Specificity of a Rust Resistance Suppressor on 7DL in the Spring Wheat Cultivar Canthatch

Mina Talajoor, Yue Jin, Anmin Wan, Xianming Chen, Sridhar Bhavani, Linda Tabe, Evans Lagudah, and Li Huang

First and eighth authors: Montana State University, Department of Plant Sciences and Plant Pathology, Bozeman 59717-3150; second author: Cereal Disease Laboratory, United State Department of Agriculture–Agricultural Research Service (USDA-ARS), St. Paul, MN 55108; third and fourth authors: Wheat Genetics, Physiology, Quality, and Disease Research Unit, USDA-ARS, Pullman, WA 99164-6430; fifth author: International Maize and Wheat Improvement Center (CIMMYT), ICRAF House, United Nations Avenue, Gigiri, P.O. Box 1041, Village Market 00621, Nairobi, Kenya; and sixth and seventh authors: CSIRO Plant Industry, GPO Box 1600, Canberra, ACT 2601, Australia.
Accepted for publication 30 November 2014.

ABSTRACT

Talajoor, M., Jin, Y., Wan, A., Chen, X., Bhavani, S., Tabe, L., Lagudah, E., and Huang, L. 2015. Specificity of a rust resistance suppressor on 7DL in the spring wheat cultivar Canthatch. Phytopathology 105:477-481.

The spring wheat 'Canthatch' has been shown to suppress stem rust resistance genes in the background due to the presence of a suppressor gene located on the long arm of chromosome 7D. However, it is unclear whether the suppressor also suppresses resistance genes against leaf rust and stripe rust. In this study, we investigated the specificity of the resistance suppression. To determine whether the suppression is genome origin specific, chromosome location specific, or rust species or race specific, we introduced 11 known rust resistance genes into the Canthatch background, including resistance to leaf, stripe, or stem rusts, originating from A, B, or D genomes and located on different chromosome homologous groups. F_1 plants of each cross were tested with the corresponding rust race, and the infection types were scored and compared with the parents. Our results show that the Canthatch 7DL suppressor only suppressed stem rust resistance genes derived from either the A or B genome, and the pattern of the suppression is gene specific and independent of chromosomal location.

Wheat is one of the most consumed cereals in the world, feeding over 40% of the world's population. Wheat cultivation mainly focuses on two major species: *Triticum turgidum durum*, called durum wheat, a tetraploid species ($2n = 4\times = 28$) that resulted from a hybridization between *T. urartu* ($2n = 2\times = 14$, genome AA) and *Aegilops speltoides* ($2n - 2\times = 14$, genome SS) (3,22); and *T. aestivum* ($2n = 6\times = 42$, genome AABBDD), called bread wheat, a hexaploid species that arose from an additional allopolyploidization event between durum and *A. tauchii* ($2n = 2\times = 14$, genome DD) (4,15).

Since its earliest domestication, wheat has been subjected to breeding and selection in an effort to enhance or incorporate beneficial traits. Breeding for disease resistance has been one of the most important activities in any breeding program. Historically, cytogeneticists and breeders have often made crosses with the diploid or tetraploid donors, to introduce new resistance (*R*) genes or traits into their desirable but otherwise susceptible cultivars. During these practices, the apparent suppression of *R* genes from donors of lower ploidy species was noted on a number of occasions following attempts to transfer the genes into bread wheat (11). In investigating this phenomenon, Kerber and Green (11) found that Tetra Canthatch, the tetraploid component ($2n = 28$ = AABB) derived from 'Canthatch', displayed an increase in resistance to races of *Puccinia graminis* f. sp. *tritici* causing stem rust, compared with its hexaploid counterpart, Canthatch ($2n = 42$ = AABBDD). The removal of the D genome somehow nullified suppression, and permitted expression of stem rust resistance (*Sr*) genes in the background (Table 1). Furthermore, Canthatch nullisomic 7D (lacking the pair of 7D chromosomes), and Canthatch ditelosomic 7DS (containing only the short arms of 7D) showed enhanced resistance to stem rust similar to Tetra Canthatch. In contrast, Canthatch ditelosomic 7DL showed susceptibility similar to Canthatch, suggesting that the suppressor was located on chromosome 7DL.

