Inheritance of Resistance to Scab in Two Wheat Cultivars from Brazil and China

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ABSTRACT

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One of the major diseases that reduces the quality of wheat is scab. Quality is reduced by the production of toxins in grain by the pathogen *Fusarium graminearum.* Various genetic sources of resistance have been identified, but the degree of resistance in most cultivars is insufficient. Although both South American and Chinese germ plasm are being used in breeding programs around the world, it is not known whether the resistance genes contributed by these sources are different. The purpose of this study was to determine the mode of inheritance and number of genes involved in the scab resistance of a wheat cultivar from Brazil and one from China, both known to possess intermediate to high resistance in regard to spread of the disease in the wheat head. The resistant Brazilian cultivar Frontana was compared to the highly resistant Chinese cultivar Ning 7840 in crosses with CNO79, a highly susceptible Mexican wheat, as the susceptible parent. Random F2-derived F7 lines from the six crosses possible (including reciprocals) among the parents were studied for their field response following inoculation with *F. graminearum* on two inoculation dates. Three methods of disease assessment were compared. The two resistant parents were shown to possess two unique dominant genes each, with all four genes being different. Combining their resistance genes may produce higher levels of resistance. The two inoculation dates provided the same gene postulations. The three disease assessment methods essentially measured the same basic process of disease spread within the head, based on the calculation of the genotypic correlations between the methods and the similarity in gene postulations. The easiest and most relevant method should therefore suffice for assessing the spread of scab through the wheat head.

Scab of wheat (*Triticum aestivum* L.) is caused by any of several *Fusarium* spp., of which the most common in developing countries is *Fusarium graminearum* Schwabe (30). The disease is most prevalent where moist and warm climatic conditions occur when wheat is flowering (17). In China alone, the area affected by scab exceeds 6 million hectares (10). In warmer wheat-growing environments, such as eastern Asia, parts of southern Africa, and South America, scab is considered one of the three most important wheat diseases (6). Although it reduces yields due to grain shriveling, the most important damage is to grain quality. Toxins such as the trichothecenes deoxynivalenol (DON) and acetyldeoxynivalenol (ADON) are produced, which obstruct protein synthesis in humans and other animals and may cause death (4,13).

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Breeding wheat for resistance to scab has high priority in countries such as China, the United States, and Brazil, and in central Europe (11,12,17). Due to its global wheat-breeding mandate, CIMMYT has been involved in breeding for resistance to this disease since 1985 (18). Sources of scab resistance have been divided into various groups according to their geographic origin: eastern Europe, Italy, China, Japan, and Brazil (11,22). To what extent these sources are genetically different is not known.

No complete resistance or immunity to scab has been observed in wheat. Various studies have shown two to five dominant genes to be involved in resistance (7,9,23,27–29). The total number of genes present in Chinese wheats is considered to be fewer than five. In the cultivar Ning 7840, two or three genes have been identified (30). Recently, Singh et al. (21) showed that three genes contribute to resistance in the Brazilian cultivar Frontana.

Besides preventing initial penetration (Type I resistance) or reducing fungal penetration or spread within a head (Type II resistance), resistant or tolerant host genotypes may also have a lower amount of toxins present (Type III resistance; 14,15,19,24,25).

The purpose of this study was to determine the mode of inheritance and number of genes involved in the scab resistance, as expressed by resistance to spread of symptoms within a head (Type II resistance), of a wheat cultivar from China and another from Brazil, both known to possess intermediate to high resistance to the disease.

MATERIALS AND METHODS

Two genetic sources of resistance to scab were studied: the old cultivar Frontana, representing the Brazilian gene pool (22,24,30); and Ning 7840, representing new, high-yielding Chinese germ plasm with a high level of resistance (8,10,11,24). Ning 7840 has the following parentage: Aurora/Anhui11//Sumai3. Seed was obtained from the CIMMYT germ plasm bank and phenotypically verified.

