Frequency of the *Ht1* Gene in Populations of Sweet Corn Selected for Resistance to *Exserohilum turcicum* Race 1

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ABSTRACT

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The possibility that the *Ht1* gene or genes tightly linked to *Ht1* convey general resistance to races of *Exserohilum turcicum* that are virulent against *Ht1* (i.e., residual resistance) could be useful in sweet corn where the *Ht1* gene is present in many commercial hybrids and breeding populations. The objective of this study was to determine if the frequency of the *Ht1* gene changed in populations of sweet corn selected for general resistance to *E. turcicum* race 1, thus conveying residual resistance. Four populations were developed with theoretical initial frequencies of the *Ht1* gene of 0, 0.25, 0.25, and 0.5. The populations were advanced by recurrent mass selection with parental control through four or five cycles of selection following inoculation with an *Ht*-virulent race of *E. turcicum* (i.e., race 1). Plants from each cycle of each population were evaluated

for severity of northern corn leaf blight (NCLB) and chlorotic lesion reactions following inoculation in field and greenhouse trials with either race 0 or 1 of *E. turcicum*. Recurrent mass selection for general resistance to *E. turcicum* race 1 reduced the severity of NCLB in all four populations of sweet corn, although the change in the most susceptible population was minimal. Percent gain per cycle was 14.5, 12.3, 14, and 3.7% for populations I, II, III, and IV, respectively. The *Ht1* gene did not convey levels of general resistance to *E. turcicum* race 1 that were substantial enough to be selected for in this population improvement program. There was no apparent selection advantage for resistance to *E. turcicum* race 1 in the populations that contained the *Ht1* gene. The frequency of the *Ht1* gene did not differ among cycles of selection within any of the populations in response to improved levels of general resistance to NCLB. The lack of change in frequency of *Ht1* in these populations and the similarity in gain per cycle among populations with and without *Ht1* lead us to conclude that the *Ht1* gene itself did not have a residual effect on resistance.

Northern corn leaf blight (NCLB), caused by *Exserohilum turcicum* (Luttrell) K.J. Leonard and E. G. Suggs, teleomorph *Setosphaeria turcica* (Pass.) K.J. Leonard and E. G. Suggs, occurs on maize (*Zea mays* L.) throughout the world, especially in humid areas with moderate temperatures. In the United States and parts of Europe, NCLB is one of the predominant diseases of sweet corn. In southern Florida, environmental conditions in the spring frequently are conducive to severe epidemics of NCLB on super-sweet hybrids with the shrunken-2 (*sh2*) endosperm mutation (31). Susceptible sugary (*su1*) sweet corn hybrids grown for processing in Illinois, Minnesota, and Wisconsin also can be infected severely in August and September (31). Severe epidemics of NCLB also have occurred on sweet corn grown in southern France.

Yield reductions due to NCLB are affected by disease severity (14,27,28,31,34). Yield reductions in sweet corn were less than 10% when disease severity within 1 week of harvest was below 25% (31), which corroborated the hypothesis of Hooker and Perkins (14) that the presence of the NCLB on lower leaves of maize did not substantially affect hybrid performance. Yield reductions from 2 to 8% occurred for each 10% increase in disease severity above 20 to 30% (27,28,34). When severity of NCLB was extensive during the grain-filling period, yield losses in field corn were as high as 50% (41). Adequate levels of host resistance can prevent reductions in yield.

Maize lines vary in reaction to NCLB from highly resistant with a few small lesions to highly susceptible with many large sporulating lesions (1,30,31,33). Resistance to NCLB differs in both type and degree. Quantitative, polygenic, partial or general

resistance reduces NCLB severity without the occurrence of categorically distinct phenotypes. General resistance appears to be under polygenic control and effective against all biotypes of the fungus. Monogenic resistance, conveyed by single genes (designated as *Ht* genes), results in distinct reactions to avirulent races of *E. turcicum*. The *Ht1* gene, identified from popcorn cv. Ladyfinger and field corn inbred GE440 (11), conditions a chlorotic lesion reaction that reduces sporulation and size of typical, necrotic, "cigar-shaped" lesions. The *Ht2* and *Ht3* genes also convey chlorotic lesion resistance (12,14). The *HtN* gene, identified from a Mexican landrace Pepitilla, increases incubation and latent periods without the occurrence of a qualitative difference in lesion type (10). A single recessive gene found in a line derived from the maize synthetic BS19 conditions a reaction to *E. turcicum* that is expressed as circular chlorotic halos (5).

