Predisposition to Diplodia Stalk Rot in Corn Affected by Three Helminthosporium Leaf Blights

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Portion of a Ph. D. thesis submitted to the University of Illinois at Urbana-Champaign.

Appreciation is extended to the U.S. Government for an A.I.D. fellowship, and to the Government of the Federal Republic of Nigeria for granting a study leave of absence.

Accepted for publication 17 June 1974.

ABSTRACT

Twenty-nine corn inbreds expressed a differential reaction to *Helminthosporium maydis* races T and O, *H. turcicum*, and *Diplodia zeae*. Total plant and ear-leaf infection was positively and highly associated for each leaf blight. The association of these with lesion size was lower, but significant.

Plants with severe infections of these leaf blights developed more stalk rot than plants with low infections. The blight reaction of the inbreds was more important than the type of leaf blight per se in determining the magnitude of stalk rot predisposition.

Phytopathology 64:1496-1499

Additional key words: male-sterile cytoplasm, gene *Ht*, northern leaf blight, southern leaf blight.

Of the several diseases of corn (*Zea mays* L.), leaf blights and stalk rots are perhaps the two most prevalent and destructive. The *Helminthosporium* leaf blights are widespread and important because of their ability to develop into large scale epiphytotics. Northern leaf blight, caused by *Helminthosporium turcicum* Pass. (*Trichometasphaeria turcica* Luttrell), is a major foliage disease of corn in many parts of the world. Prior to 1970, southern leaf blight, caused by *H. maydis* Nisikado and Miyake (*Cochliobolus heterostrophus* Drechs.), was of importance only in the warm humid climate of the tropics and subtropics. This was changed, however, by the well-known 1970 epiphytotic in the U.S. (5). The epiphytotic was due largely to the widespread distribution of race T of *H. maydis*. Another race, race O, is still of great importance in many parts of the world. Corn stalk rot is a complex disease, however, of the many fungal and bacterial organisms associated with it, *Diplodia zeae* (Schw.) Lév. has been perhaps the most widely studied in the U.S. corn belt. Diplodia stalk rot results in lodging and extensive losses in yield. Many factors are known to affect its severity (2).

This investigation was conducted to study the relative effects of the different leaf blights on predisposition of corn plants to stalk rot infection.

MATERIALS AND METHODS.—Twenty-nine corn inbred lines (Table 1) exhibiting a wide range of reaction to *H. maydis* races O and T, *H. turcicum*, and *D. zeae* were used. Several inbreds were in both normal (not male-sterile) and *cms*-T (Texas male-sterile) cytoplasm versions. Inbred WF9 was also represented in the *cms*-C and *cms*-S cytoplasms for male-sterility. Most of the inbreds were represented by their nearly isogenic counterparts containing gene *Ht* for resistance to *H. turcicum* (4).

The experimental field, in both 1971 and 1972, consisted of four separated blocks at the Agronomy South Farm, Urbana, Illinois. Plants in three blocks were inoculated with either *H. maydis* race O, *H. maydis* race T, or *H. turcicum*. The remaining block served as an uninoculated check. Each block contained 29 inbreds, in paired plots (rows), in a randomized complete block design with three replications. Plots were spaced approximately 90 cm apart and each contained 12 plants spaced about 30 cm apart. All plants in one of the paired plots were inoculated with *D. zeae*.

Four weeks after planting, the plants were artificially inoculated with leaf blight by placing in the leaf whorl a small quantity of pulverized leaf tissue infected with the appropriate pathogen and collected during the previous season. The inoculations were repeated 10 to 14 days later. Both primary and secondary infections occurred. Three to 4 wk after anthesis, individual stalks were inoculated with *D. zeae*. A water suspension of spores produced on cooked oat grain was injected into the first elongated internode above the ground.

Approximately 2 wk before mid-silk, at mid-silk, and again 2 wk after mid-silk, plants were rated on a total plant basis for percentage leaf area damaged by each leaf blight. For *H. maydis* race O and race T, lesion sizes on the ear-leaf (leaf subtending top ear) were recorded on a scale of 1 (lesions up to 3 mm in length) to 7 (lesions 21 mm or longer) with intermediate classes at 3-mm intervals. In 1972, additional ratings on the ear-leaf were taken in the form of percentage of leaf area infected by *H. maydis* race O and race T, and percentage leaf necrosis caused by *H. turcicum*. For all leaf-blight ratings a plot score was obtained by averaging three individual plant ratings.