These cultivars were intercrossed, including reciprocals, and crossed to CNO79, a highly susceptible Mexican wheat, resulting in a total of six crosses. Without imposing artificial selection, sets of randomly selected F2 plants for each of the six crosses were advanced to the F7 generation. In the F7, the number of randomly F2-derived lines available per cross varied from 63 to 125 lines per individual cross, and from 131 to 206 lines per cross combination if reciprocals were combined. The F7 lines from all crosses were planted in an alpha-lattice design with two replicates in CIMMYT's Experiment Station at Toluca (19° north latitude, 2,640-m elevation) in the central highlands of Mexico. Each line was sown in a two-row plot 1 m long, with 60 to 80 seeds per plot. To provide suitable references, 10% of the plots were parents.

No races have been identified in *F. graminearum* that significantly alter wheat genotype ranking (17,24). Also, random amplified polymorphic DNA (RAPD) and polymerase chain reaction (PCR) primers have failed to reveal much variation within *Fusarium* species (16). Hence, we prepared a mixture of five relatively aggressive isolates for inoculation purposes. In each plot, the first five heads to flower at the same time were inoculated with the isolate mixture. A small tuft of cotton soaked in a suspension of fungal spores (50,000 spores per ml) was inserted into a floret of a central spikelet with tweezers and left touching the anthers. Care was taken that the primary inoculum did not drip from the central spikelet. The inoculated head was then covered with a glassine bag to protect it from allo-infection. The method is reliable, does not damage the reproductive organs, and is well-suited for quantitative studies (2). Two weeks after the first

inoculation, five newly flowering heads in the same plots were likewise inoculated. Thus, inoculation at the same head growth stage was compared across the entries on two occasions.

Throughout the growing cycle, rainfall occurred almost daily, usually in the afternoon. Morning fog was frequent. Total rainfall for the crop cycle was about 820 mm. Mean minimum and maximum temperatures were 4.7 and 21.7°C.

About 40 to 60 days after inoculation, when the individual plants reached maturity, the heads were individually harvested, and the disease was immediately scored on each head. A spikelet was considered infected if it was partly or fully covered with white, creamy or pink mycelium. In addition, in most infected heads, a narrow strip of bright pink-orange mycelium was visible in infected spikelets at the juncture of the lemma and palea. Disease was assessed in three ways, according to Zhuping (30).

The infected spikelet rate (ISR; %), or percentage of scabbed spikelets, was calculated as follows: ISR = 100(symptomatic spikelets/total spikelets).

The reaction index (RI; 1 to 5) is the average scab severity based on grades 1 to 5, where 1 represents no spread and 5 indicates total infection of the entire head. Intermediate levels of head infection are represented by 2, 3, and 4. The RI was constructed using the following formula: RI = [G1 + (2 × G2) + (3 × G3) + (4 × G4) + (5 × G5)]/total heads. G1, G2, G3, G4, and G5 are the number of heads of grades 1, 2, 3, 4, and 5, respectively.

The disease index (DI; %) is based on grades 0 to 4 and describes the portion of infected florets per head by increments of 1/4, where 0 represents no disease, 1 indicates up to 1/4 of the spikelets are infected, and 4 means >3/4 of the spikelets are infected. The DI was constructed as above for RI.

The F7 lines were divided into two groups: (i) a homozygous susceptible group (HS) consisting of lines expressing infection levels greater than the mean value of the susceptible parent minus 2 standard errors, and (ii) the remaining group of homozygous resistant plus intermediate entries (HR + I).

Ratio analysis supported by chi-square calculations was used to estimate the number of segregating genes. In the genetic analysis, the ratio of resistant to susceptible lines among the random F2-derived F7 families should be close to 1:1 when one gene segregates. When two genes of equal effect segregate, the expected ratio is close to 1:2:1, with the central class representing lines homozygous for resistance alleles only at one of the two loci. Slight deviations from these ratios are expected because of residual heterozygosity in some F7 families. The exact ratio for the two-gene model at the F7 generation is 0.969:2.062:0.969. Similarly, higher order ratios were determined for the segregation of three to five genes and tested against the data.

