Evaluation of Elite Wheat Germ Plasm for Resistance to Tan Spot

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ABSTRACT

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Tan spot, caused by *Pyrenophora tritici-repentis*, is a serious foliar disease of wheat (*Triticum aestivum*) in North America. Control of tan spot through management practices and fungicide application is possible; however, the use of resistant varieties is the most effective and economical means of controlling tan spot. This study was conducted to determine the disease reaction of 126 elite hard red spring, white, and durum wheat varieties and advanced breeding lines collected from the northern Great Plains of the United States and Canada to individual races/toxins of *P. tritici-repentis*. Seedling evaluation of the 126 genotypes was done under controlled environmental conditions with virulent races 2, 3, and 5 of *P. tritici-repentis* and toxins Ptr ToxA and Ptr ToxB. Based on disease reactions, two resistant varieties and two advanced breeding lines adapted to the northern Great Plains were found to be resistant to all the races and insensitive to the toxins tested. Additionally, six genetically diverse lines/varieties were identified to be resistant to tan spot; however, these sources may not be well adapted to the northern Great Plains. These results suggest that the wheat germ plasm contains a broad genetic base for resistance to the most prevalent races of *P. tritici-repentis* in North America, and the resistant sources identified in this study may be utilized in wheat breeding programs to develop tan spot resistant varieties.

The fungus *Pyrenophora tritici-repentis* (Died.) Drechs. (anamorph: *Drechslera tritici-repentis* (Died.) Shoem.) causes tan spot of wheat (*Triticum aestivum* L.), an important foliar disease affecting grain quality and production (6,26). Intensified wheat production, changes in cultural practices including shifts from conventional tillage and stubble burning to reduced tillage practices, and shorter crop rotations have resulted in development of tan spot in epidemic proportions worldwide (7). Management practices such as use of nonhost plants in the crop rotation, destruction and avoidance of infested straw, stubble, and volunteer plants by either burning or burying are useful for controlling tan spot. However, stubble burning and tillage increase the risk of soil erosion and can contribute to pollution of the environment. Similarly, although the application of fungicides is effective in controlling tan spot,

their use may leave undesirable residues and is not cost-effective due to low grain prices. Therefore, resistant varieties are the most effective and economical means of controlling tan spot of wheat (7).

P. tritici-repentis induces two distinct symptoms on susceptible wheat cultivars, tan necrosis and extensive chlorosis, both of which are under independent genetic control in the host (19,24). Based on the ability to induce necrosis and/or chlorosis on a set of wheat differential cultivars, isolates of *P. tritici-repentis* are presently classified into eight races (26). In recent years, preliminary reports of occurrence of three more virulent types of *P. tritici-repentis* have been made (1,3,27). Races 1 to 5 of *P. tritici-repentis* have been reported to occur in North America (2,22), while races 6, 7, and 8 have been identified from North Africa and the Middle East region (26).

The tan spot fungus produces different host-specific toxins, which have been associated with the necrosis and chlorosis symptoms induced on susceptible wheat cultivars. One well-characterized host-selective proteinaceous toxin, Ptr ToxA (6,22), produced by races 1, 2, 7, and 8 (26), is the main factor causing the necrotic symptom in susceptible wheat culti-

vars. Two chlorosis-inducing host-specific toxins, designated as Ptr ToxB (29), isolated from race 5, and Ptr ToxC (10), isolated from race 1, have been identified and well characterized. Genetic studies have established that sensitivity to toxins Ptr ToxA, Ptr ToxB, and Ptr ToxC and susceptibility to their producer races are each controlled by the same gene (10,18,29). They also observed that toxins could be used in screening for resistance as a surrogate for spore inoculation.

Resistance to tan spot of wheat is quantitatively (11,14) or qualitatively (8,9,19,24,33) inherited. Genetic studies indicate that a single recessive gene, *tsn1*, controls resistance to necrosis caused by races 1 and 2 in both durum and common wheat and is located on the long arm of chromosome 5B (4,13). Effertz et al. (10) mapped the locus controlling insensitivity to toxin Ptr ToxC to the short arm of chromosome 1A in common wheat. Friesen and Faris (16) identified in common wheat a major quantitative trait locus (QTL) associated with insensitivity to Ptr ToxB to the distal tip of the short arm of chromosome 2B, and this QTL accounted for 69% of the phenotypic variation.