Approximately 4 wk after inoculation, 10 individual bordered plants in each plot were rated for stalk rot reaction. The stalks were split longitudinally through the inoculated area and the degree of rot spread rated according to a 0.0 to 6.0 scale where 0.0 = no rot, 1.0 = one-fourth of an internode rotted, 2.0 = one-half of an internode rotted, 3.0 = three-fourths of an internode rotted, 4.0 = one full internode rotted, 5.0 = rot spreading into adjacent internodes, and 6.0 = plant prematurely killed. Plot scores were obtained by averaging the 10 individual plant ratings.

Analyses of variance were performed for each of the disease ratings. Arcsin transformation of percentage plant and ear-leaf infections was made before performing the analyses of variance. Fisher's least significant

difference (FLSD) values were calculated for making comparisons between means (1).

RESULTS AND DISCUSSION.—*Leaf blight infection.*—The 29 inbred lines differed significantly in reaction to *H. maydis* race O, *H. maydis* race T, and *H. turcicum* in 1972 (Table 1) when disease reaction is expressed as a percentage of total plant infection.

Total plant infection was highly correlated with ear-leaf infection and with lesion size in *H. maydis* race O (r = 0.94** and 0.80**, respectively) or similarly in race T (r = 0.96** and 0.84**, respectively) and with ear-leaf necrosis in *H. turcicum* (r = 0.86**). The correlation coefficients between ear-leaf infection and lesion size were also highly significant (r = 0.74** and 0.85** for race O and race T, respectively).

For each leaf blight, ratings among the three different dates for total plant infection, ear-leaf infection, or lesion size were highly correlated (r values ranged from 0.48** to 0.98**) (3). Thus, the same trend of disease reaction among inbred lines was established by rating at any of the three dates. The strongest association was found between the second and third rating dates.

Generally, inbreds expressed a differential reaction to the three leaf blights. A cytoplasm effect on disease reaction was seen with *H. maydis* race T but not with race O or with *H. turcicum* (Table 1). Inbreds that are nearly identical, except for the type of cytoplasm they contain, expressed a similar reaction to infection by *H. maydis* race O or by *H. turcicum*. The reaction of these inbreds to *H. maydis* race T, however, was principally governed by the type of cytoplasm each inbred contained. Inbreds with *cms*-T cytoplasm had a higher percentage of leaf area damaged by *H. maydis* race T than did their counterparts with normal cytoplasm. This difference was apparent

TABLE 1. Reaction of 29 corn inbred lines to *Helminthosporium maydis* race O, *H. maydis* race T, *H. turcicum*, and *Diplodia zeae* (stalk rot pathogen) and the effect of these leaf blight pathogens in predisposing plants to stalk rot

	Plant Infection[a] (%)			Stalk rot reaction			
					D. zeae with		
	H. maydis		*H.*	*D. zeae*	*H. maydis*		*H.*
Inbred line	Race O	Race T	*turcicum*	alone	Race O	Race T	*turcicum*
Hy2	21	12	59	2.8[b]	77[c]	109[c]	139[c]
Hy2 Ht[d]	14	13	43	2.6	90	111	158
Hy2 Ht *cms*-T[e]	25	57	44	2.1	104	159	190
Oh07A	41	18	50	2.8	87	81	97
Oh07 Ht	15	9	63	2.4	99	65	166
Oh07 Ht *cms*-T	22	81	62	2.6	132	190	156
WF9 Ht	64	19	50	1.3	212	189	226
WF9 Ht *cms*-C[f]	65	14	53	1.3	238	178	224
WF9 *cms*-S[g]	66	20	59	1.6	229	108	185
WF9 Ht *cms*-T	67	74	55	2.1	117	258	120
B14A	25	1	64	1.4	163	127	149
B14A Ht	20	2	45	1.0	231	107	123
B14A *cms*-T	24	70	63	1.9	82	93	190
N28	58	17	68	1.5	125	106	108
N28 *cms*-T	58	68	63	1.8	94	178	158
B37	6	2	59	2.2	129	65	160
B37 Ht	5	3	44	2.8	96	91	111
B37 *cms*-T	2	62	51	2.0	134	109	169
Oh43 Ht	4	1	37	1.8	176	136	232
Oh43 Ht *cms*-T	4	72	43	2.1	137	219	107
W64A Ht	70	21	47	2.2	125	71	175
W64A Ht *cms*-T	70	82	58	1.3	180	245	276
C103	2	1	3	0.9	92	89	139
C103 Ht	2	2	3	0.9	212	118	144
C103 *cms*-T	1	19	13	0.9	250	159	307
C123 Ht	13	11	10	1.1	157	295	170
C123 Ht *cms*-T	17	57	10	2.1	106	176	164
187-2	56	9	66	2.8	114	100	136
187-2 Ht	63	6	58	2.3	126	91	143
Mean	31	29	46	1.9	142	139	166
FLSD 0.05[h]	26.0	19.8	6.2	0.96	ns	160.5	120.3