In addition, the number of segregating factors was determined using Cockerham's modification (5) of Wright's formula (26), in which F7 generation lines are considered fully homozygous individuals. The genotypic range (GR) between F7 lines presumably carrying all the minus factors and lines carrying all the plus factors was adjusted for by the estimate of heritability. The formula used for the calculation of the minimum number of effective factors was $n = (GR \times h^2)^2/4.27\sigma^2$, according to Singh et al. (21). Heritability estimates were calculated from the ANOVA data according to Singh and Choudhary (20). Using variance and covariance components, genotypic correlations were calculated among ISR, RI, and DI (3).

RESULTS

Scab was uniformly severe in cultivar CNO79, the susceptible check, indicating an adequate epidemic for quantitative analysis was achieved. The data are presented by cross in Table 1. By each measure of disease, Ning 7840 and Frontana were more resistant than CNO79, but the two resistant parents did not significantly differ from each other. The susceptible parent had the most scab, based on all three scales used.

In most instances, the second inoculation date showed slightly reduced levels of infection with all three methods of disease evaluation. Although differences were not always significant, and the entry × inoculation date interaction was not significant, the data are presented separately.

Table 2 lists the data for the cross of the Brazilian cultivar Frontana with the sus-

Table 1. Mean values for infected spikelet rate (ISR; %), reaction index (RI; 1 to 5), and disease index (DI; %) for the two inoculation dates (2 weeks apart) of the respective parents in each of three cross combinations (combining both reciprocal crosses), following infection with *Fusarium graminearum*

	1st inoculation date			2nd inoculation date		
Cross combination	**ISR**	**RI**	**DI**	**ISR**	**RI**	**DI**
Frontana × CNO79						
Frontana	17 a[z]	1.3 a	29 a	17 a	1.3 a	28 a
CNO79	73 b[z]	4.0 b	83 b	66 b	3.9 b	77 b
Ning 7840 × CNO79						
Ning 7840	8 a	1.3 a	23 a	17 a	1.1 a	25 a
CNO79	76 b	4.0 b	86 b	64 b	3.9 b	81 b
Frontana × Ning 7840						
Frontana	17 a	1.5 a	30 a	16 a	1.2 a	27 a
Ning 7840	15 a	1.2 a	28 a	11 a	1.2 a	26 a

[z] Letters indicate a significant difference between the two parents within a cross combination at $P = 0.01$.

Table 2. Gene estimation, based on F7 line distribution in the cross Frontana × CNO79 (both reciprocals), into two classes, according to their reaction to infection with *Fusarium graminearum*

	No. of individual lines				
Frontana × CNO79	**HR + I[z]**	**HS**	χ^2	***P* value**	**No. of genes**
1st inoculation					
ISR	159	47	0.22	>0.50	2
RI	154	46	0.33	>0.50	2
DI	152	54	0.44	>0.50	2
2nd inoculation					
ISR	152	55	0.62	>0.25	2
RI	161	46	0.45	>0.50	2
DI	154	53	0.21	>0.50	2

[z] HR + I = homozygous for parental-type resistance plus intermediate types. HS = homozygous for parental-type susceptibility.

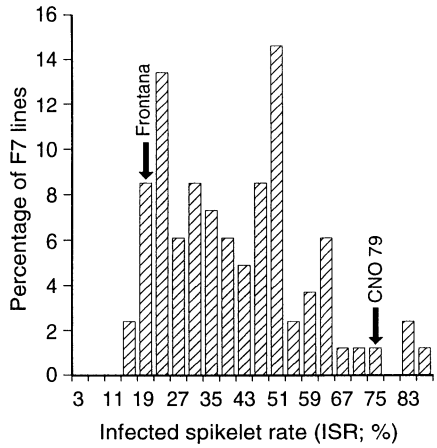

Fig. 1. Frequency distribution of F7 lines for infected spikelet rate in the cross of the resistant Brazilian cultivar Frontana with the susceptible cultivar CNO79, following infection with *Fusarium graminearum* at the first inoculation date. The level of the parents is also indicated.

ceptible Mexican cultivar CNO79. Since no significant differences were found in the ANOVA (F values varied between 1.06 and 2.57), the reciprocals were combined. The two inoculation dates are separately presented. The numbers of expected homozygous susceptible genotypes for the two inoculation dates were 49.9 and 50.1, respectively. Figure 1 depicts the distribution of the F7 lines for infected spikelet rate (ISR) at the first inoculation date. Two genes appear to be segregating in this cross. No significant transgressive segregation was observed, so both genes originated from the resistant parent Frontana.

