A New Gene in Maize Conferring the "Chlorotic Halo" Reaction to Infection by *Exserohilum turcicum*

M. L. Carson, Research Plant Pathologist, USDA-ARS and Associate Professor, Department of Plant Pathology, North Carolina State University, Raleigh 27695-7616

ABSTRACT

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During recurrent selection in the maize synthetic BS19, S_1 lines exhibiting a unique lesion phenotype in response to infection with *Exserohilum turcicum* were observed. This phenotype, dubbed "chlorotic halo," initially appears as dark orange-brown pigmented infection points that are later surrounded by a circular chlorotic halo about 1 cm in diameter. Most infection points retain this phenotype, although some later develop into the elongated, elliptical necrotic lesions typical of northern leaf blight (NLB). An inbred line derived from this initial selection was crossed to the susceptible inbred line A619 and the inheritance of the chlorotic halo reaction studied in advanced generations from this cross. Segregation ratios were consistent with the hypothesis that the chlorotic halo phenotype is controlled by a single recessive gene. This gene appears to be located on the short arm of chromosome 1 near the centromere, based upon reciprocal translocation mapping studies. The chlorotic halo reaction was expressed against races 0,1,23, and 23N of *E. turcicum* in the field. The effectiveness of the chlorotic halo gene in reducing losses to NLB remains to be demonstrated.

Additional keywords: corn, *Setosphaeria turcica*, *Zea mays*

Northern leaf blight (NLB), caused by the fungus *Exserohilum turcicum* (Pass.) K. J. Leonard & E. G. Suggs (teleomorph = *Setosphaeria turcica* (Luttrell) K. J. Leonard & E. G. Suggs; syn. = *Helminthosporium turcicum* Pass.), is a damaging disease of maize (*Zea mays* L.). It occurs worldwide virtually everywhere maize is grown (8,23,27). NLB is most prevalent and damaging when cool to moderate temperatures and moist conditions prevail during the growing season, particularly during the plant's grain-filling period (8,23,27). Extensive defoliation during this period can result in grain yield losses of 50% or more (5,19,20,32). Resistance in maize to NLB is generally classified as one of two types: major gene resistance conferred by the *Ht1, Ht2, Ht3,* or *HtN* genes that is race-specific (6,7,9–12,16, 26,28,30,33,34) and partial resistance that is under polygenic control and is effective against all pathogen biotypes (13,14,16, 31). The *Ht1, Ht2* and *Ht3* genes confer a "chlorotic lesion" type of reaction to the pathogen (7,9–12). *Ht1* was initially found in the inbred line GE440 and Ladyfinger popcorn, and was subsequently found to be present in a wide array of maize germ plasm (7,11,12). Hybrids carrying *Ht1* were used extensively during the late 1960s and 1970s until a race of *E. turcicum* virulent upon *Ht1* genotypes was discovered in Hawaii and later in the continental U.S. (1,30). *Ht2* and *Ht3* were isolated from the Australian maize inbred line NN14B and *Tripsacum floridanum*, respectively, but are probably little utilized commercially (9,10). *HtN* was initially found in the Mexican cultivar Pepitilla and successfully transferred into adapted U.S. germ plasm (6). *HtN* causes a delay of symptoms until well after anthesis (6,22). Virulence to each of these genes has been demonstrated in *E. turcicum* in the U.S. and elsewhere (1,16,17,26,28,30,33,34). Other simply inherited forms of resistance to *E. turcicum* have been found, but they are identical to, allelic to, or tightly linked to *Ht1* (11,12), their inheritance is not clear cut (25), or they are no longer extant in public germ plasm collections. Emphasis in commercial maize breeding programs in the U.S. has been on exploiting partial resistance to NLB.

During the process of recurrent selection to improve the disease resistance of the maize synthetic BS19 (3), several distinct types of resistance phenotypes were observed segregating among S1 lines from the population that had undergone two cycles of selection for NLB and Diplodia stalk rot resistance (2). One phenotype, dubbed "chlorotic halo," was fixed in an S_4 inbred line. It is characterized by infection points that develop a distinct dark orange-brown pigment and that later become surrounded by a circular chlorotic halo ca. 1 cm in diameter (Fig. 1). Most NLB infection points in this genetic stock retain the chlorotic halo phenotype until plant senescence, but some develop into typical, elongated, necrotic NLB lesions.