[a]Ratings at mid-silk.
[b]Ratings on a scale of 0.0 = no rot to 6.0 = plant prematurely killed.
[c]Percentage of ratings in the plots inoculated with *D. zeae* alone.
[d]Inbreds contain gene *Ht* for resistance to *H. turcicum* and are nearly isogenic to their counterparts lacking this gene.
[e]Inbreds have *cms*-T cytoplasm for male-sterility and are susceptible to *H. maydis* race T.
[f]Inbred has *cms*-C cytoplasm for male-sterility.
[g]Inbred has *cms*-S cytoplasm for male-sterility.
[h]FLSD = Fisher's Least Significant Difference.

throughout the rating period. Inbred WF9 with *cms*-C or *cms*-S cytoplasm for male sterility did not differ significantly from inbred WF9 Ht with normal cytoplasm but all three inbreds had less plant tissue blighted by *H. maydis* race T than did inbred WF9 Ht with *cms*-T cytoplasm.

Gene *Ht* affected the amount of leaf tissue infected by one of the leaf blight pathogens. Most inbred lines containing gene *Ht* had less infection by *H. turcicum* than their counterparts without this gene. However, gene *Ht* did not affect plant reaction to either *H. maydis* race O or race T. The difference in total plant infection between an inbred with *Ht* and its counterpart became more obvious as the season progressed.

Some of the differences among inbred lines in disease reaction were attributable to factors other than the cytoplasm and the gene *Ht*. The inbreds differed genetically and quantitatively in reaction to each leaf blight pathogen. Inbred C103 was damaged less by *H. turcicum* than any other inbred. Inbreds B14A and 187-2, on the other hand, were severely blighted by *H. turcicum*. Inbreds all having gene *Ht* also differed in their reaction to *H. turcicum*. A wide range was expressed among inbreds in reaction to *H. maydis* race O. Inbreds having *cms*-T cytoplasm also differed among each other in their reaction to *H. maydis* race T at each rating date. Inbreds C103 *cms*-T and C123 Ht *cms*-T, which were generally resistant to *H. maydis* race O, also showed less damage by *H. maydis* race T than did inbreds WF9 Ht *cms*-T and W64A Ht *cms*-T which were both very susceptible to *H. maydis* race O.

Disease ratings differed at the three dates (3). This reflected an overall increase in disease development as the season progressed. Disease increase was usually greater during the period 2 wk before mid-silk to mid-silk than during the period between mid-silk and two weeks after mid-silk for *H. maydis* race O and for *H. turcicum,* but there was little or no difference between these two periods in the development of *H. maydis* race T infection. Also, as the season progressed, the inbreds differed in the rate at which they became more severely blighted. For example, for *H. maydis* race O inbreds B14A, N28, and W64A Ht showed a greater blight increase during the period between the first and second than between the second and third ratings, whereas blight scores for either B37 or C103 did not differ significantly at the three rating dates. The rate of disease development for *H. maydis* race T was fairly uniform over the 4-wk period. There were highly significant differences among inbreds with or without the *Ht* gene in the rate of development of the northern leaf blight.

Ratings for percentage ear-leaf infection were mostly in agreement with the trend in rating for percentage total plant infection. Inbreds differed within each rating date for each disease. For *H. maydis* race O infection, there was no difference within each rating date between cytoplasm types of the same inbred, whereas inbreds containing *cms*-T cytoplasm consistently showed higher amounts of ear-leaf infection when infected with *H. maydis* race T than did their counterparts with normal or other cytoplasms for male sterility. Inbreds with or without *Ht* differed among each other in amount of ear-leaf infection by *H. turcicum*. Inbreds carrying gene *Ht* had less ear-leaf necrosis when infected with *H. turcicum* than their counterparts with the *ht* allele. There was no effect of cytoplasm on the percentage of ear-leaf that became necrotic due to *H. turcicum* infections.

