Resistance Genes in the *rp1* Region of Maize Effective Against *Puccinia sorghi* Virulent on the *Rp1-D* Gene in North America

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ABSTRACT

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Resistance in sweet corn conferred by the *Rp1-D* gene has controlled common rust, caused by *Puccinia sorghi*, in North American corn for nearly 15 years. Eleven isolates of *P. sorghi* virulent on corn with the *Rp1-D* gene were collected from Rp-resistant corn in 1999 from Wisconsin, Illinois, New York, and Minnesota. Isolates were increased on susceptible sweet corn. Urediniospores of nine isolates were bulked. Reactions of individual Rp genes in the *rp1* region and reactions of linked combinations of Rp genes in the *rp1* region (i.e., compound rust resistance genes) were evaluated against the bulked population of *P. sorghi* in several greenhouse trials. Reactions of individual and compound Rp genes also were evaluated against individual isolates of *P. sorghi*. Each trial contained at least two replicates of several lines with Rp genes and one susceptible check. Five to 10 two-leaved seedlings per line were inoculated at least twice with a suspension of urediniospores. Ten days after inoculation, rust reactions were rated: + = sporulating uredinia, – = no sporulating uredinia, and I = chlorotic or necrotic tissue surrounding small uredinia. Four single genes, *Rp1-E, Rp-G, Rp1-I,* and *Rp1-K,* and eight compound genes, *Rp1-JFC, Rp1-JC, Rp-GI, Rp-G5, Rp-GDJ, Rp-G5JD, Rp-G5JC,* and *Rp-GFJ,* conferred resistance. Additional characterization of virulence in North American populations of *P. sorghi* that are avirulent against *Rp1-D* is necessary to determine if these genes will be as widely effective as the *Rp1-D* gene has been. Two subpopulations of *P. sorghi* were detected from the bulked population after it was sequentially cultured for at least five cycles on seedlings with *Rp1-C* or with *Rp1-J*. The subpopulation cultured on *Rp1-J* was avirulent on lines with *Rp1-C/L/N, Rp1-B,* and *Rp1-M*; whereas the subpopulation cultured on *Rp1-C* was virulent on lines with each of these genes. Both subpopulations were virulent on lines with *Rp1-D*.

Common rust, caused by *Puccinia sorghi* Schwein., is an important disease of sweet corn (*Zea mays* L.) in the midwestern United States. Single, dominant resistance genes (i.e., Rp genes) have been used successfully for the past 15 years to control common rust on sweet corn in North America. Most sweet corn hybrids used for processing and grown in the Midwest in the middle or late season have Rp-resistance. Rp-resistance is characterized by hypersensitive reactions resulting in chlorotic or necrotic flecks with little or no formation of urediniospores. Many, but not all, Rp-resistant sweet corn hybrids carry the *Rp1-D* gene. The *Rp1-D* gene has been preferred over other Rp genes because it has been effective against all biotypes of *P. sorghi* in the continental United States except for an isolate collected in Illinois in 1982 (2) and an isolate collected in Kansas in 1990 (14). Virulence against other Rp genes occurs with varying frequency in North American populations of *P. sorghi* (5,6,10,14,15).

Populations of the rust fungus virulent on corn with *Rp1-D* occur throughout the world (1,3,4,6,7,10,14). In August and September 1999, isolates of *P. sorghi* were collected in Illinois, Wisconsin, Minnesota, Michigan, and New York from sweet corn hybrids known to possess Rp genes (17). Rust severity was as high as 40% in some fields of these Rp-resistant hybrids. These isolates of *P. sorghi* were virulent on greenhouse-grown seedlings with the *Rp1-D* gene (17) and on seedlings of 121 of 125 sweet corn hybrids that had Rp genes that were effective against the population of *P. sorghi* prevalent in the continental United States prior to 1999 (M. C. Pate and J. K. Pataky, *unpublished*). Control of common rust with Rp-resistance conferred by the *Rp1-D* gene will not be effective against this new population of *P. sorghi*.

