Resistance to *Puccinia polysora* in Maize Accessions

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ABSTRACT

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A number of potential sources of general and specific resistance to southern corn rust were identified from 1,890 plant introduction accessions that were screened for reactions to *Puccinia polysora* race 9. Resistance appeared to differ among four accessions on which uredinia were not observed in initial screenings. Resistance to *P. polysora* in PI 186215 (Argentine inbred 2-687) was a chlorotic fleck, hypersensitive reaction that was conditioned by a single, dominant gene that was allelic with or very closely linked to the *Rpp9* gene based on tests of allelism. All but 3 of 2,357 testcross progeny, (inbred 2-687 × *Rpp9*) × P₃ were resistant. Resistance in Ames 19016 (Va59) was effective in F₁ progeny and appeared to be dominant and simply inherited; however, this resistance appeared to be a slow-rusting or incomplete resistance that was effective in adult plants but not in young seedlings. Severity of southern rust was less than 10% on resistant progeny from crosses with Va59 compared with severity exceeding 70% on susceptible progeny. Resistance in plant introduction (PI) 186209 (Venezuelan flint) and NSL 75976 (IA DS61) were not effective in F₁ hybrid combination and, thus, probably have limited value in commercial maize. Resistance in PI 186209 may be conditioned by a single recessive gene and resistance in NSL 75976 may be co-dominant.

Additional keywords: *Rpp* genes

Southern rust, caused by *Puccinia polysora* Underw., is a troublesome foliar disease of maize (*Zea mays* L.). The disease develops mainly in tropical and subtropical areas of the world (8,12,31) and is restricted by the temperature sensitivity of the pathogen. In temperate regions, epidemics occur sporadically. The disease spreads rapidly and was destructive throughout Africa following an outbreak in 1949 (12,18). In the United States, southern rust occurs primarily in the lower Mississippi Valley and Texas, but has been found as far north as Massachusetts and Wisconsin (12,17). Epidemics developed in the United States in 1972–74, 1979, and 1981 (5,8,11,21,24). In the past few years, unusually severe outbreaks of southern rust have been observed in Missouri, Nebraska, and Wisconsin (L. E. Sweets, T. A. Jackson, and W. F. Tracy, *personal communications*, respectively). The disease also recently has increased in importance in maize-growing regions of northern China (3).

Southern rust can cause substantial yield losses (3,8,12,18,20). *P. polysora* infects leaves, leaf sheaths, and husk leaves and forms uredinia that are smaller but more

densely distributed than those of *P. sorghi* Schwein., the casual agent of common corn rust (31). Southern rust becomes progressively more severe as plants mature. Uredinia can become so numerous that leaf and sheath tissues are killed, and plants are prematurely desiccated (20). Melching (12) observed yield reductions of 24 to 37% following inoculation with *P. polysora* in Maryland. Rodriguez-Anon et al. (20) observed yield reductions of 4, 23, and 45% when near-isogenic resistant and susceptible hybrids were planted on three sequential dates in Mississippi. Raid et al. (18) observed grain yield reductions of about 18 and 39% in inoculated field trials in Pennsylvania and Maryland, respectively. Yield losses up to 50% also have been observed in Africa and China (3,8).

Host resistance is the most efficient means to control southern rust where it occurs frequently. General resistance to *P. polysora* is a component of most tropical maize-breeding programs (4). Most tropical maize has general resistance that reduces severity of infection below economically important levels (25). In an evaluation of maize germplasm adapted for temperate climates, Bailey et al. (1) observed considerable variation for slow-rusting resistance (i.e., general resistance) to *P. polysora* among 23 single-cross hybrids and 33 inbred lines. Mo17 was one of five inbred lines with area under the disease progress curve (AUDPC) values that were not significantly different from the inbred with the lowest AUDPC value.

Holland et al. (7) mapped genes from Mo 17 with minor effects on southern rust resistance to chromosome 3 (marker locus *umc26*) and chromosome 4 (marker locus *umc31*). Zummo (32) observed differences in sporulation and number, size, and plumpness of uredinia among four maize inbred lines that differed in reaction to *P. polysora*. Scott and Zummo (23) observed fewer, smaller uredinia that sporulated later on hybrids derived from crosses of slow-rusting inbreds compared with susceptible hybrids.

Several sources of simply inherited, specific resistance to *P. polysora* have been identified from different maize lines and populations; and various races of *P. polysora* have been designated. The *Rpp1* and *Rpp2* genes were identified in Kenya from maize lines AFRO 29 ('Colombia 2') and AFRO 24 ('SLP 20-4A') that originated in Colombia and Mexico, respectively (26). The *Rpp1* gene is dominant and confers complete resistance to race EA1 of *P. polysora*. The *Rpp2* gene conditions an intermediate reaction to races EA1 and EA2 and can be modified to near complete susceptibility by other host genes (8). The *Rpp1* and *Rpp2* genes are linked with about 12% recombination (27). Both genes were ineffective against a third race, EA3, recognized in Kenya in 1961 (28). The *Rpp10* and *Rpp11* genes were identified in Kenya from maize lines AFRO 761 ('Andaqui') and AFRO 600 ('Zapalote Chico') that also originated in Colombia and Mexico, respectively (28). The *Rpp10* gene was dominant and conferred complete resistance to races EA1 and EA3 and, thus, was different from the *Rpp1* gene. The *Rpp11* gene conditioned an incomplete resistance to races EA1 and EA3.