The objective of this research was to determine the inheritance of the chlorotic halo phenotype and to identify the chromosomal location of the gene(s) responsible for the trait in maize.

MATERIALS AND METHODS

An experimental S_4 maize inbred line, 357, was produced by successive generations of selfing and selection for uniformity of reaction to NLB from the original S_1 displaying the chlorotic halo phenotype. Line 357 was crossed to the highly NLB-susceptible inbred line A619. The F_2 and backcrosses were produced by selfing or crossing the F_1 to both parents, respectively. BCS_1 lines were produced by selfing individual plants in the two backcross populations.

Seeds of the parental inbred lines, the F_1, F_2, and backcross generations were planted in field trials in the summers of 1992, 1993, and 1994. BCS_1 lines were evaluated in the summer of 1994 only. The 1992 and 1993 trials were planted on 17 April and 15 April, respectively, on the Genetics Gardens plots located near Raleigh, N.C. Trials consisted of randomized complete blocks with four replications of the parental lines, F_1, F_2, and backcross generations. Forty seeds were hand planted

Fig. 1. "Chlorotic halo" symptoms observed on the maize inbred line 357 in response to infection by *Exserohilum turcicum*.

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in rows 6 m long and 0.9 m apart. A replication consisted of a single row each of the parental lines and F_1, two rows each of the backcrosses, and three rows of the F_2. Plots were not thinned and final stands were variable. The 1994 field trial was planted on 22 April at the Central Crops Research Station, Clayton, N.C., and was not replicated. This trial consisted of a single row each of the parental lines and F_1, ten rows of the F_2, eight rows each of the backcross generations, and a single row for each of 22 and 21 BCS_1 lines of the backcrosses to line 357 and A619, respectively. The planting density and row spacings were identical to the other trials.

To determine the chromosome arm on which the gene(s) responsible for the chlorotic halo phenotype of line 357 were located, line 357 was crossed with a series of reciprocal translocation stocks marked with the recessive waxy endosperm marker gene, *wx,* (obtained courtesy of E. A. Patterson, Maize Genetics Cooperative, Urbana, Ill.). Each stock carries a reciprocal translocation between chromosome 9 (site of the *wx* gene) and a portion of another chromosome arm. Because crossing-over is greatly reduced near translocation breakpoints, the *wx* gene serves as a marker for the translocated chromosomes. This set of reciprocal translocations can potentially reveal linkages with greater than 90% of the maize genome. The resulting translocation X line 357 F_1s were self pollinated and the resulting F_2 seed sorted for endosperm phenotype (waxy versus normal). Seeds of the translocation F_2s were planted in the same field and in the same manner as the 1994 genetic study above. Five rows each of waxy and normal endosperm seed from each translocation F2 were planted in this 1994 field trial, with the exception of the T8-$9_{6921}F_2$ population, of which only three rows each of the waxy and normal endosperm seed were sown due to a shortage of seed. Six rows of the backcross of the F_1 of the translocation T8-9_{6921} to line 357 were also sown.

All plants in the genetic trials were inoculated at the four- to six-leaf stage by placing 20 to 30 grains of a sorghum grain inoculum in the leaf whorl. The sorghum grain inoculum was produced by inoculating flasks of autoclaved, moistened sorghum seed with conidial suspensions prepared by washing conidia from 10-day-old lactose caseinate agar cultures of *E. turcicum* (isolate Et10; race 0). The inoculated sorghum cultures were grown for 2 weeks at room temperature and then stored at 4°C until used.

Plants in all experiments were rated for reaction type 1 to 2 weeks after anthesis when expression of the chlorotic halo phenotype in line 357 was most apparent. Plants were classified as chlorotic halo resistant if the phenotype of NLB infection points resembled that on line 357; all other plants were classified as susceptible. The fit of observed segregation ratios to expected ratios in the F_2 and backcrosses to line 357 were tested based on either a single recessive gene or an epistatic dominant-recessive two-gene interaction conditioning the chlorotic halo phenotype. BCS_1 families were classified as segregating, homozygous chlorotic halo, or homozygous susceptible, and the two models above were tested for goodness of fit to observed ratios.