Canthatch is believed to have stem rust resistance specificities *Sr5*, *Sr7a*, *Sr9g*, *Sr12*, and *Sr16* in its background; however, it displays susceptible infection types (ITs) to the stem rust races that are avirulent to some of these *Sr* genes (11). Thus, the Canthatch suppressor was deduced to be effective against some of these *Sr* genes. Several ethyl methanesulphonate- (EMS)-induced mutations at the 7DL suppressor locus were generated and found to have acquired resistance to a number of stem rust races, consistent with the deduced specificity of the wild-type suppressor for the listed *Sr* genes (9,23). However, the specificity of the suppressor with respect to resistance against leaf rust caused by *P. triticina* or stripe rust caused by *P. striiformis* f. sp. *tritici* has not been reported. The goals of this study were to further investigate the specificity of the Canthatch suppressor by determining its activity against a number of leaf and stripe rust resistance genes, as well as additional *Sr* genes, with a view to establishing its pattern of suppression.

MATERIALS AND METHODS

Plant materials. Plant material Canthatch (CItr 13345 TR04ID SD) was obtained from the United State Department of Agriculture National Plant Germplasm System. Canthatch EMS nonsuppressor mutant CTH-NS1 was kindly provided by Dr. E Kerber, and maintained at CSIRO, Canberra, Australia by Dr. Evans Lagudah. The origins of plant material containing leaf, stem, or stripe rust resistance genes are outlined in Table 1.

Plant growth conditions. Seed were planted in 10 cm pots with Sunshine Mix number 1 (Sun Gro Horticulture, Vancouver, British Columbia, Canada) in a greenhouse at the Montana State University (MSU) Plant Growth Center. Plants were grown under

Corresponding author: L. Huang; E-mail address: lhuang@montana.edu

http://dx.doi.org/10.1094/PHYTO-09-14-0261-R

a 16-h photoperiod with an average of 16 and 22°C day and night temperatures, respectively. Plant material involving temperature-sensitive *Sr6* was grown in a temperature-controlled growth chamber set to a constant temperature of 19°C. Plants were watered as needed. Micronutrients of Peter's general-purpose 20-10-20 plant food plus (Scotts-Miracle-Gro Company, Marysville, OH) were added to the water (N at 150 ppm) and fertilized starting when the plants reached two-leaf stage.

Wheat crosses. Cultivars carrying the *R* genes (Table 1 listed in brackets) studied were crossed with Canthatch as male parents to exclude false hybrids. Meanwhile, reciprocal crosses using Canthatch as a male parent were made in case the resistance was suppressed in the F_1. A video protocol detailing this process, named "crossing wheat", can be accessed from the online protocol hosted by the Department of Plant Sciences and Plant Pathology, MSU faculty page (https://vimeo.com/48607612). Approximately 20 to 50 F_1 seed were harvested for each cross and at least 15 seedlings were subsequently screened to assess the suppression of rust resistance.

Rust inoculation. Leaf rust inoculations were done at two-leaf stage with *P. triticina* race PBJJG. All leaf rust resistance (*Lr*) genes used in this study confer resistance to this race, while Canthatch is susceptible. Urediniospores were mixed in Soltrol 170 Isoparaffin (Chempoint, Bellevue, WA) at a concentration of 5 mg/ml, and sprayed onto the leaves using a Badger 350 airbrush gun and Propel propellant (Badger Air-Brush Company, Franklin Park, IL). The inoculated seedlings were then placed in a Percival I-60D dew chamber (Percival Scientific Inc., Perry, IA) with the water heated to 28°C and chamber wall at 11°C to achieve an ambient air temperature of 15 to 17°C. After 24 h of incubation, the plants were then placed back in an isolated greenhouse.

Stem rust inoculations with races TRTTF and TTKSK (Ug99) were performed at the Cereal Disease Laboratory, University of Minnesota. Inoculation protocols were followed as described by Jin et al. (7).

Plant materials for assessing stripe rust responses were sown in 10-cm pots filled with a pine bark and sand potting mix. Prior to sowing, pots were fertilized with the complete fertilizer Aquasol. Approximately 7 days later, after seedling emergence, pots were fertilized with urea. Seedlings were grown in the greenhouse at 16 to 20°C and inoculated when they were at the 1.5- to 2-leaf stage. They were inoculated with a urediniospore suspension in light mineral oil mists and incubated in humidity chambers at 10°C for 24 h in the dark, then transferred to a growth chamber with a diurnal temperature cycle that gradually changed from 4°C at 2:00 A.M. to 20°C at 2:00 P.M. with 16-h photoperiod.