Segregation for the cross of Ning 7840 with the susceptible CNO79 is presented in Table 3, where the two reciprocals are combined. Expected numbers of susceptible plants were 41.4 and 41.7, respectively, for the two inoculation dates. For the first inoculation date, there was an excess of susceptible plants for both ISR and DI. The ANOVA data of the first inoculation indicated a significant difference ($P \geq 0.05$) between reciprocals (F values varied between 4.57 and 4.66): more resistant plants occurred in the progeny when Ning 7840 was utilized as female than when it was the male. Thus, for the first inoculation, the ratio analysis was also carried out separately for the two reciprocal crosses (Table 4). Expected numbers of susceptible plants were 22.3 and 19.1, respectively. Figure 2 shows the distribution of the F7 lines for spikelet infection rate on the first inoculation date for the cross Ning 7840 × CNO79.

When Ning 7840 was used as the female parent, the two-gene model fit well, but when it was used as a male, there was a clear excess of susceptible plants, and the two-gene model no longer fit for ISR and DI. A slight maternal effect seems to be present in which CNO79 cytoplasm contributes towards susceptibility.

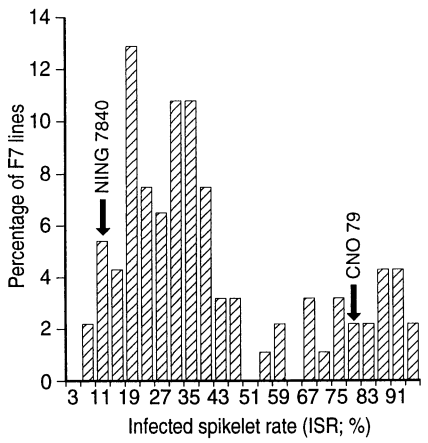

Fig. 2. Frequency distribution of F7 lines for infected spikelet rate in the cross of the resistant Chinese cultivar Ning 7840 with the susceptible cultivar CNO79, following infection with *Fusarium graminearum* at the first inoculation date. The level of the parents is also indicated.

When crossing the two resistant parents, each carrying two genes for resistance, three possible patterns of segregation exist. If the genes in both parents are the same, no segregation will be observed. If Frontana and Ning 7840 have one gene in common, but the second gene in each is unique, in which case two genes are different, segregation approaching 1:1:1:1 at the F7 level should be seen. If the two genes in Frontana are distinct from the two genes in Ning 7840, four genes will segregate, with about 1 out of 16 of the F7 lines (5.87%) carrying no resistance genes at all and being similar in susceptibility to the susceptible parent CNO79. Likewise, some lines could be more resistant than either parent.

The number of fully susceptible F7 families from the crosses between resistant parents was consistent with a model of four independent genes (Table 5). Figure 3 depicts this transgressive segregation for ISR in the cross between Frontana and Ning 7840 on the first inoculation date. The ANOVA indicated some maternal effect (F values varied between 4.99 and 6.07) on the first inoculation date.

Quantitative analysis using Wright's formula adjusted for heritability (5) was also used to estimate the number of effective factors (Table 6). The minimum number of effective factors for crosses between each of the two resistant cultivars and the susceptible CNO79 was about two. When

Table 3. Gene estimation, based on F7 line distribution in the cross Ning 7840 × CNO79 (both reciprocals), into two classes using the two-gene model, according to their reaction to infection with *Fusarium graminearum*

	No. of individual lines				
Ning 7840 × CNO79	**HR + I[z]**	**HS**	**χ^2**	***P* value**	**No. of genes**
1st inoculation					
ISR	117	54	5.03	>0.01	...
RI	126	45	0.41	>0.50	2
DI	122	49	1.83	>0.03	...
2nd inoculation					
ISR	125	47	0.90	>0.25	2
RI	137	35	1.42	>0.10	2
DI	133	39	0.23	>0.50	2

[z] HR + I = homozygous for parental-type resistance plus intermediate types. HS = homozygous for parental-type susceptibility.

