaction in Illinois inoculated test plots in 1970, and listed from resistant to susceptible regardless of cytoplasm, and to both races of *H. maydis*, were Mo17, B37, B14A, N28, and Oh07 (D. R. Smith et al., *unpublished*). These inbred lines were also used to produce segregating backcross populations that had 75% of the germplasm contributed by the inbred line and 25% contributed by the genetic stock. Resistant and susceptible plants in these segregating backcross populations were also used in field studies with race O. For field studies with race T on plants with *cms*-T cytoplasm, however, backcross-derived plants homozygous for *rhm* were compared with the original inbred lines.

Inoculum.—Conidia of races O and T of *H. maydis* were obtained from infected corn leaves. Leaf pieces with lesions were placed under conditions of high humidity in petri plates for 72 to 96 hours to induce sporulation. The sporulating tissue was then macerated in water for 1 minute in a Waring Blendor and filtered through a 246-μm (60-mesh) screen. Spore concentration was then adjusted to approximately 1,000 conidia per milliliter.

Greenhouse inoculations and disease ratings.—For greenhouse inoculations with *H. maydis* race O, the conidial suspension was sprayed at 1 atmosphere pressure (15 psi) onto plants in the three-to-four-leaf stage until they were covered with fine droplets of suspension without runoff. Inoculated plants were kept in a mist chamber for 16 hours. Disease reactions were noted 7 days after inoculation, and confirmed at 10 days. The tenth day after inoculation, individual lesions from plants which exhibited distinct lesion types were excised and immediately placed on moist filter paper in petri plates for observation of conidial production.

The same plant populations were used for evaluation against race T. All race O lesions were first excised from the seedlings and the seedlings were then inoculated with race T at about the six-leaf stage. The same methods of inoculation and observation of disease reaction and conidial production were used as described for race O.

Field inoculations and disease ratings.—Inocula of races O and T were produced as for greenhouse inoculations. Conidial suspensions were sprayed onto plants in the seven-to-eight-leaf stage during a light rain. Disease reactions were noted 10 days after inoculation and confirmed at 14 days. Since differences in varying levels of resistance in corn inbreds is most evident under field conditions, lesion length in mm was measured on the fourteenth day following inoculation. After lesions had been measured, individual lesions were excised and immediately placed on moist filter paper in petri plates for observation of conidial production.

Sporulation in lesions.—In all greenhouse and field experiments six replications consisting of single lesions from each lesion type per plant genotype were observed for conidial production.

The harvested lesions in petri plates were kept at 24 ± 2 C in the dark. Lesions were observed for conidial production at 24-hour intervals to 168 hours for greenhouse- and to 120 hours for field-produced lesions.

To estimate the number of conidia per lesion, conidia were counted to a limit of 500 or calculated to a limit of 2,000 under $\times$ 80 magnification from the sporulating lesions. The latter was accomplished by counting conidia to a maximum of 500 in approximately one-fourth the area of a sporulating lesion and multiplying the result by four. Determinations of greater than 500 conidia per one-fourth lesion were not attempted due to the inability to visually differentiate with accuracy more conidia in the counting process. Using this method, the maximum number of conidia that could be differentiated per lesion was 2,000. Therefore, the total number of conidia per lesion type per genotype was obtained by adding together the conidial counts recorded from day to day through the termination of the experiments. This value should be proportional to the actual number of conidia produced. To measure the reliability of this method of estimating the number of conidia per lesion, a second method was used. Immediately after the last day of observing conidial production, each lesion was placed in 1 ml of water in a 5-ml vial. The vial was agitated one minute. Four 0.025-ml samples were placed on a microscope slide and the total

TABLE 1. Length of 14-day-old *Helminthosporium maydis* race O chlorotic and necrotic lesions on backcross corn populations in the field

Population[w]	Lesion type[x]	Lesion length (mm)
Mo17	Chl	1.0[y] a[z]
B37	Chl	1.0 a
N28	Chl	1.0 a
B14A	Chl	1.4 ab
Oh07	Chl	1.8 bc
Mo17	Nec	2.0 c
B37	Nec	3.0 d
N28	Nec	3.8 e
B14A	Nec	4.0 ef
Oh07	Nec	4.3 f

[w]Each population had 75% of the germplasm contributed by the corn inbred line and 25% contributed by the genetic stock.