The epidemic occurrence of tan spot in recent years in major wheat-growing regions worldwide and high pathogenic variability in populations of *P. tritici-repentis* have resulted in a greater urgency to incorporate multiple resistance genes and broaden the genetic base of resistance in adapted varieties (7,18,28,34). Breeding for resistance to tan spot may be enhanced if additional resistance sources that are effective against multiple races of *P. tritici-repentis* are identified. The main objectives of this study were to (i) screen elite wheat breeding lines and currently released wheat varieties to the major virulence present in *P. tritici-repentis* in North America, (ii) identify resistance sources effective against tan spot, and (iii) provide wheat breeders with sources of resistance and effective means of evaluating tan spot resistance to allow them to transfer tan spot resistance into adapted wheat.

MATERIALS AND METHODS

Plant material. A collection of 126 elite wheat germ plasm including hard red

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spring wheat, white wheat, and durum varieties along with advanced breeding materials from both private and public wheat breeding programs of the northern Great Plains of the United States and Canada were evaluated in greenhouse studies. The different types of wheat genotypes used in this study, their pedigrees, and sources of origin are given in Table 1.

Spore inoculation. A single-spore culture of isolates Ptr 86-124 (race 2), Ptr 331-9 (race 3), and Ptr DW13 (race 5) was used to produce spore inoculum. The isolates Ptr 86-124 and Ptr 331-9 were provided to North Dakota State University wheat leaf diseases laboratory by L. Lamari, University of Manitoba, Winnipeg, Canada. The race designation of each isolate was confirmed in our laboratory. Spore inoculum was produced by the method of Lamari and Bernier (21). Throughout this study, the spore inoculum concentration was 4,000 conidia per milliliter.

Seed of the 126 wheat genotypes, including checks Glenlea, Saloumini, 6B-365, and 6B-662, was pregerminated to minimize the chances of growth variation prior to planting in plastic cones (3.8 cm in diameter and 20 cm long) filled with Fison sunshine blend #1 (Fison Horticulture, Vancouver, B.C.). The seedlings were grown in the greenhouse at an average temperature of 21°C with a 16-h photoperiod. Four seeds were planted in each cone. The seedlings were watered and fertilized as needed. Plants at the two-leaf stage were sprayed with the conidial suspension until runoff using a CO₂ pressurized hand sprayer. Following inoculation, the seedlings were incubated for 24 h under continuous leaf wetness in a mist chamber and then placed on benches in a growth chamber set at 22/17°C (day/night) temperature with a 16-h photoperiod.

Eight days after spore inoculation, the seedlings were rated for their disease reaction, using a 1 to 5 lesion-type rating scale developed by Lamari and Bernier (21). Necrotic or chlorotic symptoms observed on genotypes were recorded.

Toxin and culture filtrate infiltration. The toxin Ptr ToxA, produced from isolate Ptr 86-124 (race 2), was kindly provided by S. W. Meinhardt, Department of Plant Pathology, North Dakota State University, Fargo. The toxin was purified as described previously by Zhang et al. (36). Culture filtrate containing the toxin Ptr ToxB was produced from isolate Ptr DW13 (race 5) by the procedures of Orolaza et al. (29).

Approximately 25 µl of the toxin/culture filtrate was infiltrated into the wheat leaves using a 1-ml syringe without a needle. The infiltrated second leaf was marked by a nontoxic permanent marker. Plant reactions to the toxin/culture filtrate infiltration were recorded 4 days after infiltration. The presence or absence of necrosis/chlorosis at the site of infiltration indicated sensitiv-

ity or insensitivity to the culture filtrate, respectively.

Experimental design and statistical analysis. To evaluate the 126 wheat germ plasm for each of the three races of *P. tritici-repentis*, two experiments were conducted in the greenhouse in spring and fall of 2005 for each race. Each experiment was conducted as a randomized block design with three replicates. Each replicate consisted of the 126 wheat genotypes planted in cones. Four plants planted in a cone formed the experimental unit.

