Seedling Resistance to Tan Spot and *Stagonospora nodorum* Leaf Blotch in Wild Emmer Wheat (*Triticum dicoccoides*)

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ABSTRACT

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Tan spot and *Stagonospora nodorum* blotch (SNB), caused by *Pyrenophora tritici-repentis* and *Stagonospora nodorum*, respectively, are two destructive foliar diseases of wheat, causing significant yield reduction worldwide. The objective of this study was to evaluate 172 accessions of wild emmer wheat (*Triticum dicoccoides*) for seedling resistance to tan spot and SNB. All accessions were inoculated with *P. tritici-repentis* race 1 and a mixture of three diverse isolates of *S. nodorum*, respectively. The accessions were also evaluated for sensitivity to host-selective toxins (HSTs), including ToxA produced by both *S. nodorum* and *P. tritici-repentis* and culture filtrate produced by *S. nodorum*. A total of 34 accessions were resistant to tan spot, and 136 accessions were resistant to SNB. Among these accessions, 31 were resistant to both diseases. Significant correlations between HST insensitivity and disease resistance were observed. Our results showed that *T. dicoccoides* is a good genetic source of resistance to tan spot and SNB in wheat.

Additional keywords: *Phaeosphaeria nodorum*

Tan spot and *Stagonospora nodorum* blotch (SNB), caused by the fungi *Pyrenophora tritici-repentis* (Died.) Drechs. and *Phaeosphaeria nodorum* (E. Muller) Hedjaroude (anamorph: *Stagonospora nodorum* (Berk.) Castellani & E.G. Germano), respectively, are two destructive foliar diseases of common wheat (*Triticum aestivum* L.) and durum wheat (*T. turgidum* L. subsp. *durum*). Both diseases can cause yield losses as high as 50% (13,40). Due to the application of reduced tillage practices in cereal growing regions such as the United States, tan spot and SNB have become very common in recent years. Tan spot was identified as the most prevalent disease of wheat in Canada in 2003 (46),

and Perello et al. (38) indicated that tan spot had become more destructive in the southern Cone region of South America including Argentina, Brazil, Chile, Paraguay, and Uruguay.

Compared to the use of fungicides, breeding for disease resistance is more cost effective, socially feasible, and environmentally safe for growers. Unfortunately, due to the narrow genetic base, the majority of current bread and durum wheat cultivars are susceptible to both diseases (24,42,43,45). Rees and Platz (39) evaluated 1,400 bread wheat lines for resistance to *P. tritici-repentis* and found no complete resistance. Alam and Gustafson (1) screened 212 accessions of eight diploid and 10 polyploid species of *Aegilops* and found almost no resistance to tan spot. Some partial resistance to SNB had been detected in wild relatives of wheat, such as *T. timopheevii* (29), *T. monococcum* (28), *A. tauschii* (28), *A. speltoideus* (8), and *A. longissima* (9). Until recently, few germplasm or cultivars with good resistance were reported. Xu et al. (51) evaluated 120 elite synthetic hexaploid wheat (SHW) lines and 35 durum lines for seedling resistance to both tan spot and SNB and found that about 47 and 30% of synthetic lines showed good resistance to tan spot and SNB, respectively. Mergoum et al. (33) identified three cultivars and 12 advanced breeding lines resistant to SNB. Other germplasm resistant to tan spot and/or SNB have also been identified in other

reports (42,43,45). However, to control both diseases more effectively in the long term, the identification and utilization of novel resistance genes from wheat relatives would increase the genetic diversity and broaden the genetic base of resistance.

During the development of disease, *P. tritici-repentis* can produce several host-selective toxins (HST) that induce necrosis or chlorosis in sensitive wheat genotypes. To date, five toxins of *P. tritici-repentis*, designated Ptr ToxA (47), Ptr ToxB (31,37), Ptr ToxC (10), and two different toxins simultaneously named Ptr ToxD (30,32), have been reported. Ptr ToxA (5,47,48,49,52) and Ptr ToxB (31) have been well characterized. The dominant gene, *Tsn1*, which is located on wheat chromosome arm 5BL, controls sensitivity to Ptr ToxA (11), and genotypes without *Tsn1* are insensitive to the toxin (3). Host insensitivity to Ptr ToxA has been found to be associated with resistance to *P. tritici-repentis* races 1 and 2 (14,23).

S. nodorum also produces several HSTs such as SnToxA (18), SnTox1 (26,27), SnTox2 (16), and SnTox3 (19). Correlations between toxin insensitivity and resistance to SNB have been reported (16,18,26,27). Liu et al. (26,27) partially purified a toxin designated SnTox1 and identified the recessive host gene, *smn1*, which conferred insensitivity to the toxin. The *smn1* locus mapped to chromosome arm 1BS in the International Triticeae Mapping Initiative (ITMI) population, and explained as much as 58% of the phenotypic variation in SNB. Friesen et al. (18) reported that the gene encoding Ptr ToxA in *P. tritici-repentis* was transferred from *S. nodorum* just before 1941, and the sequence similarity between the *SnToxA* and *PtrToxA* genes was as high as 99.7%. A strong correlation between SnToxA insensitivity and SNB resistance was found, and the *tsn1* locus explained as much as 68% of the variation, indicating that a compatible *Tsn1*-SnToxA interaction plays a major role in disease development (18).

Wild emmer wheat, *T. dicoccoides* (AABB, $2n=4x=28$), distributed over the Near East Fertile Crescent, is a wild progenitor of cultivated durum and bread wheat (53). The A and B genomes of durum and bread wheat were derived from *T. dicoccoides*, and crosses between them are fully fertile, making genes from *T. dicoc-*

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coides readily transferable to cultivated wheat through homologous chromosome recombination (7,35,50). *T. dicoccoides* has been shown to be a good genetic source for resistance to powdery mildew, leaf rust, stem rust, and Fusarium head blight, as well as increased seed storage protein content (4,20,36,44). In this research, we evaluated potential sources of resistance to tan spot and SNB from *T. dicoccoides* at the seedling stage under controlled conditions.