Disease assessment. Disease responses to leaf rust were evaluated at 8 to 10 days postinoculation (dpi) when disease had fully developed on susceptible lines. Individuals were assigned an IT using a 0-to-4 scale, in which 0 indicates no visible symptoms and 4 indicates complete susceptibility (17). Primary leaves were given an IT on a scale of 0 to 4, with 0 being completely immune and 4 being completely susceptible. Stem-rust-inoculated seedlings were evaluated at 14 dpi using the same IT scale. Evaluation of disease response to stripe rust was done at 20 to 22 dpi based on a 0-to-9 scale (12).

F_1 seedlings displaying a phenotype intermediate to that of the parents were advanced to F_2 generation for further evaluation.

Genetic analysis. A χ^2 test was performed to assess goodness of fit between observed data and theoretical segregation ratios.

RESULTS

The results of Kerber and Green (11) revealed that *Sr* genes suppressed in the Canthatch background are located on three different chromosomes from two genome origins (Fig. 1). To detect any further *Sr* genes recovered by inactivating the suppressor on 7DL of Canthatch other than those previously reported, we tested Canthatch and a near-isogenic EMS mutant of Canthatch, CTH-NS, containing an inactivated suppressor, with five additional *P. graminis* f. sp. *tritici* races (Table 2). Canthatch was highly resistant to two races and was fully susceptible to TRTTF, TTKSK,

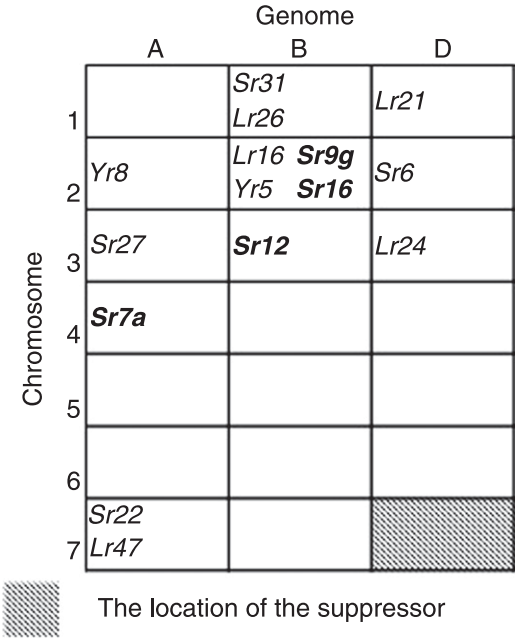

Fig. 1. Genome origin and chromosome location of the resistance genes. Genome origin and chromosome locations of the genes suppressed by the 7D suppressor (in bold) and genes that were newly introduced into Canthatch in this study are shown.

TABLE 1. Plant material used in this study[a]

Cultivar/ID	Line/accession	Gene	Origin	Pedigree	Source
Canthatch	CItr 13345			Thatcher*6/Kenya Farmer	Manitoba, Canada
CTH-NS1				EMS mutant derived from Canthatch	Evans Lagudah
ISr6-Ra	CItr 14163	*Sr6*	*Triticum aestivum*	Red Egyptian/Chinese Spring	Yue Jin, USDA
SwSr22T.B.		*Sr22*	*T. monococcum*		Yue Jin, USDA
73,214,3-1/9*LMPG		*Sr27*	*Secale cereale*		Yue Jin, USDA
Sr31/6*LMPG		*Sr31*	*Secale cereale*		Yue Jin, USDA
TcLr16	CItr 15239	*Lr16*	*T. aestivum*	Exchange/Thatcher recurrent parent	NPGS
Lovitt		*Lr21*	*Aegilops tauchii*		
Sundor	PI 495818	*Lr24*	*Thinopyrum ponticum*	3AG14/4*Condor	NPGS
Bobwhite		*Lr26*	*Secale cereale*		Robert Bowden, USDA
Scholar+Lr47		*Lr47*	*A. speltoides*		Luther Talbert
AvSYr5NIL		*Yr5*	*Triticum aestivum* subsp. *spelta*		Xianming Chen
AvSYr8NIL		*Yr8*	*T. comosum*		Xianming Chen

[a] Abbreviations: NPGS = National Plant Germplasm System, USDA = United State Department of Agriculture, and NIL = near-isogenic line.

and TMLKC. CTH-NS had a response to races QFCSC and TRTTF similar to that of Canthatch but displayed an enhanced resistance to races TMLKC and TTKSK (commonly known as Ug99). This specificity supports the observation that some *Sr* genes are suppressed in Canthatch.