The number of prevalent races of *P. polysora* in North America and elsewhere has not been documented clearly, but diversity may be limited. The sexual stage of *P. polysora* has not been described, and most epidemics are thought to result from initial inocula composed of urediniospores. Nevertheless, races are known to occur. Robert (19) designated 6 new races of *P. polysora* (i.e., PP3 to PP8) in a collection of 11 isolates from North and South America based on the differential response of 11 corn lines which presumably carried the genes *Rpp3* to *Rpp8*. An accession from Argentina, plant introduction (PI) 19802 (4F-345 CN12), was resistant to four of the six new races and to EA1 and EA2. An accession from South Africa, PI 186208

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(Boesman yellow flint), was resistant to EA2 and PP5. Ullstrup (29) identified a single dominant gene, *Rpp9*, from PI 186208. The *Rpp9* gene conferred chlorotic fleck hypersensitive resistance to the prevalent race of *P. polysora* in the U.S. Corn Belt which Ullstrup designated PP9 (i.e., race 9). The *Rpp9* gene was linked on chromosome 10S about 1.6 centimorgans from the *Rp1D* gene which confers a chlorotic fleck hypersensitive resistance to *P. sorghi*.

The *Rpp9* gene or dominant genes for southern rust resistance that may be closely linked to the *Rpp9* gene have been identified from several sources in addition to PI 186208 (3,6,7,10,22,30). Futrell et al. (6) identified a single, dominant gene for resistance to *P. polysora* race 9 from an inbred line B1138T which was derived from an open pollinated cultivar, Teko yellow, selected in Natal, South Africa. Futrell et al. (6) did not test whether the gene in B1138T was allelic with the *Rpp9* gene. Scott et al. (22) identified single dominant genes for resistance to *P. polysora* in an inbred line Mp78:5273 and in FSHmR, a broad-based population derived from about 40% tropical and 60% U.S. germplasm. The resistance genes in Mp78:5273 and FSHmR were allelic with or very closely linked to the *Rpp9* gene. Uhr (30) identified high levels of resistance to *P. polysora* in two temperate-adapted, 100% tropical maize inbreds 1416-1 and 1497-2; and Holland et al. (7) mapped a single locus conditioning southern rust resistance in both inbreds to a region of chromosome 10S between restriction fragment length polymorphism loci *bnl3.04* and *npi285* that flank the *Rp1* locus. Evaluations of test crosses of inbreds 1416-1 and 1497-2 with a known source of the *Rpp9* gene were unsuccessful. Jines et al. (10) also mapped a simply inherited, dominant gene conferring southern rust resistance in the inbred line NC 300 to the short arm of chromosome 10 between the markers *umc1380* and *bnlg1451* (bins 10.0 and 10.1, respectively). The inbred NC300 and inbreds 1416-1 and 1497-2 (7) may have the same southern rust resistance allele because their pedigrees contain double-cross tropical hybrids PX105A and PX306B (10). A single, dominant, southern rust resistance gene in the Chinese inbred line Qi319, which Chen et al. (3) designated as *RppQ*, was mapped to the same region of chromosome 10S as the *Rp1* and *Rpp9* loci. Chen et al. (3) proposed to test this southern rust resistance gene for allelism with the *Rpp9* gene.

A few field corn hybrids with race-specific resistance to southern rust have been sold commercially (7), but none of the *Rpp* genes have been used extensively in field corn or sweet corn developed for the United States. Most of the *Rpp* genes are not available in adapted temperate

germplasm. Of over 600 commercially-available, temperate sweet corn hybrids evaluated for reactions to *P. polysora*, only two, Calvary and Suregold, have chlorotic-fleck-resistant reactions (14,16). Only 18 of 379 sweet corn hybrids evaluated for reactions to *P. polysora* in 2006 had moderate levels of general resistance (16). So et al. (25) observed that essentially all temperate sweet corn hybrids were highly susceptible to *P. polysora* under environmental conditions conducive to southern rust epidemics in Hawaii. Additional resistance to *P. polysora* is necessary if temperate-based sweet corn germplasm is to be grown in subtropical climates where *P. polysora* is endemic or if *P. polysora* increases in prevalence in temperate regions in response to warmer climates.

The objectives of this study were to screen maize accessions for potential sources of resistance to *P. polysora*, determine whether resistance to *P. polysora* in resistant accessions was inherited simply, and determine whether genes conferring resistance in these accessions were allelic with the *Rpp9* gene.

MATERIALS AND METHODS

Accessions evaluated for resistance to *P. polysora* race 9. Two thousand maize accessions were obtained from the North Central Regional Plant Introduction Station (NCRPIS), Ames, IA. Accessions were selected by Mark Millard, NCRPIS maize curator, as representative of maize germplasm adapted for temperate climates from all regions of the world and representative of sweet corn and field corn germplasm from the United States. The 2,000 accessions were planted 22 May 1997 at the University of Illinois South Farm, Urbana. The trial included single rows of accessions with approximately 15 plants per accession. Rows were spaced 0.76 m apart. Reactions to *P. polysora* could not be assessed for 110 accessions due to poor germination. Of the 1,890 accessions evaluated, 889 were collected originally from the United States and 1,001 were collected from at least 48 other countries (13).