MATERIALS AND METHODS

Plant materials. A total of 172 accessions of *T. dicoccoides* were evaluated. Among them, 166 were collected from different locations in Israel and Turkey (34) and introduced to the United States by Leonard Joppa (USDA-ARS, Fargo, ND) (21). These accessions are currently maintained at the Institute of Evolution, University of Haifa, Israel and at the USDA-ARS, Northern Crop Science Laboratory, Fargo, ND. The remaining six accessions (PI272582, PI343446, PI352328, PI355459, PI428109, and PI466995) were obtained from the USDA-ARS, National Small Grains Research Facility, National Small Grains Collection, Aberdeen, ID. In addition, the International Maize and Wheat Improvement Center (CIMMYT) SHW line W-7976 and the North Dakota hard red spring wheat cultivar Grandin were used as resistant and susceptible checks, respectively.

Evaluation of reaction to *P. tritici-repentis* and Ptr ToxA. Evaluation of reaction to *P. tritici-repentis* was conducted under controlled greenhouse and growth chamber conditions using experimental procedures described by Friesen et al. (14). Seeds of all accessions were planted in super-cell Cone-tainers (Stuewe and Sons, Inc., Corvallis, OR) with sunshine SB100 (Sun Gro Horticulture, Bellevue, WA) and fertilized with Osmocote Plus 15-19-12 (Scotts Sierra Horticultural Product Company, Marysville, OH). Three seeds were planted in each cone, and each accession was planted in three cones in each of three experiments. Therefore, a total of 27 plants from each accession were tested. Cones were placed in RL98 trays (Stuewe and Sons). To eliminate any edge effect, the susceptible cultivar Grandin was planted in all the cones around the borders on each RL98 tray except for three cones for planting the resistant check W-7976.

At the two- to three-leaf stage, the secondary leaves from three plants of each accession were infiltrated with purified Ptr ToxA (provided by S. W. Meinhardt, Department of Plant Pathology, North Dakota State University, Fargo). Ptr ToxA was originally purified from *P. tritici-repentis* race 2 (isolate 86-124) as described by Zhang et al. (52). Toxin infiltration was done according to Xu et al. (51). Approximately 20 µl of toxin (10 µg ml⁻¹) was

infiltrated into the secondary leaf using a 1-ml syringe with the needle removed. The boundaries of the infiltration sites were marked with a nontoxic felt pen. Leaves were evaluated 4 days after infiltration and scored as insensitive (–) or sensitive (+) based on the absence or presence of necrosis, respectively.

After sensitivity to Ptr ToxA was tested, all plants, including those that had been infiltrated, were inoculated with conidia of the *P. tritici-repentis* isolate Pt12, a race 1 isolate obtained from a wheat field in South Dakota (17). A race 1 isolate was chosen due to its prevalence in North America (2), and because it contains the known virulence factors found in race 2 (25), the second most prevalent race found in the field (2). Preparation of inoculum and inoculations were done as described in Xu et al. (51). Briefly, the fungus was grown on V8-potato dextrose agar (PDA) plates (150 ml V8 juice, 10 g PDA, 3 g CaCO₃, 10 g agar, and 850 ml distilled water) in the dark for 4 to 5 days at 20°C. After flooding the plates with sterile distilled water, the plates were incubated under fluorescent light for 24 h at 20 to 22°C followed by 18 to 24 h in the dark at 16°C. The conidial inoculum suspension was obtained by washing the plates with distilled water and scraping the spores from the plates with a sterile inoculating loop. The final inoculum concentration was adjusted to 3,000 spores ml⁻¹. Plants were inoculated until runoff with a hand-held sprayer. Disease reaction types were rated 7 days postinoculation using the 1 to 5 lesion-type rating scale developed by Lamari and Bernier (22), with 1 being resistant, 2 moderately resistant, 3 moderately resistant/moderately susceptible, 4 susceptible, and 5 highly susceptible. Lines showing equal numbers of two lesion types were given an intermediate reaction type (e.g., reaction types 1 and 2 equal 1.5).

Evaluation of reactions to *S. nodorum* and *S. nodorum* HSTs. The experimental design used for evaluating reaction to *S. nodorum* inoculations was the same as that described for *P. tritici-repentis*. Inoculum preparation was done according to Xu et al. (51) except that three *S. nodorum* isolates, LDNSn4, BBCSn5, and Sn2000, were used to produce pycnidiospores. The fungus was grown on V8-PDA medium for 7 days, and inoculum suspension was obtained by adding sterile distilled water and scraping the spores off the plate surface with an inoculating loop and adjusting the inoculum concentration to 1×10^6 spores ml⁻¹. The inoculum suspensions of the three isolates were then mixed equally before inoculation. Isolate Sn2000, collected from a North Dakota wheat field in 1980, was chosen because it has been used to screen North Dakota germplasm and breeding lines and has been shown to be an aggressive toxin-producing isolate

(18,26,27). Isolates LDNSn4 and BBCSn5 were collected in North Dakota and Minnesota, respectively, and were used because they produce other toxins in addition to those produced by Sn2000 (T. L. Friesen, *unpublished data*). Therefore, these mixtures provide a diversity of virulence factors present in *S. nodorum*. The rating scale used for *S. nodorum* is a 0 to 5 numerical scale based on lesion type as described in Liu et al. (27), where 0 = highly resistant (absence of visible lesions); 1 = resistant (few penetration points, with lesions consisting of flecking or small dark spots); 2 = moderately resistant (lesions consisting of dark spots with little surrounding necrosis or chlorosis); 3 = moderately susceptible (dark lesions completely surrounded by necrosis or chlorosis, lesions 2 to 3 mm); 4 = susceptible (larger necrotic or chlorotic lesions, 4 mm or greater, with little coalescence); and 5 = highly susceptible (large coalescent lesions with very little green tissue remaining). Plants having equal numbers of two different lesion types were given an intermediate lesion type (e.g., lesion types 1 and 2 equal 1.5).