An analysis of variance (ANOVA) for each race causing tan spot was performed using the Statistical Analysis System version 8.2 (31). The existence of significant differences ($P < 0.05$) in disease reaction among genotypes was calculated with PROC GLM using LSD option. For the reaction of 126 genotypes to the toxin/culture filtrate, four plants per genotype were infiltrated with the toxin Ptr ToxA and culture filtrate produced by race 5 (Ptr ToxB).

RESULTS AND DISCUSSION

Analysis of variance of the disease data revealed significant ($P < 0.05$) differences among genotypes in all experiments for the three races of *P. tritici-repentis*. There was no significant ($P < 0.05$) genotype $\times$ experiment interaction for any of the races studied. The results of statistical analysis permitted combining the two experiments for each of the three races, and mean disease score of each genotype with respect to races 2, 3, and 5 and the respective LSD ($P_{0.05}$) for each race is given in Table 1.

Ten genotypes showed resistance to all races and insensitivity to all toxins of *P. tritici-repentis* tested (Table 1). Among these resistant genotypes, two recently released varieties, Knudson and Oklee, and two advanced breeding lines, N99-0241 and MN03291, are well adapted to the northern Great Plains of the United States and, hence, can be successfully used in various wheat breeding programs. In order to broaden the genetic base of resistance to tan spot, the resistance from lines 92 MREHTR28B, CIMMYT L #18, and Intros #7 and varieties Salamouni and Erik can also be used. These sources of resistance may not be well adapted to the Great Plains of North America but can be utilized for broadening the genetic base of resistant germ plasm. All durum lines screened were susceptible; however, a tetraploid accession, *T. turgidum* #283 (PI P1352519), exhibited high resistance to all races and insensitivity to all toxins of *P. tritici-repentis* tested. The pedigree of resistant sources identified in this study indicate that they come from diverse genetic background and may possibly possess different resistance genes that can be utilized in gene pyramiding and developing cultivars with broad and durable resistance to tan spot.

The majority of genotypes evaluated with races 2 and 5 and their respective toxins gave similar reactions (Table 1). Similar findings were observed by other researchers (9,19). However, a few genotypes gave differential response to races 2 and 5 spore inoculation and their toxins. Differential responses to toxin and spore inoculation have also been observed by others (17,30). Friesen et al. (15) observed that segregation of insensitivity to necrosis induced by toxin Ptr ToxA was controlled by a single gene, while the disease reaction of the recombinant inbred lines to spore inoculation by race 2 revealed that four to five genes may control resistance in the population. In the lines showing differential response, they observed that lines were insensitive to Ptr ToxA but susceptible to spore suspension and not vice versa. Similar results were observed in this study. Additional experiments need to be conducted to reveal the exact reasons for the differential response of the genotypes identified in this study.

The time required for germ plasm evaluation of tan spot reaction by toxin/culture filtrate infiltration (4 days) is half the duration required by spore suspension (8 days). The toxins/culture filtrate can be stored for a very long time and can be reused in different batches. We estimate the expense involved in disease testing using toxin/culture filtrate would be less compared with that for spore inoculation in wheat breeding programs. Reaction to the culture filtrate is clear and easily differentiates between sensitive and insensitive plants (19). Toxin/culture filtrate infiltration is less influenced by environmental conditions (35) and can easily be combined with screening for resistance to other leaf-spotting diseases (personal observation). These results indicate that toxins/culture filtrates can be used for large-scale evaluation of early generation breeding material, but final confirmation of tan spot resistance has to be done by spore inoculum to avoid selection of lines that are resistant to toxins but susceptible to spore inoculum.