Specific virulence occurs in relation to each of the *Ht* genes (23). Race 0 of *E. turcicum* is avirulent against lines with any of the *Ht* genes (23). Race 1 is virulent against lines with the *Ht1* gene. Various other combinations of virulence also occur (7,23,38).

General resistance to NCLB is expressed as reduced disease severity due to reductions in number of lesions and lesion size and an increase in incubation period (4,13,16). Jenkins and Robert (17) concluded that general resistance to NCLB was polygenic highly heritable and affected primarily by additive gene action. Using chromosomal translocation stocks, regions associated with general resistance to NCLB were identified on chromosomes 1L, 2L, 3S, 3L, 4S, 4L, 5L, 6S, 6L, 7S, 8L, and 9L (2,17,19). In several recent studies, quantitative trait loci (QTL) associated with general resistance to NCLB were identified on all 10 maize chromosomes (3,6,8,9,37,42-44).

The widespread use of the *Ht1* gene in the 1960s and 1970s resulted in an increased frequency of virulence against *Ht1* in the

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population of *E. turcicum* in the United States in the 1980s (14,24,39). Virulence against *Ht2*, *Ht3*, and *HtN* occurs at a low frequency (23,29,38). Despite the occurrence of virulence, many sweet corn hybrids grown in North America carry the *Ht1* gene.

Several authors have suggested that the combination of resistance conferred by *Ht* genes and general resistance would enhance the level of resistance and result in greater stability and longer protection against *E. turcicum* (12,13,25,35,36). Smith and Kinsey (38) suggested that an effective use of monogenic resistance might be to combine major genes in commercial hybrids since races exist that are virulent on each of the *Ht* genes. Conversely, Lipps et al. (25) concluded that in environments where NCLB epidemics are frequent and severe, there appears to be no apparent advantage in combining *Ht* resistance with high levels of general resistance. Similarly, high levels of general resistance with or without the *Ht* genes were an extremely effective method of limiting the effect of NCLB on sweet corn yield (28).

Some authors also have proposed that pyramiding major genes for resistance may produce durable resistance because residual resistance or partial resistance may be a function of ineffective resistance genes or genes closely linked to the ineffective genes (32). In a series of studies in which maize lines with or without the *Ht1* gene were inoculated with an *Ht1*-virulent isolate of *E. turcicum* (i.e., race 1), Leath and Pedersen (20–22) observed lower amounts of NCLB (e.g., area under disease progress curve, lesion size, and lesion number) on *Ht1* versions of several maize hybrids and inbreds (i.e., A632 × A619, Mo17 × A634, B37 × Mo17, and Va26) than on the *ht* versions of these lines. They interpreted these results to indicate that the *Ht1* gene or genes tightly linked to *Ht1* were responsible for the lower amount of NCLB, and that this was an example of residual resistance (32). They also suggested that quantitative genes for resistance to *E. turcicum* were transferred into recurrent parents while backcrossing the *Ht1* gene.

The possibility that an ineffective *Ht1* gene or genes tightly linked to *Ht1* may convey general resistance (i.e., residual resistance) could be useful in sweet corn where the *Ht1* gene is present in many commercial hybrids and breeding populations. If the *Ht1* gene or genes closely linked to *Ht1* contribute to general resistance, the frequency of *Ht1* should increase in populations selected for general resistance to *E. turcicum* race 1, which is virulent

against the *Ht1* gene. The objective of this study was to determine if the frequency of the *Ht1* gene changed in populations of sweet corn selected for general resistance to *E. turcicum* race 1, thus conveying residual resistance.