[x]Lesion type: Chl = chlorotic lesion; Nec = necrotic lesion.

[y]Each value is the mean of 20 lesions.

[z]Means not followed by the same letter are significantly different, $P = 0.01$, as determined by Duncan's multiple range test.

TABLE 2. Length of 14-day-old *Helminthosporium maydis* race T lesions on *cms*-T corn populations having the genotype *rhm rhm* in comparison with their *Rhm Rhm* inbred counterparts in the field

Population[x] or inbred	Genotype	Lesion length (mm)
Mo17	*rhm rhm*	4.4[y] a[z]
Oh07	*rhm rhm*	4.8 ab
Mo17	*Rhm Rhm*	5.1 ab
B14A	*rhm rhm*	5.2 bc
B37	*rhm rhm*	5.4 bcd
B37	*Rhm Rhm*	6.0 de
Oh07	*Rhm Rhm*	6.2 def
B14A	*Rhm Rhm*	7.0 f

[x]Each population had 75% of the germplasm contributed by the corn inbred line and 25% contributed by the genetic stock.

[y]Each value is the mean of 20 lesions.

[z]Means not followed by the same letter are significantly different, $P = 0.01$, as determined by Duncan's multiple range test.

number of conidia per sample was counted. The total number of conidia obtained from both methods was then compared.

Statistical analysis.—Data collected on lesion length were analyzed as a completely randomized design. Data collected for conidial production were analyzed as a split plot in time. All tests of differences between treatment means were conducted using Duncan's multiple-range test (3). Since total conidial production was computed as average totals, $\sqrt{2bEa/r}$ was used as the standard error in tests of significant differences between treatment means from the split-plot-in-time analyses (13).

RESULTS.—*Symptoms, Race O inoculations.*—Two types of disease reactions were observed in all backcross populations inoculated with *H. maydis* race O. One, designated as a necrotic-lesion type, was characterized by elongate to oval lesions with a tan-to-brown necrotic center surrounded by little or no chlorosis. The other, designated as a chlorotic-lesion type, was characterized by small circular lesions with little necrosis surrounded by a chlorotic margin. All chlorotic lesions were significantly smaller than their necrotic-lesion counterparts (Table 1). The lengths of the chlorotic lesions of the Oh07 segregating population, however, were statistically no different than the necrotic lesions of the Mo17 population. There were significant differences in necrotic-lesion sizes among the backcross populations. These differences agree closely with the ranking of the inbreds from resistant to susceptible based on earlier field data.

Race T inoculations.—In all greenhouse inoculations with *H. maydis* race T, four types of disease reactions were observed. The two most resistant reaction types were from the normal cytoplasm corn populations segregating for *rhm*. (i) One was a chlorotic-lesion type as described from the race O inoculations. (ii) Other plants expressed lesions that were small and slightly oval in shape with a tan-to-brown necrotic center surrounded by a definite margin and/or slight chlorosis. These were typical of lesions observed when normal cytoplasm corn is infected with race T. The *cms*-T corn populations inoculated with race T also exhibited two distinct lesion types. (iii) The most resistant plants expressed lesions only slightly larger than the necrotic-lesion type as described for the race O inoculation. These lesions had a shot-hole-type appearance, but the epidermis was intact and the lesions were surrounded by much chlorosis. (iv) The more susceptible-appearing plants expressed lesions that were elongate, oval-shaped, and much larger than the necrotic lesions resulting from the race O inoculations. These lesions had a tan necrotic center with little or no chlorosis surrounding the necrosis. These necrotic lesions were typical of lesion types observed when *cms*-T corn is infected with race T.

In field inoculations with *H. maydis* race T no qualitative difference in lesion type was observed on *cms*-T corn plants whether or not they possessed *rhm*. No chlorotic-shot-hole lesion was observed on plants homozygous for *rhm*. In the *cms*-T corn inbreds the lesions were surrounded by much more chlorosis than was observed in the greenhouse. This lesion type had a necrotic center and was surrounded by chlorosis. The lesions from the homozygous *rhm* populations, however, were all smaller than their non-*rhm* inbred counterparts (Table 2). Only in the B14A and Oh07 comparisons were these differences statistically significant.