Tan spot has become a major component of the leaf spotting disease complex of wheat in the northern Great Plains of the United States and Canada (20,28). Both private and public wheat-breeding programs have given major emphasis to breeding for resistance to tan spot. Screening for tan spot resistance under natural infection is not always considered reliable enough for field screening (5). Artificial infection can be obtained by spreading infected straw or by spraying spore inoculum and covering with plastic tents to maintain humidity (23). However, these methods have practical constraints like production and quantification of large amounts of inoculum, maintenance of high humidity, and disease assessment based on percentage of diseased leaf area. As a re-

Table 1. Wheat type, origin, pedigree, and disease reaction of 126 elite wheat genotypes to *Pyrenophora tritici-repentis* races 2, 3, and 5, and toxins Ptr ToxA and Ptr ToxB

Genotype	Type ^a -origin ^b	Pedigree	Race ^c			Toxin ^d	
			R-2	R-3	R-5	ToxA	ToxB
Oxen	HRSW-SD	SDY366A/SDZ004A	3.5 N	1.8 R	1.9 R	+	–
Ingot	HRSW-SD	Butte86/SD3004/Dalen	3.5 N	2.3 C	2.8 C	+	–
Norpro	HRSW-CO	N93-0119	1.9 R	1.7 R	1.4 R	+	–
Parshall	HRSW-ND	Keene//Grandin*2/Glupro	3.1 N	1.6 R	3.8 C	+	+
Reeder	HRSW-ND	IAS20*4/H567.71//Stoa/3/ND 674	2.5 N	1.5 R	3.9 C	+	+
Alsen	HRSW-ND	ND 716 (Sumai3/Wheaton//Grandin)	3.5 N	1.9 R	2.0 R	+	–
Knudson	HRSW-CO	Karl/Krona/3/Bergen//Erik/MN73167	1.6 R	1.6 R	1.8 R	–	–
Hanna	HRSW-CO	N93-2424/AC DOMAIN	3.0 N	3.1 C	3.8 C	+	+
Briggs	HRSW-SD	BW114/Bergen/SD3097	2.2 N	2.4 C	2.5 C	+	+
Granite	HRSW-MT	Unknown	1.9 R	2.2 C	2.6 C	–	–
Oklee	HRSW-MN	2375/SBF0670	1.5 R	1.4 R	1.3 R	–	–
Dapps	HRSW-ND	Kitt/Amidon//GR/Stoa sib	2.1 N	2.8 C	4.0 C	–	+
Steele-ND	HRSW-ND	Parshall/ND706	3.7 N	3.0 C	4.2 C	+	+
Fryer	HRSW-CO	Sonja/Vance//Sumai3/Dalen	2.2 N	1.6 R	2.0 R	–	–
Trooper	HRSW-MT	Keystone/Ivan	2.0 R	2.0 R	1.6 R	–	–
Banton	HRSW-MN	Buck Antorcha/HJ 9	3.1 N	3.4 C	3.9 C	+	+
Saturn	HRSW-MN	ANB'S//HEGE312-75-262/CHAT'S	2.9 N	2.1 C	1.6 R	–	–
Polaris	HRSW-MN	Lona/4/Kent/B564//SO/3/Kent/2*B564//SAP	2.2 N	1.6 R	2.6 C	–	–
2375	HRSW-ND	Olaf//Era/Suqamuxi68/3/Cis/ND487//Lark	2.8 N	2.4 C	1.9 R	+	–
Verde	HRSW-MN	MN7663/SBY35A	2.5 N	2.0 R	2.8 C	–	+
Keene	HRSW-ND	Stoa's'/3/IAS20*4//H567.71//Amidon	3.6 N	1.9 R	4.3 C	–	+
Butte 86	HRSW-ND	Butte*2/3/ND551//Butte*2/ND507	3.5 N	1.9 R	4.2 C	+	+