MATERIALS AND METHODS

Populations and selection. Two sugary/sugary enhancer (*su/se*) and two supersweet (*sh2*) populations of sweet corn were developed with different initial frequencies of the *Ht1* gene (Table 1). Each of the populations was formed in a similar manner. Four hybrids were crossed in all possible combinations in the first season. In the second season, 15 ears from each of the six double-cross hybrids per population were pollinated with pollen collected and bulked from at least 60 plants of each of the six double-cross hybrids. Populations were then formed by selecting 10 kernels from each of the 15 ears of each of the six bulk-pollinated double-cross hybrids per population. Each population was then random mated for two generations prior to selection. The two generations of random mating included about 900 and 450 individuals per population, respectively. Populations I and II were developed from sugary and sugary enhanced parents and had theoretical initial frequencies of the *Ht1* gene of 0.25 and 0, respectively (Table 1). Populations III and IV were developed from supersweet parents and had theoretical initial frequencies of the *Ht1* gene of 0.5 and 0.25, respectively (Table 1). Cycle 0 of selection consisted of the progeny from the second season of random mating.

From 1993 to 1999, populations were advanced from cycle 0 to cycle 5 by recurrent mass selection with parental control. In each cycle of selection, approximately 500 plants of each population were planted in 40 rows with about 12 plants per row. All plants in each population were inoculated two to four times with a conidial suspension of three to six isolates of *E. turcicum* race 1 (approximately 10^3 conidia per milliliter). The conidial suspension was sprayed directly into leaf whorls when plants were at the three- to seven-leaf stages. Isolates of *E. turcicum* were cultured on lactose-casein hydrolysate agar at room temperature for 1 to 3 weeks. Inoculum was prepared by blending cultures in tap water and filtering the mixture through cheesecloth.

Ear shoots of all plants were covered with glycine bags to prevent uncontrolled pollination. When nearly all plants in a population were shedding pollen, individual plants were selected for general resistance to *E. turcicum* race 1. Two different evaluators removed plants with the greatest percentage of leaf area infected. The first evaluator eliminated about 50% of the population that was most susceptible, and the second evaluator removed about 50% of the most susceptible, remaining plants. Following this procedure, selection intensity ranged from 21% to 34% among the various cycles and populations. Individuals that were not removed were then intermated by bulking pollen from all remaining plants and hand-pollinating remaining ear shoots.

Evaluation of cycles for NCLB severity and chlorotic reactions. Each available cycle of each population was evaluated for severity of NCLB and/or chlorotic lesions in field trials at Urbana, IL from 1998 to 2002. Populations were evaluated for reactions to race 1 in 1998, 1999, and 2001 and for reactions to race 0 in 1998 and 2000. In 1998, when both races were evaluated, trials were separated by at least 0.5 km. Each trial used a split plot design of a randomized complete block with two to four replicates. Each of the populations was planted in main plots, and cycles of selection were planted in subplots. Each experimental unit consisted of four to eight rows with approximately 10 to 15 plants per row. Plants at the three- to four-leaf stage were inoculated two to four times by spraying a conidial suspension directly into the plant whorl. Inoculum was prepared as described previously. In trials inoculated with race 1, each plant was rated about 3 weeks after pollination for severity of NCLB based on the percentage of leaf area infected. In trials inoculated with race 0,

TABLE 1. Hybrid parents of four sweet corn populations selected for resistance to *Exserohilum turcicum* race 1, expected *Ht1* genotype based on reaction of the S1 generation, and theoretical initial frequency of the *Ht1* gene in each population