Plants at the five- to eight-leaf stage were inoculated with *P. polysora* race 9 on 30 June and 7 July. Urediniospores were obtained from J. M. Perkins (Dekalb Genetics, Dekalb, IL) and maintained on susceptible plants grown in a greenhouse. Inoculum for this and other field trials was increased on about 600 field-grown plants of sweet corn hybrids that carried the *Rp1D* gene in order to prevent contamination of inoculum with *P. sorghi*. About 100 severely infected leaves with 60 to 80% of the upper leaf surface covered with profusely sporulating uredinia were washed in 4 liters of water and 10 ml of Tween-20 (polyoxyethylene-20) to produce a concentrated suspension of urediniospores. Urediniospore suspensions were diluted 10:1

with water. About 5 ml of diluted inoculum was sprayed directly into the leaf whorls of plants. Reactions of plants were rated on a row basis on 25 to 26 August using a 0-to-3 scale, where 0 = chlorotic flecks with no uredinia, 1 = partially resistant with a few small uredinia, 2 = moderately susceptible with moderate numbers of uredinia, and 3 = fully susceptible with an abundance of uredinia. Some rows were given two ratings (e.g., 0/1, 1/2) if all plants did not appear to have the same phenotype. Accessions with ratings of 0 and 1 were reevaluated in 1998 in a trial smaller than but similar to the trial in 1997. Three inbred lines, B37, Oh43, and W64a, into which the *Rpp9* gene had been backcrossed were obtained from D. R. Smith (Dekalb Genetics) and included as resistant checks in the 1998 trial.

Preliminary tests of allelism and inheritance. Uredinia were not observed on five accessions—Ames 19016 (Va59), NSL 75976 (IA DS61), PI 186209 (Venezuela yellow flint), PI 186215 (inbred 2-687 from Argentina), and PI 198905 (4F-374-GE 3)—and the inbred lines with the *Rpp9* gene. In order to determine whether genes conferring resistance in these accessions were allelic or closely linked to the *Rpp9* gene, the five accessions were crossed with one or more of the inbred lines carrying the *Rpp9* gene, B37*Rpp9*, Oh43*Rpp9*, and W64a*Rpp9*. The resulting F₁ crosses then were test crossed with susceptible sweet corn inbred lines (M32, M12, or Cr44). Progeny of test crosses were screened for reactions to *P. polysora* in field trials at the University of Illinois South Farm in 2000 and 2001. Resistant controls were F₁ crosses of inbred lines with the *Rpp9* gene (i.e., W64a*Rpp9* × Oh43*Rpp9* and W64a*Rpp9* × B37*Rpp9*). The sweet corn hybrid 'Bonus' was the susceptible control. Each trial included two replicates of test crosses of the five accessions (i.e., accession × *Rpp9*) × susceptible inbred, the resistant controls, and the susceptible control. The experimental design was a randomized complete block. Each replicate included different numbers of rows (ranging from 8 to 40) of each test cross depending on availability of seed, 4 rows of each resistant control, and 8 rows of the susceptible control. Each row was 3.8 m long with approximately 12 plants per row. Rows were spaced 0.76 m apart. Trials were planted 20 May 2000 and 10 May 2001. In both years, inoculum was produced as described above. Plants were inoculated between the seven-leaf to row tassel (i.e., VT) growth stages on 6, 8, 11, and 13 July 2000 and 26 June and 7, 11, and 16 July 2001. Individual plants were rated for the presence (susceptible) or absence (resistant) of uredinia near the end of August. Segregation of progeny from test-crosses were tested by χ^2 for goodness of fit to 1:0, 3:1, or 1:1 ratios (resistant:susceptible [R:S]) which would be

expected if resistance in an accession was allelic with the *Rpp9* gene, dominant and independent from the *Rpp9* gene, or recessive and independent from the *Rpp9* gene, respectively.

Inheritance of resistance in accession Ames 19016 (Va59) also was evaluated in preliminary field and greenhouse trials in 2001. The accession (P_R) was crossed with susceptible sweet corn inbred lines (P_S), M32, M12, or Cr44, to produce the F_1 generation. F_2 , BC_R , and BC_S generations were created by self pollinating the F_1 or by backcrossing the F_1 to Va59 or to a susceptible sweet corn inbred, respectively. The field trial included two replicates of a susceptible control, P_R , and the F_1 , F_2 , BC_R , and BC_S generations. The number of rows of each generation varied among generations depending on availability of seed. The experimental design was a randomized complete block. Field trials were planted, inoculated, and rated as described previously for the 2001 preliminary test of allelism.

The same generations also were evaluated in four greenhouse trials. In two of the greenhouse trials, plants were inoculated at the V2 to V3 stage as soon as leaf whorls were large enough to retain urediniospore suspensions. In the other two greenhouse trials, plants were inoculated after plants reached the six- or seven-leaf stage. Each greenhouse trial included two replicates of the generations evaluated in the field trials. The experimental design was a randomized complete block. The first two trials included 3, 3, 9, 6, 12, and 9 rows of the susceptible control, P_R , F_1 , F_2 , BC_R , and BC_S generations, respectively. The second two trials included three rows of each treatment. Plants were grown in 30-by-60-by-7-cm flats with six rows per flat and approximately eight plants per row. The pathogen was maintained on plants of the sweet corn hybrid Bonus. In each of the four trials, plants were inoculated four times within a week. Inoculum was produced and plants were inoculated similar to the previous description except that the volume of inocula and the amount of inocula retained in plant whorls was considerably less than in field trials. Plants were rated for the presence (susceptible) or absence (resistant) of uredinia about 21 days after the last inoculation. Segregation of progeny was tested by χ^2 for goodness of fit for ratios that would be expected if resistance in Va59 was inherited as a dominant gene.

Inheritance of resistance and tests of allelism with the *Rpp9* gene. To study the inheritance of resistance, four sources of resistance—Ames 19016 (Va59), NSL 75976 (IA DS61), PI 186209 (Venezuela yellow flint), and PI 186215 (inbred 2-687 from Argentina)—were crossed with a susceptible sweet corn inbred line (M32, M12, or Cr44) to produce the F_1 generation. F_2 , BC_R , and BC_S generations were

created by self pollinating the F_1 or by backcrossing the F_1 to the P_R (accession) or to P_S (a susceptible sweet corn inbred, M32, M12, or Cr44), respectively. To test for allelism with the *Rpp9* gene, each source of resistance was crossed with a field corn inbred line known to have the *Rpp9* gene (B37*Rpp9*, Oh43*Rpp9*, or W64a*Rpp9*) to produce an F_1 . Segregating generations were developed by self-pollinating F_1 progeny to produce the F_2 generation and by test crossing F_1 progeny with a susceptible sweet corn inbred (P_S).