Sporulation.—The correlation of estimates of total conidia produced on individual lesions obtained by the two methods of determination was 0.91 for race O and 0.88 for race T. These correlations were significant, $P = 0.01$. Therefore, the determination of total conidia obtained by adding the number per lesion per 24-hour interval per genotype was used for analysis.

In all split-plot-in-time analyses there were significant F tests for whole-unit treatments, time, and treatment × time interactions, $P = 0.05$. The significant influence of time was in the increase of numbers of conidia with time. The treatment × time interaction was exemplified in the difference in the magnitude of increase of conidia with time among the distinct lesion types. The influence of treatments will be examined on an experiment basis.

In experiment 1, which was conducted in the greenhouse, none of the chlorotic lesions among backcross populations differed statistically in numbers of

TABLE 3. Number of *Helminthosporium maydis* conidia produced at 24-hour intervals on lesions from greenhouse inoculations of race O or T onto normal cytoplasm or *cms*-T corn populations segregating for gene *rhm* (Experiment 2)

Population[v]	Cytoplasm[w]	Race of *H. maydis*	Lesion[x] type	Conidia produced per incubation interval (hours): 0	0-24	24-48	48-72	72-96	96-120	120-144	144-168	Total estimated number of conidia
Mo17	T	O	Chl	0[y]	0	0	0	0	0	0	0	0 a[z]
N28	N	T	Chl	0	0	0	0	0	0	0	0	0 a
N28	T	O	Chl	0	0	0	0	0	0	0	0	0 a
Mo17	N	T	Nec	0	0	0	0	0	0	0	0.3	0.3 a
Mo17	N	T	Chl	0	0	0	0	0	0	0	14	14 a
Oh07	T	O	Chl	0	0	0	0	0	4	40	66	110 a
Oh07	N	T	Chl	0	0	0	0	0	14	34	72	120 a
B37	T	O	Chl	0	0	0	0	0	0	0	134	134 a
Mo17	N	O	Chl	0	0	0	0	0	0	60	113	173 a
B37	N	T	Chl	0	0	0	0	0	12	58	184	254 a
B37	N	T	Nec	0	0	0	0	5	88	143	278	514 a
B37	N	O	Chl	0	0	0	0	0	0	69	625	694 a
Mo17	T	O	Nec	0	0	0	0	0	19	320	667	1,006 ab
Oh07	N	O	Chl	0	0	0	0	0	49	297	740	1,086 ab
N28	N	O	Chl	0	0	0	0	3	92	334	770	1,199 ab
N28	N	T	Nec	0	0	0	12	161	705	1,492	1,733	4,103 bc
B37	N	O	Nec	0	0	1	17	45	525	2,000	2,000	4,588 c
N28	T	O	Nec	0	2	2	5	144	1,008	1,683	2,000	4,844 cd
Mo17	N	O	Nec	0	0	0	1	48	1,206	1,592	2,000	4,847 cd
Oh07	N	O	Nec	0	0	2	13	277	1,667	2,000	2,000	5,959 cde
B37	T	T	CSH	0	0	15	322	856	1,485	2,000	2,000	6,678 cdef
Oh07	N	T	Nec	0	2	6	175	921	1,767	1,883	1,984	6,738 def
Oh07	T	T	CSH	0	2	59	436	1,165	1,681	1,742	1,783	6,868 def
B37	T	O	Nec	0	93	346	513	660	1,850	2,000	2,000	7,462 def
Oh07	T	O	Nec	0	9	45	608	1,710	1,867	2,000	2,000	8,239 defg
N28	N	O	Nec	0	16	37	328	1,902	2,000	2,000	2,000	8,283 efg
Mo17	T	T	Nec	0	30	154	1,108	1,704	1,750	1,800	1,834	8,380 efg
Mo17	T	T	CSH	0	1	52	672	2,000	2,000	2,000	2,000	8,725 efg
N28	T	T	CSH	0	0	137	809	1,800	2,000	2,000	2,000	8,746 efg
B37	T	T	Nec	0	42	217	1,569	1,686	1,816	2,000	2,000	9,330 efg
Oh07	T	T	Nec	0	21	98	1,237	2,000	2,000	2,000	2,000	9,356 fg
N28	T	T	Nec	0	63	445	1,900	2,000	2,000	2,000	2,000	10,408 g