The Canthatch 7DL suppressor was only reported to suppress *Sr* genes. Resistance to leaf and stripe rust was not assessed in the CTH-NS1 mutant (11), although apparent suppression of *Lr* genes in hexaploid wheat has been observed in other situations (1,2,6,16,19). It is unclear whether the suppression is specific to *Sr* genes or occurs in *Lr* and *Yr* (stripe or yellow rust resistance gene) genes as well. We screened Canthatch and CTH-NS1 with three races of *P. triticina* and five races of *P. striiformis* f. sp. *tritici*. Both the mutant and wild type were susceptible to all races of *P. triticina* and *P. striiformis* f. sp. *tritici* tested, suggesting that there is no new resistance to leaf or stripe rust recovered after inactivating the suppressor.

The published evidence indicates that enhanced resistance to stem rust in CTH-NS1 is due to the inactivation of the suppressor caused by mutation, which allows expression of the *Sr* resistance specificities that were in the wild-type Canthatch. No detectable resistance to leaf and stripe rust in the mutant led to the hypothesis that neither Canthatch nor the mutant CTH-NS1 have any *Lr* or *Yr* genes in the background that were resistant to the races that were tested. To test whether the 7D suppressor also suppresses leaf rust or stripe rust resistance genes, we crossed Canthatch with various lines carrying known *R* genes for leaf or stripe rust resistance (Table 1). We selected the additional *R* genes located on the same chromosome homologous group as the suppressor and of the same genome origin as the *Sr* genes suppressed by the suppressor (Table 1), including five *Lr* genes (TcLr16 [*Lr16*], Lovitt [*Lr21*], Sundor [*Lr24*], Bobwhite [*Lr26*], and Scholar+*Lr47* [*Lr47*]) and two *Yr* genes (*Yr5* and *Yr8*). We also included four additional *Sr* genes in our study (*Sr6*, *Sr22*, *Sr27*, and *Sr31*) to test whether any *Sr* genes are subject to suppression.

The suppressor was reported to be dominant (9) or semidominant (23); therefore, if an *R* gene was suppressed by the suppressor; the IT of the F_1 should be susceptible. To exclude false hybrids, F_1 seed from the crosses in which resistant lines were used as male parents were tested first. Each test included 15 F_1 seedlings and 10 of each their respective parents with rust races that are avirulent on the resistant parent (as conferred by the *R* gene) and virulent on Canthatch. If F_1 seedlings were resistant, then the F_1 must be true hybrids because any false F_1 from selfed female parent Canthatch should be susceptible. If the seedlings were susceptible, then F_1s from the reciprocal crosses were tested to verify that this was suppression, not false hybrids. The results are summarized in Table 3. *P. triticina* race PBJL is virulent on Canthatch (IT 3) and avirulent on *Lr16* (IT 1+), *Lr21* (IT ;), *Lr24* (IT 0;), *Lr26* (IT ;), and *Lr47* (IT ;1+). F_1 seedlings of three crosses involving *Lr16*, *Lr24*, and *Lr26* showed the same level of resistance to PBJL as the resistant parents, indicating that there is no suppression of these *Lr* genes. F_1 seedlings with *Lr21* (parental IT ;) and *Lr47* (parental IT ;) displayed an intermediate IT that was slightly higher than their resistant parent (IT 1; and IT 1 to 2, respectively) (Table 3).

P. striiformis f. sp. *tritici* race PSTv-14 was used to screen for suppression of *Yr5* in F_1 seedlings. Canthatch was fully susceptible and showed an IT of 8 on a scale of 1 to 9. The resistant parent showed an IT of 1. The F_1 seedlings were highly resistant (IT 2). *P. striiformis* f. sp. *tritici* race PST-17 was used to screen seedlings with *Yr8*. Canthatch was susceptible (IT 8) and the resistant parent and F_1 seedlings were both resistant (IT 2 to 3) (Table 3). These results suggest that the Canthatch 7DL suppressor does not suppress resistance conferred by either *Yr5* or *Yr8*.

