Evaluating Soybean Germ Plasm for Brown Stem Rot Resistance

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ABSTRACT

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Over 3,400 accessions from the USDA Soybean Germplasm Collection in maturity groups 000 to IV were evaluated between 1981 and 1984 for resistance to brown stem rot (BSR) caused by *Phialophora gregata*. Lines were evaluated in naturally infested fields at latitudes to which they were adapted. In 1985 and 1986, 13 early maturity, and 25 late maturity, putatively resistant lines were evaluated in replicated tests. The early maturity material was tested at Rosemount, MN, Hancock, WI, and Ames, IA. The late maturity material was tested at Ames, IA, and Urbana, IL. Each line also was evaluated in the greenhouse at Urbana, IL. Ratings at Hancock, WI, and in the greenhouse were based on leaf symptoms. All other ratings were based on stem symptoms. No lines were immune to BSR. In the early maturity test, no lines were as resistant as the resistant standards at all locations, but several lines were highly resistant at two or more locations. In the late maturity test, three lines (PI 424.285A, PI 424.353, and PI 424.611A) were resistant in all tests, but were not superior to previously identified sources. Many lines had inconsistent responses across environments and reasons for those interactions are discussed.

Brown stem rot (BSR), a vascular disease of soybean (*Glycine max* (L.) Merr.) caused by *Phialophora gregata* (Allington and Chamberlain) W. Gams, was first documented in Illinois in 1944 (1). This soilborne pathogen is now found throughout the soybean-producing states of the midwestern United States and in some southern states (5). Phillips (9) identified a variety of cultural types and found large differences in growth rate among isolates of *P. gregata*. He also reported differences in virulence, but there was no virulence by host interaction for the two host cultivars that he tested. Gray (4) reported two pathogenic types (type 1 and type 2) of *P. gregata* that produced vascular discoloration, but only type 1 isolates produced leaf necrosis. Sebastian (10) found a significant negative correlation between soybean yield and leaf symptom severity with up to a 16% yield loss. Despite the variability previously noted in the pathogen, Sebastian and Nickell (11) reported that the single dominant gene identified in their research provided an adequate level of resistance to BSR.

Evaluating for brown stem rot reaction in the field is difficult. Chamberlain and Bernard (2) reported large plant-to-plant and seasonal variation in disease expression in their efforts to determine the inheritance of resistance. Differences in plant spacing (8) and maturity (14) have been shown to contribute to variation in disease incidence.

Extensive screening of soybean germ plasm between 1947 and 1952 identified one strain of unknown origin, PI 84.946-2, with resistance to BSR (2). It has since been used as the source of resistance for the cultivars BSR 101, BSR 201, BSR 301, BSR 302, and Chamberlain. Tachibana and Card (13) confirmed the resistance of four other introductions (PI 86.150, PI 88.820N, PI 90.138, and PI 95.769) initially selected by Chamberlain and Bernard (*personal communication*). All of these sources of resistance are in maturity group IV. Gray (5) reported resistance in PI 437.833, a maturity group I introduction (the original report listed PI 437.823 as resistant, but the correct identification should be PI 437.833).

Since the only major screening of soybean germ plasm in the United States for BSR resistance, much has been learned about factors that influence disease incidence and pathogenic variation in *P. gregata*. The purpose of this research was to screen a large part of the northern portion of the USDA Soybean Germplasm Collection for new sources of

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resistance to BSR, particularly in maturity groups III and earlier.

MATERIALS AND METHODS

All initial screening was done in naturally infested fields. Ratings were made on leaf symptoms (interveinal necrosis) when evident, but most classification was based on browning of the vascular and pith tissue within the main stem. A total of 3,463 accessions in maturity groups 000 to IV were screened for resistance from 1981 to 1984 (Table 1) in four states. All initial tests were unreplicated. BSR 201, BSR 302, and PI 84.946-2 were used as standards in maturity groups II through IV, respectively. No sources of resistance were available in the earlier maturity groups when this work began, so those accessions with the least disease were selected for retesting. Those lines with acceptable levels of resistance in any one test were re-evaluated the following year in at least two other locations. Selection for the most resistant lines was made after each test. By 1985, all putative resistant lines had been tested in at least three, and in some cases as many as five, environments.