McNeal	HRSW-MT	PI125000/Centana//PK176/Frontiera/3/Glenman	2.9 N	1.3 R	1.6 R	–	–
Walworth	HRSW-SD	SD 3116/OXEN	3.1 N	1.7 R	1.9 R	+	–
Dandy	HRSW-MN	Unknown	3.4 N	2.0 R	2.0 R	+	–
Granger	HRSW-SD	SD8070/SD3165	2.9 N	2.1 C	2.6 C	+	+
SD3618	HRSW-SD	SD8119/SD3236	3.6 N	3.1 C	3.2 C	+	+
SD3635	HRSW-SD	SD8070/SD3118//SD3225	3.6 N	3.1 C	3.1 C	+	+
SD3668	HRSW-SD	SD8089/SD8070	3.2 N	1.8 R	2.1 C	+	+
SD3687	HRSW-SD	SD3305/KS91W005-1-4//SD8089	3.4 N	2.3 C	1.9 R	+	–
SD3746	HRSW-SD	SD93380/SD3335//Oxen	3.5 N	3.1 C	3.9 C	+	+
SD3827	HRSW-SD	W94-320/SD3367//SD3335	3.6 N	2.6 C	2.8 C	+	–
SD3851	HRSW-SD	ND 2897/SD3219//SD3414	2.6 N	2.5 C	2.5 C	+	+
SD3854	HRSW-SD	ND 2897/SD3219//SD3414	3.3 N	2.0 R	4.1 C	+	+
SD3860	HRSW-SD	AZ99FHB-RC	3.2 N	2.1 C	2.2 C	–	–
SD3868	HRSW-SD	ND 2897/SD3479//SD3411	3.3 N	1.8 R	2.5 C	+	+
SD3870	HRSW-SD	FHBCOMP	3.1 N	1.8 R	3.9 C	+	+
BZ 998-447W	WW-MT	Spillman/WestBred 906R	3.0 N	2.0 R	2.9 C	–	+
CA-902-701	OW-MT	Keystone/Granite	2.1 N	2.8 C	1.8 R	–	–
CA-901-580W	WW-MT	White Sharpshooter/Lars	3.0 N	1.8 R	1.8 R	–	–
N99-0241	HRSW-CO	N93-0090/Hamer	1.9 R	1.4 R	1.8 R	–	–
96S0404-03	HRSW-CO	Hamer//RHT21/Lars	1.9 R	1.5 R	2.5 C	–	+
98S0051-1	HRSW-CO	N93-0243//CHISCAB#7/N93-0338	2.1 N	1.3 R	3.6 C	+	+
98S0127-06	HRSW-CO	N97-0214/3/N93-0338//Sumai3/Dalen	2.5 N	1.8 R	2.1 C	+	+
97S0254-8-1	HRSW-CO	N94-0236/IVAN//Verde	2.0 R	2.5 C	2.2 C	–	–
MN97369	HRSW-MN	MN97369-98*4/Pavon	3.0 N	1.5 R	3.8 C	+	+
MN97448	HRSW-MN	HJ98/SHARP	3.3 N	1.8 R	2.2 C	+	+
MN97695	HRSW-MN	MN92387/SBE0303-23	3.2 N	1.9 R	4.0 C	+	+
MN99017	HRSW-MN	Fujian 5114/Grandin	2.5 N	2.5 C	4.3 C	+	+
MN99436-6	HRSW-MN	Norlander/HJ98	3.4 N	1.7 R	1.7 R	+	–
MN00232-2-2	HRSW-MN	MN2540W/KULM//ND2710	3.0 N	2.2 C	3.9 C	+	+
MN00258-2	HRSW-MN	INGOT/HJ98	3.2 N	2.3 C	1.9 R	+	–
MN00261-4	HRSW-MN	MN95286/MN94155//Verde	2.3 N	1.8 R	1.6 R	+	–
MN01261-8	HRSW-MN	MN95229/ND694	2.9 N	1.8 R	3.8 C	–	+
MN01281	HRSW-MN	SD-AD B9/MN93434(2375/Grandin)	3.1 N	1.8 R	2.4 C	+	+
MN01333-A	HRSW-MN	MN97008/MN97695	3.1 N	1.7 R	2.0 R	+	–
MN01108-6	HRSW-MN	MN95229//MF9606/Aberdeen 9	3.4 N	2.7 C	3.7 C	+	+
MN01116	HRSW-MN	MN95211(PAR11(MRNC/BUC//BLD/PSN)/MN90124)// LizCimmyt9/MN95023	3.4 N	1.8 R	3.8 C	+	+
MN01180-4	HRSW-MN	LizCimmyt17/Parshall/HJ98	2.4 N	1.5 R	1.5 R	–	–

(continued on next page)

^a The different types of wheat: HRSW, hard red spring wheat; OW, other wheat; and WW, white wheat.