Because SnToxA is functionally identical to Ptr ToxA (18), the results from Ptr ToxA infiltration were used to infer reactions to SnToxA. Isolate Sn2000 has been shown to produce SnToxA (18), SnTox1 (26,27), and several additional toxins in culture (T. L. Friesen, *unpublished data*); however, only SnToxA has been purified. Therefore, culture filtrate of Sn2000KO6-1, a genetically modified strain of the isolate Sn2000 with a disrupted *ToxA* gene (18), was used for investigation of the reaction to *S. nodorum* toxins in the absence of SnToxA. Culture filtrates were produced as described by Liu et al. (26). Briefly, 200 µl of Sn2000KO6-1 spore suspension was added to 60 ml of liquid Fries medium (5 g of ammonium tartrate, 1.0 g of ammonium nitrate, 0.5 g of magnesium sulfate, 1.3 g of potassium phosphate [dibasic], 2.6 g of potassium phosphate [monobasic], 30.0 g of sucrose, 1.0 g of yeast extract, dissolved in 1,000 ml of water) and placed on an orbital shaker at 80 rpm for 48 h followed by 3 weeks of stationary growth. Both growth steps took place in the dark at room temperature. Culture filtrate was filtered through two layers of cheesecloth followed by vacuum filtration through a Whatman No. 1 filter and a 0.45-µm Whatman filter.

Statistical analysis. Statistical analysis was done using the Statistical Analysis System version 9.1 (41). Bartlett's χ^2 was calculated to test the homogeneity of variance among experiments. A two-sample *t* test was used to test the difference of average reactions to *P. tritici-repentis* and *S. nodorum* according to the reactions to HSTs. The least significant difference (LSD) was used to test for significant differences among the accessions. Regression

analysis was performed to evaluate the association of sensitivity to HSTs with average reaction to *P. tritici-repentis* and *S. nodorum*. For regression analysis, a value of 1 was given to accessions that were sensitive to HSTs, and a value of 0 for accessions that were insensitive.

RESULTS

Reaction to *P. tritici-repentis* and Ptr ToxA. Average tan spot reaction types in all *T. dicoccoides* accessions ranged from 1.2 to 4.7 with susceptible (Grandin) and resistant (W-7976) controls having average disease reaction types of 4.5 and 1.3, respectively (Fig. 1). Bartlett's χ^2 test showed homogeneity among three experiments ($\chi^2_{df=2} = 0.26$, $P = 0.88$); therefore, results from the three experiments were combined (Tables 1 and 2). Almost half of the *T. dicoccoides* accessions were susceptible, with average disease reactions ranging from 3.0 to 4.0. Among the susceptible lines, 12 were highly susceptible with average disease reactions greater than 4.0. Therefore, the majority of the accessions of *T. dicoccoides* tested were susceptible to tan spot. However, 34 accessions had an average disease reaction of less than 2, and of these, 21 accessions had an average disease reaction lower than 1.5, demonstrating that *T. dicoccoides* is a potential source of tan spot resistance.

Of the 172 accessions tested, 149 were insensitive to Ptr ToxA and 23 were sensitive (Tables 1 and 2, Figs. 1 and 2), indicating that only 23 of the lines carried the dominant *Tsn1* allele, which confers sensitivity to Ptr ToxA (11). All 34 resistant accessions were insensitive to Ptr ToxA (Tables 1 and 2, Figs. 1 and 2). Ptr ToxA insensitive accessions showed an average tan spot reaction type of 2.89, whereas accessions sensitive to Ptr ToxA had an average reaction of 3.41. A two-sample *t* test indicated that the difference between these two average disease reactions (0.52) was highly significant ($P < 0.0001$) (Table 3), demonstrating that the absence of *Tsn1* can significantly increase resistance to tan spot. Simple linear regression analysis between insensitivity to Ptr ToxA and resistance to tan spot showed a 3% association ($R^2 = 0.03$, $P < 0.0001$), indicating a weak but significant association between Ptr ToxA insensitivity and tan spot resistance. Therefore, genes other than *Tsn1/tsn1* are most likely also involved in governing susceptibility/resistance to tan spot in *T. dicoccoides*.

Reaction to *S. nodorum*, SnToxA, and *S. nodorum* culture filtrates. Bartlett's χ^2 test for homogeneity among experiments of the *S. nodorum* inoculations showed that three experiments were homogeneous ($\chi^2_{df=2} = 0.98$, $P = 0.61$). Therefore, the disease reaction types from the three experiments were combined. Reactions to *S. nodorum* among the *T. dicoccoides* accessions were quite different from those of *P.*

tritici-repentis (Tables 1 and 2). Of the 172 accessions, 136 had average disease reaction types less than 2.0, and 80 of these were less than 0.5, indicating that most of the *T. dicoccoides* accessions were resistant to SNB. In addition, 31 of the 136 SNB-resistant accessions had tan spot reaction types less than 2.0 (Table 2), indicating that these accessions are promising sources for resistance to both tan spot and SNB.