Fifty-one lines were selected for replicated tests in 1985. Ten lines in groups 000 to 0 were tested at Rosemount, MN, and Hancock, WI. Fifteen lines in groups I and II were screened at the same two locations plus Arlington, WI, and Ames, IA. Sixteen lines in groups III and IV were tested at Ames, IA, and at two locations at Urbana, IL. Ten additional lines selected from initial testing at Urbana in 1984 were included at the two locations at Urbana. The cultivars Clay and McCall were selected as susceptible standards for maturity groups 000 to 0. No resistant lines are known in those maturity groups. The cultivars BSR 101 and BSR 201 were the resistant standards, and Hodgson 78 and Corsoy 79 were the susceptible standards in maturity groups I and II, respectively (Table 2). Cumberland was the susceptible standard for the maturity groups III and IV tests, and the experimental line A8, PI 84.946-2, and PI 86.150 were the resistant standards (Table 3). Plots consisted of one row, 3 m long. Tests at all locations were replicated three times in a randomized complete block design.

The results from 1985 were used to eliminate some susceptible accessions and in 1986, 13 early maturing accessions were evaluated at Ames, IA, Hancock, WI, and Rosemount, MN (Table 4), and 25 late maturing accessions were evaluated at Ames, IA, and two locations at Urbana, IL (Table 5). The same standards were used as in 1985 except that Elgin, a susceptible cultivar, was added to the early test (Table 2) and Chamberlain, a BSR-resistant cultivar, was added to the late test at the Ames location (Table 3). Plot size, experimental design, and number of replications at each location were the same as in 1985.

Ratings at the Rosemount, Urbana, and Arlington locations were based on the discoloration of the vascular and pith tissue of the main stem approximately 1–2 wk before growth stage R7 (3) according to the following scale: 1 = no discoloration, 2 = discoloration below the second node, 3 = discoloration up to the fifth node, 4 = discoloration up to the eighth node, 5 = discoloration to the top of the plant. The one exception was site 2 at Urbana in 1985 where the actual number of nodes infected was counted at growth stage R6 (Table 3). Ratings at the Ames location were also based on the discoloration of the vascular and pith tissue but are expressed as a percentage of the total plant height at approximately growth stage R7. For those locations where ratings were based on stem symptoms, a minimum of 10 stems were

Table 1. Location of initial tests and identification of accessions screened for brown stem rot resistance at each location

Location	Year	Maturity group	Inclusive PI and FC numbers of lines tested	No. tested
Urbana, IL	1981	IV	PI 273.483C to PI 427.008J[a]	769
Urbana, IL	1982	II	PI 427.137 to PI 438.186	378
Rosemount, MN	1983	000	PI 437.178 to PI 445.826	28
Rosemount, MN	1983	00	PI 430.491 to PI 445.836	153
Rosemount, MN	1983	0	PI 427.138 to PI 445.833	400
Ames, IA	1983	I	PI 437.170 to PI 437.949	290
Hancock, WI	1983	II	PI 438.192 to PI 445.814	77
Urbana, IL	1983	III	FC 02.108 to FC 31.684	13
Urbana, IL	1983	III	PI 54.583 to PI 90.723	338
Rosemount, MN	1984	I	PI 437.951 to PI 445.837	276
Urbana, IL	1984	III	PI 91.083 to PI 475.882C[b]	542
Urbana, IL	1984	IV	FC 03.548 to FC 33.243	18
Urbana, IL	1984	IV	PI 19.986 to PI 83.881	181
Total				3,463

[a] These are all accessions contained in the early group IV evaluation as reported in USDA Tech. Bull. 1718. They include 35 accessions now classified as group III.
[b] Only 542 of the 667 accessions of this group were rated because of Phytophthora root rot damage to the remaining accessions.