Phenotypes were evaluated in two trials at the University of Illinois South Farms in 2005. The two trials were planted 17 May and 13 June. Each trial included two replicates of generations from crosses with each source of resistance. Each row was 3.8 m long with about 20 plants per row. Rows were spaced 0.76 m apart. Each trial included 416 rows. Each replicate was blocked by sources of resistance and generations. The number of rows of each generation varied among populations due to availability of seed; however, each replicate usually contained one to three rows of a susceptible control, P_R , both F_1 generations ($P_R \times P_S$ and $P_R \times Rpp9$), BC_R , and B37*Rpp9*, Oh43*Rpp9*, or W64a*Rpp9*. Each replicate also included 7 to 15 rows of BC_S , F_2 , ($P_R \times Rpp9$) $\times P_S$, and ($P_R \times Rpp9$) F_2 . Plants in the trial planted on 17 May were inoculated from the seven-leaf to row tassel (VT) growth stages on 6, 7, 9, 12, and 26 July. Plants in the trial planted on 13 June were inoculated from the four- to eight-leaf growth stages on 6, 7, 9, 12, 20, and 25 July. Inoculum was produced and plants were inoculated as described previously. If tassels had emerged, urediniospore suspensions were sprayed behind leaf sheaths. Phenotypes were scored at least 20 days after the final inoculation. Each plant was placed in one of three categories: S = susceptible phenotypes with numerous, profusely sporulating uredinia (leaf area infected > 25%); I = intermediate phenotypes with a few sporulating uredia (leaf area infected < 25%); and R = resistant phenotypes without sporulating uredia. Intermediate phenotypes were grouped with susceptible phenotypes except for crosses with Ames 19016 (Va59), for which severity was less than 10% on plants placed in the intermediate category; thus, they were grouped with resistant phenotypes.

Segregation was analyzed by χ^2 goodness-of-fit tests. If a single, dominant gene conditioned resistance in any of the accessions, the expected ratios of R:S plants would be 1:0, 1:1, and 3:1 in the BC_R , BC_S , and F_2 generation, respectively. If a single, recessive gene conditioned resistance in any of the accessions, the expected ratios of R:S plants would be 1:1, 0:1, and 1:3 in the BC_R , BC_S and F_2 generation, respectively. If the resistance in each of the four sources was allelic to the

Rpp9 gene, all F_2 and test cross progeny from crosses with *Rpp9* would be resistant. If a single, dominant gene independent from the *Rpp9* gene conditioned resistance in any of the accessions, F_2 and test cross progeny from crosses with *Rpp9* would segregate 3:1 and 15:1, respectively. If a single, recessive gene independent from the *Rpp9* gene conditioned resistance in any of the accessions, F_2 and test cross progeny from crosses with *Rpp9* would segregate 13:3 and 1:1, respectively.

RESULTS

Sources of resistance in maize accessions. About 97% of the 1,890 accessions evaluated were susceptible to *P. polysora*. Of the 55 accessions that were rated 0.5 to 1 and appeared to have some partial resistance to southern rust, 30 were collected from South America, 12 from the United States, and 7 from Africa (Table 1). No attempt was made to separate levels of resistance among these lines or to evaluate resistance contributed by these lines to F_1 hybrids when crossed with a susceptible tester.

Uredinia were not observed on six accessions which had a chlorotic-fleck-resistant phenotype similar to the resistance conditioned by the *Rpp9* gene. These included Ames 19016 (Va59), NSL 75976 (IA DS61), PI 186209 (Venezuela yellow flint), PI 186215 (Argentina inbred 2-687), PI 198905 (4F-374 GE 3), and PI 186191 (Uruguay inbred 627). All of these except PI 186191 were crossed with inbred lines carrying the *Rpp9* gene. All but PI 186191 and PI 198905 also were crossed with susceptible sweet corn inbreds.

Preliminary tests of allelism and inheritance. Based on segregation of progeny from testcrosses (i.e., [accessions $\times Rpp9$ line] $\times P_S$), the resistance in PI 186215 appeared to be allelic with the *Rpp9* gene; Ames 19016 appeared to have dominant resistance that was independent of the *Rpp9* gene; and NSL 75976, PI 186209, and PI 198905 appeared to be susceptible or have resistance that was recessive (Table 2). All but 3 of 1,707 test cross progeny from (inbred 2-687 $\times Rpp9$ line) $\times P_S$ were resistant. Therefore, resistance to *P. polysora* in inbred 2-687 appeared to be allelic with or very closely linked to the *Rpp9* gene. Testcross progeny from (Va59 $\times Rpp9$ line) $\times P_S$ segregated 823:300 (R:S) which was not significantly different from a ratio of 3:1 ($P = 0.185$). A 3:1 ratio of segregation would occur if resistance in Va59 was dominant and independent of the *Rpp9* gene. Test cross progeny from (PI 186209 $\times Rpp9$ line) $\times P_S$ and (PI 198905 $\times Rpp9$ line) $\times P_S$ segregated in a ratio that was not significantly different from 1:1 ($P = 0.406$ and 0.085 , respectively). Segregation from the testcross (NSL 75976 $\times Rpp9$ line) $\times P_S$ was slightly different from 1:1 due to more resistant than susceptible progeny. Segregation of 1:1 would be expected in test cross prog-

eny if resistance was inherited only from the *Rpp9* gene; thus, these accessions likely have resistance that is recessive and not expressed in test cross progeny.