[v]Each population had 75% of the germplasm contributed by the corn inbred line and 25% contributed by the genetic stock.
[w]Cytoplasm: N = normal cytoplasm; T = *cms*-T cytoplasm.
[x]Lesion type: Chl = chlorotic lesion; CSH = chlorotic-shot-hole lesion; Nec = necrotic lesion.
[y]Each value is the mean of six replications.
[z]Means not followed by the same letter are significantly different, $P = 0.01$, as determined by Duncan's multiple range test.

TABLE 4. Number of *Helminthosporium maydis* conidia produced at 24-hour intervals on lesions from field inoculations of race O onto normal cytoplasm corn populations segregating for gene *rhm*

Population[w]	Lesion type[x]	Conidia produced per incubation interval (hours) 0	0-24	24-48	48-72	72-96	96-120	Total estimated number of conidia
B14A	Chl	0[y]	0	0	0	0	0	0 a[z]
Mo17	Chl	0	0	0	0	0	2	2 a
N28	Chl	0	0	0	0	0	4	4 a
B37	Chl	0	0	0	0	0	6	6 a
Oh07	Chl	0	0	0	0	4	32	36 a
Mo17	Nec	0	0	0	6	96	255	357 a
B37	Nec	0	0	0	28	220	523	771 ab
B14A	Nec	0	0	1	20	164	678	863 ab
Oh07	Nec	0	1	2	70	615	959	1,647 b
N28	Nec	0	0	1	36	490	1,450	1,977 b

[w]Each population had 75% of the germplasm contributed by the corn inbred line and 25% contributed by the genetic stock.
[x]Lesion type: Chl = chlorotic lesion; Nec = necrotic lesion.
[y]Each value is the mean of six replications.
[z]Means not followed by the same letter are significantly different, $P = 0.01$, as determined by Duncan's multiple range test.

conidia produced. The chlorotic-shot-hole lesions had fewer conidia than their necrotic-lesion counterparts. All differences in conidial production on chlorotic versus necrotic lesions resulting from race O inoculations were significant. In comparisons of chlorotic versus necrotic lesions of normal cytoplasm corn populations inoculated with race T, no significant difference in sporulation was observed. Only in the CI64 *cms*-T backcross population

TABLE 5. Number of *Helminthosporium maydis* conidia produced at 24-hour intervals on lesions from field inoculations of race T onto *cms*-T corn populations having the genotype *rhm rhm* in comparison with their *Rhm Rhm* inbred counterparts

Population[x] or inbred	Genotype	Conidia produced per incubation interval (hours)						Total estimated number of conidia
		0	0-24	24-48	48-72	72-96	96-120	
Mo17	*rhm rhm*	0[y]	31	60	309	882	1,783	3,065[z]
B14A	*rhm rhm*	0	22	111	351	1,196	1,650	3,330
Oh07	*rhm rhm*	0	1	62	297	1,817	2,000	4,177
B14A	*Rhm Rhm*	0	2	84	353	1,750	2,000	4,189
Mo17	*Rhm Rhm*	0	29	190	1,060	1,591	1,900	4,770
Oh07	*Rhm Rhm*	0	1	224	785	1,817	2,000	4,827
B37	*rhm rhm*	0	19	92	816	2,000	2,000	4,927
B37	*Rhm Rhm*	0	5	100	980	1,900	2,000	4,985

[x]Each population had 75% of the germplasm contributed by the corn inbred line and 25% contributed by the genetic stock.
[y]Each value is the mean of six replications.
[z]No significant differences were observed between means, $P = 0.01$ or 0.05, as determined by Duncan's multiple range test.

were significant differences in sporulation observed. In this population, there were significant differences in sporulation of chlorotic-shot-hole versus necrotic lesions. The necrotic lesions on *cms*-T corn inoculated with race T had the greatest number of conidia.