^b Origins of the genotypes studied: CAN, Canada; CO, Colorado; KS, Kansas; MEX, Mexico; MN, Minnesota; MT, Montana; ND, North Dakota; SD, South Dakota; and WA, Washington.

^c Plants were rated on a 1 to 5 lesion type rating scale (23) with 1 to 2 considered resistant (R) and 3 to 5 considered susceptible with necrosis (N) or chlorosis (C) symptoms.

^d Reactions to toxin/culture filtrate were scored as + and –, indicating sensitivity and insensitivity, respectively. Toxin Ptr ToxA is produced by race 2 and induces necrosis, while Ptr ToxB is produced by race 5 and causes chlorosis.

Table 1. (continued from previous page)

Genotype	Type ^a -origin ^b	Pedigree	Races ^c			Toxins ^d	
			R-2	R-3	R-5	ToxA	ToxB
MN01278-7	HRSW-MN	MN94200/SD-AD B61	3.7 N	1.8 R	1.8 R	+	-
MN02255	HRSW-MN	ND721/MN97448	3.5 N	1.9 R	3.9 C	-	+
MN02258	HRSW-MN	ND724/MN97448	2.5 N	1.9 R	2.3 C	+	-
MN02296-A	HRSW-MN	Verde/MN93415/HJ98	2.0 R	1.5 R	3.4 C	+	+
MN03098	HRSW-MN	MN97803-10/DANDY-1	2.2 N	3.0 C	1.8 R	-	-
MN03119	HRSW-MN	Alsen-1/MN97803-10	3.2 N	2.0 R	1.7 R	-	-
MN03130	HRSW-MN	MN97695-4/MN97448-17	3.2 N	2.0 R	1.6 R	+	-
MN03148	HRSW-MN	MN99390-1/MN97803-10	2.8 N	1.8 R	3.6 C	-	+
MN03239	HRSW-MN	MN98543/MN97448	2.8 N	1.9 R	3.9 C	-	+
MN03243	HRSW-MN	01X019-3/MN97803-10	2.6 N	3.0 C	4.0 C	+	+
MN03279	HRSW-MN	MN98224/MN97448	2.1 N	1.7 R	1.6 R	-	-
MN03291	HRSW-MN	MN98389/MN98021	2.0 R	1.4 R	1.9 R	-	-
MN03308	HRSW-MN	MN95002/Parshall/MN97518	1.6 R	1.3 R	3.5 C	-	+
MN03328	HRSW-MN	Alsen-1/MN97448-17	2.7 N	1.9 R	2.5 C	+	+
WA007925	OW-WA	Spillman/WPB906R//Sunstar II	3.1 N	2.1 C	1.7 R	-	-
WA007931	WW-WA	PI591045/3/Tanager 'S'/Torim 73//Spillman	3.7 N	2.3 C	1.6 R	+	-
MT 0245	HRSW-MT	MT9433/Reeder	3.1 N	2.0 R	2.0 R	-	-
MT 0249	HRSW-MT	Reeder/MT9433	3.2 N	1.9 R	4.0 C	-	+
ND 747	HRSW-ND	ND2831//Parshall/ND706	3.9 N	1.9 R	3.9 C	+	+
ND 751	HRSW-ND	ND2709/3/GR*3//Ramsey/ND622/4/ND688/ND674	2.7 N	1.7 R	1.7 R	-	-
ND 752	HRSW-ND	ND2891/ND721	3.0 N	2.0 R	4.3 C	-	+
ND 753	HRSW-ND	ND721/Parshall	3.0 N	1.8 R	4.0 C	-	+
ND 755	HRSW-ND	ND2849/ND721	3.2 N	1.7 R	4.2 C	+	+
ND 756	HRSW-ND	ND2849/ND721	2.3 N	1.7 R	4.4 C	-	+
ND 800	HRSW-ND	ND741 SELN (Parshall/ND706)	3.3 N	2.1 C	4.3 C	+	+
ND 801	HRSW-ND	ND2849/ND721	2.3 N	1.5 R	4.0 C	-	+
ND 802	HRSW-ND	ND721/ND725	3.5 N	1.8 R	2.3 C	-	+
ND 803	HRSW-ND	ND2831/FO.2808	2.4 N	1.5 R	3.6 C	-	+