The *rp1* region of maize chromosome 10 is a complex structure of rust resistance genes (10). In the 1960s, Hooker and coworkers identified 14 genes that were given the *Rp1* designation (*Rp1-A* to *Rp1-N*) because they mapped to a single locus (6,19). Two genes that were more than one map unit from *rp1* were designated *Rp5* and *Rp6* (6,19). Subsequently, *Rp1-G* was designated *Rp-G* because it was about two map units from *rp1* (11). These Rp genes and other Rp genes on chromosomes 3 and 4 were backcrossed by Hooker into field corn inbred lines R168 and B14. These lines have been used to monitor virulence in populations of *P. sorghi* (4–7,10,14–16). Some of these genes also have been incorporated into sweet corn germ plasm.

Hooker observed that the Rp complex and nearby regions of chromosome 10 may have evolved through repeated duplication with subsequent structural modification of individual genes (7,8). In the past decade, Hulbert (10) used restriction fragment length polymorphism (RFLP) markers and a collection of 11 biotypes of *P. sorghi* with different virulence phenotypes to analyze the structural complexity of the *rp1* region. All but one of the previously characterized Rp genes were identified, but several could not be differentiated based on reactions to this collection of *P. sorghi*. The *Rp1-E, Rp1-I,* and *Rp-K* genes had the same phenotypes in response to this collection of *P. sorghi* as did the *Rp1-A* and *Rp1-F* genes and the *Rp1-C, Rp1-L,* and *Rp1-N* genes (10). By using flanking markers and this collection of rust isolates to identify recombination events, Hu and Hulbert (9) developed a set of "compound genes" for rust resistance in which two or more Rp genes in the *rp1* region were linked in coupling phase. Theoretically, these compound Rp genes should be more durable than single Rp genes because multiple genes for virulence are necessary for *P. sorghi* to overcome all component genes of compound rust resistance. Nonetheless, compound Rp genes can be manipulated in breeding programs as if they were single genes because they are very closely linked. Hulbert et al. (12,13) released the *Rp1-DJ, Rp-GFJ, Rp-GDJ,* and *Rp1-JFC* compound Rp genes in *su* and *sh2* sweet corn backgrounds. These compound genes and other compound rust resistance genes have been incorporated rapidly into elite sweet corn inbreds because of their potential usefulness if virulence against *Rp1-D* becomes frequent in North America.

Control of common rust with Rp-resistance will require that sweet corn hybrids be developed from inbreds with effective single or compound Rp genes if biotypes of *P. sorghi* that were virulent on *Rp1-D* in the Midwest in 1999 become prevalent in North America. The objective of this re-

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search was to determine which compound and single Rp genes in the *rp1* region were effective against the population of *P. sorghi* that was virulent against *Rp1-D* and widely prevalent in the Midwest in 1999.

MATERIALS AND METHODS

Isolates of *P. sorghi*. Isolates of *P. sorghi* from Rp-resistant sweet corn and field corn were collected in September 1999 from Mendota, Rock Falls, and Dekalb, IL; Sun Prairie, Madison, and Ripon, WI; and Rochester, Stanton, and Le Sueur, MN. Inoculum of these isolates was increased on seedlings of two or three susceptible sweet corn hybrids (e.g., Goldilocks, Snow White, Stylepak, Crisp n Sweet 710) that were inoculated with urediniospores from about 40 to 400 uredinia per isolate. Ten to 20 pieces of leaf tissue about 1 cm^2 with sporulating uredinia were suspended in 50 to 200 ml of water to which one or two drops of Tween 20 were added. After thoroughly agitating to release urediniospores, the spore suspension and leaf tissue were placed in whorls of four- to six-leaved seedlings growing in a greenhouse. Inoculated seedlings were placed on different greenhouse benches or spaced far apart to prevent contamination between isolates.

When *P. sorghi* began sporulating on seedlings about 7 days after inoculation, urediniospores were collected with cyclone spore collectors (ERI Machine Shop, Iowa State University, Ames). Ten two-leaved seedlings of one susceptible and five Rp-resistant sweet corn hybrids were inoculated with urediniospores collected for each isolate. All isolates were virulent on hybrids with the *Rp1-D* gene. Half of the second cycle of urediniospores of each isolate then were bulked. To evaluate reactions of Rp genes, seedlings were inoculated with the bulked collection of urediniospores. Urediniospores and infected leaves were collected from susceptible plants in each trial and used as inocula in subsequent trials.