There were differences in lesion size among inbreds infected with *H. maydis* race O or with race T (3). Inbreds B37 and C103 which showed a low percentage of total plant infection by *H. maydis* race O also reacted to race T with smaller lesions than inbreds such as WF9 Ht, N28, and W64A Ht, which showed a high percentage of leaf area infected. As was true for total plant infection, there was no significant difference in lesion size between individual inbreds with different cytoplasms in their reaction to *H. maydis* race O. On the contrary, lesions were larger in inbreds having *cms*--T than their counterparts having other cytoplasms when infected with *H. maydis* race T. Inbreds having *cms*-T cytoplasm also differed in lesion size: lesions on inbred Oh43 Ht *cms*-T were significantly larger than those on any other line with *cms*-T cytoplasm. Also, C103 *cms*-T which reacted with less leaf area damage showed the smallest lesion of any inbred in *cms*-T cytoplasm. There was a greater tendency for inbreds with larger lesions to differ significantly between rating dates than those with smaller lesions. Gene *Ht* did not affect the lesion size of inbreds infected by either *H. maydis* race O or race T.

The same type of data for leaf infection were obtained in 1971 (3).

Stalk rot reaction in check plots.—There were highly significant differences among inbred lines in their reaction to stalk rot in the check plots where plants were not inoculated with any leaf blight pathogen in both 1971 (3) and 1972 (Table 1). Inbreds B14A, N28, C103 and C123 Ht were similar in stalk rot reaction but had significantly lower stalk rot scores than inbreds Hy2, Oh07A, and 187-2. Generally, neither the type of cytoplasm nor the presence of gene *Ht* affected the stalk rot reaction in the absence of leaf blight infection.

Stalk rot reaction of plants with prior leaf blight infection.—When stalk rots are expressed as percentage of rot scores in the check plots of the same inbred, the lines differed in their reaction to stalk rot when previously infected with each leaf blight pathogen (Table 1). Similar data on stalk rot were obtained in 1971 and 1972 (3). These differences were, however, not significant among inbreds infected with *H. maydis* race O. Stalk rot was generally more severe in corn infected with leaf blight than when not infected.

Most inbreds with *cms*-T cytoplasm infected with *H. maydis* race T had more stalk rot than their counterparts with other cytoplasm types (Table 1). Based on a 2-yr mean (3), inbreds with *cms*-T cytoplasm were 16.23% more predisposed to stalk rot by *H. maydis* race T infection than were their counterparts in normal cytoplasm.

The effect of gene *Ht* in predisposition to stalk rot by northern leaf blight depends on the residual genotype of the specific inbred line. Thus, gene *Ht* reduced the extent of predisposition in B14A and B37 compared with their *ht* counterparts, but it had no such effect in Hy2, Oh07A, and C103. However, based on a 2-yr mean (3), there was 8.11% more stalk rot infection in inbreds without *Ht* than in their counterparts with this gene.

Generally, *H. turcicum* was more important in increasing stalk rot severity than either *H. maydis* race O

or race T. But the reaction of an inbred to a particular leaf blight was more important than the type of leaf blight per se in determining the extent of the rot increase. The apparent differences among inbreds in the extent of predisposition probably indicates that factors other than leaf blight reaction may also be important. Perhaps there is an interplay between genetic factors for stalk rot and leaf blight reactions and/or that corn strains have inherently different mechanisms of withstanding stress.

The physiological state of the plant is among the many and diverse factors that influence the development of corn stalk rots. The pith condition and the level of soluble solids content, mostly sucrose, of the stalk have been related to stalk rot infection (6). The higher stalk rot scores in plants with a greater leaf area damaged by a leaf-blight pathogen supports the hypothesis that the low photosynthate resulting from a reduction in functional leaf area by leaf blight subsequently leaves the plants with fewer metabolites. This low level of requisite metabolites reduces the physiological activity of the plant, thereby hastening senescence. Thus, the spread of *D. zeae,* a facultative saprophyte, is uninhibited in the senescent or dead stalk tissue. The trends in sugar content are negatively correlated with stalk rot reaction. Also, Wysong and Hooker (6) found that clipping corn leaves reduced the soluble solids content of the plant. It is probable that the pathotoxin produced by *H. maydis* race T also hastens stalk senescence and, thus, predisposes plants with *cms*-T cytoplasm to increased stalk rot severity. This needs further investigation.

Agricultural crops are frequently subject to several diseases simultaneously. Stalk rot susceptibility was increased as a consequence of leaf blight infection. This may be viewed as another example of predisposition to disease caused by a stress factor. In this instance, the stress is another disease. Since the effects of these diseases were cumulative, resistance to several diseases must be integrated into the same plant in any effective crop improvement program.

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