ND 804	HRSW-ND	ND744 SELN	3.1 N	1.7 R	2.1 C	+	+
ND 805	HRSW-ND	ND2857/3/KITT/Amidon//GR/Stoa SIB	1.7 R	2.1 C	3.7 C	-	+
ND 806	HRSW-ND	SD3348/Reeder/ND721	2.7 N	1.6 R	3.5 C	-	+
ND 807	HRSW-ND	ND2831//Parshall/ND706	3.1 N	1.5 R	1.9 R	+	-
ND 808	HRSW-ND	ND2857/3/Kitt/Amidon//GR/Stoa SIB	1.9 R	2.1 C	3.9 C	-	+
ND 809	HRSW-ND	ND2831//Parshall/ND706/3/ND721	3.4 N	1.8 R	3.9 C	-	+
ND 810	HRSW-ND	ND694/GR*2/RL4137//Amidon	2.6 N	1.6 R	3.9 C	-	+
ND 811	HRSW-ND	ND724/2*Reeder	1.9 R	1.4 R	3.4 C	-	+
NDSW0207	WW-ND	9734/AC Vista	3.2 N	2.2 C	4.1 C	-	+
NDSW0217	WW-ND	Sunstate/9734	3.5 N	3.0 C	2.6 C	+	+
NDSW0345	WW-ND	MT9904/Alsen	3.5 N	2.4 C	4.2 C	+	+
NDSW0347	WW-ND	Reeder//IDO560/9712	3.7 N	1.8 R	4.2 C	+	+
NDSW0431	WW-ND	BaiHuo/Kanto107//Express/3/MTHW9420	4.0 N	2.6 C	2.2 C	+	+
Erik	HRSW-KS	Kitt/2/Waldron/Era	1.3 R	1.3 R	1.5 R	-	-
Glenlea	OW-CAN	Pemina*2/Bage/2/CB100	4.3 N	2.0 R	1.9 R	+	-
6B-365	OW-CAN	Unknown	1.7 R	4.1 C	1.9 R	-	-
6B-662	OW-CAN	Unknown	1.7 R	1.7 R	4.0 C	-	+
ND 495	HRSW-ND	Justin*2/3/ND259/Conley//ND112	4.5 N	2.3 C	2.5 C	+	+
Conway	HRSW-CAN	Chris/Siete-Cerros-66//Neepawa/Opal	4.0 N	1.8 R	4.1 C	+	+
Katepwa	HRSW-CAN	Neepawa*6/RL-2938/3/Neepawa*6//CI-8154/2*Frocor	4.0 N	1.8 R	4.4 C	+	+
Kenyon	HRSW-CAN	Neepawa*5/Buck Manantial	4.3 N	2.4 C	3.8 C	+	+
Coulter	Durum-CAN	DT-188/Dt-224//DT-182	4.3 N	4.1 N	4.3 N	+	-
4B-160	OW-CAN	Unknown	1.7 R	2.9 N	2.5 N	-	-
<i>T. turgidum</i> #283	OW-CAN	PI352519	1.3 R	1.4 R	1.4 R	-	-
Intros #7	OW-MEX	Unknown	1.4 R	1.3 R	1.3 R	-	-
92 MREHTR28B	OW-CAN	Car853/Coc//Vee'S'/3/E7408/Pam'S'//Hor	1.6 R	1.3 R	1.3 R	-	-
2000 Spelt #20	OW-CAN	PI348771/Oberkum	1.4 R	1.2 R	1.5 R	-	-
CIMMYT L #18	OW-MEX	Unknown	1.5 R	1.3 R	1.4 R	-	-
Saloumini	OW-CAN	Unknown	1.4 R	1.4 R	1.3 R	-	-
Mountrail	Durum-ND	D8479/Renville	2.2 N	3.2 N	2.2 N	-	-
Ben	Durum-ND	D8024/Monroe	3.8 N	3.6 N	2.3 N	+	-
Lebsock	Durum-ND	Munich/D8469	2.7 N	3.0 N	1.9 R	+	-
Pierce	Durum-ND	D86117/D88289	3.6 N	3.2 N	2.0 R	+	-
Maier	Durum-ND	D8193/D8335	3.1 N	2.7 N	2.5 N	+	-
Plaza	Durum-ND	DT606/D8291	3.0 N	2.4 N	2.7 N	+	-
Dilse	Durum-ND	Maier/D88273	3.2 N	2.9 N	3.3 N	+	-
Grenora	Durum-ND	D901260/D901419	2.6 N	3.1 N	2.5 N	+	-
Divide	Durum-ND	Ben/D901282//Belzer	2.9 N	3.0 N	3.0 N	+	-
Alkabo	Durum-ND	D901247/D89263	2.1 N	2.5 N	3.0 N	+	-
LSD (0.05)			0.5	0.6	0.5		