Reactions of Rp genes to bulked isolates. Reactions of lines with the 14 Rp genes originally designated *Rp1*, and lines with 11 compound Rp genes were evaluated in several greenhouse trials at the University of Illinois, Urbana and at Kansas State University, Manhattan. Each trial included two to five replicates of lines with Rp genes and at least one susceptible check that did not have a known Rp gene. Each experimental unit was 5 to 10 seedlings grown in a 10-cm-diameter pot in a greenhouse. The first two trials at Illinois included two replicates of two sources of R168 inbreds with each of the Rp genes at *rp1*, at least two different sweet corn lines with one of six different Rp genes (*Rp1-D, Rp1-E, Rp1-F, Rp-G, Rp1-I,* or *Rp1-K*), and at least two different sweet corn lines with 1 of 11 different compound Rp genes, including *Rp1-DJ, Rp1-JF, Rp1-JFC, Rp1-JC, Rp1-D5, Rp-GI, Rp-G5, Rp-GDJ, Rp-G5JD, Rp-G5JC,* and *Rp-GFJ*. All of these resistance genes in a field corn inbred background, H95, also were evaluated in a trial at Kansas. In eight subsequent trials at Illinois, reactions were evaluated in two to five replicates of an assortment of sweet corn inbreds and breeding lines with various single or compound Rp genes at *rp1*. Many of the sweet corn lines were at various stages of backcrossing and/or selfing; therefore, resistant and susceptible segregates occurred in lines with effective Rp genes. These trials also included several rust resistant lines (e.g., sweet corn breeding lines, PI accessions, etc.) for which the gene conferring Rp-resistance was not known.

In each trial, two- to four-leaved seedlings were inoculated as described above. Seedlings were inoculated at least twice in each trial. Ten to 14 days after inoculation, rust reactions were rated: – = no sporulating uredinia, + = sporulating uredinia, and I = chlorotic or necrotic tissue surrounding small, sporulating uredinia. Because most sweet corn breeding lines were segregating for Rp genes, effectiveness of the Rp genes in each line was determined from consistency of segregating or homogeneous resistant (– or I) phenotypes among replicates and multiple trials.

Reactions of Rp genes to individual isolates. Urediniospores of each of the nine isolates of *P. sorghi* that were combined to form the bulked population and two additional isolates from DeForest, WI, and Hall, NY, were increased on and collected from susceptible sweet corn hybrids as described above. Reactions to each isolate were evaluated in separate trials. Each trial included two replicates of the R168 inbreds or sweet corn lines with 10 of the 14 Rp genes originally designated *Rp1* (all except *Rp1-B, Rp1-H, Rp1-L,* and *Rp1-J*) and two replicates of lines with seven of the compound rust genes (*Rp1-JF, Rp1-JFC, Rp-GI, Rp-G5, Rp-GDJ, Rp-G5JC,* and *Rp-GFJ*). Two sources of each Rp-resistance were used in each trial except for *Rp1-M,* for which seed was limited. Seedlings were inoculated and rated as described above. Effectiveness of Rp genes was determined, as described above, from consistency of segregating or homogeneous resistant (– or I) phenotypes among replicates, multiple sources of the Rp genes, and multiple trials.

Serial inoculation of lines with *Rp1-C* or *Rp1-J*. A dent corn inbred line, H95, with either the *Rp1-C* or the *Rp1-J* gene was serially inoculated as described above in greenhouse trials in Kansas to produce two subpopulations of *P. sorghi* from the bulked population. After at least five asexual generations of serial inoculation on H95 *Rp1-C* or H95 *Rp1-J*, virulence phenotypes of the two subpopulations, designated I99C or I99J, respectively, were determined as described above.

RESULTS

Reactions of Rp genes to bulked isolates. Four Rp genes, *Rp1-E, Rp1-I, Rp1-K,* and *Rp-G,* conferred resistance in all trials (Table 1). Since some lines carrying Rp genes were segregating, as many as 60% of the seedlings were susceptible for any given source of a gene; however, reactions of segregating and nonsegregating sources were consistent over replicates and trials.

Seven compound rust genes, *Rp1-JFC, Rp-GI, Rp-G5, Rp-GDJ, Rp-G5JD, Rp-G5JC,* and *Rp-GFJ*, were effective in all trials (Table 1). The *Rp1-JC* compound gene was effective in six of seven trials. All effective compound genes except *Rp-G5JD* were segregating in sweet corn lines. At least three sweet corn lines presumed to carry the compound genes *Rp1-JC, Rp-GDJ,* and *Rp-G5JC* based on resistant reactions in field trials in 1999 were susceptible in all greenhouse trials in which they were assayed. Because these lines were consistently susceptible while others consistently segregated for resistance, we believe that either they do not carry the compound gene or an effective component of the compound gene was lost, possibly as a result of a crossover event.