Resistance in Va59 appeared to be conditioned by a single, dominant gene based

on field trials in 2001; however, this resistance was not expressed in subsequent greenhouse trials of seedlings (Table 3). All but four F₁ progeny and all but one BC_R progeny were resistant in field trials, as expected. Segregation of BC_S and F₂

progeny in field trials was not significantly different from ratios of 1:1 and 3:1, respectively ($P = 0.699$ and 0.388 , respectively). In two greenhouse trials inoculated at a seedling stage (i.e., prior to four-leaf stage), 93 of 96 P_R plants, 226 of 238 F₁ plants, and 138 of 140 BC_R plants were susceptible. Uredinia were not observed on approximately 10% (7 of 73) of the susceptible control plants which apparently escaped infection. Likewise, the percentage of resistant progeny was not significantly greater than 10% for any of the other generations evaluated in these two trials. Therefore, all resistant plants likely were escapes, and all plants in the trial were susceptible. In the two greenhouse trials inoculated after plants had at least six leaves, only one susceptible control plant escaped infection. All 70 plants of Va59 were resistant, but 18 of 69 F₁ plants and 11 of 68 BC_R plants were susceptible. Progeny of the BC_S and F₂ generations segregated in ratios that were similar ($P = 0.017$ and 0.093) to 1:1 and 3:1, respectively.

Inheritance of resistance and tests of allelism with the *Rpp9* gene. Southern rust was more severe in the late-planted trials than in the early-planted trials in 2005, probably due to the growth stage at which plants initially were inoculated. In rows of susceptible check plants, severity exceeded 40% leaf area infected in the early-planted trials and 70% in the late-planted trials. All plants of the four PI accessions and the three inbreds with the *Rpp9* gene were resistant except for four plants of NSL 75976 (IA DS61), which may have been due to a planting error (Tables 4 and 5). Inheritance of resistance and allelism with the *Rpp9* gene differed among the four PI accessions.

PI 186215 (Argentine inbred 2-687). Segregation of progeny in the F₂ and BC_S generations of the inheritance study was not significantly different ($P > 0.35$) from the ratios expected if resistance in PI 1862215 (inbred 2-687) was conditioned by a single, dominant gene (Table 4). The resistant reaction was a hypersensitive, chlorotic-fleck response to *P. polysora*. There were no intermediate responses. All 651 testcross progeny and 483 F₂ progeny in the test of allelism with the *Rpp9* gene were resistant (Table 5). The resistance in PI 186215 appears to be allelic with the *Rpp9* gene. In all trials (2000, 2001, and 2005) only 3 of 2,357 test cross progeny were susceptible. These three plants probably represent planting or pollination errors.

Ames 19016 (Va59). Segregation of F₂ progeny of Ames 19016 × P_S in the early-planted inheritance trial and segregation of BC_S progeny in both inheritance trials were not significantly different ($P > 0.01$) from those expected if resistance was conferred by a single, dominant gene (Table 4). However, F₂ progeny from the late-

Table 1. Maize accessions with resistance to *Puccinia polysora* race 9 in field trials in Urbana, IL in 1997 and 1998

Rating and accession ^a	Source	Accession name
Accessions rated 0		
Ames 19016	Virginia	Va59
NSL 75976	Iowa	IA DS61
PI 186209	Venezuela	Yellow Flint
PI 186215	Argentina	Inbred 2-687
PI 198905	Argentina	4F-374 GE 3
PI 186191	Uruguay	Inbred 627
Accessions rated 0.5		
PI 484024	United States	Puerto Rico 13
PI 515053	Peru	Loreto 9
PI 515055	Peru	Loreto 11
PI 515097	Peru	San Martin 126
PI 553058	Iowa	OC5
PI 558524	Missouri	Mo6
PI 596502	Iowa	OC15
Accessions rated 1		
Ames 13103	Mississippi	Kyles Long Ear
Ames 19004	Virginia	Va21A
Ames 19006	Virginia	Va22B
Ames 19284	Iowa	C103
Ames 19467	Mexico	Guerrero 3
NSL 174429	California	INB 101LFY
PI 162703	Argentina	Maiz Amarillo Klein
PI 162926	Paraguay	Mkt., Asuncion
PI 171915	Turkey	Niksar, Tokat
PI 177651	Syria	Arnet, Damascus
PI 186182	Uruguay	Inbred 378
PI 186230	Uruguay	Inbred 334
PI 194047	Ethiopia	No. 8815
PI 194387	Ethiopia	No. 9179
PI 194388	Ethiopia	No. 9180
PI 197503	Ethiopia	Various markets
PI 198888	Argentina	4F-35 BK
PI 198893	Argentina	4F-240 BX 16
PI 200183	Israel	No. 162
PI 200304	Yugoslavia	No. 1122
PI 200307	Yugoslavia	No. 1126
PI 217407	Iowa	Ladyfinger
PI 221750	South Africa	E292
PI 221806	South Africa	A256-1
PI 221821	South Africa	E680
PI 240326	Bolivia	No. 9
PI 368830	Yugoslavia	Pukanki Zolti 1294
PI 443762	Columbia	Antioquia 308
PI 443803	Columbia	Antioquia 373
PI 443806	Columbia	Antioquia 377
PI 443986	Columbia	Atlatnico 308
PI 444018	Columbia	Bolivar 308
PI 444029	Columbia	Bolivar 326
PI 444831	Columbia	Huil 1351
PI 444888	Columbia	Magdalena 310
PI 444929	Columbia	Magdalena 362
PI 445526	Columbia	Tolima 403
PI 445578	Columbia	Valle 343
PI 450051	Brazil	Comum 094-R2
PI 515042	Peru	Lima 86
PI 515052	Peru	Loreto 8
PI 515063	Peru	San Martin 7
PI 515064	Peru	San Martin 9
PI 558532	Missouri	Mo17
PI 571725	Peru	San Martin 111
PI 576309	Columbia	Narino 628
PI 577828	Bolivia	BOZM 1155
PI 587136	Kentucky	Ky228