Populations and hybrid parents	Reaction of S1	<i>Ht1</i> genotype	Theoretical frequency (p) of the <i>Ht1</i> gene in the population
Population I (<i>su/se</i>) ^a			0.25
Classic	Susceptible	<i>ht1 ht1</i>	
Dandy	Segregating	<i>Ht1 ht1</i>	
More	Segregating	<i>Ht1 ht1</i>	
Sugar Buns	Susceptible	<i>ht1 ht1</i>	
Population II (<i>su/se</i>)			0
Classic	Susceptible	<i>ht1 ht1</i>	
Merlin	Susceptible	<i>ht1 ht1</i>	
Snopak	Susceptible	<i>ht1 ht1</i>	
Sugar Buns	Susceptible	<i>ht1 ht1</i>	
Population III (<i>sh2</i>)			0.5
Sch 5007	Segregating	<i>Ht1 ht1</i>	
Sch 5434	Segregating	<i>Ht1 ht1</i>	
Sch 7710	Segregating	<i>Ht1 ht1</i>	
Crisp and Sweet 710	Segregating	<i>Ht1 ht1</i>	
Population IV (<i>sh2</i>)			0.25
FMX 235	Susceptible	<i>ht1 ht1</i>	
Sch 4055	Segregating	<i>Ht1 ht1</i>	
Sch 4175	Resistant	<i>Ht1 Ht1</i>	
Sunset	Segregating	<i>ht1 ht1</i>	

^a Endosperm type: *su/se* = sugary and sugary enhanced, *sh2* = shrunken-2.

the number of plants with chlorotic and necrotic lesions was counted, and each plant with necrotic lesions was rated for NCLB severity.

Chlorotic lesion reactions also were evaluated in greenhouse trials using a split plot design of a randomized complete block with four replicates. Treatments assigned to main plots and subplots were the same as field trials. Each experimental unit consisted of a six-row flat with 12 plants per row. Approximately 288 seedlings of each cycle of each population were evaluated. Two weeks after planting, plants were inoculated twice by spraying a conidial suspension of race 0 directly into the whorls. Inoculum was prepared as described previously. The number of plants with necrotic and chlorotic lesions was counted.

Data in each trial were analyzed by analysis of variance (ANOVA) with $P = 0.05$, and cycles were compared by mean separation values. Mean severity of NCLB also was regressed on cycles of selection for each population. Improvement in NCLB resistance (i.e., decreases in NCLB severity) in each population was estimated from slope coefficients (b_1) of linear regressions. Since severity of NCLB differed somewhat among each trial, gain per cycle was estimated as a percentage of the best estimate of NCLB severity of the initial cycle of selection (C_0) of each population by dividing the slope coefficient by the intercept coefficient, i.e., percentage gain per cycle = $(b_1/b_0) \times 100$. Within populations, slope coefficients were compared among all possible trials. Data for each population was combined from all trials (races 0 and 1) except for populations I and III inoculated with race 1 in 2001 for which NCLB development was poor and slope coefficients were significantly lower than those from some of the other trials (described below). Mean severity of NCLB from all trials was then regressed on cycles of selection for each population, and gain per cycle was calculated as described previously.

The proportion of plants with chlorotic lesions was estimated for each cycle of each population from data collected from the two field trials and the greenhouse trial in which plants were inoculated with *E. turcicum* race 0. Since the *Ht1* gene is completely dominant, heterozygotes (i.e., *Ht1ht1*) had the same chlorotic lesion phenotype as resistant homozygotes (i.e., *Ht1Ht1*). Therefore, the frequency of the *Ht1* gene (p) in each cycle of each population was calculated from the proportion of plants with chlorotic reactions (C) using the equation $p = 1 - \sqrt{1-C}$.

RESULTS

Severity of NCLB differed among the five field trials (Table 2). Except for population IV, NCLB was least severe in the trial inoculated with race 1 in 2001 and most severe in the trials inoculated with races 0 or 1 in 1998. Mean NCLB severity ranged from about 4 to 48% among cycles across all populations and trials. Severity of NCLB differed among populations and cycles in each trial (Table 2).

Changes in NCLB severity with cycles of selection. Mean severity of NCLB decreased as cycles of selection increased for all populations in all trials (Table 2). Frequency distributions of NCLB severity on individual plants were relatively normal for the initial cycle of selection (C_0) of each population, but were skewed with a high frequency of resistant individuals in C4 and C5 of populations I, II, and III (Fig. 1). The mean of C4 from population IV was lower than the mean from C_0 , but the distribution of plants in C4 was not as skewed as the distributions for populations I, II, and III (Fig. 1). Within populations, slope coefficients from regressions of NCLB severity on cycles of selection did not differ among trials except for populations I and III inoculated with race 1 in 2001 (Table 3). Percent gain per cycle ranged from about 4 to 19% among all trials of all populations (Table 3).