Crosses with *Sr31* were screened with *P. graminis* f. sp. *tritici* race TRTTF. Canthatch was susceptible (IT 3 to 3+) and *Sr31* was resistant (IT 2– to 2). F_1 was also resistant (IT 1 to 2). Race TTKSK was used to screen *Sr22* (IT 2–) and *Sr27* (IT ;). Canthatch was susceptible (IT 3 to 3+). F_1s with *Sr22* showed an IT 2. F_1s with *Sr27* showed an IT ;. These results indicate that the Canthatch suppressor does not suppress *Sr22*, *Sr27*, or *Sr31*.

Race TMLKC was used to screen *Sr6*. Canthatch displayed an IT of 3+C to 4 while *Sr6* was highly resistant (IT 0;). F_1s with *Sr6* showed a slightly higher response than the resistant parent (IT 1;).

The intermediate ITs observed in crosses between Canthatch and *Lr47* or *Lr21* could be explained by dosage effect of the *R* gene or, possibly, by suppression. To determine the mechanism behind the intermediate phenotype, we screened the F_2 generation and analyzed the segregation ratios. We tested the F_2 seedling ITs against two segregation ratios. If the *R* genes were simply displaying a dosage effect, then the intermediate phenotype would still be considered a resistant reaction and segregate with a ratio of 3:1 resistant to susceptible. If the *R* gene was, indeed, being suppressed, then the intermediate phenotype would be the interaction between the suppressor and the *R* gene. If the intermediate IT phenotype is considered to be a susceptible reaction, a segregation ratio of 3:13 resistant to susceptible should be expected. There would be a substantially lower number of highly resistant individuals (IT ;) which are homozygous for both the *R* gene and nonsuppressor loci.

We tested goodness of fit for the two hypothesized segregation ratios. To test the 3:1 ratio, we combined the observed resistant and observed intermediate, assuming that the *R* gene was expressed but displayed a dosage effect. To test the 3:13 ratio, we assumed that the intermediate IT we observed was due to partial suppression of resistance by the suppressor. Thus, we combined the observed intermediate and observed susceptible categories. In all four cases, we rejected the hypothesis that the expected ITs segregate in a 3:13 ratio (Table 4). Additionally, in all four cases, we failed to reject our hypothesis that the observed ITs fit a 3:1 ratio. These results suggest that the *R* genes were expressed and showed a dosage effect.

DISCUSSION

Resistance being suppressed or lost in a new polyploid background has been reported for *Lr* genes (1,2,6,16,19), *Sr* genes

TABLE 2. Infection types (ITs) of Canthatch and CTH-NS seedlings with races of *Puccinia* spp.

Pathogen[b]	Race	IT[a]	
		Canthatch	CTH-NS
P. graminis f. sp. *tritici*	TRTTF[c]	3 to 3+	3–
	TTKSK[c]	3 to 3+	;23 to 22+C
	TMLKC[d]	3–C to 4	0; to ;1–
	QFCSC[d]	0;	0;
	BCCBC[d]	0;	0;
P. triticina	PBJJG[d]	3	3– to 3C
	TFBG[d]	3 to 3+	3 to 3+
	TFBJ[d]	3	3 to 3+C
P. striiformis f. sp. *tritici*	PSTv-14[e]	8	8
	PSTv-37[e]	8	8
	PSTv-40[e]	8	8
	PSTv-51[e]	7 to 8	8
	PST-17[e]	8	8

[a] For *Puccinia triticina* and *P. graminis* f. sp. *tritici* races, an IT scale of 0 (immune) to 4 (highest susceptible) was used, as described by McIntosh et al. (17). For *P. striiformis* f. sp. *tritici* races, an IT scale of 0 (immune) to 9 (highest susceptible) was used, as described by Line and Qayoum (12). Variations in IT between individuals are given as a range. IT classes are further qualified with a + (more than average) or a – (less than average). A semicolon (;) indicates the presence of hypersensitive flecks.

[b] Pathogens used for IT assessment.

[c] Assessment of disease conducted at Cereal Disease Laboratory located at the University of Minnesota.

[d] Assessment of disease conducted in the Montana State University Plant Growth Center.

[e] Assessment of disease conducted at Washington State University.

(2,6,11), stripe rust resistance genes (15,23), and powdery mildew resistance genes (20). It is generally believed that loss of resistance in a new genetic background is most likely due to a direct suppression of the *R* genes or suppression of a gene involved in the resistance response pathway.