Table 2. Ratings for brown stem rot severity for the resistant (R) and susceptible (S) standards used for maturity groups 0, I, and II

Entry	Ames 1986	Arlington 1985	Hancock 1985	Hancock 1986	Rosemount 1985	Rosemount 1986	Greenhouse 1984	Greenhouse 1985
Hodgson 78 (S)	62.8[a]	3.0[b]	4.0[c]	7.0[c]	3.7[b]	3.5[b]	···	···
Hardin (S)	···	···	···	7.7	···	···	···	···
BSR 101 (R)	15.9	2.0	0.0	0.0	1.1	1.8	···	···
Elgin (S)	81.4	···	···	1.0	···	2.5	···	···
Corsoy 79 (S)	83.2	5.0	8.0	7.3	2.0	1.4	4.1[d]	4.1[d]
BSR 201 (R)	35.4	2.0	0.0	0.0	1.3	1.1	0.1	0.0
Century (S)	···	···	···	···	···	···	5.4	5.4
LSD (0.05)	18.7	0.7	1.8	2.1	1.6	1.1	···	···

[a] Percent of total main stem height showing symptoms.
[b] Scale of 1–5, where 1 = no main stem discoloration and 5 = main stem discoloration to the top of the plant.
[c] Leaf symptom rating on the Horsfall-Barratt scale of 0 = no symptoms to 11 = complete necrosis.
[d] Mean number of nodes showing leaf symptoms.

Table 3. Ratings for brown stem rot severity for the resistant (R) and susceptible (S) standards used for maturity groups III and IV

Entry	Ames 1986	Urbana Site 1 1985	Urbana Site 2 1985	Urbana 1986	Greenhouse 1985
Cumberland (S)	81.8[a]	2.2[b]	7.4[c]	3.1[b]	4.2[d]
A8 (R)	25.9	1.0	0.2	1.4	···
Chamberlain (R)	47.8	···	···	···	···
PI 84.946-2 (R)	43.5	1.4	0.8	1.2	···
PI 86.150 (R)	21.0	1.0	1.2	1.1	···
BSR 302 (R)	···	···	···	···	0.0
LSD (0.05)	18.7	0.6	3.0	0.8	···

[a] Percent of total main stem height showing symptoms.
[b] Scale of 1–5, where 1 = no main stem discoloration and 5 = main stem discoloration to the top of the plant.
[c] Mean number of internodes showing stem browning.
[d] Mean number of nodes showing leaf symptoms.

examined per plot. At the Hancock location, resistance was evaluated using foliage symptoms between growth stages R6 and R7. This was the only location where leaf necrosis occurred. Ratings were based on the Horsfall-Barratt system with 0 = no symptoms and 11 = complete necrosis (7).

In addition to the field testing, each accession was screened in the greenhouse using the technique of Sebastian and Nickell (11). *P. gregata* was isolated from an infected soybean plant at Urbana, IL. One single-spore isolate was selected for its ability to produce leaf symptoms in a BSR-susceptible soybean plant. Seeds from each entry were germinated in sand in 10-cm plastic pots. Plants that were 12 days old were removed, rinsed in water, and roots were blotted on paper towels. Groups of five healthy, uniform plants were dipped in 50 ml of inoculum at 1.2×10^6 propagules ml^{-1} and transplanted to 15-cm-diameter, steam-sterilized clay pots containing steam-treated 1:1 sand:soil mix. Plants were maintained under a 15-hr photoperiod at an average of 600 $\mu mol \cdot m^{-2} \cdot s^{-1}$ at midday and 18–24 C. Each pot received 150 ml of water twice daily and was fertilized weekly. The ratings were based on the number of nodes displaying leaf symptoms and were the mean of five plants grown in a single pot. Ratings were made when the plants were approximately 5 wk old. BSR 201 was the resistant standard

Table 4. Classification for brown stem rot resistance for soybean introductions screened in 1985 and 1986 in maturity groups 0 to II[a]