When population I was inoculated with *E. turcicum* race 1, mean severity ranged from 25.2% for C_0 to 10.7% for C3 in 1998, from 24.8% for C_0 to 12.4% for C4 in 1999 and from 9.1%

for C_0 to 3.1% for C5 in 2001 (Table 2). When population I was inoculated with *E. turcicum* race 0, mean severity ranged from 27.2% for C_0 to 17.1% for C3 in 1998 and from 19.7% for C_0 to 5.3% for C5 in 2000 (Table 2). Slope coefficients ranged from -1.2 to -4.3 among the five trials (Table 3). Slope coefficients were not significantly different from each other except for the slope of -1.2 from the trial inoculated with race 1 in 2001 when disease development was poor (i.e., mean severity of NCLB ranged from about 3 to 9% among cycles). Percent gain per cycle ranged from about 12 to 19% among the five trials (Table 3).

When population II was inoculated with *E. turcicum* race 1, mean severity ranged from 31.7% for C_0 to 19.1% for C3 in 1998, from 26.1% for C_0 to 18.0% for C4 in 1999, and from 22.6% for C_0 to 7.4% for C5 in 2001 (Table 2). When population II was inoculated with *E. turcicum* race 0, mean severity ranged from 26.5% for C_0 to 18.5% for C3 in 1998 and from 23.7% for C_0 to 11.2% for C5 in 2000 (Table 2). Slope coefficients that ranged from -2.2 to -4.1 among the five trials were not significantly different from each other (Table 3). Percent gain per cycle ranged from about 8 to 14% among the five trials (Table 3).

When population III was inoculated with *E. turcicum* race 1, mean severity ranged from 25.9% for C_0 to 19.7% for C3 in 1998 and from 9.6% for C_0 to 3.7% for C5 in 2001 (Table 2). When population III was inoculated with *E. turcicum* race 0, mean severity ranged from 27.9% for C_0 to 24.3% for C3 in 1998 and from 28.3% for C_0 to 12.6% for C5 in 2000 (Table 2). Slope coefficients ranged from -1.6 to -3.8 among the five trials (Table 3). Slope coefficients were not significantly different from each other except for the two most extreme slopes of -1.6 and -3.8 . Similar to population I, the least negative slope for population III

TABLE 2. Mean severity of northern corn leaf blight (NCLB; %) for four or five cycles of selection of four sweet corn populations inoculated with *Exserohilum turcicum* races 0 or 1 from 1998 to 2001

Populations and cycles	NCLB severity (% leaf area infected)				
	Race 0		Race 1		
	1998	2000	1998	1999	2001
Population I					
0	27.2	19.7	25.2	24.8	9.1
1	19.7	18.5	15.0	20.6	7.4
2	17.7	17.0	15.3	20.7	4.8
3	17.1	11.8	10.7	16.1	4.6
4	...	7.5	...	12.4	3.3
5	...	5.3	...	...	3.1
Population II					
0	26.5	23.7	31.7	26.1	22.6
1	25.5	19.5	27.1	26.0	21.9
2	18.2	16.0	24.0	23.4	18.5
3	18.5	14.0	19.1	19.9	11.6
4	...	11.5	...	18.0	9.4
5	...	11.2	...	...	7.4
Population III					
0	27.9	28.3	25.9	...	9.6
1	23.8	20.6	22.3	...	8.1
2	24.3	18.7	19.7	...	7.9
3	...	14.3	...	...	4.3
4	...	12.6	...	...	3.7
5	...	...	...	...	...
Population IV					
0	42.9	45.1	41.6	34.7	47.6
1	38.4	43.2	41.2	30.6	44.4
2	37.0	38.6	37.2	29.2	39.9
3	...	38.6	...	27.8	38.8
4	...	37.5	...	...	36.4
5	...	...	...	...	...
BLSD ^b	8.0	2.7	5.6	4.1	4.6

^a NCLB severity—percent leaf area infected as a mean of all plants in a cycle of selection of a population when plants were rated individually.