One common feature of all reported suppression in polyploid wheat is that the suppressors and suppressed genes are always from different genome. Our findings agree with these reports in that none of the genes located on D genome were suppressed by the 7D suppressor, supporting the view that suppression may result from interactions across genomes, possibly between genes of similar DNA sequence (which could result in gene silencing) or similar function (which could lead to competition at the protein level).

Bread wheat is a hexaploid containing three different genomes, thus possessing three homologs for most gene loci. Where investigated, additive expression was detected at most of the loci in which all three homologs are expressed in wheat (14). However, nonadditive expression patterns were also reported; for example, there were complex interactions between homologous genes encoding an esterase on homologous group 6 chromosomes. The expression of the 6A esterase homolog was suppressed by the 6D esterase homolog, whereas the expression of the 6B homolog was not (14). Similarly, there are a number of examples in the literature in which the introduction of a disease *R* gene has been found to be suppressed by another homolog. For example, *Lr* gene *Lr23* is specifically suppressed by the ortholog located on 2DS (19). The powdery mildew resistance gene, *Pm8*, is located on a 1RS segment of rye (*Secale cereale*), and was suppressed by a suppressor located on 1AS after *Pm8* was transferred in hexaploid wheat by the 1B.1RS translocation (20,21). Thus, the suppression of *Lr23* (16,19) and the *Pm8* specificities (21) appear to be the result of direct interactions between very similar genes at homologous positions in the different genomes of wheat. McIntosh et al. (18) noted that the suppression of *Pm8* requires a full-length copy of *Pm3*, indicating that the *Pm3* gene must be expressed for suppression to occur. More recent studies on *Pm8* interaction with *Pm3*, showed a heteromeric complex between the two proteins and points to a posttranslational mechanism involvement in the suppression effect (5).

A characteristic of direct suppression of the *R* genes is that the suppressor and suppressee are located on the same homologous group; for example, group 2 in the case of *Lr23*. However, this is not the case for most of the disease resistance suppression reported in wheat (1,2,6,8,13), including the stem rust resistance suppression by the Canthatch 7D suppressor (11). A previous study indicated that the Canthatch suppressor inhibits the function of four *Sr* genes located on three different homologous groups and two different genomes (11). Among the four genes thought to be suppressed in Canthatch, *Sr9g* and *Sr16* locate on 2B, *Sr12*, on chromosome 3B, and *Sr7a* on 4A (11). The different homologous locations of the 7D suppressor and its suppressed genes argues against the possibility of direct suppression of the *R* genes by their cross-genome homologs, and suggests the hypothesis that the targets of the suppressor may be a component of a defense response pathway shared by the affected *R* genes. Alternatively, direct suppression may be possible if the four postulated suppressed genes share closely related encoding sequences with the Canthatch suppressor.

TABLE 3. Infection types (ITs) of F_1 seedlings and parents with races of *Puccinia* spp.[a]

Pathogen[c]	Race	*R* gene[d]	IT[b]		
			Resistant parent	Canthatch	F_1
P. triticina	PBJL[e]	*Lr16*	1^+	3	1^+
	PBJL[e]	*Lr21*	;	3	1;
	PBJL[e]	*Lr24*	0;	3	;
	PBJL[e]	*Lr26*	;	3	;
	PBJL[e]	*Lr47*	;1^+	3	1 to 2
P. striiformis f. sp. *tritici*	PSTv-14[f]	*Yr5*	1	8	2
	PST-17[f]	*Yr8*	2 to 2^-3	8	2 to 3
P. graminis f. sp. *tritici*	TRTTF[g]	*Sr31*	2^- to 2	3 to 3^+	1 to 2
	TTKSK[g]	*Sr22*	2^-	3 to 3^+	2
	TTKSK[g]	*Sr27*	;	3 to 3^+	;
	TMLKC[g]	*Sr6*	0;	3^+C to 4	1;N

[a] F_1s were derived from the crosses in which resistant lines were used as male parents.
[b] For *Puccinia triticina* and *P. graminis* f. sp. *tritici* races, an IT scale of 0 (immune) to 4 (highest susceptible) was used, as described by McIntosh et al. (17). For *P. striiformis* f. sp. *tritici* races, an IT scale of 0 (immune) to 9 (highest susceptible) was used, as described by Line and Qayoum (12). Variations in IT between individuals are given as a range. IT classes are further qualified with a + (more than average) or a − (less than average). A semicolon (;) indicates the presence of hypersensitive flecks.
[c] Pathogens used for IT assessment.
[d] Resistance gene.
[e] Assessment of disease conducted in the Montana State University Plant Growth Center.
[f] Assessment of disease conducted at Washington State University.
[g] Assessment of disease conducted at Cereal Disease Laboratory located at the University of Minnesota.