Table 4. Gene estimation, based on F7 line distribution in the two reciprocals of the cross Ning 7840 × CNO79, into two classes using the two-gene model, according to their reaction to infection with *Fusarium graminearum*

	No. of individual lines				
1st inoculation	**HR + I[z]**	**HS**	**χ^2**	***P* value**	**No. of genes**
Ning 7840 × CNO79					
ISR	69	24	0.14	>0.50	2
RI	72	21	0.15	>0.50	2
DI	70	23	0.01	>0.90	2
CNO79 × Ning 7840					
ISR	48	30	8.52	>0.00	...
RI	55	23	1.17	>0.25	2
DI	47	31	10.23	>0.00	...

[z] HR + I = homozygous for parental-type resistance plus intermediate types. HS = homozygous for parental-type susceptibility.

Table 5. Gene estimation, based on F7 line distribution in the cross Frontana × Ning 7840 (and reciprocal), into two classes using the four-gene model, according to their reaction to infection with *Fusarium graminearum*

	No. of individual lines				
Frontana × Ning 7840	**HR + I[z]**	**HS**	**χ^2**	***P* value**	**No. of genes**
1st inoculation					
ISR	127	4	1.88	>0.10	4
RI	127	4	1.88	>0.10	4
DI	124	7	0.07	>0.75	4
2nd inoculation					
ISR	124	7	0.07	>0.75	4
RI	126	5	1.00	>0.25	4
DI	126	5	1.00	>0.25	4

[z] HR + I = homozygous for parental-type resistance plus intermediate types. HS = homozygous for parental-type susceptibility.

the Brazilian and Chinese resistant cultivars were intercrossed, the minimum number of effective factors was calculated at just under four.

Three methods of scoring the disease spread were used in the experiments. The genotypic correlations among these methods were high to very high (Table 7), and there was little difference among them. There was a great difference between crosses, however, probably due to larger variability among F7 lines in the crosses between the resistant and the susceptible parent.

DISCUSSION

Frontana, from Brazil, and Ning 7840, from China, are known to have medium to high levels of resistance to scab, and they have been widely used in breeding aimed at increasing resistance. The two cultivars were shown to carry two to three genes each (21,30), but whether these genes are different and may be pyramided was unknown.

This study indicates that each cultivar contains two genes for resistance for fungal spread within the wheat head (Type II resistance), and that the four genes are different from one another.

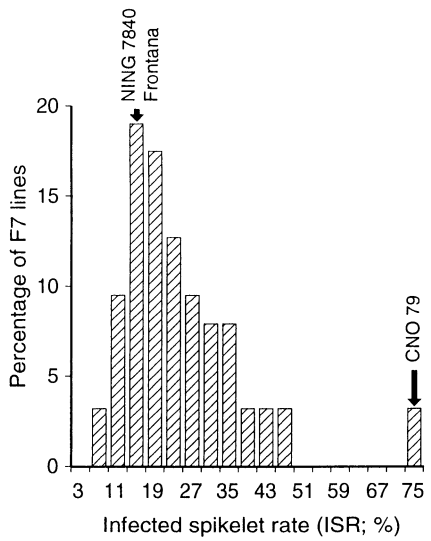

Fig. 3. Frequency distribution of F7 lines for infected spikelet rate in the cross of the resistant Brazilian cultivar Frontana with the resistant Chinese cultivar Ning 7840, following infection with *Fusarium graminearum* at the first inoculation date. The level of the parents is also indicated, plus that of the susceptible CNO79.