Reactions to toxins produced by *S. nodorum* were strongly associated with SNB disease reactions. The average SNB reaction types (0 to 5 scale), separated based on reaction to SnToxA, were 0.84 for insensitive (SnToxA⁻) accessions and

3.17 for sensitive (SnToxA⁺) accessions, and the difference (2.33) between these two classes is highly significant ($P < 0.0001$, Table 3). Only two accessions with disease reaction types less than 2 were SnToxA sensitive (Tables 1 and 2, Fig. 3).

For Sn2000KO6-1 culture filtrate infiltrations, 64 of the 172 accessions were insensitive (Tables 1 and 2). The difference of the average SNB reaction types between accessions that were culture filtrate sensitive (CF⁺) versus insensitive (CF⁻) was 0.71 ($P < 0.0001$) (Table 3). Sixty of the 64 insensitive accessions had average SNB reaction types less than 2, and all 24 accessions with average SNB reaction types greater than 2.5 were sensitive (Tables 1

Fig. 1. Reaction of *Pyrenophora tritici-repentis* inoculation (A) and Ptr ToxA infiltration (B). Resistant and susceptible checks: (a) Grandin (susceptible and Ptr ToxA sensitive) and (b) W7976 (resistant and Ptr ToxA insensitive); *Triticum dicoccoides* accessions: (c) B-35 (resistant and Ptr ToxA insensitive) and (d) 18-1 (susceptible and Ptr ToxA sensitive).

Table 1. Reaction to inoculation of conidia and infiltration of host selective toxins produced by *Pyrenophora tritici-repentis* (Ptr) and *Stagonospora nodorum* (Sn) in 172 *Triticum dicoccoides* accessions

Disease rating	Number of accessions				
	Reaction to Ptr ^a		Reaction to Sn ^b		
	Inoculation	Ptr ToxA ⁻	Inoculation	SnToxA ⁻	CF ⁻
0 – 0.5			80	79	35
0.5 – 1			28	28	14
1 – 1.5	21	21	17	17	9
1.5 – 2	13	13	11	10	2
2 – 2.5	10	10	12	9	4
2.5 – 3	33	32	6	1	0
3 – 3.5	54	38	8	1	0
3.5 – 4	29	24	5	4	0
4 – 4.5	11	10	3	0	0
4.5 – 5	1	1	2	0	0

^a Ptr ToxA⁻ means Ptr ToxA insensitive.

^b SnToxA⁻ and CF⁻ means SnToxA and CF insensitive, respectively; CF = culture filtrate produced by Sn2000KO6-1.

and 2, Fig. 3), indicating that toxins produced in the Sn2000KO6-1 culture filtrate were important for disease development. To compare the association between SnToxA- and culture filtrate-insensitivity to that of *S. nodorum* resistance separately, the average SNB scores among the four classes involving reaction to culture filtrate (CF) and SnToxA (SnToxA⁻CF⁻ versus SnToxA⁻CF⁺, SnToxA⁺CF⁻ versus SnToxA⁺CF⁺, SnToxA⁻CF⁻ versus SnToxA⁺CF⁻, and SnToxA⁻CF⁺ versus SnToxA⁺CF⁺) were analyzed using a two-sample *t* test. All comparisons were significantly different at the $P < 0.0001$ level of probability (Table 3). Therefore, both SnToxA and other toxins present in the Sn2000KO6-1 culture filtrate play a significant role in the development of SNB among the *T. dicoccoides* accessions.

Results from regression analysis confirmed the association between reactions to HSTs and *S. nodorum*. Associations between SnToxA and SNB ($R^2 = 0.40$, $P < 0.0001$) and between SNB and culture filtrate ($R^2 = 0.07$, $P < 0.0001$) were 40 and 7%, respectively, suggesting that resistance to SNB in *T. dicoccoides* is largely governed by *tsn1* on chromosome 5B, as well as additional loci at other genomic regions that govern insensitivity to toxins present in the Sn2000KO6-1 culture filtrate. Multiple regression analysis of SNB severity and reaction to SnToxA and culture filtrate showed the association was increased to 43% ($R^2 = 0.43$, $P < 0.0001$). The average disease reactions of accessions insensitive to both SnToxA and culture filtrate is 0.63, which is significantly different from that of accessions sensitive to at least one toxin ($P < 0.0001$, Table 3). Therefore, the resistance conferred by toxin insensitivity loci such as *tsn1* on chromosome 5B and that of others appears to be additive.

DISCUSSION

Here, we identified a number of *T. dicoccoides* accessions with good levels of resistance to tan spot and SNB. These accessions can be used for the development of disease-resistant germplasm. The 31 accessions that showed good resistance to both diseases will be especially useful.

Correlation between insensitivity to Ptr ToxA and resistance to tan spot in hexaploid wheat has been identified. Friesen et al. (14) found that the Ptr ToxA reaction accounted for 24.4% of the variation in tan spot using a Ptr ToxA producing race 2 isolate. Xu et al. (51) found a 10% association between insensitivity to Ptr ToxA and resistance to tan spot among 120 SHW lines. Our research indicated a weak but significant association between Ptr ToxA insensitivity and disease resistance ($R^2 = 0.03$, $P < 0.0001$), but all accessions with high levels of resistance were Ptr ToxA insensitive (Table 2). This demonstrates that insensitivity to Ptr ToxA can increase

Table 2. Seedling reactions to *Pyrenophora tritici-repentis* (Ptr) and *Stagonospora nodorum* (Sn) in 172 *Triticum dicoccoides* accessions