Sporulation on lesions from populations where the inbreds were chosen for varying levels of disease resistance to *H. maydis* in experiment 2 (Table 3) was similar to observations from experiment 1. None of the chlorotic lesions among backcross populations differed significantly in numbers of conidia produced. The chlorotic lesions always had fewer conidia than their necrotic-lesion counterparts. These differences were significant, except in the Mo17 and B37 populations of normal cytoplasm plants inoculated with race T. The necrotic lesions of the N28 and Oh07 normal cytoplasm populations had significantly more conidia than their chlorotic-lesion counterparts resulting from the race T inoculations. There was no significant difference in sporulation between chlorotic-shot-hole lesions when compared to their necrotic-lesion counterparts resulting from race T inoculation on *cms*-T corn populations. In general, the *cms*-T corn populations having necrotic lesions resulting from the race T inoculations had the greatest number of conidia.

Results from the inoculation of race O in the field agreed with greenhouse observations (Table 4). None of the chlorotic lesions among self-pollinated backcross populations differed statistically in sporulation. All chlorotic lesions had fewer conidia than their necrotic-lesion counterparts. These differences were significant within the more susceptible populations of Oh07 and N28.

From inoculations of race T in the field, the homozygous *rhm cms*-T corn populations had fewer conidia than their *cms*-T inbred counterparts (Table 5). However, none of these differences were significant.

DISCUSSION.—Monogenic chlorotic-lesion resistance in corn to *H. maydis* race O was expressed in the form of lesion type, lesion size, and reduced sporulation within lesions. These results are in agreement with those of Craig and Daniel-Kalio (1) working with the original source of resistance and an African isolate of *H. maydis*. Against race O, gene *rhm* was always expressed as a chlorotic-lesion form of resistance. This is to be expected, since race O does not exhibit specialized pathogenicity to different corn cytoplasms (5, 6, 11). Using infection rate, van der Plank (14) postulated that cultivars with both vertical and horizontal resistance would have less disease than cultivars that possess either of these types of resistance separately. Although the comparisons in this investigation are not isogenic, this pattern is apparent when lesion size and rate of sporulation are used to measure disease resistance. When *rhm* (vertical resistance) is present in corn populations with different levels of disease resistance (horizontal resistance), smaller lesions and lower sporulation rates are observed than when either vertical or horizontal resistance occurs alone. These results indicate that to develop corn plants with maximum resistance to race O, vertical and horizontal resistance should be combined.

Gene *rhm* can easily be incorporated into corn inbreds in a backcrossing program. It has been released in the form of a homozygous stock by the University of Illinois Agricultural Experiment Station and the United States Department of Agriculture (10).

The expression of chlorotic-lesion resistance against race T was less pronounced than that observed against race O. There were differences in lesion type in greenhouse experiments and in lesion size in field experiments, but no consistent differences in sporulation. Plant cytoplasm was an important factor in expression of resistance to race T. This is to be expected because of the specialized pathogenicity of race T. Following van der Plank's hypothesis, the results obtained from the race T inoculations followed a pattern similar to that observed against race O. In this case, an additional form of vertical resistance was superimposed upon the resistance in the nature of plant cytoplasm. The degree of expression of *rhm* in corn with normal cytoplasm to race T was influenced by the level of resistance contributed by other genetic systems. In normal-cytoplasm corn populations segregating for gene *rhm*, two distinct lesion types were observed. The differences in sporulation between these lesion types were significant for the more susceptible inbreds, N28 and Oh07, but were of a lesser magnitude than for race O inoculations, because normal cytoplasm alone has a high level of resistance to race T. In the *cms*-T populations segregating for *rhm*, differences of lesion size and sporulation rate were not consistent. Thus, plant cytoplasm conditions the strongest form of vertical resistance against race T. Gene *rhm* alone cannot overcome the susceptibility to race T conditioned by *cms*-T cytoplasm.

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