^b Bayesian least significant difference ($k = 100$) for comparison of mean severity of all cycles of all populations in a trial.

was from the trial inoculated with race 1 in 2001 when disease development was poor (i.e., mean severity of NCLB ranged from about 4 to 10% among cycles). Percent gain per cycle ranged from about 7 to 16% among the five trials (Table 3).

When population IV was inoculated with *E. turcicum* race 1, mean severity ranged from 41.6% for C0 to 37.2% for C3 in 1998, from 34.7% for C0 to 27.8% for C4 in 1999, and from 47.6% for C0 to 36.4% for C5 in 2001 (Table 2). When population IV was inoculated with *E. turcicum* race 0, mean severity ranged from 42.9% for C0 to 37% for C3 in 1998 and from 45.1% for C0 to 37.5% for C4 in 2000 (Table 2). Slope coefficients ranging from -2.0 to -3.0 among the five trials were not significantly different from each other (Table 3). Percent gain per cycle ranged from about 4 to 7% among the five trials (Table 3).

Based on similar slope coefficients among trials within populations, data were combined from all trials, except populations I and III inoculated with race 1 in 2001. Mean severity differed among cycles of selection for each population (Table 4). Slope coefficients from regressions of severity on cycles were -3.3, -3.2, -3.7, and -1.6 for populations I, II, III, and IV, respectively (Fig. 2). Percent gain per cycle was 14.5, 12.3, 14, and 3.7% for populations I, II, III, and IV, respectively (Fig. 2).

Frequency of the *Ht1* gene. The proportion of plants inoculated with race 0 that had chlorotic lesion reactions (i.e., *Ht* resistance) ranged from 0.42 to 0.51 for C0 to C5 of population I, from 0.66 to 0.71 for C1 to C4 of population III, and from 0.32 to 0.39 for C0 to C4 for population IV (Table 4). None of the plants in population II had chlorotic lesions. There were no significant

Fig. 1. Frequency distributions of severity of northern corn leaf blight (NCLB) on individual plants from four sweet corn populations selected for four or five cycles for resistance to *Exserohilum turcicum* race 1.

TABLE 3. Simple linear regression coefficients from the regression of severity of northern corn leaf blight on cycles of selection, coefficients of determination, and percent gain per cycle for four populations of sweet corn inoculated with *Exserohilum turcicum* races 0 or 1 from 1998 to 2001

Population, year, and race	b_0	b_1	r^2	Gain per cycle (%)
Population I				
1998 race 1	23.0	-4.3	0.83	18.8
1999 race 1	24.8	-2.9	0.94	11.7
2001 race 1	8.4	-1.2	0.90	14.4
1998 race 0	25.3	-3.2	0.80	12.8
2000 race 0	21.1	-3.1	0.95	14.9
Population II				
1998 race 1	31.6	-4.1	0.99	13.0
1999 race 1	27.2	-2.2	0.94	8.3
2001 race 1	23.9	-3.4	0.95	14.4
1998 race 0	26.8	-3.1	0.82	11.6
2000 race 0	22.3	-2.5	0.94	11.4
Population III				
1998 race 1	25.7	-3.1	0.99	12.0
2001 race 1	9.9	-1.6	0.91	16.0
1998 race 0	27.1	-1.8	0.66	6.7
2000 race 0	26.4	-3.8	0.93	14.2
Population IV				
1998 race 1	42.2	-2.2	0.81	5.2
1999 race 1	33.9	-2.2	0.91	6.5
2001 race 1	47.0	-2.8	0.96	6.0
1998 race 0	42.4	-3.0	0.91	7.0
2000 race 0	44.5	-2.0	0.86	4.4