Accession ^a	Tan spot mean	Ptr/Sn ToxA ^b	SNB ^c mean	Culture filtrate ^{b,d}
Checks				
W7976	1.30 ± 0.29	–	1.00 ± 0.00	+
Grandin	4.50 ± 0.00	+	4.30 ± 0.29	+
Resistant to both tan spot and SNB				
20-35	1.20 ± 0.29	–	0.30 ± 0.58	+
16-1	1.20 ± 0.29	–	0.70 ± 0.29	–
I-15	1.20 ± 0.29	–	0.80 ± 0.29	–
I-29	1.20 ± 0.29	–	1.20 ± 0.29	–
B-35	1.20 ± 0.29	–	1.30 ± 0.76	–
A-82	1.30 ± 0.29	–	0.20 ± 0.29	–
I-20	1.30 ± 0.29	–	1.20 ± 0.58	+
I-18	1.30 ± 0.29	–	1.30 ± 0.29	–
I-50	1.30 ± 0.29	–	1.30 ± 0.29	+
32-45	1.30 ± 0.58	–	1.20 ± 2.02	–
B-34	1.30 ± 0.58	–	1.30 ± 0.76	–
B-6	1.30 ± 0.58	–	1.70 ± 1.04	–
16-29	1.50 ± 0.00	–	0.80 ± 0.29	+
L-1	1.50 ± 0.00	–	1.50 ± 0.87	–
A-33	1.50 ± 0.50	–	0.20 ± 0.29	+
B-8	1.50 ± 0.50	–	0.20 ± 0.29	–
A-61	1.50 ± 0.50	–	0.70 ± 0.29	–
I-17	1.50 ± 0.50	–	0.70 ± 0.29	–
I-45	1.50 ± 0.50	–	1.80 ± 0.29	+
K-50	1.70 ± 0.29	–	0.20 ± 0.29	+
A-52	1.70 ± 0.29	–	0.30 ± 0.29	–
B-31	1.70 ± 0.29	–	0.50 ± 0.50	–
L-29	1.70 ± 0.29	–	0.80 ± 0.29	+
L-33	1.70 ± 0.29	–	1.00 ± 0.00	+
18-48	1.80 ± 0.29	–	0.30 ± 0.29	+
L-43	1.80 ± 0.29	–	1.20 ± 0.58	+
A-56	2.00 ± 0.00	–	0.80 ± 0.29	–
F-25	2.00 ± 0.50	–	0.20 ± 0.29	+
18-24	2.00 ± 0.50	–	0.70 ± 0.29	–
L-34	2.00 ± 0.50	–	1.00 ± 0.00	+
L-40	2.00 ± 0.50	–	1.00 ± 0.50	+
Resistant to tan spot only				
B-32	1.30 ± 0.29	–	2.30 ± 0.29	–
I-4	1.50 ± 0.50	–	2.30 ± 0.29	+
32-29	1.70 ± 0.29	–	2.20 ± 0.29	–
Resistant to SNB only				
C-27	2.30 ± 0.29	–	0.00 ± 0.00	–
17-22	2.70 ± 0.29	–	0.00 ± 0.00	+
15-17	2.80 ± 0.29	–	0.00 ± 0.00	–
18-49	2.80 ± 0.29	–	0.00 ± 0.00	+
15-32	3.30 ± 0.29	–	0.00 ± 0.00	+
F-28	3.80 ± 0.29	–	0.00 ± 0.00	+
F-18	4.20 ± 0.29	–	0.00 ± 0.00	+
15-19	2.50 ± 0.00	–	0.20 ± 0.29	–
15-2	2.50 ± 0.00	–	0.20 ± 0.29	+
15-37	2.50 ± 0.00	–	0.20 ± 0.29	+
19-25	2.50 ± 0.00	–	0.20 ± 0.29	+
19-29	2.70 ± 0.29	–	0.20 ± 0.29	–
14-2	2.80 ± 0.29	–	0.20 ± 0.29	+
15-43	2.80 ± 0.29	–	0.20 ± 0.29	–
19-45	2.80 ± 0.29	–	0.20 ± 0.29	–
19-50	2.80 ± 0.29	–	0.20 ± 0.29	–
14-45	3.00 ± 0.00	–	0.20 ± 0.29	+
36-33	3.00 ± 0.00	–	0.20 ± 0.29	–
14-13	3.00 ± 0.50	–	0.20 ± 0.29	+
15-1	3.00 ± 0.50	–	0.20 ± 0.29	–
19-20	3.00 ± 0.50	–	0.20 ± 0.29	–
19-36	3.00 ± 0.50	–	0.20 ± 0.29	–
19-51	3.00 ± 0.50	–	0.20 ± 0.29	–
19-7	3.00 ± 0.50	–	0.20 ± 0.29	+

(continued on next page)

^a Accessions: W-7976 and Grandin are the resistant and susceptible checks, respectively; PI stands for plant identification, and the PI accessions were from the USDA National Small Grains Collection in Aberdeen, ID, USA; all others are accession numbers of the Institute of Evolution, University of Haifa, Israel, and were introduced to the United States by Leonard Joppa (USDA-ARS, Fargo, ND).

^b Reaction to Ptr/Sn ToxA and culture filtrate: insensitive (–), sensitive (+).

^c *Stagonospora nodorum* blotch.

^d Culture filtrate was produced by Sn2000KO6-1, a strain derived from Sn2000 by mutating the *ToxA* gene.