Entry	Maturity group	Origin	Ames 1986	Arlington 1985	Hancock		Rosemount		Greenhouse	
					1985	1986	1985	1986	1984	1985
PI 427.138	0	S. Korea	S (79)[b]	···	R (0.3)[d]	R (1.7)[d]	S (2.7)[e]	R (2.2)[e]	···	R (0.0)[e]
PI 437.936	0	N.E. China	S (73)	···	R (1.0)	R (1.3)	S (2.7)	S (3.5)	···	R (0.0)
PI 437.327	I	U.S.S.R.	S (55)	S (4.0)[c]	R (0.0)	R (1.7)	S (2.9)	S (3.3)	···	R (0.0)
PI 437.366	I	U.S.S.R.	S (62)	S (4.0)	R (0.0)	R (0.0)	S (2.4)	S (3.2)	···	R (0.0)
PI 437.570	I	China	S (45)	···	R (1.3)	S (6.7)	S (3.8)	S (3.3)	···	R (1.0)
PI 437.833	I	N.E. China	S (52)	···	···	R (2.0)	···	S (2.8)	R (0.0)[e]	R (0.1)
PI 437.934A	I	N.E. China	R (34)	S (3.0)	R (0.0)	R (0.3)	S (2.8)	R (1.9)	···	R (0.0)
PI 437.475	II	U.S.S.R.	S (75)	R (2.0)	R (0.3)	R (0.7)	R (1.6)	S (3.2)	R (0.0)	I (1.6)
PI 437.497	II	U.S.S.R.	S (70)	R (2.0)	R (0.3)	R (0.0)	S (3.4)	S (3.1)	R (0.0)	R (0.0)
PI 437.900	II	N.E. China	S (70)	S (3.0)	R (0.0)	R (0.3)	S (2.8)	S (3.2)	R (0.0)	R (0.0)
PI 438.222	II	N.E. China	S (87)	S (3.0)	R (1.0)	S (3.0)	R (2.0)	S (2.4)	R (0.0)	R (0.0)
PI 437.685D	II	China	S (70)	S (5.0)	R (0.0)	R (0.0)	S (2.5)	S (3.4)	R (0.0)	R (0.0)
PI 438.490A	II	U.S.S.R.	I (61)	S (5.0)	R (0.7)	R (2.0)	R (1.7)	S (2.4)	···	R (0.0)

[a] Lines were classified as R (resistant) or S (susceptible) if they were not statistically different from the resistant or susceptible standards, respectively, in the same maturity group (Table 2). Lines classified as I (intermediate) were statistically different from both the resistant and susceptible standards.
[b] Percent of total main stem height showing symptoms.
[c] Scale of 1–5, where 1 = no main stem discoloration and 5 = main stem discoloration to the top of the plant.
[d] Leaf symptom rating on the Horsfall-Barratt scale of 0 = no symptoms to 11 = complete necrosis.
[e] Mean number of nodes showing leaf symptoms.

Table 5. Classification for brown stem rot resistance for soybean introductions screened in 1985 and 1986 in maturity groups III and IV[a]

Entry	Maturity group	Origin	Ames 1986[b]	Urbana		Urbana 1986	Greenhouse 1985
				Site 1 1985	Site 2 1985		
PI 68.621	III	N.E. China	S (86)[c]	R (1.2)[d]	R (3.6)[e]	R (1.7)[d]	R (0.0)[f]
PI 68.710	III	N.E. China	S (80)	R (1.3)	R (0.5)	S (2.4)	R (0.0)
PI 70.076	III	N.E. China	S (82)	R (1.4)	R (3.0)	I (2.1)	R (0.0)
PI 70.515	III	N.E. China	S (73)	R (1.4)	R (2.1)	I (2.3)	R (0.0)
PI 84.908-2	III	Unknown	S (87)	R (1.0)	R (1.4)	S (2.8)	S (3.0)
PI 86.026-1	III	Unknown	S (82)	R (1.3)	R (1.9)	R (1.5)	R (0.0)
PI 86.144	III	Japan	S (86)	R (1.1)	R (0.9)	I (2.1)	R (0.2)
PI 87.619	III	Korea	S (67)	R (1.0)	R (0.5)	I (2.0)	R (0.0)
PI 88.818	III	Korea	S (85)	R (1.2)	···	R (1.5)	S (3.8)
PI 90.499-1	III	Unknown	S (71)	R (1.3)	R (2.9)	R (1.7)	R (0.0)
PI 90.573	III	N.E. China	S (86)	R (1.1)	R (3.8)	I (2.1)	R (0.0)
PI 93.565A	III	Unknown	S (85)	R (1.6)	R (3.8)	I (2.2)	R (0.0)
PI 196.157	III	Japan	S (76)	R (1.0)	R (2.5)	I (2.1)	R (0.0)
PI 398.311	III	S. Korea	R (49)	R (1.1)	R (0.4)	I (2.2)	R (0.0)
PI 398.755	III	S. Korea	S (64)	R (1.3)	R (3.7)	I (2.1)	R (0.0)
PI 398.930	III	S. Korea	S (62)	R (1.4)	R (2.1)	R (1.5)	R (0.0)
PI 416.862	III	Japan	S (66)	R (1.1)	R (3.1)	R (1.3)	R (0.0)
PI 423.826A	III	S. Korea	S (79)	R (1.2)	R (1.3)	I (2.1)	R (0.0)
PI 424.353	III	S. Korea	R (53)	R (1.2)	R (0.0)	R (1.7)	R (0.0)
PI 424.368A	III	S. Korea	S (64)	R (1.1)	R (1.3)	I (2.1)	R (0.0)
PI 424.373	III	S. Korea	S (64)	R (1.3)	R (1.0)	R (1.9)	R (0.0)
PI 423.930A	IV	Japan	S (62)	R (1.0)	R (0.0)	S (1.5)	R (0.0)
PI 424.285A	IV	S. Korea	R (41)	R (1.1)	R (1.2)	R (1.4)	R (0.0)
PI 424.386B	IV	S. Korea	R (45)	R (1.1)	R (0.8)	I (2.2)	R (0.0)
PI 424.611A	IV	S. Korea	R (46)	R (1.0)	R (1.3)	R (1.8)	R (0.0)