^a Rating: 0 = chlorotic-fleck reaction, no uredinia; 0.5 = very few, very small uredinia or segregating for chlorotic fleck reactions; and 1 = few, small uredinia, mostly on lower leaves.

planted trial did not fit a 3:1 ratio because of a larger number of susceptible plants than expected. Also, about 10% of the progeny in the F₁ and BC_R generations were susceptible in the late-planted trial, even though all of these plants should have been resistant. The resistance conferred by Ames 19016 was not as complete as that conferred by the *Rpp9* gene. F₁ progeny and resistant progeny in segregating generations often had an intermediate reaction with less than 10% leaf area infected. Possibly, some plants with intermediate and susceptible reactions were misclassified, particularly in the late-planted trial, in which southern rust was more severe. Alternatively, the resistance of Ames 19016 may be related to plant growth stage (i.e., adult-plant resistance) and a greater number of plants inoculated as seedlings in the late-planted trial were susceptible.

Based on the test of allelism, the resistance in Ames 19016 appears to be different than that conferred by the *Rpp9* gene (Table 5). Segregation of test cross and F₂ progeny in the early-planted trial support the hypothesis that resistance in Ames 19016 is conferred by a single, dominant gene that is independent of the *Rpp9* gene. In the late-planted allelism trial, the number of susceptible plants among test cross and F₂ progeny was larger than expected, which was similar to reactions of F₂ progeny in the late-planted inheritance trial.

PI 186209 (Venezuelan flint). Segregation of F₂ progeny in the late-planted inheritance trial and BC_R progeny in both inheritance trials was not significantly different ($P > 0.01$) from expected if resistance in PI 186209 was conditioned by a single, recessive gene (Table 4). The number of resistant plants among F₂ progeny in the early-planted trial was larger than expected based on a single, recessive gene hypothesis. The resistant reaction of PI 186209 was similar to the hypersensitive, chlorotic-fleck reaction of the *Rpp9* gene. Segregation of test cross progeny in both allelism trials fit a 1:1 ratio ($P = 0.96$ and 0.28 , respectively), which would be expected if resistance was conferred only by the *Rpp9* gene and a recessive gene from PI 186209 was not expressed (Table 5). Segregation in the F₂ generation fit a 13:3 ratio ($P = 0.03$ and 0.04), which would be expected if PI 186209 carried a recessive gene for a chlorotic-fleck reaction; however, the segregation in the F₂ generation of both allelism trials more closely fit ($P = 0.32$ and 0.44) a 3:1 ratio that would be expected if resistance was conferred only by the *Rpp9* gene.

NSL 75976 (IA DS61). Although NSL 75976 appeared to be resistant in initial screening of germplasm and in the inheritance trials, few of the progeny from crosses with NSL 75976 were resistant (Table 4). Only 1 F₂ progeny of NSL 75976 × P_S was resistant in the late-

planted trial and only 11 of 374 progeny were resistant in the early-planted trial. Likewise, none of the BC_R progeny were resistant in the late-planted trial and only 7 of 52 progeny were resistant in the early-planted trial. Possibly, resistance in NSL 75976 is co-dominant or polygenic. If this resistance is co-dominant, progeny with intermediate phenotypes may have been classified as susceptible based on our method of scoring rust reactions. Segregation of test cross progeny and F₂ progeny in the late-planted allelism trial were not significantly different ($P > 0.28$) from 1:1 and 3:1, which would be expected if resistance in these generations was inherited solely from the *Rpp9* gene (Table 5).

DISCUSSION

A number of potential sources of general and specific resistance to *P. polysora* were identified from 1,890 PI accessions that were representative of temperate-adapted maize germplasm collected throughout the world. The number of uredinia was reduced and southern rust was less severe on 55 accessions that appeared to have general resistance. Many of those accessions were from South America. Quantitative trait loci with minor effects on southern rust resistance have been mapped to chromosomes 3 and 4 in one of these sources, Mo17 (7).

Uredinia did not form on six accessions that presumably carried specific resistance to *P. polysora* race 9. Resistance to *P. poly-*

Table 2. Segregation of progeny from testcrosses of plant introduction accessions that were potential sources of resistance to *Puccinia polysora* and field corn inbred lines carrying the *Rpp9* gene

Crosses and controls tested and PI accession sources of resistance	Hypothesized ratio ^a	Reaction of progeny		χ^{2b}	<i>P</i> ^c
	R:S	R	S		
(Accession × <i>Rpp9</i> line) × susceptible tester (M32, M12, or Cr44)					
PI 186215 (Inbred 2-687)	1:0	1,703	3		
Ames 19016 (Va59)	3:1	823	300	1.76	0.185
NSL 75976 (IA DS 61)	1:1	478	399	7.12	0.008
PI 186209 (Yellow Flint)	1:1	135	149	0.69	0.406
PI 198905 (4F-374 GE 3)	1:1	146	177	2.98	0.085
Controls: <i>Rpp9</i> -resistant and susceptible					
W64a <i>Rpp9</i> × Oh43 <i>Rpp9</i>		156	0		
W64a <i>Rpp9</i> × B37 <i>Rpp9</i>		153	0		
Bonus (susceptible check)		3	300		

^a Hypothesized ratios for testcrosses: 1:0, allelic or very closely linked to *Rpp9*; 3:1, single, dominant gene independent of *Rpp9*; 1:1, no resistance or recessive resistance.