TABLE 4. Mean severity of northern corn leaf blight (NCLB; %) for four or five cycles of selection of four sweet corn populations inoculated with *Exserohilum turcicum* races 0 and 1 from 1998 to 2001, proportion of plants with chlorotic lesion-resistant reactions when inoculated with *E. turcicum* race 0, and estimated frequency of the *Ht1* gene

Populations and cycles	Mean severity	Proportion chlorotic	<i>Ht1</i> gene frequency
Population I			
0	22.9	0.47	0.27
1	17.7	0.45	0.26
2	17.7	0.51	0.30
3	13.1	0.44	0.25
4	9.8	0.42	0.24
5	5.3	0.42	0.24
BLSD ^b	4.15	...	...
Population II			
0	25.7	0	0
1	23.6	0	0
2	19.8	0	0
3	16.6	0	0
4	13.2	0	0
5	10.0	0	0
BLSD	2.98	...	...
Population III			
0	27.7	0.45	0.26
1	21.7	0.66	0.42
2	19.9	0.68	0.43
3	14.3	0.71	0.47
4	12.6	0.66	0.42
BLSD	8.81	...	...
Population IV			
0	41.8	0.32	0.18
1	40.3	0.39	0.22
2	36.9	0.38	0.21
3	35.5	0.33	0.18
4	36.5	0.34	0.19
BLSD	5.62	...	...

^a NCLB severity—percent leaf area infected of all plants in a cycle of selection of a population when plants were rated individually.

^b Bayesian least significant difference ($k = 100$) for comparison of mean severity of all cycles of a population.

differences among cycles in the proportion of plants with chlorotic lesions for populations I or IV. In population III, the initial cycle (C0) had a significantly lower proportion of plants with chlorotic lesions than C1 to C4 (Table 4); however, sample size of the C0 was relatively low due to poor germination ($n = 96$ and 180 for field and greenhouse trials, respectively). Approximately 55 and 40% of the plants in the C0 of population III were chlorotic in field and greenhouse trials, respectively.

The frequency of the *Ht1* gene did not differ among cycles of selection except for C0 of population III (Table 4). The regression of frequency of *Ht1* on cycles was not significant for any of the populations (Fig. 3). The theoretical frequency of *Ht1* in the initial cycle of selection was 0.25 in populations I and IV and 0.5 in population III. The estimated mean frequency of *Ht1* for all cycles of populations I, III, and IV was 0.26, 0.45, and 0.20, respectively, excluding C0 of population III for which the estimated frequency of *Ht1* was 0.26 (i.e., 0.33 and 0.23 from field and greenhouse trials, respectively).

DISCUSSION

Recurrent mass selection for general resistance to *E. turcicum* race 1 reduced the severity of NCLB in all four populations of sweet corn, although the change in the most susceptible population was minimal. Improvement in NCLB resistance through recurrent selection was expected based on the results of previous

Fig. 2. Simple linear regression of severity of northern corn leaf blight (NCLB) on cycles of selection and percent gain per cycle for four sweet corn populations selected for resistance to *Exserohilum turcicum* race 1 for four or five cycles.

Fig. 3. Frequency of the *Ht1* gene in three sweet corn populations selected for resistance to *Exserohilum turcicum* race 1 for four or five cycles of selection.

studies (15,16,18,26). Jenkins and Robert (16) and Jenkins et al. (18) observed substantial increase in NCLB resistance following two or three cycles of recurrent selection. Hughes and Hooker (15) concluded that general resistance to NCLB was conditioned by a relatively low number of genes in the lines they evaluated and improvement could be accomplished by simple procedures such as phenotypic recurrent selection. Miles et al. (26) improved resistance to NCLB by modified ear-to-row selection and half-sib family selection using a selection index that included other maize leaf diseases. Similar to the conclusions of Jenkins and Robert (16), Jenkins et al. (18), Hughes and Hooker (15), and Miles et al. (26), we observed rapid gain in NCLB resistance from just a few cycles of mass selection in three of four populations.