[a] At all locations, except Ames, lines were classified as R (resistant) or S (susceptible) if they were not statistically different from the resistant or susceptible standards, respectively, in the same maturity group (Table 3). Lines classified as I (intermediate) were statistically different from both the resistant and susceptible standards.
[b] At Ames all lines had ratings significantly larger than PI 86.150. Susceptible or resistant classification at the Ames location was based on comparisons with PI 84.946-2.
[c] Percent of total main stem height showing symptoms.
[d] Scale of 1–5, where 1 = no main stem discoloration and 5 = main stem discoloration to the top of the plant.
[e] Mean number of internodes showing stem browning.
[f] Mean number of nodes showing leaf symptoms.

and Corsoy 79 and Century were the susceptible standards for the early maturing lines (Table 2). Cumberland and BSR 302 were the susceptible and resistant standards, respectively, for the late maturing lines (Table 3).

Data from each location were subjected to an analysis of variance. Lines were declared resistant or susceptible if they were not significantly different from the resistant or susceptible standards, respectively. Lines that were significantly different from both the resistant and susceptible standards were classified as intermediate. An LSD test at the 0.05 probability level was used to test for differences.

RESULTS

The level of infection for the replicated tests was moderate to high in the susceptible genotypes in most location-year combinations. Data collected at Ames in 1985 were not included because of the high experimental error due to variability in disease incidence. Data from one site at Urbana in 1986 were not included because of few disease symptoms. At all other sites, statistically significant differences in disease ratings were found between resistant and susceptible standards (Tables 2 and 3), but the reaction of all susceptible cultivars was not the same at all locations. Corsoy 79 was highly susceptible at all test sites, except for Rosemount where it was not significantly different from the resistant standards in either year. Elgin was only tested in three environments, but showed a susceptible reaction at Ames and Rosemount and a resistant reaction at Hancock. Only at the Ames location were significant differences found among resistant standards. In the early maturity test (Table 2), BSR 101 had a significantly lower rating than BSR 201, which could be a function of time of maturity because Hodgson 78 had a significantly lower score than either of the two group II susceptible standards. In the late maturity test (Table 3), PI 86.150 had a lower rating than PI 84.946-2, as has been previously observed (13), and also lower than Chamberlain. A8 was also significantly better than either PI 84.946-2 or Chamberlain.

No introductions were rated as immune. In the early maturity test, none of the potential new sources of resistance were classified as resistant in all environments. Tests conducted in the greenhouse at Urbana and in the field at Hancock, where resistance was based on leaf symptoms, identified all but one and two of the lines, respectively, as consistently resistant (Table 4). The stem ratings at the Rosemount location showed more variability with five lines classified differently between years. Four lines were consistently near the statistical boundary between susceptibility and resistance, but a major difference in rating occurred for PI 437.475. Data from only 1 yr is available for the Ames and Arlington locations. Most lines were susceptible at both locations. PI 437.833, previously reported as being resistant, was resistant at Hancock and in the greenhouse at Urbana, but not at Ames or Rosemount.