^b Chi-square value from a goodness-of-fit test based on the hypothesized ratio.

^c Probability associated with the chi-square value from the goodness-of-fit test.

Table 3. Segregation analysis for chlorotic fleck resistance to *Puccinia polysora* among crosses of Ames 19016 (Va59) and susceptible sweet corn inbreds evaluated in field and greenhouse trials in 2001

Trials and generations	Hypothesized ratio ^a	Reaction of progeny		χ^2 ^b	<i>P</i> ^c
	R:S	R	S		
Field trials					
Bonus (susceptible check)	0:1	0	42		
P _R	1:0	22	0		
F _I	1:0	69	4		
BC _R	1:0	102	1		
BC _S	1:1	86	81	0.15	0.699
F ₂	3:1	101	28	0.75	0.388
Greenhouse trials: <4 leaves ^d					
Bonus (susceptible check)	0:1	7	67		
P _R	1:0	3	93		
F _I	1:0	12	226		
BC _R	1:0	2	138		
BC _S	1:1	35	315	225	<0.001
F ₂	3:1	2	207	611	<0.001
Greenhouse trials: >6 leaves ^d					
Bonus (susceptible check)	0:1	1	53		
P _R	1:0	70	0		
F _I	1:0	51	18		
BC _R	1:0	57	11		
BC _S	1:1	45	25	5.71	0.017
F ₂	3:1	57	11	2.82	0.093

^a Hypothesized ratios based on a single dominant gene for resistance.

^b Chi-square value from a goodness-of-fit test based on the hypothesized ratio.

^c Probability associated with the chi-square value from the goodness-of-fit test.

^d Greenhouse trials in which plants were inoculated with *P. polysora* as soon as leaf whorls were large enough to retain urediniospore suspensions (i.e., <4 leaves) or when plants had at least 6 leaves.

Table 4. Analysis of segregation to determine inheritance of resistance to *Puccinia polysora* in four plant introduction accessions in early and late planted field trials in 2005

PI source of resistance, trials, and generations	Hypothesized ratio ^c	Early planted trial ^a				Late planted trial ^b			
		Reaction of progeny		χ^{2d}	<i>P</i> ^e	Reaction of progeny		χ^{2d}	<i>P</i> ^e
	R:S	R	S			R	S		
PI 186215 (Argentine inbred 2-687)									
Bonus (susceptible check)	0:1	0	23			0	32		
P _R (PI 186215)	1:0	12	0			...	...		
F ₁	1:0	23	3			27	0		
BC _S	1:1	115	121	0.15	0.696	124	110	0.84	0.36
F ₂	3:1	151	48	0.08	0.775	156	45	0.73	0.394
Ames 19016 (Va59)									
Bonus (susceptible check)	0:1	0	53			0	85		
P _R (Ames 19016)	1:0	117	0			107	0		
F ₁	1:0	111	1			117	12		
BC _R	1:0	56	2			61	6		
BC _S	1:1	67	89	3.1	0.078	92	125	5.0	0.025
F ₂	3:1	204	80	1.52	0.217	183	167	96.3	<0.001
PI 186209 (Venezuelan flint)									
Bonus (susceptible check)	0:1	0	70			0	62		
P _R (PI 186209)	1:0	7	0			...	...		
BC _R	1:1	13	26	4.3	0.037	10	24	5.8	0.016
BC _S	0:1	10	107			3	110		
F ₂	1:3	109	190	20.9	<0.001	64	184	0.1	0.077
NSL 75976 (IA DS61)									
Bonus (susceptible check)	0:1	0	48			0	67		
P _R (NSL 75976)	1:0	20	4			18	0		
F ₁	1:0	0	19			0	23		
BC _R	1:1	7	45	27.8	<0.001	0	60		<0.001
BC _S	0:1	2	283			2	302		
F ₂	1:3	11	363	97.1	<0.001	1	429	141	<0.001

^a Early planted trials inoculated while plants were between the 7-leaf and row tassel growth stages.

^b Late planted trials inoculated while plants were between the 4-leaf and 8-leaf growth stages.

^c Hypothesized ratios for PI 186215 and Ames 19016 based on a single, dominant gene for resistance; ratios for PI 186209 and NSL 75976 based on a single, recessive gene for resistance.

^d Chi-square value from a goodness-of-fit test based on the hypothesized ratio.

^e Probability associated with the chi-square value from the goodness-of-fit test.

sora appeared to differ among four of these six accessions. The Argentine inbred 2-687 (PI 186215) conferred a chlorotic-fleck, hypersensitive-type resistance conditioned by a single, dominant gene. Similar to several other sources of chlorotic-fleck resistance (3,6,7,10,22,30), the resistance in inbred 2-687 appeared to be allelic with or linked to the *Rpp9* gene originally identified from South African accession PI 186208 (19,29).