TABLE 4. F_2 segregation ratio χ^2 test

R gene[a]	Observed			Total	Expected		Ratio tested	*P* value
	Resistant	Intermediate	Susceptible		Resistant	Susceptible		
Lr21	23	24	12	59	44.25	14.75	3:1	0.41
					11.06	47.94	3:13	6.84E-05
Lr47	34	45	30	109	81.75	27.25	3:1	0.54
					20.44	88.56	3:13	8.74E-04
Lr16	103	11	39	153	114.75	38.25	3:1	0.89
					28.69	124.31	3:13	1.84E-53
Sr6	40	72	32	144	108.00	36.00	3:1	0.44
					27.00	117.00	3:13	5.51E-03

[a] Resistance gene.

The 7D suppressor did not show any evidence of suppression of leaf rust or stripe rust resistance, because the mutant CTH-NS did not display any resistance to any of the *P. triticina* and *P. striiformis* f. sp. *tritici* races tested. Consistent with this, none of the *Lr* or *Yr* genes crossed into the Canthatch background were suppressed (Table 3). Thus, our results add to published evidence that the Canthatch 7DL suppressor is specific to stem rust resistance. Furthermore, the lack of suppression of four additional *Sr* genes, spread across the three genomes of wheat (Tables 1 and 3), supports the view that the suppressor acts only on specific *Sr* genes. The Canthatch nonsuppressor mutant CTH-NS showed enhanced resistance to stem rust races TTKSK and TMLCK (Table 2) but F_1 plants containing *Sr6*, *Sr22*, or *Sr27* showed full resistance to these rust races (Table 3). Together, these data indicate that the suppressor, although not active against *Sr22*, *Sr27*, or *Sr6*, is active against uncharacterized *R* genes in the Canthatch background that are effective against TTKSK or TMLKC. The specificity of the suppressor for a subset of *Sr* genes may imply that the suppressor targets a feature of a resistance response pathway shared by only the suppressed *Sr* genes. Because none of the genes suppressed by the 7D suppressor has been cloned and no study of their pathways has been reported, further investigation is required to test this hypothesis.

Canthatch was developed by crossing *Sr7a* from 'Kenya Farmer' wheat into 'Thatcher' (the recurrent parent). Thatcher was also confirmed to have the 7DL suppressor allele (10). Thatcher is commonly used to develop near-isogenic lines with various *Lr* genes for identifying various rust races, and the source of *Lr16* used in this study was in the Thatcher background. Furthermore, the durum wheat 'Iumillo' is a parent of Thatcher and shows a higher level of resistance to races of *P. graminis* f. sp. *tritici* that is not seen in Thatcher or Canthatch (11). The main factor influencing the detection of the Canthatch 7DL suppressor in Thatcher is the antisuppressing effect of the adult plant *R* gene *Lr34* (10). When *Lr34* is present in the Thatcher background, it permits expression of stem rust resistance despite the presence of the 7DL suppressor (10). Although *Lr34* confers broad-spectrum resistance to a variety of pathogens, it does not confer seedling resistance to stem rust. However, Kerber and Aung (10) discovered that Canthatch + *Lr34* displayed stem rust resistance to races that are virulent on the existing *Sr* genes in the background, suggesting that *Lr34* permitted expression of additional *Sr* genes in the Canthatch background.

The presence of suppressors and their apparent ubiquity among polyploid species seems to be the result of interactions that occur when addition of an extra genome introduces extra copies of homologous genes whose products interfere with each other at either the nucleic acid or protein levels. For example, similar proteins with differing activities could either compensate for each other or compete with each other for the same binding partner. As a result, when a gene is relocated into a polyploid background, the expression could be shut down due to the existence of the homologs that compensate the function, or the gene product could lose its binding advantage due to the competition of a decoy homolog. The complexity of intergenome interactions could result in different patterns of resistance suppression. Further investigation of the mode of action of the 7D Canthatch suppressor and other suppressors in wheat would be a valuable tool to "unlock" the genome and enable breeders to utilize new sources of resistance.

ACKNOWLEDGMENTS

This study was supported by the NSF BREAD program (IOS-0965429).

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