Table 2. (continued from previous page)

Accession ^a	Tan spot mean	Ptr/Sn ToxA ^b	SNB ^c mean	Culture filtrate ^{b,d}
14-47	3.20 ± 0.29	—	0.20 ± 0.29	+
15-18	3.20 ± 0.29	—	0.20 ± 0.29	—
32-46	3.20 ± 0.29	—	0.20 ± 0.29	+
F-3	3.20 ± 0.29	—	0.20 ± 0.29	+
K-45	3.20 ± 0.29	—	0.20 ± 0.29	+
T-7-40	3.20 ± 0.29	—	0.20 ± 0.29	—
17-2	3.20 ± 0.76	—	0.20 ± 0.29	+
19-23	3.20 ± 0.76	—	0.20 ± 0.29	+
15-52	3.30 ± 0.29	—	0.20 ± 0.29	—
18-15	3.30 ± 0.29	—	0.20 ± 0.29	—
15-26	3.30 ± 0.58	—	0.20 ± 0.29	+
19-6	3.30 ± 0.58	—	0.20 ± 0.29	+
C-55	3.50 ± 0.00	—	0.20 ± 0.29	—
18-20	3.50 ± 0.50	+	0.20 ± 0.29	—
14-36	3.70 ± 0.29	—	0.20 ± 0.29	+
F-38	3.70 ± 0.58	—	0.20 ± 0.29	+
F-65	3.80 ± 0.29	—	0.20 ± 0.29	+
C-21	4.20 ± 0.58	—	0.20 ± 0.29	+
F-43	4.30 ± 0.29	—	0.20 ± 0.29	+
C-30	2.80 ± 0.29	—	0.30 ± 0.29	—
PI272582	2.80 ± 0.29	—	0.30 ± 0.29	—
19-24	2.80 ± 0.58	—	0.30 ± 0.29	+
PI343446	3.00 ± 0.50	—	0.30 ± 0.29	—
B-13	3.20 ± 0.58	—	0.30 ± 0.29	—
F-7	3.30 ± 0.29	—	0.30 ± 0.29	+
14-34	3.50 ± 0.50	—	0.30 ± 0.29	+
14-35	3.70 ± 1.04	—	0.30 ± 0.29	—
F-37	3.80 ± 0.29	—	0.30 ± 0.29	+
F-81	3.80 ± 0.29	—	0.30 ± 0.29	+
14-25	4.00 ± 0.50	—	0.30 ± 0.29	—
14-40	4.00 ± 0.50	—	0.30 ± 0.29	—
K-36	4.20 ± 0.29	—	0.30 ± 0.29	—
C-19	4.50 ± 0.50	—	0.30 ± 0.29	+
32-44	2.30 ± 0.29	—	0.30 ± 0.58	+
17-1	2.50 ± 0.00	—	0.30 ± 0.58	+
A-35	2.50 ± 0.00	—	0.30 ± 0.58	+
18-37	2.70 ± 0.29	—	0.50 ± 0.00	+
L-10	3.00 ± 0.50	—	0.50 ± 0.00	+
15-55	3.20 ± 0.29	—	0.50 ± 0.00	—
A-57	3.20 ± 0.29	—	0.50 ± 0.00	+
K-32	3.20 ± 0.29	—	0.50 ± 0.00	—
PI352328	3.20 ± 0.58	—	0.50 ± 0.00	—
I-39	3.50 ± 0.50	—	0.50 ± 0.00	—
14-49	3.70 ± 0.29	—	0.50 ± 0.00	+
B-16	3.70 ± 1.04	—	0.50 ± 0.00	—
PI466995	4.00 ± 0.50	—	0.50 ± 0.00	+
C-7	3.80 ± 1.04	—	0.50 ± 0.50	+
B-40	2.50 ± 0.50	—	0.70 ± 0.29	+
PI355459	2.80 ± 0.29	—	0.70 ± 0.29	+
L-25	3.50 ± 0.00	—	0.70 ± 0.29	—
G-47	3.80 ± 1.04	—	0.70 ± 0.29	—
C-36	4.20 ± 1.04	—	0.70 ± 0.29	+
B-19	2.70 ± 0.58	—	0.70 ± 0.76	—
32-14	3.20 ± 0.29	—	0.70 ± 1.15	—
G-56	2.20 ± 0.58	—	0.80 ± 0.58	+
G-59	3.50 ± 0.00	—	0.80 ± 0.58	+
F-54	3.80 ± 0.29	—	0.80 ± 0.58	+
C-13	4.20 ± 0.76	—	0.80 ± 0.58	+
B-37	2.70 ± 0.76	—	0.80 ± 0.76	—
A-51	3.20 ± 0.29	—	1.00 ± 0.00	—
L-28	2.70 ± 0.29	—	1.00 ± 0.50	+
C-52	3.80 ± 0.58	—	1.00 ± 0.50	+
K-19	4.00 ± 0.87	—	1.00 ± 0.50	—
18-16	4.20 ± 0.29	—	1.00 ± 1.73	—
K-46	3.20 ± 0.29	—	1.20 ± 0.58	—
20-11	3.50 ± 0.50	—	1.20 ± 0.58	+
G-58	3.80 ± 0.76	—	1.20 ± 0.76	+
PI428109	3.00 ± 0.00	—	1.30 ± 0.76	+
G-52	4.70 ± 0.29	—	1.30 ± 0.76	—
20-42	3.30 ± 0.76	—	1.30 ± 1.15	+
L-61	3.20 ± 0.29	—	1.50 ± 1.00	—
G-50	4.30 ± 0.29	—	1.50 ± 1.50	+
32-25	3.80 ± 0.35	—	1.70 ± 0.29	+

(continued on next page)

resistance to tan spot in *T. dicoccoides*. Faris and Friesen (12) identified race non-specific resistance coming from chromosomes 1B and 3B rather than *tsn1* in a wheat population segregating for Ptr ToxA sensitivity (*Tsn1*), indicating that Ptr ToxA was not a detectable factor during tan spot development in their population. Among the *T. dicoccoides* accessions investigated in this research, it is possible that resistance in a few accessions is largely due to the presence of the recessive allele at the *Tsn1* locus, whereas in most other accessions resistance may be due to genes at other loci. Therefore, genes other than *tsn1* could play more important roles in governing tan spot resistance in *T. dicoccoides*.