Plants in the reciprocal translocation mapping experiment were classified as previously described. To determine if an association existed between the chromosome arm(s) involved in the reciprocal translocation and the chlorotic halo character, two tests were made for each translocation F_2. First, a chi-square test of homogeneity was conducted to determine if segregation ratios for the chlorotic halo trait were homogeneous in the waxy and the normal F_2s. A significant reduction in the frequency of chlorotic halo plants in the waxy versus normal endosperm plants indicates that one or both of the chromosome arms involved in the translocation carry gene(s) involved in the chlorotic halo reaction. Secondly, a chi-square test of homogeneity of segregation ratios for the chlorotic halo reaction among fully fertile (homozygous normal chromosomes) and semisterile (translocation heterozygotes) plants was conducted within translocation F_2s from normal endosperm seed. The fertility of plants was determined by partially husking ears and examining kernel set. Fertile plants had few or no missing kernels whereas semisterile plants had ca. 50% of normal kernel numbers. A few plants were either barren (no kernels) or had such poor kernel set that classification was not possible and were omitted from the analysis. A significantly higher frequency of chlorotic halo plants in the fully fertile versus semisterile plants indicates that the chromosome arm involved in the reciprocal translocation with the *wx* marked chromosome 9 carries a gene(s) for the chlorotic halo reaction.

The chlorotic halo source inbred line 357 and a set of differential inbred lines carrying the *Ht1, Ht2, Ht3,* and *HtN* gene were inoculated in a 1994 field trial with *E. turcicum* races 0, 1, 23, and 23N. The trial consisted of four sets of single-row plots of the differentials and line 357, with each set being inoculated with a single race. Details of planting, row size and spacing, and inoculation were as described for the other 1994 field trials. Plots were rated for NLB reaction at anthesis.

Table 1. Test of genetic ratios in crosses of the maize inbred 357 ('chlorotic halo' source) and A619. Data are from field experiments conducted at Raleigh, N.C., (1992 and 1993) or Clayton, N.C. (1994)

Population	Year	Expected ratio (R:S)	Observed ratio (R:S)	Chi-square
357	1992	R	98:0	
	1993	R	89:0	
	1994	R	38:0	
A619	1992	S	0:70	
	1993	S	0:73	
	1994	S	0:33	
F1	1992	S	0:96	
	1993	S	0:97	
	1994	S	0:40	
F2	1992	1:3	57:187	0.35
		3:13		3.24
	1993	1:3	59:258	6.90**[a]
		3:13		0.00
	1994	1:3	75:260	1.29
		3:13		2.82
(357XA619)	1992	1:1	112:103	0.38
X357	1993	1:1	99:107	0.40
	1994	1:1	158:144	0.65
(357XA619)	1992	all S	0:201	
XA619	1993	all S	0:182	
	1994	all S	0:295	
BCR self[b]	1994	1seg.:1allR	14:8	1.64
		3seg.:1allR		1.52
BCS self[b]	1994	1seg.:1allS	8:13	1.19
		1seg.:3allS		1.92

[a] ** indicates chi-square value is significant at the 0.01 level.

[b] BCR self and BCS self indicate S_1 progenies derived from the (357 × A619) × 357 and (357 × A619) × A619 populations, respectively.

RESULTS

The chlorotic halo source, line 357, consistently expressed the chlorotic halo reaction in all three years (Table 1), indicating the inbred was homozygous and uniform in expression of the trait. The chlorotic halo phenotype was consistently expressed in response to inoculation with *E. turcicum* and was not found on non-inoculated plants of line 357 except late in the season when secondary inoculum was abundant in the nursery. These observa-

tions and the observation that these chlorotic halos develop directly from NLB infection sites indicate that this is a reaction to infection by *E. turcicum* and not a genetic leafspot or lesion mimic. Plants of line 357 also developed normal symptoms of southern leaf blight (*Cochliobolus heterostrophus*), common rust (*Puccinia sorghi*), and southern rust (*P. polysora*) indicating the chlorotic halo symptom is specific to *E. turcicum*.

Segregation ratios in the F_2 were consistent with both the single recessive gene (1:3) or dominant-recessive two-gene epistatic models (3:13) with the exception of the 1993 test where an excess of susceptible plants resulted in a significant deviation from the 1:3 expected ratio, but a perfect fit to the 3:13 ratio (Table 1). Classification of plants in 1993 was complicated by drought stress during the classification period. Leaves were often rolled and exhibited varying degrees of "leaf firing" that made classification difficult. Segregation ratios in the backcross to line 357 were consistent with a 1:1 ratio in all years; this expected ratio is the same for both genetic models. Segregation of BCS_1 families was also consistent with the ratios expected for both genetic models.