sult, assessment of tan spot resistance on a lesion-type rating scale is preferred (5,23).

Screening wheat seedlings by artificial inoculation in the greenhouse permits examination of large populations for resistance under uniform disease pressure. A positive correlation has been reported between assessment of resistance at seedling stage and rating of adult plants in the field (12). The fact that tan spot in wheat fields occurs in association with other foliar diseases makes it more difficult and error prone to do field screening for tan spot resistance (20,23). Hence, when breeding for tan spot resistance, seedling evaluation of plants under controlled conditions is the preferred methodology.

To date, the fungus *P. tritici-repentis* has eight races. Races 2, 3, and 5 are all reported to have single virulence genes and are reported as the base races, i.e., combination of virulence of races 2, 3, and 5 have resulted in the development of other virulent races (26). Race 1 is the most prevalent race of *P. tritici-repentis* in the northern Great Plains of North America (2,22,25) and is reported to have the virulence of both races 2 and 3 (26). By screening the wheat germ plasm individually with races 2 and 3, we can predict the expected reaction of a genotype to race 1. Additionally, races 2 and 3 induce only one disease symptom, i.e., either necrosis or chlorosis in hexaploid and/or tetraploid wheat, thereby making the disease scoring easy, precise, and accurate. Results of this study can be used to predict the reaction of a genotype to other virulent races that are combinations of races 2, 3, and 5.

All susceptible hexaploid wheat genotypes tested with race 5 developed chlorosis, while durum wheat genotypes exhibited necrosis (Table 1). Upon infiltration of culture filtrate of race 5 to susceptible hexaploid wheat, genotypes showed chlorotic symptoms while susceptible durum exhibited insensitive reaction, indicating culture filtrate lacks the factor present in the spore suspension, which causes necrosis. These results indicate race 5 isolates may possibly have at least two disease inducing factors, one causing necrosis in durum and the other, toxin Ptr ToxB, inducing chlorosis in hexaploid wheat. Similar views have been suggested by Gamba and Lamari (18) and Singh and Hughes (32).

Our results indicate that the most genotypes are resistant to *P. tritici-repentis* race 3 followed by race 5 and race 2 (Table 1). In recent years, race 5 isolates have increased in prevalence among the *P. tritici-repentis* population, with a single isolate found in the Manitoba-Saskatchewan border (25) and a significantly higher proportion of this race detected in North Dakota (2). Most wheat breeding programs in the northern Great Plains use race 1 isolates and natural

inoculum as a means of screening for tan spot resistance (5,8). Although race 1 isolates induce both necrosis and chlorosis, similar to the combined symptoms induced by races 2 and 3, they do not have the virulence of race 5. Hence, we may not be able to eliminate susceptibility for race 5 in tan spot resistance breeding. It is recommended that future tan spot screening should include multiple race screening or a mixture of races 1 and 5 to develop tan spot resistant varieties.

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