Particularly obvious was the transgressive segregation of plants as susceptible as CNO79, the highly susceptible parent, in the cross between the two resistant parents. Progenies more resistant than the parents were identified, with infection levels of less than 5% ISR. However, these values were not significantly different from those of the parents, which had ISR values of 8 to 17%. The reason for the lack of significant differences at the resistant tail of the distribution lies in the inoculation method used to measure resistance to disease spread within the head. The inoculum was directly placed on the stigma of a floret in the central spikelet at the time of flowering. This is the ideal time and place for infection. In all heads observed of the parents and the F7 lines, the inoculated spikelet itself always developed disease symptoms. Progeny lines with higher levels of resistance than the parents would not only need to show resistance to spread of the fungus from the inoculated spikelet (Type II resistance), but also to infection itself (Type I resistance). In that case, the inoculated spikelet would have remained free of symptoms, and disease values would likely have been significantly lower than those of the parents. However, Type I resistance (immunity) to disease was not the objective of this study and probably does not exist in wheat (17). Therefore, the type of resistance studied and the method of inoculation used did not allow the detection of F7 lines significantly more resistant than the parents.

The qualitative analysis showed that although two genes operate in each of the resistant cultivars, a small maternal effect on disease expression was present on the first inoculation date when Ning 7840 was used as the female parent. Other researchers have not found maternal effects (17).

In the quantitative analysis, the minimum numbers of effective factors were very close to those from the analysis of genetic ratios. Each cultivar contained close to two genes, and the cross between the two resistant lines indicated 3.5 to 4.0 genes to be segregating.

In a study by Singh et al. (21), three genes were postulated for Frontana. In the present study, quantitative analysis data from the second inoculation date gave several estimates for the minimum number of factors in Frontana that were at the 2.4 to 2.5 level. A reason for the difference in estimates may be that in this study CNO79 was used as the susceptible parent, while Singh et al. (21) used Inia 66 and Opata 85. Data obtained from the two studies are in general agreement. Bai et al. (1) also found few genes of major effect to be operating in resistant materials.

The data are presented separately for the two inoculation dates. One reason was to illustrate the presence of a slight maternal effect in the crosses with Ning 7840 in the first inoculation date. However, in addition, the consistency of the data between the two inoculation dates stresses the importance of the individual heads, rather than the plant or the plot, being in the same stage of growth at the time of inoculation. Thus, on a plot basis, this allows the experimenter some flexibility in carrying out the inoculations. Later heads from later tillers can be used, but they should all be inoculated at anthesis.

From the genotypic correlation coefficients and the similarity of gene postulations obtained, it can be concluded that the three methods of assessing resistance essentially scored the same basic process of disease spread within a head. The simplest method of scoring disease will suffice to obtain conclusive data. The infected spikelet rate, which is equivalent to the percentage of scabbed spikelets, can be determined on an individual head basis and therefore may be desirable in certain cases, such as when screening large numbers of populations consisting of unique individuals (e.g., an F2 population). However, this requires precise counting of the total number of spikelets and the number of scabbed spikelets. The reaction index and the disease index use visual estimates of the portion of the head bearing scabbed spikelets but are based on the mean of a number of heads. Hence, the reaction index or the disease index may be the trait of choice for more precisely evaluating resistance at the advanced line stage or when identifying potential parents.

Table 6. Estimation, based on F7 line distribution, of the number of effective factors contributing resistance following infection with *Fusarium graminearum*

	Frontana × CNO79	Ning 7840 × CNO79	Frontana × Ning 7840
1st inoculation			
ISR	1.9	2.1	3.8
RI	1.5	1.9	3.5
DI	1.3	2.1	3.7
2nd inoculation			
ISR	2.5	1.9	3.2
RI	1.8	2.3	3.5
DI	2.4	2.3	3.4

Table 7. Genotypic coefficients of correlation between infected spikelet rate (ISR), reaction index (RI), and disease index (DI) following infection with *Fusarium graminearum*

	Frontana × CNO79	Ning 7840 × CNO79	Frontana × Ning 7840
1st inoculation			
ISR/RI	1.00	0.82	0.64
ISR/DI	1.00	0.83	0.64
RI/DI	0.99	0.82	0.64
2nd inoculation			
ISR/RI	0.99	0.82	0.63
ISR/RI	1.00	0.83	0.63
RI/DI	0.99	0.81	0.63

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