Tests of independence of segregation ratios among plants derived from waxy versus normal endosperm seed in reciprocal translocation F_2 populations resulted in significant deviations in seven of the 24 F_2s (Table 2), implicating portions of chromosomes 1, 3, 4, 9 and 10. When the independence of segregation ratios among fertile versus semisterile plants derived from normal endosperm F_2 seed was tested, only two translocation F_2s involving portions of chromosome 1 exhibited significant heterogeneity (Table 2).

The chlorotic halo reaction was expressed against all four races of *E. turcicum* with which it was tested (Table 3). Races 0 and 1 gave predictable reactions on the differentials, but races 23 and 23N did not exhibit virulence in accordance with their virulence formulae (16).

DISCUSSION

Chlorotic halo appears to be a unique reaction in maize to infection by *Exserohilum turcicum*. Not only does this phenotype differ from the chlorotic lesion phenotype typical of that caused by the dominant genes *Ht1, Ht2,* and *Ht3* and the delay in symptom expression caused by *HtN*, but it also differs in its apparent inheritance. Notwithstanding the significant deviation observed 1 year in the F_2, the simplest explanation of the data is that the chlorotic halo reaction is controlled by a single recessive gene in line 357. This does not mean that the expression of chlorotic halo may not be modified by other genes in the maize genome. The expression of *Ht1, Ht2,* and *Ht3* is also influenced by the level of polygenic, partial resistance in the plant (18,21). It is also not known to what extent environment may affect the expression of this trait, since these experiments were confined to the hot, humid summers typical of the southeastern U.S. The influence of light and temperature on the expression of virulence to *Ht2* and *Ht3* is well documented (4,15,29). The chlorotic halo trait needs to be evaluated in a variety of genetic backgrounds and in a variety of climates.

The single recessive gene controlling the chlorotic halo reaction appears to be located on chromosome 1 of maize, most probably on the short arm near the centromere. The significant heterogeneity in segregation ratios between plants derived from waxy versus normal endosperm seed in several translocation F_2s other than those involving chromosome 1 could be an indication of modifying genes affecting expression of the chlorotic halo phenotype. One possible explanation could be the presence on chromosome 9 of a modifier gene loosely linked to the *wx* locus. In all but two of the translocation F_2s, the frequency of chlorotic halo plants was lower in plants derived from waxy endosperm seed than in those from normal seed, and in seven of these F_2s this deviation was significant. Although the segre-

Table 2. Chi square values for tests of independence of ratios of chlorotic halo resistant and susceptible reactions in seeds grown from normal vs. waxy endosperm seed in reciprocal translocation × 357 F_2 populations; and semi-sterile vs. fertile plants grown from normal endosperm seed in those same populations

	Chi-square	
Reciprocal translocation and location of breakpoints	**Normal vs. waxy**	**Semi-sterile vs. fertile**
T1-9c (1S.48, 9L.22)	22.54**[a]	13.68**
T1-9_{5622} (1L.10, 9L.12)	13.92**	14.24**
T1-9_{8389} (1L.74, 9L.13)	0.07	0.00
T2-9c (2S.49, 9S.33)	0.04	0.49
T2-9b (2S.18, 9L.22)	0.30	0.00
T2-9d (2L.83, 9L.27)	1.10	0.08
T3-9_{8447} (3S.44, 9L.14)	12.69**	0.14
T3-9_{8562} (3L.65, 9L.22)	12.50**	0.52
T4-9e (4S.53, 99L.26)	0.16	0.17
T4-9b (4L.90, 9L.29)	6.45*	0.00
T4-9_{5657} (4L.33, 9S.25)	2.77	0.17
T5-9_{4871} (5L.71, 9S.38)	2.09	0.65
T5-9a (5L.69, 9S.17)	0.16	0.12
T6-9_{4778} (6S.80, 9L.30)	2.82	0.00
T6-9b (6L.10, 9S.37)	3.49	0.01
T7-9_{4363} (7cent., 9cent.)	0.55	0.31
T7-9a (7L.63, 99S.07)	3.75	0.01
T8-9d (8L.09, 9S.33)	0.21	3.73
T8-9_{6673} (8L.35, 9S.31)	0.32	0.01
T8-9_{6921} (8L.85, 9L.15)	1.18	1.90[b]
INV9a (9S.70, 9L.90)[c]	5.12*	0.01
T9-10_{8630} (9S.28, 10L.37)	4.79*	0.16
T9-$10_{059\text{-}10}$ (9S.31, 10L.53)	3.65	0.00
T9-10b (9S.13, 10S.40)	5.30*	0.84

[a] *, ** indicate chi-square value is significant at the 0.05 and 0.01 level, respectively.
[b] The difference in segregation ratios between semi-sterile and fertile plants was measured in the test-cross [(translocation × 357) × 357] population, not the F_2.
[c] The waxy-marked pericentric inversion stock was used instead of a reciprocal translocation.