Toxin insensitivity related to SNB disease resistance has also been identified in hexaploid wheat. Liu et al. (26,27) reported a strong correlation between insensitivity to SnTox1 and resistance to SNB. The *Snn1* locus, which governs SnTox1 sensitivity, coincided with a major QTL that explained up to 58% of the phenotypic variation. Friesen et al. (18) reported a strong correlation (34 to 68%) between SnToxA insensitivity and SNB resistance. Our research also revealed a significant association between insensitivity to HSTs and SNB resistance in *T. dicoccoides* with 40% of the SNB resistance being associated with SnToxA insensitivity and 7% of the SNB resistance being associated with culture filtrate insensitivity. Our culture filtrate was derived from Sn2000KO6-1, which has a disrupted form of the *ToxA* gene (18). Other than SnTox1, Sn2000KO6-1 likely produces additional toxins (T. L. Friesen, unpublished data), and the association between SNB resistance and culture filtrate insensitivity could be due to SnTox1 or other unidentified toxins also produced by Sn2000KO6-1.

SNB resistance in *T. dicoccoides* was primarily governed by the *tsn1* locus, but other loci governing insensitivity to toxins in the culture filtrate were involved as well. A stronger association between SNB resistance and SnToxA insensitivity indicates that *tsn1* had greater impact than other loci governing insensitivity to toxins produced in Sn2000KO6-1 culture filtrates. Multiple regression analysis and comparisons of average SNB reactions among toxin-sensitive and insensitive classes indicated that resistance conferred by the *tsn1* locus and other toxin insensitivity loci are additive.

Genomic regions related to disease resistance but not associated with known toxin insensitivity loci have been reported. As previously mentioned, Faris and Friesen (12) identified tan spot race non-specific resistance QTL on chromosomes 1B and 3B, but the *tsn1* locus on 5B was not associated with disease. In addition to *Tsc2* on 2BS, which governs sensitivity to Ptr ToxB, Friesen and Faris (15) found QTL on chromosomes 2A and 4A associ-

Table 2. (continued from previous page)

Accession ^a	Tan spot mean	Ptr/Sn ToxA ^b	SNB ^c mean	Culture filtrate ^{b,d}
20-37	3.70 ± 0.29	—	1.70 ± 0.76	+
20-8	3.50 ± 0.50	—	1.70 ± 1.04	+
I-40	2.70 ± 0.29	—	2.00 ± 0.00	+
L-56	2.70 ± 0.29	—	2.00 ± 0.00	+
20-38	3.80 ± 0.58	—	2.00 ± 0.00	+
I-44	3.80 ± 1.04	—	2.00 ± 0.00	+
20-20	4.20 ± 0.76	—	2.00 ± 0.50	+
Susceptible to both tan spot and SNB				
G-11	3.30 ± 0.29	+	2.00 ± 1.32	—
T-7-18	3.30 ± 0.29	—	2.20 ± 0.29	+
20-22	3.00 ± 0.50	—	2.20 ± 0.58	+
20-9	3.20 ± 0.29	—	2.20 ± 1.04	+
G-15	3.30 ± 0.29	+	2.30 ± 0.29	—
20-34	3.70 ± 0.76	+	2.30 ± 0.29	+
G-42	4.20 ± 0.29	+	2.30 ± 0.29	—
20-31	3.50 ± 0.50	—	2.30 ± 0.76	+
G-61	3.80 ± 1.04	—	2.30 ± 1.61	+
32-30	3.30 ± 0.35	—	2.50 ± 0.00	+
36-25	2.70 ± 0.76	+	2.70 ± 0.58	+
G-22	3.50 ± 0.50	+	2.80 ± 0.29	+
18-56	3.80 ± 0.29	+	2.80 ± 0.29	+
36-26	3.20 ± 0.58	+	3.00 ± 0.00	+
36-40	3.20 ± 0.29	+	3.00 ± 0.87	+
T-7-21	3.00 ± 0.00	—	3.00 ± 1.32	+
36-38	3.20 ± 0.58	+	3.20 ± 0.29	+
36-22	3.30 ± 0.29	+	3.20 ± 0.58	+
36-39	3.50 ± 0.50	+	3.20 ± 1.04	+
36-12	3.20 ± 0.29	+	3.30 ± 0.29	+
36-17	3.20 ± 0.76	+	3.30 ± 0.58	+
T-7-5	3.20 ± 0.29	—	3.30 ± 1.04	+
T-7-1	3.80 ± 0.29	+	3.30 ± 1.61	+
36-37	3.20 ± 0.29	+	3.50 ± 0.00	+
T-7-8	3.00 ± 0.00	—	3.70 ± 0.76	+
T-7-7	3.70 ± 0.29	—	3.70 ± 0.76	+
T-7-45	3.30 ± 0.29	+	3.80 ± 0.58	+
T-7-15	3.20 ± 0.29	—	3.80 ± 1.15	+
T-7-35	3.30 ± 0.29	—	4.00 ± 0.50	+
36-21	3.20 ± 0.29	+	4.20 ± 0.29	+
18-10	3.20 ± 0.58	+	4.20 ± 0.29	+
18-1	3.80 ± 0.58	+	4.20 ± 0.29	+
T-7-26	3.50 ± 0.00	+	5.00 ± 0.00	+
T-7-30	3.70 ± 0.29	+	5.00 ± 0.00	+
LSD _{0.05}	0.71		0.87

Fig. 2. Histogram of average tan spot disease reaction type frequency comparison of Ptr ToxA sensitive (+) and insensitive (–) accessions in *Triticum dicoccoides* at the seedling stage.