None of the late-maturing lines tested at Ames was as resistant to BSR as PI 86.150 or A8. Five lines had ratings not significantly different from that of PI 84.946-2 (Table 5). None of the lines tested in the field at Urbana in 1985 were susceptible. The greenhouse tests, conducted with an isolate of the fungus recovered from the Urbana location, indicated only two susceptible lines, PI 84.908-2 and PI 88.818. Urbana field data from 1986 would suggest that PI 84.908-2 is susceptible, but PI 88.818 was consistently resistant in both years of field testing. In 1986 at Urbana, no infection was found at one site, but at the second site infection was greater than in 1985. Although only three strains were as susceptible as the susceptible standards, only 10 of the 25 were as resistant as the resistant standards. Nine entries were declared resistant in all four tests conducted at Urbana and three of those (PI 424.285A, PI 424.353, and PI 424.611A) were also among those shown to be resistant at Ames (Table 5).

DISCUSSION

The lack of immunity to BSR, variability in *P. gregata*, and the environmental influence on disease symptoms make screening for resistance a difficult task. As shown in Tables 4 and 5, ratings of individual lines were seldom the same in all environments.

The data collected on leaf symptoms in the greenhouse declared more lines resistant than those from any of the field tests. In this procedure, the possibility of influences by other disease organisms is eliminated or greatly reduced and leaf symptoms can be consistently produced on susceptible cultivars. Under greenhouse conditions, Sebastian et al (12) found that the heritability of leaf symptoms was two to three times greater than the heritability of stem symptoms. Stem browning observed in the less controlled field environments may be only partly due to *P. gregata*. The only field location at which leaf symptoms could be consistently observed was Hancock. The results from Hancock agreed more closely with the greenhouse screening than those obtained from any other test. PI 437.833 was declared resistant when foliage symptoms were the basis for classification, but it was not resistant when stem browning was used as the rating criterion. The data from Ames may also provide evidence for the influence of other factors besides *P. gregata*. A8 had a significantly better rating than either Chamberlain or PI 84.946-2, even though the resistance to BSR in both A8 and Chamberlain was derived from PI 84.946-2. The apparent superior resistance of A8 to BSR may be due to its resistance to other stem browning organisms such as *Acremonium* sp. (6).

Another possible explanation for the variable disease reactions involves the timing of symptom expression. The greenhouse ratings are made on plants that are 5 wk old. The most susceptible genotypes can be easily distinguished from resistant genotypes at this time, but we do not know if additional distinctions could be made among the putative resistant types if those plants were allowed to grow in the presence of the disease organism for as long as the field-rated plants in this study. Part of the variability observed in the field may be due to the rate of pathogenesis and symptom development allowed by each genotype and the interaction of this rate with the environment.

A third explanation for the conflicting results presented here is pathogenic variability within *P. gregata*. Gray (4) and Phillips (9) have previously noted that variability does exist within this organism. Sebastian and Nickell (11) identified two genes for resistance in PI 84.946-2, but also found that the transfer of only one of those genes would confer resistance to BSR. The potential confounding factors discussed in the first part of this section make it impossible to ascertain from these data what role pathogen variability is having in these results. However, the apparent genotype by location interaction in these data provides sources of diversity for both the plant and pathogen that could be used to test this theory. Since the completion of this research, strains of *P. gregata* have been collected from the same locations used in this study. Greenhouse evaluation revealed that several isolates caused intermediate to susceptible disease reactions on the normally resistant strain PI 437.833 (David B. Willmot, *personal communication*). These results suggest that variability for virulence, or an ability to overcome host resistance, exists in the pathogen. Moreover, variability for aggressiveness, or the severity of disease reaction, is wide. This would indicate that pathogen variability is a probable cause for the differential reactions observed among locations (Tables 4 and 5).

None of the potential new sources of resistance identified in this study is superior to the known sources. These strains do expand the range of putative origin for sources of resistance (Tables 4 and 5). Of the five published sources of resistance, two are of unknown origin (PI 84.946-2 and PI 90.138), two are from Korea (PI 88.820N and PI 95.769), and one is from Japan (PI 86.150). In addition to expanding the number of

resistant lines known from these two countries, the lines identified here also originated from China and the U.S.S.R. All the introductions listed from the U.S.S.R. in Table 4 come from Primorskaya Province which forms part of the eastern boundary of northern China. Until the variability of the pathogen is better understood and the genetics of resistance in these introductions is elucidated, the value of these new sources of resistance will not be fully known.

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