The R168 lines with the *Rp1-L* gene were resistant in trials at Illinois, but H95 lines with *Rp1-L* were susceptible in a trial at Kansas. In subsequent trials in Kansas, the R168 *Rp1-L* lines were resistant (– reactions) to subpopulations I99C and I99J, but they were susceptible to two isolates, IN-1 and IN-2, that were virulent against *Rp-G* (10). Also, the R168 *Rp1-L* lines were identical to *Rp-G* when tested with an *Rp1* probe. Therefore, we believe that the R168 *Rp1-L* lines actually have the *Rp-G* gene.

Lines with eight Rp genes were susceptible to the bulked population of *P. sorghi* (Table 1). Susceptible reactions were observed on lines with *Rp1-A, Rp1-B, Rp1-C, Rp1-D, Rp1-F, Rp1-H, Rp1-J,* and *Rp-5*. Uredinia were not observed on about 5% of the seedlings carrying these genes and about 2% of the susceptible sweet corn hybrid checks included in these trials. These plants probably escaped infection.

Three compound genes, *Rp1-DJ, Rp1-JF,* and *Rp-5D,* did not confer resistance when inoculated with the bulked population (Table 1). Uredinia were not observed on about 3% of the seedlings of these lines, which is similar to the percentage of susceptible checks presumed to have escaped infection.

Reactions to subpopulations from serial inoculations. Lines carrying the *Rp1-C, Rp1-N,* and *Rp1-M* genes generally were scored as susceptible when inoculated with the bulked population, but intermediate reaction types were observed sometimes (Table 1). When seedlings of H95 lines homozygous for these genes were evaluated under high inoculum density, a

mixture of reaction types was observed. All seedlings had numerous, well-developed uredinia and variable numbers of chlorotic flecks. Urediniospores harvested from lines with *Rp1-M* were used to reinoculated seedlings with *Rp1-M*, and urediniospores from those seedlings were used to inoculate seedlings with *Rp1-M* again. Fully susceptible reactions were observed when lines with *Rp1-M* were inoculated with urediniospores that had been cycled through two uredinial generations on seedlings with *Rp1-M*. Seedlings with the *Rp1-C* and *Rp1-N* genes had mixed infection types when inoculated with this cycled inoculum. When lines carrying the *Rp1-J* gene or the *Rp1-JF* and *Rp1-DJ* compound genes were inoculated with urediniospores cycled on *Rp1-M*, resistant reactions were observed.

After sequentially culturing the bulked population of *P. sorghi* on seedlings with *Rp1-C* or with *Rp1-J* for at least five cycles, two distinct virulence phenotypes were observed. The I99J subpopulation was avirulent on lines with *Rp1-C/L/N*, *Rp1-B*, and *Rp1-M*; whereas the I99C subpopulation was virulent on lines with each of these genes but avirulent on lines with *Rp1-H* and *Rp1-J*. Both subpopulations were virulent on lines with *Rp1-D*.

Reactions of Rp genes to individual isolates. When lines with 10 single Rp genes or seven compound Rp genes were inoculated in Illinois with 11 individual isolates of *P. sorghi*, results were similar to trials using the bulked population of *P. sorghi*. Four single Rp genes, *Rp1-E*, *Rp1-I*, *Rp1-K*, and *Rp-G*, and seven compound Rp genes, *Rp1-JFC*, *Rp-GI*, *Rp-G5*, *Rp-GDJ*, *Rp-G5JC*, and *Rp-GFJ*, conferred resistance to all 11 isolates (Table 2). One compound gene, *Rp1-JF*, and five single Rp genes, *Rp1-A*, *Rp1-C*, *Rp1-D*, *Rp1-F*, and *Rp1-N*, were ineffective (Table 2). Reactions of *Rp1-M* varied among isolates, as there were only a few plants assayed per isolate and several had intermediate reactions.