ated with resistance to tan spot in the ITMI population when inoculating with Ptr race 5. Liu et al. (27) found several genomic regions other than the *snm1* locus that were associated with SNB resistance. Friesen et al. (16) also identified SNB resistance from chromosome 5A in addition to toxin insensitivity loci. Our results also showed

that the resistance/susceptibility to tan spot or SNB in *T. dicoccoides* was not limited to toxin insensitivity/sensitivity loci. Of the 16 accessions with tan spot reaction types greater than 4.0, only two accessions were sensitive to Ptr ToxA (Table 2). For SNB, some accessions with average disease reaction types around 2.5 (e.g., 18-56

[2.8], 20-34 [2.3], 36-25 [2.7], and G-22 [2.8]) were sensitive to both SnToxA and the Sn2000KO6-1 culture filtrate (Table 2). Therefore, the resistance to tan spot or SNB in some *T. dicoccoides* accessions could be governed by genes other than the known toxin insensitivity genes. It is possible that the additional resistance loci are actually those conferring insensitivity to undetectable toxins; however, it is just as likely that there are resistance QTL conferring moderate levels of resistance that have no association with toxin insensitivity. Therefore, the *T. dicoccoides* accessions that have resistance/susceptibility unrelated to known toxin insensitivities/sensitivities would be especially useful for either identifying new toxins or characterizing different types of resistance in this host–pathogen system.

T. dicoccoides has been shown to be a good genetic source for resistance to powdery mildew, leaf rust, stem rust, and Fusarium head blight, as well as increased seed storage protein content (4,20,36,44). However, *T. dicoccoides* does have some drawbacks due to its exotic nature including shattering, later-maturing, and nonfree threshing traits (6). According to the results presented here, elimination of toxin sensitivities will increase the level of both SNB and tan spot resistance but may have a greater effect on resistance to SNB because it is more strongly associated with insensitivity to HSTs produced by its corresponding fungus than is tan spot. Furthermore, elimination of ToxA sensitivity (*Tsn1*) could increase resistance to both diseases. Other than insensitivity to toxins, *T. dicoccoides* likely harbors resistance to both SNB and tan spot that will increase the base of resistance in tetraploid wheat. Evidence of this is found in lines that are toxin sensitive but still maintain a high or moderate level of resistance. These include Ptr ToxA-sensitive accession 36-25 having an average tan spot reaction type of 2.7, SnToxA-sensitive accessions 18-20 and G-11 having average SNB reaction types of 0.2 and 2.0, respectively, and several accessions with SNB reaction types of around 2.5 (18-56, 20-34, 36-25, and G-22) being sensitive to both SnToxA and *S. nodorum* culture filtrate.

In summary, from 172 *T. dicoccoides* accessions, we identified a number of accessions with good resistance to tan spot and SNB, and 31 accessions showed good resistance to both diseases. Based on the association analysis between HST insensitivity and resistance to the pathogen, the resistance in *T. dicoccoides* involves toxin insensitivity loci as well as other uncharacterized genes, and it is therefore a potentially new source of resistance for cultivated wheat. Data in this article present useful information for the selection of parental lines for either producing resistant germplasm or for developing mapping populations for the characterization of

Table 3. Two sample *t* test to compare the difference of average reactions to *Pyrenophora tritici-repentis* (Ptr) and *Stagonospora nodorum* (Sn) according to the reactions to host selective toxins (HSTs) in 172 *Triticum dicoccoides* accessions

Comparison ^a	Mean1 ^b	Mean2 ^b	Difference ^c	<i>t</i> value	<i>P</i> ^d
Reaction to <i>P. tritici-repentis</i>					
Ptr ToxA ⁻ vs. Ptr ToxA ⁺	2.89 (149)	3.41 (23)	0.52	6.87	<0.0001
Reaction to <i>S. nodorum</i>					
SnToxA ⁻ vs. SnToxA ⁺	0.84 (149)	3.17 (23)	2.33	18.28	<0.0001
CF ⁻ vs. CF ⁺	0.70 (64)	1.41 (108)	0.71	7.37	<0.0001
SnToxA ⁻ CF ⁻ vs. SnToxA ⁻ CF ⁺	0.63 (60)	0.98 (89)	0.35	4.16	<0.0001
SnToxA ⁺ CF ⁻ vs. SnToxA ⁺ CF ⁺	1.71 (4)	3.54 (18)	1.83	6.38	<0.0001
SnToxA ⁻ CF ⁺ vs. SnToxA ⁺ CF ⁻	0.63 (60)	1.71 (4)	1.08	5.00	<0.0001
SnToxA ⁻ CF ⁺ vs. SnToxA ⁺ CF ⁺	0.98 (89)	3.54 (18)	2.56	16.35	<0.0001

^a Insensitivity and sensitivity to host selective toxins are indicated by – and +, respectively; CF = culture filtrate from Sn2000KO6-1.

^b Mean1 and Mean2 are the average disease reaction types of the first and second components in the comparison, respectively. Numbers of accessions used for each mean are indicated in parentheses.

^c Difference is calculated by Mean2 minus Mean1.

^d Probability associated with the *t* value from two-sample *t* test.

Fig. 3. Histogram of average *Stagonospora nodorum* blotch (SNB) disease reaction type frequency comparison of Ptr ToxA and/or Sn2000KO6-1 culture filtrate (CF) sensitive (+) and insensitive (–) accessions in *Triticum dicoccoides* at the seedling stage. **A**, Ptr ToxA sensitive and insensitive; **B**, culture filtrate sensitive and insensitive; and **C**, both Ptr ToxA and culture filtrate sensitive and insensitive.

resistance found in these accessions. Data presented here also provide evidence for the usefulness of screening early germplasm with both purified toxins and crude culture filtrates as a preliminary screen for

disease resistance. The *T. dicoccoides* accessions are currently maintained and can be obtained by contacting Steven Xu at the USDA-ARS, Northern Crop Science Laboratory, Fargo, ND.

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