Resistance in Va59 (Ames 19016) was effective in F₁ progeny and appeared to be dominant and simply inherited; however, in inheritance and allelism trials, this resistance appeared to be a slow-rusting or incomplete resistance that was effective in adult plants but not in young seedlings. Hu et al. (9) also have observed differences in reactions of three-leaved seedlings and adult plants to *P. polysora*. In adult plants, fewer *P. polysora* uredinia formed on BC₂F₂ families homozygous for the *Rp1-DJ* compound gene for resistance to *P. sorghi* than on BC₂F₂ families that carried the susceptible alleles (*rp1/rp1*). At the three-leaf growth stage, resistance to *P. polysora* did not differ among *Rp1-DJ* and *rp1/rp1* families. The *Rp1-DJ* compound gene occurs in the same chromosomal region as the *Rpp9* gene. However, based on our tests of allelism, the resistance in Va59 is not linked with the *Rpp9* or *Rp1-*

DJ compound genes. The resistance in Va59 may be an additional source that can be useful in commercial maize in situations in which infection occurs after seedling stages, such as recent outbreaks of southern rust in the U.S. Corn Belt.

The resistance in PI 186209 (Venezuelan flint) and NSL 75976 (IA DS61) was not effective in hybrid combination (F₁ progeny) and, thus, probably has limited value in commercial maize. Resistance in PI 186209 may be conditioned by a single recessive gene and resistance in NSL 75976 may be co-dominant.

Resistant lines identified from this collection of accessions were not evaluated for reactions to multiple races of *P. polysora* or to populations of *P. polysora* common in tropical regions of the world. Isolates of *P. polysora* appear to differ in specific virulence (2,19,28) even though the aecial stage is unknown and teliospores are not known to be functional (8) which, in theory, would slow the development of virulent races. Nevertheless, simply inherited resistance to *P. polysora* has not been durable in Africa or Hawaii (25,28). Similarly, Roberts (17) observed in the early 1960s that four-leaved seedlings of the source of the *Rpp9* gene, PI 186208, were susceptible to 8 of 13 *P. polysora* isolates evaluated, including isolates from Florida and Mexico. Subsequently, the *Rpp9* gene

or a closely linked gene or genes was effective in trials in Florida, Mississippi, North Carolina, and China from the 1970s through 2006 (3,6,7,10,16,22,30). Possibly, the *Rpp9* gene or other simply inherited resistance to *P. polysora*, such as the resistance in Va59, may be effective for a prolonged period in situations where selection pressure is not intense, such as the U.S. Corn Belt. The *Rp1-D* gene, which conveys a similar type of chlorotic-fleck, hypersensitive resistance to *P. sorghi*, effectively controlled common rust in sweet corn in North America from the mid 1980s until 1999, when a virulent race occurred (15).

We did not extensively evaluate an Argentine accession, PI 198905 (4F-374 GE 3), or an accession from Uruguay, PI 186191 (inbred 627), that were resistant in our initial trial, due to a limited quantity of seed harvested from pollinations. Also, we did not evaluate an Argentine accession, PI 198902 (4F-345 CN 12), that was resistant to 9 of 13 isolates screened by Roberts (19). These and other accessions, particularly those from South America, may carry southern rust resistance genes other than the *Rpp9* gene. Larger collections of *Rpp* genes and collections of virulent isolates of *P. polysora* would provide resources necessary to improve our understanding of the genetic mechanisms and molecular biology

Table 5. Analysis of segregation to test resistance to *Puccinia polysora* in four plant introduction accessions for allelism with the *Rpp9* gene in early and late planted field trials in 2005

PI source of resistance, trials, and generations	Hypothesized ratio ^c	Early planted trial ^a				Late planted trial ^b			
		Reaction of progeny				Reaction of progeny			
	R:S	R	S	χ^2 ^d	<i>P</i> ^e	R	S	χ^2 ^d	<i>P</i> ^e
B37 <i>Rpp9</i>	1:0	60	0			67	0		
Oh43 <i>Rpp9</i>	1:0	45	0			44	0		
W64a <i>Rpp9</i>	1:0	16	0			8	0		
PI 186215 (Argentinean inbred 2-687)									
P _R × <i>Rpp9</i> line	1:0	58	0			56	0		
(P _R × <i>Rpp9</i> line) × P _S	1:0	331	0			320	0		
(P _R × <i>Rpp9</i> line) F ₂	1:0	245	0			238	0		
Ames 19016 (Va59)									
P _R × <i>Rpp9</i> line	1:0	20	0			26	0		
(P _R × <i>Rpp9</i> line) × P _S	3:1	228	65	0.68	0.421	273	171	43.2	<0.001
(P _R × <i>Rpp9</i> line) F ₂	15:1	342	41	12.9	0.003	397	79	86.9	<0.001
PI 186209 (Venezuelan flint)									
(P _R × <i>Rpp9</i> line) × P _S	1:1	173	174	0	0.957	158	178	1.2	0.275
(P _R × <i>Rpp9</i> line) F ₂	13:3	320	95	4.7	0.031	265	80	4.5	0.035
NSL 75976 (IA DS61)									
P _R × <i>Rpp9</i> line	1:0	40	0			47	0		
(P _R × <i>Rpp9</i> line) × P _S	1:1	172	189	0.81	0.371	208	188	1.0	0.315
(P _R × <i>Rpp9</i> line) F ₂	3:1	362	77	13	<0.001	279	105	1.1	0.289

^a Early planted trials inoculated while plants were between the 7-leaf and row tassel growth stages.

^b Late planted trials inoculated while plants were between the 4-leaf and 8-leaf growth stages.

^c Hypothesized ratios for PI 186215 based on a single gene allelic with the *Rpp9* gene; ratios for Ames 19016 based on a single, dominant gene independent from the *Rpp9* gene; ratios for PI 186209 based on a single, recessive gene for resistance; ratios for NSL 75976 based on resistance solely from the *Rpp9* gene.

^d Chi-square value from a goodness-of-fit test based on the hypothesized ratio.

^e Probability associated with the chi-square value from the goodness-of-fit test.

associated with this host–pathogen interaction.

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