Table 3. Reactions of differential maize inbred lines and the chlorotic halo source inbred 357 to infection by four races of *Exserohilum turcicum*. Data are from a 1994 field trial at Clayton, NC

	Race			
Differential maize inbred	**0**	**1**	**23**	**23N**
A632	S	S	S	S
B37	S	S	S	S
A619Ht1	R[a]	S	R	R
A619Ht2	R	R	R	R
A619Ht3	R	R	R	R
B37Ht1	R	S	R	R
B37Ht2	R	R	R	R
B37Ht3	R	R	R	R
B68HtN	R[b]	R	R	R
A632HtN	R	R	R	R
Line 357	R[c]	R	R	R

[a] Resistant reaction in *Ht1*, *Ht2*, and *Ht3* differentials is expressed as a chlorotic lesion reaction.
[b] Resistant reaction in *HtN* differentials is expressed as a fleck reaction that only becomes a necrotic, susceptible-type lesion well after anthesis.
[c] Resistant in reaction in line 357 is expressed as a 'chlorotic halo' reaction as in Figure 1.

gation of the chlorotic halo reaction was sometimes not independent of the *wx* marker gene in reciprocal translocation F_2s involving chromosomes other than chromosome 1, segregation was not independent of semisterility in only two translocation F_2s, both of which have translocation breakpoints within ca. 40 cM of each other on chromosome 1. Because semisterility is a direct marker of plants heterozygous for the translocation, using it rather than the linked *wx* locus phenotype as a marker is probably a more reliable means of classifying plants. This proposed chromosomal location of the gene controlling the chlorotic halo reaction differs from the location of other major genes for resistance to *E. turcicum*. *Ht1* is located on the long arm of chromosome 2, *Ht2* and *HtN* are located on the long arm of chromosome 8, and *Ht3* has not been mapped although it is apparently unlinked to the other *Ht* loci (24,35).

In addition to its unique expression, recessive inheritance, and probable chromosomal location, the chlorotic halo reaction was expressed against all North American races of *E. turcicum* in a field trial. However, differential inbred lines carrying the *Ht2, Ht3,* and *HtN* also expressed resistance to all races, including races 23 and 23N, under the hot, humid conditions prevalent in this trial. It is conceivable that virulence to the chlorotic halo reaction may also not be expressed under conditions of high temperatures and light as has been reported for *Ht2, Ht3,* and *HtN* (4,15, 28,29).

Throughout this paper, chlorotic halo has deliberately been referred to as a disease reaction and not a disease resistance character. It remains to be conclusively demonstrated that this trait will actually reduce NLB development and that the low level of NLB observed on line 357 is due to the chlorotic halo character and not a high background level of partial resistance. Because some normal susceptible necrotic lesions are produced on line 357, it is conceivable that it could sustain significant damage during a severe NLB epidemic. Efforts to backcross chlorotic halo into a range of genetic backgrounds have been initiated so the effect of chlorotic halo in near-isogenic backgrounds may be evaluated. Even if it does confer adequate resistance to all races of *E. turcicum*, the chlorotic halo trait may have limited commercial value. Its recessive inheritance means that both parental inbred lines would need to carry the gene for the hybrid to be resistant. Further, backcross breeding needed to transfer the trait is considered conservative; in the time needed to backcross the trait into an inbred line, the commercial life of that line may have expired. Also, most commercial breeding programs have abandoned the widespread use of the other *Ht* genes due to the appearance of new virulences in the *E. turcicum* population. Perhaps the best use of the chlorotic halo trait would be in combination with other *Ht* genes as well as genes for partial resistance in a gene-pyramiding scheme involving a synthetic source population from which to derive inbred lines with high levels of resistance to all races of *E. turcicum*.

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