DISCUSSION

Four individual genes in the *rp1* region (*Rp1-E*, *Rp1-I*, *Rp1-K*, and *Rp-G*) and eight compound Rp genes (*Rp1-JFC*, *Rp1-JC*, *Rp-GI*, *Rp-G5*, *Rp-GDJ*, *Rp-G5JD*, *Rp-G5JC*, and *Rp-GFJ*) conferred resistance to the population of *P. sorghi* that was widespread in the Midwest in 1999 and virulent on sweet corn hybrids that carried the *Rp1-D* gene. Since many of these single and compound Rp genes are in advanced stages of backcrossing or inbreeding in several commercial sweet corn breeding programs, an assortment of resistant hybrids may be available in a year or two. The long-term effectiveness of these Rp-hybrids will depend on the rapidity with which virulence becomes frequent and widespread in North American populations of *P. sorghi*. In Hawaii, virulence against *Rp1-D* occurred about 4 years after the conversion of Hawaiian corn to *Rp1-D* resistance (3). Virulence against *Rp1-D* was followed in less than 2 years by virulence against *Rp-G* and *Rp-Td*, an Rp-resistance gene from *Tripsacum dactyloides* (1,3). None of the Rp genes are currently effective in Hawaii (3).

Except for the *Rp1-D* and *Rp1-F* genes, the Rp genes that have been most effective against populations of *P. sorghi* prevalent in the continental United States in the past 35 years were the ones that were effective against the population of *P. sorghi* that was widespread in the Midwest in 1999 and virulent on *Rp1-D*. In 1966, seven genes near the *rp1* region (*Rp1-B*, *Rp1-D*, *Rp1-F*, *Rp-G*, *Rp1-I*, *Rp1-K*, and *Rp-5*) conditioned effective resistance to naturally occurring biotypes of *P. sorghi* in a nursery in Urbana (6). In 1984 and 1986, six genes near the *rp1* region (*Rp1-D*, *Rp1-E*, *Rp1-F*, *Rp-G*, *Rp1-I*, and *Rp1-K*) conditioned resistance to populations of *P. sorghi* in Urbana, while *Rp1-A* and *Rp1-B* conditioned

Table 1. Reactions from greenhouse trials of maize lines with 1 of 15 single Rp genes in the *rp1* region or 1 of 11 compound Rp genes inoculated with a bulked population of isolates of *Puccinia sorghi* collected in 1999 from Mendota, Rock Falls, and Dekalb, IL; Sun Prairie, Madison, and Ripon, WI; and Rochester, Stanton, and Le Sueur, MN

	Reaction in no. of trials[a]		Percent resistant plants[b]	
Rp genes	**R**	**S**	**–**	**I**
Single Rp genes				
Rp1-A	0	5	0	0
-F	0	4	0	0
Rp1-B	0	3	0	0
Rp1-C	1	4	50	50
-L	6	1	95	5
-N	4	2	80	10
Rp1-D	0	10	0	0
Rp1-E	5	0	98	2
-I	7	0	100	0
-K	5	0	100	0
Rp1-H	0	1	0	0
-J	0	3	0	0
Rp1-M	2	2	50	50
Rp-G	8	0	99	1
Rp-5	0	2	0	0
Compound Rp genes				
Rp1-DJ	0	4	0	0
Rp1-JF	0	3	0	0
Rp1-JFC	7	0	75	25
Rp1-JC	6	1	60	40
Rp-5D	0	4	0	0
Rp-GI	4	0	99	1
Rp-G5	3	0	99	1
Rp-GDJ	7	0	100	0
Rp-G5JD	3	0	100	0
Rp-G5JC	5	0	100	0
Rp-GFJ	5	0	100	0

[a] R = resistant or S = susceptible reactions based on consistent reactions of lines in replicates and trials considering segregation of lines that were not homozygous.

[b] Percent resistant plants from all trials rated: – = no sporulating uredinia, and I = chlorotic or necrotic tissue surrounding small, sporulating uredinia.