Among the three populations with similar levels of NCLB in C0 (i.e., populations I, II, and III), gain per cycle was similar for the two populations with the *Ht1* gene (populations I and III) and the population (population II) without the *Ht1* gene. Thus, there was no apparent selection advantage for resistance to *E. turcicum* race 1 in the populations that contained the *Ht1* gene.

The frequency of the *Ht1* gene did not differ among cycles of selection within any of the populations in response to improved levels of general resistance to NCLB, although the frequency of *Ht1* was anomalously low in C0 of population III. Except for C0 of population III, estimates of the frequency of the *Ht1* gene in all cycles of each population were similar to the theoretical frequency that was estimated from the *Ht1* genotype of the parents of each population. Thus, there was no consistent evidence based on change in gene frequency that the observed improvement in general resistance to NCLB was associated with a "residual" effect of the *Ht1* gene or genes closely linked to *Ht1*.

The *Ht1* gene maps to the long arm of chromosome 2. Based on results from translocation mapping studies (2,17,19) and QTL mapping studies (3,6,8,9,37,42–44), genes for general resistance to NCLB appear to be located on all 10 maize chromosomes, including chromosome 2L. Different gene regions or QTL have been associated with NCLB resistance in different populations of maize. In some populations, clusters of QTL for general NCLB resistance were found near the *Ht2* and *HtN* genes on chromosome 8 (42); however, these clusters were not found in all maize populations. A similar situation may occur with genes for general resistance and the *Ht1* gene. It is possible that clusters of genes for general resistance occur in the region of the *Ht1* gene in some maize lines but not in others, which could explain why Leath and Pedersen (20–22,32) observed residual resistance associated with *Ht1*, and we did not. The gains in general resistance to NCLB in the sweet corn populations evaluated in these trials appear to be the result of QTL that are not closely located to the *Ht1* gene. Similarly, the lack of change in frequency of *Ht1* in these populations and the similarity in gain per cycle among populations with and without *Ht1* lead us to conclude that the *Ht1* gene itself did not convey a residual effect.

The resistance to NCLB that was selected by inoculating these populations with *E. turcicum* race 1 appeared to be race non-specific. Within each population, gain per cycle was similar for resistance to race 0 and race 1 even though populations were not selected for reactions to race 0. Average gain per cycle ranged from 7.8 to 13.9% for race 1 and from 5.4 to 12.5% for race 0.

The populations were developed for these studies to examine different hypotheses. Populations I and II contained at least 50% genetic similarity (i.e., two of four parents were similar) but differed for the presence of the *Ht1* gene. These two populations had similar initial levels of general resistance to NCLB. Improvement in NCLB resistance was similar among the two populations probably because the same QTL for resistance were being selected; however, the presence of the *Ht1* gene did not provide a selection advantage to population I. Populations I and III had similar initial levels of general NCLB resistance and theoretical initial frequencies of the *Ht1* gene of 0.25 and 0.5, respectively. Improve-

ment due to selection was similar for these two populations in spite of different frequencies of the *Ht1* gene. Population IV was created from extremely susceptible parents of which two were heterozygous for *Ht1*. Although general resistance to NCLB was improved in population IV, gain per cycle was less than in the other three populations probably because there were fewer QTL for resistance in population IV. In spite of substantially lower levels of general resistance in this population, the frequency of the *Ht1* gene did not change. Thus, we observed that when the genetic background of populations was similar, when the frequency of the *Ht1* gene differed among populations, and when the level of general resistance differed among populations, there was no change in the frequency of the *Ht1* gene when the populations were selected for general resistance to *E. turcicum* race 1.

Many commercial sweet corn hybrids, inbreds, and breeding materials presently carry the *Ht1* gene. The chlorotic lesion reaction conditioned by this gene supplements general resistance when *E. turcicum* race 0 is prevalent (13,28,33,36,40). The *Ht1* gene also may confer limited levels of resistance when populations of *E. turcicum* are comprised of a mixture of races 0 and 1. However, based on the results from this study, the *Ht1* gene does not appear to convey levels of general resistance to a virulent race (i.e., residual resistance) that are substantial enough to be selected for in a population improvement breeding program.

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