Table 2. Reactions of maize lines with 1 of 10 single Rp genes in the *rp1* region or 1 of 7 compound Rp genes inoculated with 11 isolates of *Puccinia sorghi* collected in 1999 from Mendota, Rock Falls, and Dekalb, IL; Sun Prairie, Madison, Ripon, and DeForest, WI; Rochester, Stanton, and Le Sueur, MI; and Hall, NY

	Reactions[a] to no. of isolates[b]	
Rp genes	**Resistant**	**Susceptible**
Single Rp genes		
Rp1-A	0	11
-F	0	11
Rp1-C	0	11
-N	0	7
Rp1-D	0	11
Rp1-E	11	0
-I	11	0
-K	10	0
Rp1-M	5	4
Rp-G	11	0
Compound Rp genes		
Rp1-JF	0	8
Rp1-JFC	8	0
Rp-GI	10	0
Rp-G5	10	0
Rp-GDJ	10	0
Rp-G5JC	9	0
Rp-GFJ	10	0

[a] Resistant or susceptible reactions to each of 11 isolates of *P. sorghi* based on consistent reactions of lines in replicates and trials considering segregation of lines that were not homozygous.

[b] All Rp genes were not tested against all isolates due to poor germination and limited amounts of seed.

resistance only in 1986 (15). Rust severity was low on lines with the *Rp1-M* gene, but uredinia were fully developed (i.e., type 4 uredinia). R168 lines with the Rp genes were planted in a total of 23 trials at 14 different locations from 1987 to 1991 (5). Virulence was not observed against *Rp1-D*. For *Rp1-E, Rp1-F, Rp-G, Rp1-I,* and *Rp1-K*, virulence was observed in 1, 3, 4, 10, and 6 trials, respectively. In trials at St. Paul, MN, in 1987 to 1989 and 1991, virulence against *Rp1-A, Rp1-D, Rp1-E, Rp1-F, Rp-G, Rp1-I,* and *Rp1-K* was infrequent (5).

Examination of the Rp genes in near-isogenic lines developed by Hooker and coworkers with current collections of rust isolates did not distinguish between *Rp1-A* and *Rp1-F*, between *Rp1-H* and *Rp1-J*, among *Rp1-E, Rp1-I,* and *Rp1-K*, or among *Rp1-C, Rp1-L,* and *Rp1-N* (10). Reactions of lines with these genes in response to inoculation with the bulked population of *P. sorghi* collected in 1999 grouped these resistance genes in a similar manner except for the R168 lines with *Rp1-L*, which is probably *Rp-G* rather than *Rp1-L*.

Most of the compound Rp genes that were effective against the bulked population from 1999 contain one of two effective single Rp genes, *Rp-G* or *Rp1-I*. Since the *Rp-GI* compound gene has both of these effective Rp genes, it may be more durable than other compound genes, although the prolonged effectiveness of this combination of Rp-resistance in North America will depend on the absence of virulence against both resistance genes. Virulence against each of these individual Rp genes has been observed in some trials (5,6).

The high level of resistance of lines with the *Rp1-JC* and *Rp1-JFC* compound genes is particularly interesting since the *Rp1-C* and *Rp1-J* genes were susceptible individually to the bulked population. The resistance of these compound genes appears to be due to heterogeneity for virulence against the individual genes in the bulked population from 1999. The bulked population included subpopulations that were virulent against *Rp1-C* or against *Rp1-J*, but isolates with virulence against both of these genes apparently were not collected.

The pattern of virulence for the bulked population in this study was similar to, but not exactly the same as, an isolate of *P. sorghi*, HI1, collected from Hawaii (14). A similar virulence phenotype also has been observed from South America (Steve Grier, *personal communication*). The need to develop sweet corn hybrids with compound or single Rp genes that are effective against biotypes that comprised the bulked population from 1999 depends on whether the occurrence of these biotypes in the Midwest in 1999 was an unusual event or if these biotypes are established where *P. sorghi* inocula for the continental United States overwinter. A population of *P. sorghi* that infected a set of Rp lines grown in a nursery in Los Mochis, Mexico, in March 2000 had the same pattern of virulence as the bulked population from 1999 (16). Rust severity in the Mexican trial was over 50% on some sweet corn hybrids with the *Rp1-D* (J. K. Pataky, *personal observation*). In April and May 2000, isolates of *P. sorghi* with this pattern of virulence were collected from Texas and Florida (18), but rust severity was substantially less on sweet corn hybrids with *Rp1-D* than on hybrids without *Rp1-D*.

Additional characterization of virulence in North American populations of *P. sorghi* that are avirulent against *Rp1-D* is necessary to determine if genes that were effective in these trials will be widely effective. In the long term, combinations of Rp genes or higher levels of general (i.e., partial) rust resistance will probably provide the most effective and durable control.

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